DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS

FIFTH EDITION

DSM-5™

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DSM-5 Classification

Before each disorder name, ICD-9-CM codes are provided, followed by ICD-10-CM codes in parentheses. Blank lines indicate that either the ICD-9-CM or the ICD-10-CM code is not applicable. For some disorders, the code can be indicated only according to the subtype or specifier.

ICD-9-CM codes are to be used for coding purposes in the United States through September 30, 2014. ICD-10-CM codes are to be used starting October 1, 2014.

Following chapter titles and disorder names, page numbers for the corresponding text or criteria are included in parentheses.

Note for all mental disorders due to another medical condition: Indicate the name of the other medical condition in the name of the mental disorder due to [the medical condition]. The code and name for the other medical condition should be listed first immediately before the mental disorder due to the medical condition.

Neurodevelopmental Disorders (31)

Intellectual Disabilities (33)

319	**(___.__)**	Intellectual Disability (Intellectual Developmental Disorder) (33) *Specify* current severity:
	(F70)	Mild
	(F71)	Moderate
	(F72)	Severe
	(F73)	Profound
315.8	**(F88)**	Global Developmental Delay (41)
319	**(F79)**	Unspecified Intellectual Disability (Intellectual Developmental Disorder) (41)

Communication Disorders (41)

315.39 (F80.9)	Language Disorder (42)
315.39 (F80.0)	Speech Sound Disorder (44)
315.35 (F80.81)	Childhood-Onset Fluency Disorder (Stuttering) (45) **Note:** Later-onset cases are diagnosed as 307.0 (F98.5) adult-onset fluency disorder.
315.39 (F80.89)	Social (Pragmatic) Communication Disorder (47)
307.9 (F80.9)	Unspecified Communication Disorder (49)

Autism Spectrum Disorder (50)

299.00 (F84.0) Autism Spectrum Disorder (50)
Specify if: Associated with a known medical or genetic condition or environmental factor; Associated with another neurodevelopmental, mental, or behavioral disorder
Specify current severity for Criterion A and Criterion B: Requiring very substantial support, Requiring substantial support, Requiring support
Specify if: With or without accompanying intellectual impairment, With or without accompanying language impairment, With catatonia (use additional code 293.89 [F06.1])

Attention-Deficit/Hyperactivity Disorder (59)

___.__ (___.__) Attention-Deficit/Hyperactivity Disorder (59)
Specify whether:
314.01 (F90.2) Combined presentation
314.00 (F90.0) Predominantly inattentive presentation
314.01 (F90.1) Predominantly hyperactive/impulsive presentation
Specify if: In partial remission
Specify current severity: Mild, Moderate, Severe

314.01 (F90.8) Other Specified Attention-Deficit/Hyperactivity Disorder (65)

314.01 (F90.9) Unspecified Attention-Deficit/Hyperactivity Disorder (66)

Specific Learning Disorder (66)

___.__ (___.__) Specific Learning Disorder (66)
Specify if:
315.00 (F81.0) With impairment in reading (*specify* if with word reading accuracy, reading rate or fluency, reading comprehension)
315.2 (F81.81) With impairment in written expression (*specify* if with spelling accuracy, grammar and punctuation accuracy, clarity or organization of written expression)
315.1 (F81.2) With impairment in mathematics (*specify* if with number sense, memorization of arithmetic facts, accurate or fluent calculation, accurate math reasoning)
Specify current severity: Mild, Moderate, Severe

Motor Disorders (74)

315.4 (F82) Developmental Coordination Disorder (74)

307.3 (F98.4) Stereotypic Movement Disorder (77)
Specify if: With self-injurious behavior, Without self-injurious behavior
Specify if: Associated with a known medical or genetic condition, neurodevelopmental disorder, or environmental factor
Specify current severity: Mild, Moderate, Severe

Tic Disorders

307.23 (F95.2) Tourette's Disorder (81)

307.22 (F95.1) Persistent (Chronic) Motor or Vocal Tic Disorder (81)
Specify if: With motor tics only, With vocal tics only

307.21 (F95.0) Provisional Tic Disorder (81)

307.20 (F95.8) Other Specified Tic Disorder (85)

307.20 (F95.9) Unspecified Tic Disorder (85)

Other Neurodevelopmental Disorders (86)

315.8 (F88) Other Specified Neurodevelopmental Disorder (86)

315.9 (F89) Unspecified Neurodevelopmental Disorder (86)

Schizophrenia Spectrum and Other Psychotic Disorders (87)

The following specifiers apply to Schizophrenia Spectrum and Other Psychotic Disorders where indicated:

[a]*Specify* if: The following course specifiers are only to be used after a 1-year duration of the disorder: First episode, currently in acute episode; First episode, currently in partial remission; First episode, currently in full remission; Multiple episodes, currently in acute episode; Multiple episodes, currently in partial remission; Multiple episodes, currently in full remission; Continuous; Unspecified

[b]*Specify* if: With catatonia (use additional code 293.89 [F06.1])

[c]*Specify* current severity of delusions, hallucinations, disorganized speech, abnormal psychomotor behavior, negative symptoms, impaired cognition, depression, and mania symptoms

301.22 (F21) Schizotypal (Personality) Disorder (90)

297.1 (F22) Delusional Disorder[a, c] (90)
Specify whether: Erotomanic type, Grandiose type, Jealous type, Persecutory type, Somatic type, Mixed type, Unspecified type
Specify if: With bizarre content

298.8 (F23) Brief Psychotic Disorder[b, c] (94)
Specify if: With marked stressor(s), Without marked stressor(s), With postpartum onset

295.40 (F20.81) Schizophreniform Disorder[b, c] (96)
Specify if: With good prognostic features, Without good prognostic features

295.90 (F20.9) Schizophrenia[a, b, c] (99)

___.__ (___.__) Schizoaffective Disorder[a, b, c] (105)
Specify whether:

295.70 (F25.0) Bipolar type

295.70 (F25.1) Depressive type

___.__ (___.__) Substance/Medication-Induced Psychotic Disorder[c] (110)
Note: See the criteria set and corresponding recording procedures for substance-specific codes and ICD-9-CM and ICD-10-CM coding.
Specify if: With onset during intoxication, With onset during withdrawal

___.__ (___.__) Psychotic Disorder Due to Another Medical Condition[c] (115)
Specify whether:

293.81 (F06.2) With delusions

293.82 (F06.0) With hallucinations

Bipolar and Related Disorders (123)

The following specifiers apply to Bipolar and Related Disorders where indicated:
[a]*Specify:* With anxious distress (*specify* current severity: mild, moderate, moderate-severe, severe); With mixed features; With rapid cycling; With melancholic features; With atypical features; With mood-congruent psychotic features; With mood-incongruent psychotic features; With catatonia (use additional code 293.89 [F06.1]); With peripartum onset; With seasonal pattern

Depressive Disorders (155)

The following specifiers apply to Depressive Disorders where indicated:
[a]*Specify:* With anxious distress (*specify* current severity: mild, moderate, moderate-severe, severe); With mixed features; With melancholic features; With atypical features; With mood-congruent psychotic features; With mood-incongruent psychotic features; With catatonia (use additional code 293.89 [F06.1]); With peripartum onset; With seasonal pattern

Anxiety Disorders (189)

Obsessive-Compulsive and Related Disorders (235)

Trauma- and Stressor-Related Disorders (265)

Dissociative Disorders (291)

Somatic Symptom and Related Disorders (309)

Feeding and Eating Disorders (329)

The following specifiers apply to Feeding and Eating Disorders where indicated:
[a]*Specify* if: In remission
[b]*Specify* if: In partial remission, In full remission
[c]*Specify* current severity: Mild, Moderate, Severe, Extreme

Elimination Disorders (355)

Sleep-Wake Disorders (361)

The following specifiers apply to Sleep-Wake Disorders where indicated:
[a]*Specify* if: Episodic, Persistent, Recurrent
[b]*Specify* if: Acute, Subacute, Persistent
[c]*Specify* current severity: Mild, Moderate, Severe

780.52 (G47.00) Insomnia Disorder[a] (362)
Specify if: With non–sleep disorder mental comorbidity, With other medical comorbidity, With other sleep disorder

780.54 (G47.10) Hypersomnolence Disorder[b, c] (368)
Specify if: With mental disorder, With medical condition, With another sleep disorder

___.__ (___.__) Narcolepsy[c] (372)
Specify whether:

347.00 (G47.419) Narcolepsy without cataplexy but with hypocretin deficiency

347.01 (G47.411) Narcolepsy with cataplexy but without hypocretin deficiency

347.00 (G47.419) Autosomal dominant cerebellar ataxia, deafness, and narcolepsy

347.00 (G47.419) Autosomal dominant narcolepsy, obesity, and type 2 diabetes

347.10 (G47.429) Narcolepsy secondary to another medical condition

Breathing-Related Sleep Disorders (378)

327.23 (G47.33) Obstructive Sleep Apnea Hypopnea[c] (378)

___.__ (___.__) Central Sleep Apnea (383)
Specify whether:

327.21 (G47.31) Idiopathic central sleep apnea

786.04 (R06.3) Cheyne-Stokes breathing

780.57 (G47.37) Central sleep apnea comorbid with opioid use
Note: First code opioid use disorder, if present.
Specify current severity

___.__ (___.__) Sleep-Related Hypoventilation (387)
Specify whether:

327.24 (G47.34) Idiopathic hypoventilation

327.25 (G47.35) Congenital central alveolar hypoventilation

327.26 (G47.36) Comorbid sleep-related hypoventilation
Specify current severity

___.__ (___.__) Circadian Rhythm Sleep-Wake Disorders[a] (390)
Specify whether:

307.45 (G47.21) Delayed sleep phase type (391)
Specify if: Familial, Overlapping with non-24-hour sleep-wake type

307.45 (G47.22) Advanced sleep phase type (393)
Specify if: Familial

307.45 (G47.23) Irregular sleep-wake type (394)

307.45 (G47.24) Non-24-hour sleep-wake type (396)

307.45 (G47.26) Shift work type (397)

307.45 (G47.20) Unspecified type

Parasomnias (399)

__.__ (__.__) Non–Rapid Eye Movement Sleep Arousal Disorders (399)
Specify whether:

307.46 (F51.3) Sleepwalking type
Specify if: With sleep-related eating, With sleep-related sexual behavior (sexsomnia)

307.46 (F51.4) Sleep terror type

307.47 (F51.5) Nightmare Disorder[b, c] (404)
Specify if: During sleep onset
Specify if: With associated non–sleep disorder, With associated other medical condition, With associated other sleep disorder

327.42 (G47.52) Rapid Eye Movement Sleep Behavior Disorder (407)

333.94 (G25.81) Restless Legs Syndrome (410)

__.__ (__.__) Substance/Medication-Induced Sleep Disorder (413)
Note: See the criteria set and corresponding recording procedures for substance-specific codes and ICD-9-CM and ICD-10-CM coding.
Specify whether: Insomnia type, Daytime sleepiness type, Parasomnia type, Mixed type
Specify if: With onset during intoxication, With onset during discontinuation/withdrawal

780.52 (G47.09) Other Specified Insomnia Disorder (420)

780.52 (G47.00) Unspecified Insomnia Disorder (420)

780.54 (G47.19) Other Specified Hypersomnolence Disorder (421)

780.54 (G47.10) Unspecified Hypersomnolence Disorder (421)

780.59 (G47.8) Other Specified Sleep-Wake Disorder (421)

780.59 (G47.9) Unspecified Sleep-Wake Disorder (422)

Sexual Dysfunctions (423)

The following specifiers apply to Sexual Dysfunctions where indicated:
[a]*Specify* whether: Lifelong, Acquired
[b]*Specify* whether: Generalized, Situational
[c]*Specify* current severity: Mild, Moderate, Severe

302.74 (F52.32) Delayed Ejaculation[a, b, c] (424)

302.72 (F52.21) Erectile Disorder[a, b, c] (426)

302.73 (F52.31) Female Orgasmic Disorder[a, b, c] (429)
Specify if: Never experienced an orgasm under any situation

302.72 (F52.22) Female Sexual Interest/Arousal Disorder[a, b, c] (433)

302.76 (F52.6) Genito-Pelvic Pain/Penetration Disorder[a, c] (437)

Substance-Related and Addictive Disorders (481)

The following specifiers and note apply to Substance-Related and Addictive Disorders where indicated:

[a]*Specify* if: In early remission, In sustained remission

[b]*Specify* if: In a controlled environment

[c]*Specify* if: With perceptual disturbances

[d]The ICD-10-CM code indicates the comorbid presence of a moderate or severe substance use disorder, which must be present in order to apply the code for substance withdrawal.

Substance-Related Disorders (483)

Alcohol-Related Disorders (490)

___.__	(___.__)	Alcohol Use Disorder[a, b] (490) *Specify* current severity:
305.00	**(F10.10)**	Mild
303.90	**(F10.20)**	Moderate
303.90	**(F10.20)**	Severe
303.00	(___.__)	Alcohol Intoxication (497)
	(F10.129)	With use disorder, mild
	(F10.229)	With use disorder, moderate or severe
	(F10.929)	Without use disorder
291.81	(___.__)	Alcohol Withdrawal[c, d] (499)
	(F10.239)	Without perceptual disturbances
	(F10.232)	With perceptual disturbances
___.__	(___.__)	Other Alcohol-Induced Disorders (502)
291.9	**(F10.99)**	Unspecified Alcohol-Related Disorder (503)

Caffeine-Related Disorders (503)

305.90	**(F15.929)**	Caffeine Intoxication (503)
292.0	**(F15.93)**	Caffeine Withdrawal (506)
___.__	(___.__)	Other Caffeine-Induced Disorders (508)
292.9	**(F15.99)**	Unspecified Caffeine-Related Disorder (509)

Cannabis-Related Disorders (509)

___.__	(___.__)	Cannabis Use Disorder[a, b] (509) *Specify* current severity:
305.20	**(F12.10)**	Mild
304.30	**(F12.20)**	Moderate
304.30	**(F12.20)**	Severe

304.60 (F18.20) Moderate
304.60 (F18.20) Severe

292.89 (___.__) Inhalant Intoxication (538)
(F18.129) With use disorder, mild
(F18.229) With use disorder, moderate or severe
(F18.929) Without use disorder

___.__ (___.__) Other Inhalant-Induced Disorders (540)

292.9 (F18.99) Unspecified Inhalant-Related Disorder (540)

Opioid-Related Disorders (540)

___.__ (___.__) Opioid Use Disorder[a] (541)
Specify if: On maintenance therapy, In a controlled environment
Specify current severity:
305.50 (F11.10) Mild
304.00 (F11.20) Moderate
304.00 (F11.20) Severe

292.89 (___.__) Opioid Intoxication[c] (546)
Without perceptual disturbances
(F11.129) With use disorder, mild
(F11.229) With use disorder, moderate or severe
(F11.929) Without use disorder
With perceptual disturbances
(F11.122) With use disorder, mild
(F11.222) With use disorder, moderate or severe
(F11.922) Without use disorder

292.0 (F11.23) Opioid Withdrawal[d] (547)

___.__ (___.__) Other Opioid-Induced Disorders (549)

292.9 (F11.99) Unspecified Opioid-Related Disorder (550)

Sedative-, Hypnotic-, or Anxiolytic-Related Disorders (550)

___.__ (___.__) Sedative, Hypnotic, or Anxiolytic Use Disorder[a, b] (550)
Specify current severity:
305.40 (F13.10) Mild
304.10 (F13.20) Moderate
304.10 (F13.20) Severe

292.89 (___.__) Sedative, Hypnotic, or Anxiolytic Intoxication (556)
(F13.129) With use disorder, mild
(F13.229) With use disorder, moderate or severe
(F13.929) Without use disorder

292.0 (___.__) Sedative, Hypnotic, or Anxiolytic Withdrawal[c, d] (557)
(F13.239) Without perceptual disturbances
(F13.232) With perceptual disturbances

292.9 (__.__) Unspecified Stimulant-Related Disorder (570)
(F15.99) Amphetamine or other stimulant
(F14.99) Cocaine

Tobacco-Related Disorders (571)

__.__ (__.__) Tobacco Use Disorder[a] (571)
Specify if: On maintenance therapy, In a controlled environment
Specify current severity:
305.1 **(Z72.0)** Mild
305.1 **(F17.200)** Moderate
305.1 **(F17.200)** Severe

292.0 **(F17.203)** Tobacco Withdrawal[d] (575)

__.__ (__.__) Other Tobacco-Induced Disorders (576)

292.9 **(F17.209)** Unspecified Tobacco-Related Disorder (577)

Other (or Unknown) Substance–Related Disorders (577)

__.__ (__.__) Other (or Unknown) Substance Use Disorder[a, b] (577)
Specify current severity:
305.90 **(F19.10)** Mild
304.90 **(F19.20)** Moderate
304.90 **(F19.20)** Severe

292.89 (__.__) Other (or Unknown) Substance Intoxication (581)
(F19.129) With use disorder, mild
(F19.229) With use disorder, moderate or severe
(F19.929) Without use disorder

292.0 **(F19.239)** Other (or Unknown) Substance Withdrawal[d] (583)

__.__ (__.__) Other (or Unknown) Substance–Induced Disorders (584)

292.9 **(F19.99)** Unspecified Other (or Unknown) Substance–Related Disorder (585)

Non-Substance-Related Disorders (585)

312.31 **(F63.0)** Gambling Disorder[a] (585)
Specify if: Episodic, Persistent
Specify current severity: Mild, Moderate, Severe

Neurocognitive Disorders (591)

__.__ (__.__) Delirium (596)
[a]**Note:** See the criteria set and corresponding recording procedures for substance-specific codes and ICD-9-CM and ICD-10-CM coding.
Specify whether:
__.__ (__.__) Substance intoxication delirium[a]
__.__ (__.__) Substance withdrawal delirium[a]
292.81 (__.__) Medication-induced delirium[a]
293.0 **(F05)** Delirium due to another medical condition

293.0 (F05) Delirium due to multiple etiologies
Specify if: Acute, Persistent
Specify if: Hyperactive, Hypoactive, Mixed level of activity

780.09 (R41.0) Other Specified Delirium (602)

780.09 (R41.0) Unspecified Delirium (602)

Major and Mild Neurocognitive Disorders (602)

Specify whether due to: Alzheimer's disease, Frontotemporal lobar degeneration, Lewy body disease, Vascular disease, Traumatic brain injury, Substance/medication use, HIV infection, Prion disease, Parkinson's disease, Huntington's disease, Another medical condition, Multiple etiologies, Unspecified

[a]*Specify* Without behavioral disturbance, With behavioral disturbance. *For possible major neurocognitive disorder and for mild neurocognitive disorder, behavioral disturbance cannot be coded but should still be indicated in writing.*

[b]*Specify* current severity: Mild, Moderate, Severe. *This specifier applies only to major neurocognitive disorders (including probable and possible).*

Note: As indicated for each subtype, an additional medical code is needed for probable major neurocognitive disorder or major neurocognitive disorder. An additional medical code should *not* be used for possible major neurocognitive disorder or mild neurocognitive disorder.

Major or Mild Neurocognitive Disorder Due to Alzheimer's Disease (611)

__.__ (__.__) Probable Major Neurocognitive Disorder Due to Alzheimer's Disease[b]
Note: Code first **331.0 (G30.9)** Alzheimer's disease.

294.11 (F02.81) With behavioral disturbance

294.10 (F02.80) Without behavioral disturbance

331.9 (G31.9) Possible Major Neurocognitive Disorder Due to Alzheimer's Disease[a, b]

331.83 (G31.84) Mild Neurocognitive Disorder Due to Alzheimer's Disease[a]

Major or Mild Frontotemporal Neurocognitive Disorder (614)

__.__ (__.__) Probable Major Neurocognitive Disorder Due to Frontotemporal Lobar Degeneration[b]
Note: Code first **331.19 (G31.09)** frontotemporal disease.

294.11 (F02.81) With behavioral disturbance

294.10 (F02.80) Without behavioral disturbance

331.9 (G31.9) Possible Major Neurocognitive Disorder Due to Frontotemporal Lobar Degeneration[a, b]

331.83 (G31.84) Mild Neurocognitive Disorder Due to Frontotemporal Lobar Degeneration[a]

Major or Mild Neurocognitive Disorder With Lewy Bodies (618)

__.__ (__.__) Probable Major Neurocognitive Disorder With Lewy Bodies[b]
Note: Code first **331.82 (G31.83)** Lewy body disease.

294.11 (F02.81) With behavioral disturbance

294.10 (F02.80) Without behavioral disturbance

331.9 (G31.9) Possible Major Neurocognitive Disorder With Lewy Bodies[a, b]

331.83 (G31.84) Mild Neurocognitive Disorder With Lewy Bodies[a]

Major or Mild Vascular Neurocognitive Disorder (621)

___.__ (___.__) Probable Major Vascular Neurocognitive Disorder[b]
Note: No additional medical code for vascular disease.

290.40 (F01.51) With behavioral disturbance

290.40 (F01.50) Without behavioral disturbance

331.9 (G31.9) Possible Major Vascular Neurocognitive Disorder[a, b]

331.83 (G31.84) Mild Vascular Neurocognitive Disorder[a]

Major or Mild Neurocognitive Disorder Due to Traumatic Brain Injury (624)

___.__ (___.__) Major Neurocognitive Disorder Due to Traumatic Brain Injury[b]
Note: For ICD-9-CM, code first **907.0** late effect of intracranial injury without skull fracture. For ICD-10-CM, code first **S06.2X9S** diffuse traumatic brain injury with loss of consciousness of unspecified duration, sequela.

294.11 (F02.81) With behavioral disturbance

294.10 (F02.80) Without behavioral disturbance

331.83 (G31.84) Mild Neurocognitive Disorder Due to Traumatic Brain Injury[a]

Substance/Medication-Induced Major or Mild Neurocognitive Disorder[a] (627)
Note: No additional medical code. See the criteria set and corresponding recording procedures for substance-specific codes and ICD-9-CM and ICD-10-CM coding.
Specify if: Persistent

Major or Mild Neurocognitive Disorder Due to HIV Infection (632)

___.__ (___.__) Major Neurocognitive Disorder Due to HIV Infection[b]
Note: Code first **042 (B20)** HIV infection.

294.11 (F02.81) With behavioral disturbance

294.10 (F02.80) Without behavioral disturbance

331.83 (G31.84) Mild Neurocognitive Disorder Due to HIV Infection[a]

Major or Mild Neurocognitive Disorder Due to Prion Disease (634)

___.__ (___.__) Major Neurocognitive Disorder Due to Prion Disease[b]
Note: Code first **046.79 (A81.9)** prion disease.

294.11 (F02.81) With behavioral disturbance

294.10 (F02.80) Without behavioral disturbance

331.83 (G31.84) Mild Neurocognitive Disorder Due to Prion Disease[a]

Major or Mild Neurocognitive Disorder Due to Parkinson's Disease (636)

___.__ (___.__) Major Neurocognitive Disorder Probably Due to Parkinson's Disease[b]
Note: Code first **332.0 (G20)** Parkinson's disease.

294.11 (F02.81) With behavioral disturbance

294.10 (F02.80) Without behavioral disturbance

331.9 (G31.9) Major Neurocognitive Disorder Possibly Due to Parkinson's Disease[a, b]

331.83 (G31.84) Mild Neurocognitive Disorder Due to Parkinson's Disease[a]

Major or Mild Neurocognitive Disorder Due to Huntington's Disease (638)

___.__ (___.__) Major Neurocognitive Disorder Due to Huntington's Disease[b]
Note: Code first **333.4 (G10)** Huntington's disease.

294.11 (F02.81) With behavioral disturbance

294.10 (F02.80) Without behavioral disturbance

331.83 (G31.84) Mild Neurocognitive Disorder Due to Huntington's Disease[a]

Major or Mild Neurocognitive Disorder Due to Another Medical Condition (641)

___.__ (___.__) Major Neurocognitive Disorder Due to Another Medical Condition[b]
Note: Code first the other medical condition.

294.11 (F02.81) With behavioral disturbance

294.10 (F02.80) Without behavioral disturbance

331.83 (G31.84) Mild Neurocognitive Disorder Due to Another Medical Condition[a]

Major or Mild Neurocognitive Disorder Due to Multiple Etiologies (642)

___.__ (___.__) Major Neurocognitive Disorder Due to Multiple Etiologies[b]
Note: Code first all the etiological medical conditions (with the exception of vascular disease).

294.11 (F02.81) With behavioral disturbance

294.10 (F02.80) Without behavioral disturbance

331.83 (G31.84) Mild Neurocognitive Disorder Due to Multiple Etiologies[a]

Unspecified Neurocognitive Disorder (643)

799.59 (R41.9) Unspecified Neurocognitive Disorder[a]

Personality Disorders (645)

Cluster A Personality Disorders

301.0 (F60.0) Paranoid Personality Disorder (649)

301.20 (F60.1) Schizoid Personality Disorder (652)

301.22 (F21) Schizotypal Personality Disorder (655)

Cluster B Personality Disorders

301.7 (F60.2) Antisocial Personality Disorder (659)

301.83 (F60.3) Borderline Personality Disorder (663)

301.50 (F60.4) Histrionic Personality Disorder (667)

301.81 (F60.81) Narcissistic Personality Disorder (669)

Cluster C Personality Disorders

301.82 (F60.6) Avoidant Personality Disorder (672)

301.6 (F60.7) Dependent Personality Disorder (675)

301.4 (F60.5) Obsessive-Compulsive Personality Disorder (678)

Other Personality Disorders

310.1 (F07.0) Personality Change Due to Another Medical Condition (682)
Specify whether: Labile type, Disinhibited type, Aggressive type, Apathetic type, Paranoid type, Other type, Combined type, Unspecified type

301.89 (F60.89) Other Specified Personality Disorder (684)

301.9 (F60.9) Unspecified Personality Disorder (684)

Paraphilic Disorders (685)

The following specifier applies to Paraphilic Disorders where indicated:
[a]*Specify* if: In a controlled environment, In full remission

302.82 (F65.3) Voyeuristic Disorder[a] (686)

302.4 (F65.2) Exhibitionistic Disorder[a] (689)
Specify whether: Sexually aroused by exposing genitals to prepubertal children, Sexually aroused by exposing genitals to physically mature individuals, Sexually aroused by exposing genitals to prepubertal children and to physically mature individuals

302.89 (F65.81) Frotteuristic Disorder[a] (691)

302.83 (F65.51) Sexual Masochism Disorder[a] (694)
Specify if: With asphyxiophilia

302.84 (F65.52) Sexual Sadism Disorder[a] (695)

302.2 (F65.4) Pedophilic Disorder (697)
Specify whether: Exclusive type, Nonexclusive type
Specify if: Sexually attracted to males, Sexually attracted to females, Sexually attracted to both
Specify if: Limited to incest

302.81 (F65.0) Fetishistic Disorder[a] (700)
Specify: Body part(s), Nonliving object(s), Other

302.3 (F65.1) Transvestic Disorder[a] (702)
Specify if: With fetishism, With autogynephilia

302.89 (F65.89) Other Specified Paraphilic Disorder (705)

302.9 (F65.9) Unspecified Paraphilic Disorder (705)

Other Mental Disorders (707)

294.8 (F06.8) Other Specified Mental Disorder Due to Another Medical Condition (707)

294.9 (F09) Unspecified Mental Disorder Due to Another Medical Condition (708)

300.9 (F99) Other Specified Mental Disorder (708)

300.9 (F99) Unspecified Mental Disorder (708)

Medication-Induced Movement Disorders and Other Adverse Effects of Medication (709)

332.1 (G21.11) Neuroleptic-Induced Parkinsonism (709)

332.1 (G21.19) Other Medication-Induced Parkinsonism (709)

333.92 (G21.0) Neuroleptic Malignant Syndrome (709)

333.72 (G24.02) Medication-Induced Acute Dystonia (711)

333.99 (G25.71) Medication-Induced Acute Akathisia (711)

333.85 (G24.01) Tardive Dyskinesia (712)

333.72 (G24.09) Tardive Dystonia (712)

333.99 (G25.71) Tardive Akathisia (712)

333.1 (G25.1) Medication-Induced Postural Tremor (712)

333.99 (G25.79) Other Medication-Induced Movement Disorder (712)

___.__ (___.__) Antidepressant Discontinuation Syndrome (712)

995.29 (T43.205A) Initial encounter

995.29 (T43.205D) Subsequent encounter

995.29 (T43.205S) Sequelae

___.__ (___.__) Other Adverse Effect of Medication (714)

995.20 (T50.905A) Initial encounter

995.20 (T50.905D) Subsequent encounter

995.20 (T50.905S) Sequelae

Other Conditions That May Be a Focus of Clinical Attention (715)

Relational Problems (715)

Problems Related to Family Upbringing (715)

V61.20 (Z62.820) Parent-Child Relational Problem (715)

V61.8 (Z62.891) Sibling Relational Problem (716)

V61.8 (Z62.29) Upbringing Away From Parents (716)

V61.29 (Z62.898) Child Affected by Parental Relationship Distress (716)

Other Problems Related to Primary Support Group (716)

V61.10 (Z63.0) Relationship Distress With Spouse or Intimate Partner (716)

V61.03 (Z63.5) Disruption of Family by Separation or Divorce (716)

V61.8 (Z63.8) High Expressed Emotion Level Within Family (716)

V62.82 (Z63.4) Uncomplicated Bereavement (716)

Abuse and Neglect (717)

Child Maltreatment and Neglect Problems (717)

Child Physical Abuse (717)

Child Physical Abuse, Confirmed (717)

995.54 (T74.12XA) Initial encounter

995.54 (T74.12XD) Subsequent encounter

Child Physical Abuse, Suspected (717)

995.54 (T76.12XA) Initial encounter

995.54 (T76.12XD) Subsequent encounter

Other Circumstances Related to Child Physical Abuse (718)

V61.21 (Z69.010) Encounter for mental health services for victim of child abuse by parent

V61.21 (Z69.020) Encounter for mental health services for victim of nonparental child abuse

V15.41 (Z62.810) Personal history (past history) of physical abuse in childhood

V61.22 (Z69.011) Encounter for mental health services for perpetrator of parental child abuse

V62.83 (Z69.021) Encounter for mental health services for perpetrator of nonparental child abuse

Child Sexual Abuse (718)

Child Sexual Abuse, Confirmed (718)

995.53 (T74.22XA) Initial encounter

995.53 (T74.22XD) Subsequent encounter

Child Sexual Abuse, Suspected (718)

995.53 (T76.22XA) Initial encounter

995.53 (T76.22XD) Subsequent encounter

Other Circumstances Related to Child Sexual Abuse (718)

V61.21 (Z69.010) Encounter for mental health services for victim of child sexual abuse by parent

V61.21 (Z69.020) Encounter for mental health services for victim of nonparental child sexual abuse

V15.41 (Z62.810) Personal history (past history) of sexual abuse in childhood

V61.22 (Z69.011) Encounter for mental health services for perpetrator of parental child sexual abuse

V62.83 (Z69.021) Encounter for mental health services for perpetrator of nonparental child sexual abuse

Child Neglect (718)

Child Neglect, Confirmed (718)

995.52 (T74.02XA) Initial encounter

995.52 (T74.02XD) Subsequent encounter

Child Neglect, Suspected (719)

995.52 (T76.02XA) Initial encounter

995.52 (T76.02XD) Subsequent encounter

Other Circumstances Related to Child Neglect (719)

V61.21 (Z69.010) Encounter for mental health services for victim of child neglect by parent

V61.21 (Z69.020) Encounter for mental health services for victim of nonparental child neglect

V15.42 (Z62.812) Personal history (past history) of neglect in childhood

V61.22 (Z69.011) Encounter for mental health services for perpetrator of parental child neglect

V62.83 (Z69.021) Encounter for mental health services for perpetrator of nonparental child neglect

Child Psychological Abuse (719)

Child Psychological Abuse, Confirmed (719)

995.51 (T74.32XA) Initial encounter

995.51 (T74.32XD) Subsequent encounter

Child Psychological Abuse, Suspected (719)

995.51 (T76.32XA) Initial encounter

995.51 (T76.32XD) Subsequent encounter

Other Circumstances Related to Child Psychological Abuse (719)

V61.21 (Z69.010) Encounter for mental health services for victim of child psychological abuse by parent

V61.21 (Z69.020) Encounter for mental health services for victim of nonparental child psychological abuse

V15.42 (Z62.811) Personal history (past history) of psychological abuse in childhood

V61.22 (Z69.011) Encounter for mental health services for perpetrator of parental child psychological abuse

V62.83 (Z69.021) Encounter for mental health services for perpetrator of nonparental child psychological abuse

Adult Maltreatment and Neglect Problems (720)

Spouse or Partner Violence, Physical (720)

Spouse or Partner Violence, Physical, Confirmed (720)

995.81 (T74.11XA) Initial encounter

995.81 (T74.11XD) Subsequent encounter

Spouse or Partner Violence, Physical, Suspected (720)

995.81 (T76.11XA) Initial encounter

995.81 (T76.11XD) Subsequent encounter

Other Circumstances Related to Spouse or Partner Violence, Physical (720)

V61.11 (Z69.11) Encounter for mental health services for victim of spouse or partner violence, physical

V15.41 (Z91.410) Personal history (past history) of spouse or partner violence, physical

V61.12 (Z69.12) Encounter for mental health services for perpetrator of spouse or partner violence, physical

Spouse or Partner Violence, Sexual (720)

Spouse or Partner Violence, Sexual, Confirmed (720)

995.83 (T74.21XA) Initial encounter

995.83 (T74.21XD) Subsequent encounter

Spouse or Partner Violence, Sexual, Suspected (720)

995.83 (T76.21XA) Initial encounter

995.83 (T76.21XD) Subsequent encounter

Other Circumstances Related to Spouse or Partner Violence, Sexual (720)

V61.11 (Z69.81) Encounter for mental health services for victim of spouse or partner violence, sexual

V15.41 (Z91.410) Personal history (past history) of spouse or partner violence, sexual

V61.12 (Z69.12) Encounter for mental health services for perpetrator of spouse or partner violence, sexual

Spouse or Partner, Neglect (721)

Spouse or Partner Neglect, Confirmed (721)

995.85 (T74.01XA) Initial encounter

995.85 (T74.01XD) Subsequent encounter

Spouse or Partner Neglect, Suspected (721)

995.85 (T76.01XA) Initial encounter

995.85 (T76.01XD) Subsequent encounter

Other Circumstances Related to Spouse or Partner Neglect (721)

V61.11 (Z69.11) Encounter for mental health services for victim of spouse or partner neglect

V15.42 (Z91.412) Personal history (past history) of spouse or partner neglect

V61.12 (Z69.12) Encounter for mental health services for perpetrator of spouse or partner neglect

Spouse or Partner Abuse, Psychological (721)

Spouse or Partner Abuse, Psychological, Confirmed (721)

995.82 (T74.31XA) Initial encounter

995.82 (T74.31XD) Subsequent encounter

Spouse or Partner Abuse, Psychological, Suspected (721)

995.82 (T76.31XA) Initial encounter

995.82 (T76.31XD) Subsequent encounter

Other Circumstances Related to Spouse or Partner Abuse, Psychological (721)

V61.11 (Z69.11) Encounter for mental health services for victim of spouse or partner psychological abuse

V15.42 (Z91.411) Personal history (past history) of spouse or partner psychological abuse

V61.12 (Z69.12) Encounter for mental health services for perpetrator of spouse or partner psychological abuse

Adult Abuse by Nonspouse or Nonpartner (722)

Adult Physical Abuse by Nonspouse or Nonpartner, Confirmed (722)

995.81 (T74.11XA) Initial encounter

995.81 (T74.11XD) Subsequent encounter

Adult Physical Abuse by Nonspouse or Nonpartner, Suspected (722)

995.81 (T76.11XA) Initial encounter

995.81 (T76.11XD) Subsequent encounter

Adult Sexual Abuse by Nonspouse or Nonpartner, Confirmed (722)

995.83 (T74.21XA) Initial encounter

995.83 (T74.21XD) Subsequent encounter

Adult Sexual Abuse by Nonspouse or Nonpartner, Suspected (722)

995.83 (T76.21XA) Initial encounter

995.83 (T76.21XD) Subsequent encounter

Adult Psychological Abuse by Nonspouse or Nonpartner, Confirmed (722)

995.82 (T74.31XA) Initial encounter

995.82 (T74.31XD) Subsequent encounter

Adult Psychological Abuse by Nonspouse or Nonpartner, Suspected (722)

995.82 (T76.31XA) Initial encounter

995.82 (T76.31XD) Subsequent encounter

Other Circumstances Related to Adult Abuse by Nonspouse or Nonpartner (722)

V65.49 (Z69.81) Encounter for mental health services for victim of nonspousal adult abuse

V62.83 (Z69.82) Encounter for mental health services for perpetrator of nonspousal adult abuse

Educational and Occupational Problems (723)

Educational Problems (723)

V62.3 (Z55.9) Academic or Educational Problem (723)

Occupational Problems (723)

V62.21 (Z56.82) Problem Related to Current Military Deployment Status (723)

V62.29 (Z56.9) Other Problem Related to Employment (723)

Housing and Economic Problems (723)

Housing Problems (723)

V60.0 (Z59.0) Homelessness (723)

V60.1 (Z59.1) Inadequate Housing (723)

V60.89 (Z59.2) Discord With Neighbor, Lodger, or Landlord (723)

V60.6 (Z59.3) Problem Related to Living in a Residential Institution (724)

Economic Problems (724)

V60.2 (Z59.4) Lack of Adequate Food or Safe Drinking Water (724)

V60.2 (Z59.5) Extreme Poverty (724)

V60.2 (Z59.6) Low Income (724)

V60.2 (Z59.7) Insufficient Social Insurance or Welfare Support (724)

V60.9 (Z59.9) Unspecified Housing or Economic Problem (724)

Other Problems Related to the Social Environment (724)

V62.89 (Z60.0) Phase of Life Problem (724)

V60.3 (Z60.2) Problem Related to Living Alone (724)

V62.4 (Z60.3) Acculturation Difficulty (724)

V62.4 (Z60.4) Social Exclusion or Rejection (724)

V62.4 (Z60.5) Target of (Perceived) Adverse Discrimination or Persecution (724)

V62.9 (Z60.9) Unspecified Problem Related to Social Environment (725)

Problems Related to Crime or Interaction With the Legal System (725)

V62.89 (Z65.4) Victim of Crime (725)

V62.5 (Z65.0) Conviction in Civil or Criminal Proceedings Without Imprisonment (725)

V62.5 (Z65.1) Imprisonment or Other Incarceration (725)

V62.5 (Z65.2) Problems Related to Release From Prison (725)

V62.5 (Z65.3) Problems Related to Other Legal Circumstances (725)

Other Health Service Encounters for Counseling and Medical Advice (725)

V65.49 (Z70.9) Sex Counseling (725)

V65.40 (Z71.9) Other Counseling or Consultation (725)

Problems Related to Other Psychosocial, Personal, and Environmental Circumstances (725)

V62.89 (Z65.8) Religious or Spiritual Problem (725)

V61.7 (Z64.0) Problems Related to Unwanted Pregnancy (725)

V61.5 (Z64.1) Problems Related to Multiparity (725)

V62.89 (Z64.4) Discord With Social Service Provider, Including Probation Officer, Case Manager, or Social Services Worker (725)

V62.89 (Z65.4) Victim of Terrorism or Torture (725)

V62.22 (Z65.5) Exposure to Disaster, War, or Other Hostilities (725)

V62.89 (Z65.8) Other Problem Related to Psychosocial Circumstances (725)

V62.9 (Z65.9) Unspecified Problem Related to Unspecified Psychosocial Circumstances (725)

Other Circumstances of Personal History (726)

V15.49 (Z91.49) Other Personal History of Psychological Trauma (726)

V15.59 (Z91.5) Personal History of Self-Harm (726)

V62.22 (Z91.82) Personal History of Military Deployment (726)

V15.89 (Z91.89) Other Personal Risk Factors (726)

V69.9 (Z72.9) Problem Related to Lifestyle (726)

V71.01 (Z72.811) Adult Antisocial Behavior (726)

V71.02 (Z72.810) Child or Adolescent Antisocial Behavior (726)

Problems Related to Access to Medical and Other Health Care (726)

V63.9 (Z75.3) Unavailability or Inaccessibility of Health Care Facilities (726)

V63.8 (Z75.4) Unavailability or Inaccessibility of Other Helping Agencies (726)

Nonadherence to Medical Treatment (726)

V15.81 (Z91.19) Nonadherence to Medical Treatment (726)

278.00 (E66.9) Overweight or Obesity (726)

V65.2 (Z76.5) Malingering (726)

V40.31 (Z91.83) Wandering Associated With a Mental Disorder (727)

V62.89 (R41.83) Borderline Intellectual Functioning (727)

Preface

The American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM) is a classification of mental disorders with associated criteria designed to facilitate more reliable diagnoses of these disorders. With successive editions over the past 60 years, it has become a standard reference for clinical practice in the mental health field. Since a complete description of the underlying pathological processes is not possible for most mental disorders, it is important to emphasize that the current diagnostic criteria are the best available description of how mental disorders are expressed and can be recognized by trained clinicians. DSM is intended to serve as a practical, functional, and flexible guide for organizing information that can aid in the accurate diagnosis and treatment of mental disorders. It is a tool for clinicians, an essential educational resource for students and practitioners, and a reference for researchers in the field.

Although this edition of DSM was designed first and foremost to be a useful guide to clinical practice, as an official nomenclature it must be applicable in a wide diversity of contexts. DSM has been used by clinicians and researchers from different orientations (biological, psychodynamic, cognitive, behavioral, interpersonal, family/systems), all of whom strive for a common language to communicate the essential characteristics of mental disorders presented by their patients. The information is of value to all professionals associated with various aspects of mental health care, including psychiatrists, other physicians, psychologists, social workers, nurses, counselors, forensic and legal specialists, occupational and rehabilitation therapists, and other health professionals. The criteria are concise and explicit and intended to facilitate an objective assessment of symptom presentations in a variety of clinical settings—inpatient, outpatient, partial hospital, consultation-liaison, clinical, private practice, and primary care—as well in general community epidemiological studies of mental disorders. DSM-5 is also a tool for collecting and communicating accurate public health statistics on mental disorder morbidity and mortality rates. Finally, the criteria and corresponding text serve as a textbook for students early in their profession who need a structured way to understand and diagnose mental disorders as well as for seasoned professionals encountering rare disorders for the first time. Fortunately, all of these uses are mutually compatible.

These diverse needs and interests were taken into consideration in planning DSM-5. The classification of disorders is harmonized with the World Health Organization's *International Classification of Diseases* (ICD), the official coding system used in the United States, so that the DSM criteria define disorders identified by ICD diagnostic names and code numbers. In DSM-5, both ICD-9-CM and ICD-10-CM codes (the latter scheduled for adoption in October 2014) are attached to the relevant disorders in the classification.

Although DSM-5 remains a categorical classification of separate disorders, we recognize that mental disorders do not always fit completely within the boundaries of a single disorder. Some symptom domains, such as depression and anxiety, involve multiple diagnostic categories and may reflect common underlying vulnerabilities for a larger group of disorders. In recognition of this reality, the disorders included in DSM-5 were reordered into a revised organizational structure meant to stimulate new clinical perspectives. This new structure corresponds with the organizational arrangement of disorders planned for ICD-11 scheduled for release in 2015. Other enhancements have been introduced to promote ease of use across all settings:

- **Representation of developmental issues related to diagnosis.** The change in chapter organization better reflects a lifespan approach, with disorders more frequently diagnosed in childhood (e.g., neurodevelopmental disorders) at the beginning of the manual and disorders more applicable to older adulthood (e.g., neurocognitive disorders) at the end of the manual. Also, within the text, subheadings on development and course provide descriptions of how disorder presentations may change across the lifespan. Age-related factors specific to diagnosis (e.g., symptom presentation and prevalence differences in certain age groups) are also included in the text. For added emphasis, these age-related factors have been added to the criteria themselves where applicable (e.g., in the criteria sets for insomnia disorder and posttraumatic stress disorder, specific criteria describe how symptoms might be expressed in children). Likewise, gender and cultural issues have been integrated into the disorders where applicable.
- **Integration of scientific findings from the latest research in genetics and neuroimaging.** The revised chapter structure was informed by recent research in neuroscience and by emerging genetic linkages between diagnostic groups. Genetic and physiological risk factors, prognostic indicators, and some putative diagnostic markers are highlighted in the text. This new structure should improve clinicians' ability to identify diagnoses in a disorder spectrum based on common neurocircuitry, genetic vulnerability, and environmental exposures.
- **Consolidation of autistic disorder, Asperger's disorder, and pervasive developmental disorder into autism spectrum disorder.** Symptoms of these disorders represent a single continuum of mild to severe impairments in the two domains of social communication and restrictive repetitive behaviors/interests rather than being distinct disorders. This change is designed to improve the sensitivity and specificity of the criteria for the diagnosis of autism spectrum disorder and to identify more focused treatment targets for the specific impairments identified.
- **Streamlined classification of bipolar and depressive disorders.** Bipolar and depressive disorders are the most commonly diagnosed conditions in psychiatry. It was therefore important to streamline the presentation of these disorders to enhance both clinical and educational use. Rather than separating the definition of manic, hypomanic, and major depressive episodes from the definition of bipolar I disorder, bipolar II disorder, and major depressive disorder as in the previous edition, we included all of the component criteria within the respective criteria for each disorder. This approach will facilitate bedside diagnosis and treatment of these important disorders. Likewise, the explanatory notes for differentiating bereavement and major depressive disorders will provide far greater clinical guidance than was previously provided in the simple bereavement exclusion criterion. The new specifiers of anxious distress and mixed features are now fully described in the narrative on specifier variations that accompanies the criteria for these disorders.
- **Restructuring of substance use disorders for consistency and clarity.** The categories of substance abuse and substance dependence have been eliminated and replaced with an overarching new category of substance use disorders—with the specific substance used defining the specific disorders. "Dependence" has been easily confused with the term "addiction" when, in fact, the tolerance and withdrawal that previously defined dependence are actually very normal responses to prescribed medications that affect the central nervous system and do not necessarily indicate the presence of an addiction. By revising and clarifying these criteria in DSM-5, we hope to alleviate some of the widespread misunderstanding about these issues.
- **Enhanced specificity for major and mild neurocognitive disorders.** Given the explosion in neuroscience, neuropsychology, and brain imaging over the past 20 years, it was critical to convey the current state-of-the-art in the diagnosis of specific types of disorders that were previously referred to as the "dementias" or organic brain diseases. Biological markers identified by imaging for vascular and traumatic brain disorders and

specific molecular genetic findings for rare variants of Alzheimer's disease and Huntington's disease have greatly advanced clinical diagnoses, and these disorders and others have now been separated into specific subtypes.

- **Transition in conceptualizing personality disorders.** Although the benefits of a more dimensional approach to personality disorders have been identified in previous editions, the transition from a categorical diagnostic system of individual disorders to one based on the relative distribution of personality traits has not been widely accepted. In DSM-5, the categorical personality disorders are virtually unchanged from the previous edition. However, an alternative "hybrid" model has been proposed in Section III to guide future research that separates interpersonal functioning assessments and the expression of pathological personality traits for six specific disorders. A more dimensional profile of personality trait expression is also proposed for a trait-specified approach.
- **Section III: new disorders and features.** A new section (Section III) has been added to highlight disorders that require further study but are not sufficiently well established to be a part of the official classification of mental disorders for routine clinical use. Dimensional measures of symptom severity in 13 symptom domains have also been incorporated to allow for the measurement of symptom levels of varying severity across all diagnostic groups. Likewise, the WHO Disability Assessment Schedule (WHODAS), a standard method for assessing global disability levels for mental disorders that is based on the International Classification of Functioning, Disability and Health (ICF) and is applicable in all of medicine, has been provided to replace the more limited Global Assessment of Functioning scale. It is our hope that as these measures are implemented over time, they will provide greater accuracy and flexibility in the clinical description of individual symptomatic presentations and associated disability during diagnostic assessments.
- **Online enhancements.** DSM-5 features online supplemental information. Additional cross-cutting and diagnostic severity measures are available online (www.psychiatry.org/dsm5), linked to the relevant disorders. In addition, the Cultural Formulation Interview, Cultural Formulation Interview—Informant Version, and supplementary modules to the core Cultural Formulation Interview are also included online at www.psychiatry.org/dsm5.

These innovations were designed by the leading authorities on mental disorders in the world and were implemented on the basis of their expert review, public commentary, and independent peer review. The 13 work groups, under the direction of the DSM-5 Task Force, in conjunction with other review bodies and, eventually, the APA Board of Trustees, collectively represent the global expertise of the specialty. This effort was supported by an extensive base of advisors and by the professional staff of the APA Division of Research; the names of everyone involved are too numerous to mention here but are listed in the Appendix. We owe tremendous thanks to those who devoted countless hours and invaluable expertise to this effort to improve the diagnosis of mental disorders.

We would especially like to acknowledge the chairs, text coordinators, and members of the 13 work groups, listed in the front of the manual, who spent many hours in this volunteer effort to improve the scientific basis of clinical practice over a sustained 6-year period. Susan K. Schultz, M.D., who served as text editor, worked tirelessly with Emily A. Kuhl, Ph.D., senior science writer and DSM-5 staff text editor, to coordinate the efforts of the work groups into a cohesive whole. William E. Narrow, M.D., M.P.H., led the research group that developed the overall research strategy for DSM-5, including the field trials, that greatly enhanced the evidence base for this revision. In addition, we are grateful to those who contributed so much time to the independent review of the revision proposals, including Kenneth S. Kendler, M.D., and Robert Freedman, M.D., co-chairs of the Scientific Review Committee; John S. McIntyre, M.D., and Joel Yager, M.D., co-chairs of the Clinical and Public Health Committee; and Glenn Martin, M.D., chair of the APA Assem-

bly review process. Special thanks go to Helena C. Kraemer, Ph.D., for her expert statistical consultation; Michael B. First, M.D., for his valuable input on the coding and review of criteria; and Paul S. Appelbaum, M.D., for feedback on forensic issues. Maria N. Ward, M.Ed., RHIT, CCS-P, also helped in verifying all ICD coding. The Summit Group, which included these consultants, the chairs of all review groups, the task force chairs, and the APA executive officers, chaired by Dilip V. Jeste, M.D., provided leadership and vision in helping to achieve compromise and consensus. This level of commitment has contributed to the balance and objectivity that we feel are hallmarks of DSM-5.

We especially wish to recognize the outstanding APA Division of Research staff—identified in the Task Force and Work Group listing at the front of this manual—who worked tirelessly to interact with the task force, work groups, advisors, and reviewers to resolve issues, serve as liaisons between the groups, direct and manage the academic and routine clinical practice field trials, and record decisions in this important process. In particular, we appreciate the support and guidance provided by James H. Scully Jr., M.D., Medical Director and CEO of the APA, through the years and travails of the development process. Finally, we thank the editorial and production staff of American Psychiatric Publishing—specifically, Rebecca Rinehart, Publisher; John McDuffie, Editorial Director; Ann Eng, Senior Editor; Greg Kuny, Managing Editor; and Tammy Cordova, Graphics Design Manager—for their guidance in bringing this all together and creating the final product. It is the culmination of efforts of many talented individuals who dedicated their time, expertise, and passion that made DSM-5 possible.

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December 19, 2012

SECTION I
DSM-5 Basics

This section is a basic orientation to the purpose, structure, content, and use of DSM-5. It is not intended to provide an exhaustive account of the evolution of DSM-5, but rather to give readers a succinct overview of its key elements. The introductory section describes the public, professional, and expert review process that was used to extensively evaluate the diagnostic criteria presented in Section II. A summary of the DSM-5 structure, harmonization with ICD-11, and the transition to a non-axial system with a new approach to assessing disability is also presented. "Use of the Manual" includes "Definition of a Mental Disorder," forensic considerations, and a brief overview of the diagnostic process and use of coding and recording procedures.

Introduction

The creation of the fifth edition of *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) was a massive undertaking that involved hundreds of people working toward a common goal over a 12-year process. Much thought and deliberation were involved in evaluating the diagnostic criteria, considering the organization of every aspect of the manual, and creating new features believed to be most useful to clinicians. All of these efforts were directed toward the goal of enhancing the clinical usefulness of DSM-5 as a guide in the diagnosis of mental disorders.

Reliable diagnoses are essential for guiding treatment recommendations, identifying prevalence rates for mental health service planning, identifying patient groups for clinical and basic research, and documenting important public health information such as morbidity and mortality rates. As the understanding of mental disorders and their treatments has evolved, medical, scientific, and clinical professionals have focused on the characteristics of specific disorders and their implications for treatment and research.

While DSM has been the cornerstone of substantial progress in reliability, it has been well recognized by both the American Psychiatric Association (APA) and the broad scientific community working on mental disorders that past science was not mature enough to yield fully validated diagnoses—that is, to provide consistent, strong, and objective scientific validators of individual DSM disorders. The science of mental disorders continues to evolve. However, the last two decades since DSM-IV was released have seen real and durable progress in such areas as cognitive neuroscience, brain imaging, epidemiology, and genetics. The DSM-5 Task Force overseeing the new edition recognized that research advances will require careful, iterative changes if DSM is to maintain its place as the touchstone classification of mental disorders. Finding the right balance is critical. Speculative results do not belong in an official nosology, but at the same time, DSM must evolve in the context of other clinical research initiatives in the field. One important aspect of this transition derives from the broad recognition that a too-rigid categorical system does not capture clinical experience or important scientific observations. The results of numerous studies of comorbidity and disease transmission in families, including twin studies and molecular genetic studies, make strong arguments for what many astute clinicians have long observed: the boundaries between many disorder "categories" are more fluid over the life course than DSM-IV recognized, and many symptoms assigned to a single disorder may occur, at varying levels of severity, in many other disorders. These findings mean that DSM, like other medical disease classifications, should accommodate ways to introduce dimensional approaches to mental disorders, including dimensions that cut across current categories. Such an approach should permit a more accurate description of patient presentations and increase the validity of a diagnosis (i.e., the degree to which diagnostic criteria reflect the comprehensive manifestation of an underlying psychopathological disorder). DSM-5 is designed to better fill the need of clinicians, patients, families, and researchers for a clear and concise description of each mental disorder organized by explicit diagnostic criteria, supplemented, when appropriate, by dimensional measures that cross diagnostic boundaries, and a brief digest of information about the diagnosis, risk factors, associated features, research advances, and various expressions of the disorder.

Clinical training and experience are needed to use DSM for determining a diagnosis. The diagnostic criteria identify symptoms, behaviors, cognitive functions, personality traits, physical signs, syndrome combinations, and durations that require clinical expertise to differentiate from normal life variation and transient responses to stress. To facilitate a thorough

examination of the range of symptoms present, DSM can serve clinicians as a guide to identify the most prominent symptoms that should be assessed when diagnosing a disorder. Although some mental disorders may have well-defined boundaries around symptom clusters, scientific evidence now places many, if not most, disorders on a spectrum with closely related disorders that have shared symptoms, shared genetic and environmental risk factors, and possibly shared neural substrates (perhaps most strongly established for a subset of anxiety disorders by neuroimaging and animal models). In short, we have come to recognize that the boundaries between disorders are more porous than originally perceived.

Many health profession and educational groups have been involved in the development and testing of DSM-5, including physicians, psychologists, social workers, nurses, counselors, epidemiologists, statisticians, neuroscientists, and neuropsychologists. Finally, patients, families, lawyers, consumer organizations, and advocacy groups have all participated in revising DSM-5 by providing feedback on the mental disorders described in this volume. Their monitoring of the descriptions and explanatory text is essential to improve understanding, reduce stigma, and advance the treatment and eventual cures for these conditions.

A Brief History

The APA first published a predecessor of DSM in 1844, as a statistical classification of institutionalized mental patients. It was designed to improve communication about the types of patients cared for in these hospitals. This forerunner to DSM also was used as a component of the full U.S. census. After World War II, DSM evolved through four major editions into a diagnostic classification system for psychiatrists, other physicians, and other mental health professionals that described the essential features of the full range of mental disorders. The current edition, DSM-5, builds on the goal of its predecessors (most recently, DSM-IV-TR, or Text Revision, published in 2000) of providing guidelines for diagnoses that can inform treatment and management decisions.

DSM-5 Revision Process

In 1999, the APA launched an evaluation of the strengths and weaknesses of DSM based on emerging research that did not support the boundaries established for some mental disorders. This effort was coordinated with the World Health Organization (WHO) Division of Mental Health, the World Psychiatric Association, and the National Institute of Mental Health (NIMH) in the form of several conferences, the proceedings of which were published in 2002 in a monograph entitled *A Research Agenda for DSM-V*. Thereafter, from 2003 to 2008, a cooperative agreement with the APA and the WHO was supported by the NIMH, the National Institute on Drug Abuse (NIDA), and the National Institute on Alcoholism and Alcohol Abuse (NIAAA) to convene 13 international DSM-5 research planning conferences, involving 400 participants from 39 countries, to review the world literature in specific diagnostic areas to prepare for revisions in developing both DSM-5 and the *International Classification of Diseases*, 11th Revision (ICD-11). Reports from these conferences formed the basis for future DSM-5 Task Force reviews and set the stage for the new edition of DSM.

In 2006, the APA named David J. Kupfer, M.D., as Chair and Darrel A. Regier, M.D., M.P.H., as Vice-Chair of the DSM-5 Task Force. They were charged with recommending chairs for the 13 diagnostic work groups and additional task force members with a multidisciplinary range of expertise who would oversee the development of DSM-5. An additional vetting process was initiated by the APA Board of Trustees to disclose sources of income and thus avoid conflicts of interest by task force and work group members. The full disclosure of all income and research grants from commercial sources, including the pharmaceutical industry, in the previous 3 years, the imposition of an income cap from all commercial sources, and the publication of disclosures on a Web site set a new standard for the

field. Thereafter, the task force of 28 members was approved in 2007, and appointments of more than 130 work group members were approved in 2008. More than 400 additional work group advisors with no voting authority were also approved to participate in the process. A clear concept of the next evolutionary stage for the classification of mental disorders was central to the efforts of the task force and the work groups. This vision emerged as the task force and work groups recounted the history of DSM-IV's classification, its current strengths and limitations, and strategic directions for its revision. An intensive 6-year process involved conducting literature reviews and secondary analyses, publishing research reports in scientific journals, developing draft diagnostic criteria, posting preliminary drafts on the DSM-5 Web site for public comment, presenting preliminary findings at professional meetings, performing field trials, and revising criteria and text.

Proposals for Revisions

Proposals for the revision of DSM-5 diagnostic criteria were developed by members of the work groups on the basis of rationale, scope of change, expected impact on clinical management and public health, strength of the supporting research evidence, overall clarity, and clinical utility. Proposals encompassed changes to diagnostic criteria; the addition of new disorders, subtypes, and specifiers; and the deletion of existing disorders.

In the proposals for revisions, strengths and weaknesses in the current criteria and nosology were first identified. Novel scientific findings over the previous two decades were considered, leading to the creation of a research plan to assess potential changes through literature reviews and secondary data analyses. Four principles guided the draft revisions: 1) DSM-5 is primarily intended to be a manual to be used by clinicians, and revisions must be feasible for routine clinical practice; 2) recommendations for revisions should be guided by research evidence; 3) where possible, continuity should be maintained with previous editions of DSM; and 4) no a priori constraints should be placed on the degree of change between DSM-IV and DSM-5.

Building on the initial literature reviews, work groups identified key issues within their diagnostic areas. Work groups also examined broader methodological concerns, such as the presence of contradictory findings within the literature; development of a refined definition of mental disorder; cross-cutting issues relevant to all disorders; and the revision of disorders categorized in DSM-IV as "not otherwise specified." Inclusion of a proposal for revision in Section II was informed by consideration of its advantages and disadvantages for public health and clinical utility, the strength of the evidence, and the magnitude of the change. New diagnoses and disorder subtypes and specifiers were subject to additional stipulations, such as demonstration of reliability (i.e., the degree to which two clinicians could independently arrive at the same diagnosis for a given patient). Disorders with low clinical utility and weak validity were considered for deletion. Placement of conditions in "Conditions for Further Study" in Section III was contingent on the amount of empirical evidence generated on the diagnosis, diagnostic reliability or validity, presence of clear clinical need, and potential benefit in advancing research.

DSM-5 Field Trials

The use of field trials to empirically demonstrate reliability was a noteworthy improvement introduced in DSM-III. The design and implementation strategy of the DSM-5 Field Trials represent several changes over approaches used for DSM-III and DSM-IV, particularly in obtaining data on the precision of kappa reliability estimates (a statistical measure that assesses level of agreement between raters that corrects for chance agreement due to prevalence rates) in the context of clinical settings with high levels of diagnostic comorbidity. For DSM-5, field trials were extended by using two distinctive designs: one in large, diverse medical-academic settings, and the other in routine clinical practices. The former capitalized on the need for large sample sizes to test hypotheses on reliability and clinical utility of a range of diagnoses in a

variety of patient populations; the latter supplied valuable information about how proposed revisions performed in everyday clinical settings among a diverse sample of DSM users. It is anticipated that future clinical and basic research studies will focus on the validity of the revised categorical diagnostic criteria and the underlying dimensional features of these disorders (including those now being explored by the NIMH Research Domain Criteria initiative).

The medical-academic field trials were conducted at 11 North American medical-academic sites and assessed the reliability, feasibility, and clinical utility of select revisions, with priority given to those that represented the greatest degree of change from DSM-IV or those potentially having the greatest public health impact. The full clinical patient populations coming to each site were screened for DSM-IV diagnoses or qualifying symptoms likely to predict several specific DSM-5 disorders of interest. Stratified samples of four to seven specific disorders, plus a stratum containing a representative sample of all other diagnoses, were identified for each site. Patients consented to the study and were randomly assigned for a clinical interview by a clinician blind to the diagnosis, followed by a second interview with a clinician blind to previous diagnoses. Patients first filled out a computer-assisted inventory of cross-cutting symptoms in more than a dozen psychological domains. These inventories were scored by a central server, and results were provided to clinicians before they conducted a typical clinical interview (with no structured protocol). Clinicians were required to score the presence of qualifying criteria on a computer-assisted DSM-5 diagnostic checklist, determine diagnoses, score the severity of the diagnosis, and submit all data to the central Web-based server. This study design allowed the calculation of the degree to which two independent clinicians could agree on a diagnosis (using the intraclass kappa statistic) and the agreement of a single patient or two different clinicians on two separate ratings of cross-cutting symptoms, personality traits, disability, and diagnostic severity measures (using intraclass correlation coefficients) along with information on the precision of these estimates of reliability. It was also possible to assess the prevalence rates of both DSM-IV and DSM-5 conditions in the respective clinical populations.

The routine clinical practice field trials involved recruitment of individual psychiatrists and other mental health clinicians. A volunteer sample was recruited that included generalist and specialty psychiatrists, psychologists, licensed clinical social workers, counselors, marriage and family therapists, and advanced practice psychiatric mental health nurses. The field trials provided exposure of the proposed DSM-5 diagnoses and dimensional measures to a wide range of clinicians to assess their feasibility and clinical utility.

Public and Professional Review

In 2010, the APA launched a unique Web site to facilitate public and professional input into DSM-5. All draft diagnostic criteria and proposed changes in organization were posted on www.dsm5.org for a 2-month comment period. Feedback totaled more than 8,000 submissions, which were systematically reviewed by each of the 13 work groups, whose members, where appropriate, integrated questions and comments into discussions of draft revisions and plans for field trial testing. After revisions to the initial draft criteria and proposed chapter organization, a second posting occurred in 2011. Work groups considered feedback from both Web postings and the results of the DSM-5 Field Trials when drafting proposed final criteria, which were posted on the Web site for a third and final time in 2012. These three iterations of external review produced more than 13,000 individually signed comments on the Web site that were received and reviewed by the work groups, plus thousands of organized petition signers for and against some proposed revisions, all of which allowed the task force to actively address concerns of DSM users, as well as patients and advocacy groups, and ensure that clinical utility remained a high priority.

Expert Review

The members of the 13 work groups, representing expertise in their respective areas, collaborated with advisors and reviewers under the overall direction of the DSM-5 Task

Force to draft the diagnostic criteria and accompanying text. This effort was supported by a team of APA Division of Research staff and developed through a network of text coordinators from each work group. The preparation of the text was coordinated by the text editor, working in close collaboration with the work groups and under the direction of the task force chairs. The Scientific Review Committee (SRC) was established to provide a scientific peer review process that was external to that of the work groups. The SRC chair, vice-chair, and six committee members were charged with reviewing the degree to which the proposed changes from DSM-IV could be supported with scientific evidence. Each proposal for diagnostic revision required a memorandum of evidence for change prepared by the work group and accompanied by a summary of supportive data organized around validators for the proposed diagnostic criteria (i.e., antecedent validators such as familial aggregation, concurrent validators such as biological markers, and prospective validators such as response to treatment or course of illness). The submissions were reviewed by the SRC and scored according to the strength of the supportive scientific data. Other justifications for change, such as those arising from clinical experience or need or from a conceptual reframing of diagnostic categories, were generally seen as outside the purview of the SRC. The reviewers' scores, which varied substantially across the different proposals, and an accompanying brief commentary were then returned to the APA Board of Trustees and the work groups for consideration and response.

The Clinical and Public Health Committee (CPHC), composed of a chair, vice-chair, and six members, was appointed to consider additional clinical utility, public health, and logical clarification issues for criteria that had not yet accumulated the type or level of evidence deemed sufficient for change by the SRC. This review process was particularly important for DSM-IV disorders with known deficiencies for which proposed remedies had neither been previously considered in the DSM revision process nor been subjected to replicated research studies. These selected disorders were evaluated by four to five external reviewers, and the blinded results were reviewed by CPHC members, who in turn made recommendations to the APA Board of Trustees and the work groups.

Forensic reviews by the members of the APA Council on Psychiatry and Law were conducted for disorders frequently appearing in forensic environments and ones with high potential for influencing civil and criminal judgments in courtroom settings. Work groups also added forensic experts as advisors in pertinent areas to complement expertise provided by the Council on Psychiatry and Law.

The work groups themselves were charged with the responsibility to review the entire research literature surrounding a diagnostic area, including old, revised, and new diagnostic criteria, in an intensive 6-year review process to assess the pros and cons of making either small iterative changes or major conceptual changes to address the inevitable reification that occurs with diagnostic conceptual approaches that persist over several decades. Such changes included the merger of previously separate diagnostic areas into more dimensional spectra, such as that which occurred with autism spectrum disorder, substance use disorders, sexual dysfunctions, and somatic symptom and related disorders. Other changes included correcting flaws that had become apparent over time in the choice of operational criteria for some disorders. These types of changes posed particular challenges to the SRC and CPHC review processes, which were not constructed to evaluate the validity of DSM-IV diagnostic criteria. However, the DSM-5 Task Force, which had reviewed proposed changes and had responsibility for reviewing the text describing each disorder contemporaneously with the work groups during this period, was in a unique position to render an informed judgment on the scientific merits of such revisions. Furthermore, many of these major changes were subject to field trial testing, although comprehensive testing of all proposed changes could not be accommodated by such testing because of time limitations and availability of resources.

A final recommendation from the task force was then provided to the APA Board of Trustees and the APA Assembly's Committee on DSM-5 to consider some of the clinical utility and feasibility features of the proposed revisions. The assembly is a deliberative

body of the APA representing the district branches and wider membership that is composed of psychiatrists from throughout the United States who provide geographic, practice size, and interest-based diversity. The Committee on DSM-5 is a committee made up of a diverse group of assembly leaders.

Following all of the preceding review steps, an executive "summit committee" session was held to consolidate input from review and assembly committee chairs, task force chairs, a forensic advisor, and a statistical advisor, for a preliminary review of each disorder by the assembly and APA Board of Trustees executive committees. This preceded a preliminary review by the full APA Board of Trustees. The assembly voted, in November 2012, to recommend that the board approve the publication of DSM-5, and the APA Board of Trustees approved its publication in December 2012. The many experts, reviewers, and advisors who contributed to this process are listed in the Appendix.

Organizational Structure

The individual disorder definitions that constitute the operationalized sets of diagnostic criteria provide the core of DSM-5 for clinical and research purposes. These criteria have been subjected to scientific review, albeit to varying degrees, and many disorders have undergone field testing for interrater reliability. In contrast, the classification of disorders (the way in which disorders are grouped, which provides a high-level organization for the manual) has not generally been thought of as scientifically significant, despite the fact that judgments had to be made when disorders were initially divided into chapters for DSM-III.

DSM is a medical classification of disorders and as such serves as a historically determined cognitive schema imposed on clinical and scientific information to increase its comprehensibility and utility. Not surprisingly, as the foundational science that ultimately led to DSM-III has approached a half-century in age, challenges have begun to emerge for clinicians and scientists alike that are inherent in the DSM structure rather than in the description of any single disorder. These challenges include high rates of comorbidity within and across DSM chapters, an excessive use of and need to rely on "not otherwise specified" (NOS) criteria, and a growing inability to integrate DSM disorders with the results of genetic studies and other scientific findings.

As the APA and the WHO began to plan their respective revisions of the DSM and the *International Classification of Disorders* (ICD), both considered the possibility of improving clinical utility (e.g., by helping to explain apparent comorbidity) and facilitating scientific investigation by rethinking the organizational structures of both publications in a linear system designated by alphanumeric codes that sequence chapters according to some rational and relational structure. It was critical to both the DSM-5 Task Force and the WHO International Advisory Group on the revision of the ICD-10 Section on Mental and Behavioral Disorders that the revisions to the organization enhance clinical utility and remain within the bounds of well-replicated scientific information. Although the need for reform seemed apparent, it was important to respect the state of the science as well as the challenge that overly rapid change would pose for the clinical and research communities. In that spirit, revision of the organization was approached as a conservative, evolutionary diagnostic reform that would be guided by emerging scientific evidence on the relationships between disorder groups. By reordering and regrouping the existing disorders, the revised structure is meant to stimulate new clinical perspectives and to encourage researchers to identify the psychological and physiological cross-cutting factors that are not bound by strict categorical designations.

The use of DSM criteria has the clear virtue of creating a common language for communication between clinicians about the diagnosis of disorders. The official criteria and disorders that were determined to have accepted clinical applicability are located in Section II of the manual. However, it should be noted that these diagnostic criteria and their

relationships within the classification are based on current research and may need to be modified as new evidence is gathered by future research both within and across the domains of proposed disorders. "Conditions for Further Study," described in Section III, are those for which we determined that the scientific evidence is not yet available to support widespread clinical use. These diagnostic criteria are included to highlight the evolution and direction of scientific advances in these areas to stimulate further research.

With any ongoing review process, especially one of this complexity, different viewpoints emerge, and an effort was made to consider various viewpoints and, when warranted, accommodate them. For example, personality disorders are included in both Sections II and III. Section II represents an update of the text associated with the same criteria found in DSM-IV-TR, whereas Section III includes the proposed research model for personality disorder diagnosis and conceptualization developed by the DSM-5 Personality and Personality Disorders Work Group. As this field evolves, it is hoped that both versions will serve clinical practice and research initiatives.

Harmonization With ICD-11

The groups tasked with revising the DSM and ICD systems shared the overarching goal of harmonizing the two classifications as much as possible, for the following reasons:

- The existence of two major classifications of mental disorders hinders the collection and use of national health statistics, the design of clinical trials aimed at developing new treatments, and the consideration of global applicability of the results by international regulatory agencies.
- More broadly, the existence of two classifications complicates attempts to replicate scientific results across national boundaries.
- Even when the intention was to identify identical patient populations, DSM-IV and ICD-10 diagnoses did not always agree.

Early in the course of the revisions, it became apparent that a shared organizational structure would help harmonize the classifications. In fact, the use of a shared framework helped to integrate the work of DSM and ICD work groups and to focus on scientific issues. The DSM-5 organization and the proposed linear structure of the ICD-11 have been endorsed by the leadership of the NIMH Research Domain Criteria (RDoC) project as consistent with the initial overall structure of that project.

Of course, principled disagreements on the classification of psychopathology and on specific criteria for certain disorders were expected given the current state of scientific knowledge. However, most of the salient differences between the DSM and the ICD classifications do not reflect real scientific differences, but rather represent historical by-products of independent committee processes.

To the surprise of participants in both revision processes, large sections of the content fell relatively easily into place, reflecting real strengths in some areas of the scientific literature, such as epidemiology, analyses of comorbidity, twin studies, and certain other genetically informed designs. When disparities emerged, they almost always reflected the need to make a judgment about where to place a disorder in the face of incomplete—or, more often, conflicting—data. Thus, for example, on the basis of patterns of symptoms, comorbidity, and shared risk factors, attention-deficit/hyperactivity disorder (ADHD) was placed with neurodevelopmental disorders, but the same data also supported strong arguments to place ADHD within disruptive, impulse-control, and conduct disorders. These issues were settled with the preponderance of evidence (most notably validators approved by the DSM-5 Task Force). The work groups recognize, however, that future discoveries might change the placement as well as the contours of individual disorders and, furthermore, that the simple and linear organization that best supports clinical practice

may not fully capture the complexity and heterogeneity of mental disorders. The revised organization is coordinated with the mental and behavioral disorders chapter (Chapter V) of ICD-11, which will utilize an expanded numeric–alphanumeric coding system. However, the official coding system in use in the United States at the time of publication of this manual is that of the *International Classification of Diseases, Ninth Revision, Clinical Modification* (ICD-9-CM)—the U.S. adaptation of ICD-9. *International Classification of Diseases, Tenth Revision, Clinical Modification* (ICD-10-CM), adapted from ICD-10, is scheduled for implementation in the United States in October 2014. Given the impending release of ICD-11, it was decided that this iteration, and not ICD-10, would be the most relevant on which to focus harmonization. However, given that adoption of the ICD-9-CM coding system will remain at the time of the DSM-5 release, it will be necessary to use the ICD-9-CM codes. Furthermore, given that DSM-5's organizational structure reflects the anticipated structure of ICD-11, the eventual ICD-11 codes will follow the sequential order of diagnoses in the DSM-5 chapter structure more closely. At present, both the ICD-9-CM and the ICD-10-CM codes have been indicated for each disorder. These codes will not be in sequential order throughout the manual because they were assigned to complement earlier organizational structures.

Dimensional Approach to Diagnosis

Structural problems rooted in the basic design of the previous DSM classification, constructed of a large number of narrow diagnostic categories, have emerged in both clinical practice and research. Relevant evidence comes from diverse sources, including studies of comorbidity and the substantial need for not otherwise specified diagnoses, which represent the majority of diagnoses in areas such as eating disorders, personality disorders, and autism spectrum disorder. Studies of both genetic and environmental risk factors, whether based on twin designs, familial transmission, or molecular analyses, also raise concerns about the categorical structure of the DSM system. Because the previous DSM approach considered each diagnosis as categorically separate from health and from other diagnoses, it did not capture the widespread sharing of symptoms and risk factors across many disorders that is apparent in studies of comorbidity. Earlier editions of DSM focused on excluding false-positive results from diagnoses; thus, its categories were overly narrow, as is apparent from the widespread need to use NOS diagnoses. Indeed, the once plausible goal of identifying homogeneous populations for treatment and research resulted in narrow diagnostic categories that did not capture clinical reality, symptom heterogeneity within disorders, and significant sharing of symptoms across multiple disorders. The historical aspiration of achieving diagnostic homogeneity by progressive subtyping within disorder categories no longer is sensible; like most common human ills, mental disorders are heterogeneous at many levels, ranging from genetic risk factors to symptoms.

Related to recommendations about alterations in the chapter structure of DSM-5, members of the diagnostic spectra study group examined whether scientific validators could inform possible new groupings of related disorders within the existing categorical framework. Eleven such indicators were recommended for this purpose: shared neural substrates, family traits, genetic risk factors, specific environmental risk factors, biomarkers, temperamental antecedents, abnormalities of emotional or cognitive processing, symptom similarity, course of illness, high comorbidity, and shared treatment response. These indicators served as empirical guidelines to inform decision making by the work groups and the task force about how to cluster disorders to maximize their validity and clinical utility.

A series of papers was developed and published in a prominent international journal (*Psychological Medicine*, Vol. 39, 2009) as part of both the DSM-5 and the ICD-11 developmental processes to document that such validators were most useful for suggesting large groupings of disorders rather than for "validating" individual disorder diagnostic criteria. The regrouping of mental disorders in DSM-5 is intended to enable future research to en-

hance understanding of disease origins and pathophysiological commonalities between disorders and provide a base for future replication wherein data can be reanalyzed over time to continually assess validity. Ongoing revisions of DSM-5 will make it a "living document," adaptable to future discoveries in neurobiology, genetics, and epidemiology.

On the basis of the published findings of this common DSM-5 and ICD-11 analysis, it was demonstrated that clustering of disorders according to what has been termed *internalizing* and *externalizing* factors represents an empirically supported framework. Within both the internalizing group (representing disorders with prominent anxiety, depressive, and somatic symptoms) and the externalizing group (representing disorders with prominent impulsive, disruptive conduct, and substance use symptoms), the sharing of genetic and environmental risk factors, as shown by twin studies, likely explains much of the systematic comorbidities seen in both clinical and community samples. The adjacent placement of "internalizing disorders," characterized by depressed mood, anxiety, and related physiological and cognitive symptoms, should aid in developing new diagnostic approaches, including dimensional approaches, while facilitating the identification of biological markers. Similarly, adjacencies of the "externalizing group," including disorders exhibiting antisocial behaviors, conduct disturbances, addictions, and impulse-control disorders, should encourage advances in identifying diagnoses, markers, and underlying mechanisms.

Despite the problem posed by categorical diagnoses, the DSM-5 Task Force recognized that it is premature scientifically to propose alternative definitions for most disorders. The organizational structure is meant to serve as a bridge to new diagnostic approaches without disrupting current clinical practice or research. With support from DSM-associated training materials, the National Institutes of Health other funding agencies, and scientific publications, the more dimensional DSM-5 approach and organizational structure can facilitate research across current diagnostic categories by encouraging broad investigations within the proposed chapters and across adjacent chapters. Such a reformulation of research goals should also keep DSM-5 central to the development of dimensional approaches to diagnosis that will likely supplement or supersede current categorical approaches in coming years.

Developmental and Lifespan Considerations

To improve clinical utility, DSM-5 is organized on developmental and lifespan considerations. It begins with diagnoses thought to reflect developmental processes that manifest early in life (e.g., neurodevelopmental and schizophrenia spectrum and other psychotic disorders), followed by diagnoses that more commonly manifest in adolescence and young adulthood (e.g., bipolar, depressive, and anxiety disorders), and ends with diagnoses relevant to adulthood and later life (e.g., neurocognitive disorders). A similar approach has been taken, where possible, within each chapter. This organizational structure facilitates the comprehensive use of lifespan information as a way to assist in diagnostic decision making.

The proposed organization of chapters of DSM-5, after the neurodevelopmental disorders, is based on groups of internalizing (emotional and somatic) disorders, externalizing disorders, neurocognitive disorders, and other disorders. It is hoped that this organization will encourage further study of underlying pathophysiological processes that give rise to diagnostic comorbidity and symptom heterogeneity. Furthermore, by arranging disorder clusters to mirror clinical reality, DSM-5 should facilitate identification of potential diagnoses by non–mental health specialists, such as primary care physicians.

The organizational structure of DSM-5, along with ICD harmonization, is designed to provide better and more flexible diagnostic concepts for the next epoch of research and to serve as a useful guide to clinicians in explaining to patients why they might have received multiple diagnoses or why they might have received additional or altered diagnoses over their lifespan.

Cultural Issues

Mental disorders are defined in relation to cultural, social, and familial norms and values. Culture provides interpretive frameworks that shape the experience and expression of the symptoms, signs, and behaviors that are criteria for diagnosis. Culture is transmitted, revised, and recreated within the family and other social systems and institutions. Diagnostic assessment must therefore consider whether an individual's experiences, symptoms, and behaviors differ from sociocultural norms and lead to difficulties in adaptation in the cultures of origin and in specific social or familial contexts. Key aspects of culture relevant to diagnostic classification and assessment have been considered in the development of DSM-5.

In Section III, the "Cultural Formulation" contains a detailed discussion of culture and diagnosis in DSM-5, including tools for in-depth cultural assessment. In the Appendix, the "Glossary of Cultural Concepts of Distress" provides a description of some common cultural syndromes, idioms of distress, and causal explanations relevant to clinical practice.

The boundaries between normality and pathology vary across cultures for specific types of behaviors. Thresholds of tolerance for specific symptoms or behaviors differ across cultures, social settings, and families. Hence, the level at which an experience becomes problematic or pathological will differ. The judgment that a given behavior is abnormal and requires clinical attention depends on cultural norms that are internalized by the individual and applied by others around them, including family members and clinicians. Awareness of the significance of culture may correct mistaken interpretations of psychopathology, but culture may also contribute to vulnerability and suffering (e.g., by amplifying fears that maintain panic disorder or health anxiety). Cultural meanings, habits, and traditions can also contribute to either stigma or support in the social and familial response to mental illness. Culture may provide coping strategies that enhance resilience in response to illness, or suggest help seeking and options for accessing health care of various types, including alternative and complementary health systems. Culture may influence acceptance or rejection of a diagnosis and adherence to treatments, affecting the course of illness and recovery. Culture also affects the conduct of the clinical encounter; as a result, cultural differences between the clinician and the patient have implications for the accuracy and acceptance of diagnosis as well as for treatment decisions, prognostic considerations, and clinical outcomes.

Historically, the construct of the culture-bound syndrome has been a key interest of cultural psychiatry. In DSM-5, this construct has been replaced by three concepts that offer greater clinical utility:

1. *Cultural syndrome* is a cluster or group of co-occurring, relatively invariant symptoms found in a specific cultural group, community, or context (e.g., *ataque de nervios*). The syndrome may or may not be recognized as an illness within the culture (e.g., it might be labeled in various ways), but such cultural patterns of distress and features of illness may nevertheless be recognizable by an outside observer.
2. *Cultural idiom of distress* is a linguistic term, phrase, or way of talking about suffering among individuals of a cultural group (e.g., similar ethnicity and religion) referring to shared concepts of pathology and ways of expressing, communicating, or naming essential features of distress (e.g., *kufungisisa*). An idiom of distress need not be associated with specific symptoms, syndromes, or perceived causes. It may be used to convey a wide range of discomfort, including everyday experiences, subclinical conditions, or suffering due to social circumstances rather than mental disorders. For example, most cultures have common bodily idioms of distress used to express a wide range of suffering and concerns.
3. *Cultural explanation or perceived cause* is a label, attribution, or feature of an explanatory model that provides a culturally conceived etiology or cause for symptoms, illness, or distress (e.g., *maladi moun*). Causal explanations may be salient features of folk classifications of disease used by laypersons or healers.

These three concepts (for which discussion and examples are provided in Section III and the Appendix) suggest cultural ways of understanding and describing illness experiences that can be elicited in the clinical encounter. They influence symptomatology, help seeking, clinical presentations, expectations of treatment, illness adaptation, and treatment response. The same cultural term often serves more than one of these functions.

Gender Differences

Sex and gender differences as they relate to the causes and expression of medical conditions are established for a number of diseases, including selected mental disorders. Revisions to DSM-5 included review of potential differences between men and women in the expression of mental illness. In terms of nomenclature, *sex differences* are variations attributable to an individual's reproductive organs and XX or XY chromosomal complement. *Gender differences* are variations that result from biological sex as well as an individual's self-representation that includes the psychological, behavioral, and social consequences of one's perceived gender. The term *gender differences* is used in DSM-5 because, more commonly, the differences between men and women are a result of both biological sex and individual self-representation. However, some of the differences are based on only biological sex.

Gender can influence illness in a variety of ways. First, it may exclusively determine whether an individual is at risk for a disorder (e.g., as in premenstrual dysphoric disorder). Second, gender may moderate the overall risk for development of a disorder as shown by marked gender differences in the prevalence and incidence rates for selected mental disorders. Third, gender may influence the likelihood that particular symptoms of a disorder are experienced by an individual. Attention-deficit/hyperactivity disorder is an example of a disorder with differences in presentation that are most commonly experienced by boys or girls. Gender likely has other effects on the experience of a disorder that are indirectly relevant to psychiatric diagnosis. It may be that certain symptoms are more readily endorsed by men or women, and that this contributes to differences in service provision (e.g., women may be more likely to recognize a depressive, bipolar, or anxiety disorder and endorse a more comprehensive list of symptoms than men).

Reproductive life cycle events, including estrogen variations, also contribute to gender differences in risk and expression of illness. Thus, a specifier for postpartum onset of mania or major depressive episode denotes a time frame wherein women may be at increased risk for the onset of an illness episode. In the case of sleep and energy, alterations are often normative postpartum and thus may have lower diagnostic reliability in postpartum women.

The manual is configured to include information on gender at multiple levels. If there are gender-specific symptoms, they have been added to the diagnostic criteria. A gender-related specifier, such as perinatal onset of a mood episode, provides additional information on gender and diagnosis. Finally, other issues that are pertinent to diagnosis and gender considerations can be found in the section "Gender-Related Diagnostic Issues."

Use of Other Specified and Unspecified Disorders

To enhance diagnostic specificity, DSM-5 replaces the previous NOS designation with two options for clinical use: *other specified disorder* and *unspecified disorder*. The other specified disorder category is provided to allow the clinician to communicate the specific reason that the presentation does not meet the criteria for any specific category within a diagnostic class. This is done by recording the name of the category, followed by the specific reason. For example, for an individual with clinically significant depressive symptoms lasting 4 weeks but whose symptomatology falls short of the diagnostic threshold for a major depressive episode, the clinician would record "other specified depressive disorder, depressive episode with insufficient symptoms." If the clinician chooses not to specify the

reason that the criteria are not met for a specific disorder, then "unspecified depressive disorder" would be diagnosed. Note that the differentiation between other specified and unspecified disorders is based on the clinician's decision, providing maximum flexibility for diagnosis. Clinicians do not have to differentiate between other specified and unspecified disorders based on some feature of the presentation itself. When the clinician determines that there is evidence to specify the nature of the clinical presentation, the other specified diagnosis can be given. When the clinician is not able to further specify and describe the clinical presentation, the unspecified diagnosis can be given. This is left entirely up to clinical judgment.

For a more detailed discussion of how to use other specified and unspecified designations, see "Use of the Manual" in Section I.

The Multiaxial System

Despite widespread use and its adoption by certain insurance and governmental agencies, the multiaxial system in DSM-IV was not required to make a mental disorder diagnosis. A nonaxial assessment system was also included that simply listed the appropriate Axis I, II, and III disorders and conditions without axial designations. DSM-5 has moved to a nonaxial documentation of diagnosis (formerly Axes I, II, and III), with separate notations for important psychosocial and contextual factors (formerly Axis IV) and disability (formerly Axis V). This revision is consistent with the DSM-IV text that states, "The multiaxial distinction among Axis I, Axis II, and Axis III disorders does not imply that there are fundamental differences in their conceptualization, that mental disorders are unrelated to physical or biological factors or processes, or that general medical conditions are unrelated to behavioral or psychosocial factors or processes." The approach of separately noting diagnosis from psychosocial and contextual factors is also consistent with established WHO and ICD guidance to consider the individual's functional status separately from his or her diagnoses or symptom status. In DSM-5, Axis III has been combined with Axes I and II. Clinicians should continue to list medical conditions that are important to the understanding or management of an individual's mental disorder(s).

DSM-IV Axis IV covered psychosocial and environmental problems that may affect the diagnosis, treatment, and prognosis of mental disorders. Although this axis provided helpful information, even if it was not used as frequently as intended, the DSM-5 Task Force recommended that DSM-5 should not develop its own classification of psychosocial and environmental problems, but rather use a selected set of the ICD-9-CM V codes and the new Z codes contained in ICD-10-CM. The ICD-10 Z codes were examined to determine which are most relevant to mental disorders and also to identify gaps.

DSM-IV Axis V consisted of the Global Assessment of Functioning (GAF) scale, representing the clinician's judgment of the individual's overall level of "functioning on a hypothetical continuum of mental health–illness." It was recommended that the GAF be dropped from DSM-5 for several reasons, including its conceptual lack of clarity (i.e., including symptoms, suicide risk, and disabilities in its descriptors) and questionable psychometrics in routine practice. In order to provide a global measure of disability, the WHO Disability Assessment Schedule (WHODAS) is included, for further study, in Section III of DSM-5 (see the chapter "Assessment Measures"). The WHODAS is based on the International Classification of Functioning, Disability and Health (ICF) for use across all of medicine and health care. The WHODAS (version 2.0), and a modification developed for children/adolescents and their parents by the Impairment and Disability Study Group were included in the DSM-5 field trial.

Online Enhancements

It was challenging to determine what to include in the print version of DSM-5 to be most clinically relevant and useful and at the same time maintain a manageable size. For this reason, the inclusion of clinical rating scales and measures in the print edition is limited to those considered most relevant. Additional assessment measures used in the field trials are available online (www.psychiatry.org/dsm5), linked to the relevant disorders. The Cultural Formulation Interview, Cultural Formulation Interview—Informant Version, and supplementary modules to the core Cultural Formulation Interview are also available online at www.psychiatry.org/dsm5.

DSM-5 is available as an online subscription at PsychiatryOnline.org as well as an e-book. The online component contains modules and assessment tools to enhance the diagnostic criteria and text. Also available online is a complete set of supportive references as well as additional helpful information. The organizational structure of DSM-5, its use of dimensional measures, and compatibility with ICD codes will allow it to be readily adaptable to future scientific discoveries and refinements in its clinical utility. DSM-5 will be analyzed over time to continually assess its validity and enhance its value to clinicians.

Use of the Manual

The introduction contains much of the history and developmental process of the DSM-5 revision. This section is designed to provide a practical guide to using DSM-5, particularly in clinical practice. The primary purpose of DSM-5 is to assist trained clinicians in the diagnosis of their patients' mental disorders as part of a case formulation assessment that leads to a fully informed treatment plan for each individual. The symptoms contained in the respective diagnostic criteria sets do not constitute comprehensive definitions of underlying disorders, which encompass cognitive, emotional, behavioral, and physiological processes that are far more complex than can be described in these brief summaries. Rather, they are intended to summarize characteristic syndromes of signs and symptoms that point to an underlying disorder with a characteristic developmental history, biological and environmental risk factors, neuropsychological and physiological correlates, and typical clinical course.

Approach to Clinical Case Formulation

The case formulation for any given patient must involve a careful clinical history and concise summary of the social, psychological, and biological factors that may have contributed to developing a given mental disorder. Hence, it is not sufficient to simply check off the symptoms in the diagnostic criteria to make a mental disorder diagnosis. Although a systematic check for the presence of these criteria as they apply to each patient will assure a more reliable assessment, the relative severity and valence of individual criteria and their contribution to a diagnosis require clinical judgment. The symptoms in our diagnostic criteria are part of the relatively limited repertoire of human emotional responses to internal and external stresses that are generally maintained in a homeostatic balance without a disruption in normal functioning. It requires clinical training to recognize when the combination of predisposing, precipitating, perpetuating, and protective factors has resulted in a psychopathological condition in which physical signs and symptoms exceed normal ranges. The ultimate goal of a clinical case formulation is to use the available contextual and diagnostic information in developing a comprehensive treatment plan that is informed by the individual's cultural and social context. However, recommendations for the selection and use of the most appropriate evidence-based treatment options for each disorder are beyond the scope of this manual.

Although decades of scientific effort have gone into developing the diagnostic criteria sets for the disorders included in Section II, it is well recognized that this set of categorical diagnoses does not fully describe the full range of mental disorders that individuals experience and present to clinicians on a daily basis throughout the world. As noted previously in the introduction, the range of genetic/environmental interactions over the course of human development affecting cognitive, emotional and behavioral function is virtually limitless. As a result, it is impossible to capture the full range of psychopathology in the categorical diagnostic categories that we are now using. Hence, it is also necessary to include "other specified/unspecified" disorder options for presentations that do not fit exactly into the diagnostic boundaries of disorders in each chapter. In an emergency department setting, it may be possible to identify only the most prominent symptom expressions associated with a particular chapter—for example, delusions, hallucinations,

mania, depression, anxiety, substance intoxication, or neurocognitive symptoms—so that an "unspecified" disorder in that category is identified until a fuller differential diagnosis is possible.

Definition of a Mental Disorder

Each disorder identified in Section II of the manual (excluding those in the chapters entitled "Medication-Induced Movement Disorders and Other Adverse Effects of Medication" and "Other Conditions That May Be a Focus of Clinical Attention") must meet the definition of a mental disorder. Although no definition can capture all aspects of all disorders in the range contained in DSM-5, the following elements are required:

> A mental disorder is a syndrome characterized by clinically significant disturbance in an individual's cognition, emotion regulation, or behavior that reflects a dysfunction in the psychological, biological, or developmental processes underlying mental functioning. Mental disorders are usually associated with significant distress or disability in social, occupational, or other important activities. An expectable or culturally approved response to a common stressor or loss, such as the death of a loved one, is not a mental disorder. Socially deviant behavior (e.g., political, religious, or sexual) and conflicts that are primarily between the individual and society are not mental disorders unless the deviance or conflict results from a dysfunction in the individual, as described above.

The diagnosis of a mental disorder should have clinical utility: it should help clinicians to determine prognosis, treatment plans, and potential treatment outcomes for their patients. However, the diagnosis of a mental disorder is not equivalent to a need for treatment. Need for treatment is a complex clinical decision that takes into consideration symptom severity, symptom salience (e.g., the presence of suicidal ideation), the patient's distress (mental pain) associated with the symptom(s), disability related to the patient's symptoms, risks and benefits of available treatments, and other factors (e.g., psychiatric symptoms complicating other illness). Clinicians may thus encounter individuals whose symptoms do not meet full criteria for a mental disorder but who demonstrate a clear need for treatment or care. The fact that some individuals do not show all symptoms indicative of a diagnosis should not be used to justify limiting their access to appropriate care.

Approaches to validating diagnostic criteria for discrete categorical mental disorders have included the following types of evidence: antecedent validators (similar genetic markers, family traits, temperament, and environmental exposure), concurrent validators (similar neural substrates, biomarkers, emotional and cognitive processing, and symptom similarity), and predictive validators (similar clinical course and treatment response). In DSM-5, we recognize that the current diagnostic criteria for any single disorder will not necessarily identify a homogeneous group of patients who can be characterized reliably with all of these validators. Available evidence shows that these validators cross existing diagnostic boundaries but tend to congregate more frequently within and across adjacent DSM-5 chapter groups. Until incontrovertible etiological or pathophysiological mechanisms are identified to fully validate specific disorders or disorder spectra, the most important standard for the DSM-5 disorder criteria will be their clinical utility for the assessment of clinical course and treatment response of individuals grouped by a given set of diagnostic criteria.

This definition of mental disorder was developed for clinical, public health, and research purposes. Additional information is usually required beyond that contained in the DSM-5 diagnostic criteria in order to make legal judgments on such issues as criminal responsibility, eligibility for disability compensation, and competency (see "Cautionary Statement for Forensic Use of DSM-5" elsewhere in this manual).

Criterion for Ciinical Significance

There have been substantial efforts by the DSM-5 Task Force and the World Health Organization (WHO) to separate the concepts of mental disorder and disability (impairment in social, occupational, or other important areas of functioning). In the WHO system, the International Classification of Diseases (ICD) covers all diseases and disorders, while the International Classification of Functioning, Disability and Health (ICF) provides a separate classification of global disability. The WHO Disability Assessment Schedule (WHODAS) is based on the ICF and has proven useful as a standardized measure of disability for mental disorders. However, in the absence of clear biological markers or clinically useful measurements of severity for many mental disorders, it has not been possible to completely separate normal and pathological symptom expressions contained in diagnostic criteria. This gap in information is particularly problematic in clinical situations in which the patient's symptom presentation by itself (particularly in mild forms) is not inherently pathological and may be encountered in individuals for whom a diagnosis of "mental disorder" would be inappropriate. Therefore, a generic diagnostic criterion requiring distress or disability has been used to establish disorder thresholds, usually worded "the disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning." The text following the revised definition of a mental disorder acknowledges that this criterion may be especially helpful in determining a patient's need for treatment. Use of information from family members and other third parties (in addition to the individual) regarding the individual's performance is recommended when necessary.

Elements of a Diagnosis

Diagnostic Criteria and Descriptors

Diagnostic criteria are offered as guidelines for making diagnoses, and their use should be informed by clinical judgment. Text descriptions, including introductory sections of each diagnostic chapter, can help support diagnosis (e.g., providing differential diagnoses; describing the criteria more fully under "Diagnostic Features").

Following the assessment of diagnostic criteria, clinicians should consider the application of disorder subtypes and/or specifiers as appropriate. Severity and course specifiers should be applied to denote the individual's current presentation, but only when the full criteria are met. When full criteria are not met, clinicians should consider whether the symptom presentation meets criteria for an "other specified" or "unspecified" designation. Where applicable, specific criteria for defining disorder severity (e.g., mild, moderate, severe, extreme), descriptive features (e.g., with good to fair insight; in a controlled environment), and course (e.g., in partial remission, in full remission, recurrent) are provided with each diagnosis. On the basis of the clinical interview, text descriptions, criteria, and clinician judgment, a final diagnosis is made.

The general convention in DSM-5 is to allow multiple diagnoses to be assigned for those presentations that meet criteria for more than one DSM-5 disorder.

Subtypes and Specifiers

Subtypes and specifiers (some of which are coded in the fourth, fifth, or sixth digit) are provided for increased specificity. *Subtypes* define mutually exclusive and jointly exhaustive phenomenological subgroupings within a diagnosis and are indicated by the instruction "*Specify* whether" in the criteria set. In contrast, *specifiers* are not intended to be mutually exclusive or jointly exhaustive, and as a consequence, more than one specifier may be given. Specifiers are indicated by the instruction "*Specify*" or "*Specify if*" in the criteria set. Specifiers provide an opportunity to define a more homogeneous subgrouping of

individuals with the disorder who share certain features (e.g., major depressive disorder, with mixed features) and to convey information that is relevant to the management of the individual's disorder, such as the "with other medical comorbidity" specifier in sleep-wake disorders. Although a fifth digit is sometimes assigned to code a subtype or specifier (e.g., 294.11 [F02.81] major neurocognitive disorder due to Alzheimer's disease, with behavioral disturbance) or severity (296.21 [F32.0] major depressive disorder, single episode, mild), the majority of subtypes and specifiers included in DSM-5 cannot be coded within the ICD-9-CM and ICD-10-CM systems and are indicated only by including the subtype or specifier after the name of the disorder (e.g., social anxiety disorder [social phobia], performance type). Note that in some cases, a specifier or subtype is codable in ICD-10-CM but not in ICD-9-CM. Accordingly, in some cases the 4th or 5th character codes for the subtypes or specifiers are provided only for the ICD-10-CM coding designations.

A DSM-5 diagnosis is usually applied to the individual's current presentation; previous diagnoses from which the individual has recovered should be clearly noted as such. Specifiers indicating *course* (e.g., in partial remission, in full remission) may be listed after the diagnosis and are indicated in a number of criteria sets. Where available, *severity specifiers* are provided to guide clinicians in rating the intensity, frequency, duration, symptom count, or other severity indicator of a disorder. Severity specifiers are indicated by the instruction "*Specify* current severity" in the criteria set and include disorder-specific definitions. *Descriptive features specifiers* have also been provided in the criteria set and convey additional information that can inform treatment planning (e.g., obsessive-compulsive disorder, with poor insight). Not all disorders include course, severity, and/or descriptive features specifiers.

Medication-Induced Movement Disorders and Other Conditions That May Be a Focus of Clinical Attention

In addition to important psychosocial and environmental factors (see "The Multiaxial System" in the "Introduction" elsewhere in this manual), these chapters in Section II also contain other conditions that are not mental disorders but may be encountered by mental health clinicians. These conditions may be listed as a reason for clinical visit in addition to, or in place of, the mental disorders listed in Section II. A separate chapter is devoted to medication-induced disorders and other adverse effects of medication that may be assessed and treated by clinicians in mental health practice such as akathisia, tardive dyskinesia, and dystonia. The description of neuroleptic malignant syndrome is expanded from that provided in DSM-IV-TR to highlight the emergent and potentially life-threatening nature of this condition, and a new entry on antidepressant discontinuation syndrome is provided. An additional chapter discusses other conditions that may be a focus of clinical attention. These include relational problems, problems related to abuse and neglect, problems with adherence to treatment regimens, obesity, antisocial behavior, and malingering.

Principal Diagnosis

When more than one diagnosis for an individual is given in an inpatient setting, the principal diagnosis is the condition established after study to be chiefly responsible for occasioning the admission of the individual. When more than one diagnosis is given for an individual in an outpatient setting, the reason for visit is the condition that is chiefly responsible for the ambulatory care medical services received during the visit. In most cases, the principal diagnosis or the reason for visit is also the main focus of attention or treatment. It is often difficult (and somewhat arbitrary) to determine which diagnosis is the principal diagnosis or the reason for visit, especially when, for example, a substance-related diagnosis such as alcohol use disorder is accompanied by a non-substance-related diagnosis such as schizophrenia. For example, it may be unclear which diagnosis should

be considered "principal" for an individual hospitalized with both schizophrenia and alcohol use disorder, because each condition may have contributed equally to the need for admission and treatment. The principal diagnosis is indicated by listing it first, and the remaining disorders are listed in order of focus of attention and treatment. When the principal diagnosis or reason for visit is a mental disorder due to another medical condition (e.g., major neurocognitive disorder due to Alzheimer's disease, psychotic disorder due to malignant lung neoplasm), ICD coding rules require that the etiological medical condition be listed first. In that case, the principal diagnosis or reason for visit would be the mental disorder due to the medical condition, the second listed diagnosis. In most cases, the disorder listed as the principal diagnosis or the reason for visit is followed by the qualifying phrase "(principal diagnosis)" or "(reason for visit)."

Provisional Diagnosis

The specifier "provisional" can be used when there is a strong presumption that the full criteria will ultimately be met for a disorder but not enough information is available to make a firm diagnosis. The clinician can indicate the diagnostic uncertainty by recording "(provisional)" following the diagnosis. For example, this diagnosis might be used when an individual who appears to have a major depressive disorder is unable to give an adequate history, and thus it cannot be established that the full criteria are met. Another use of the term *provisional* is for those situations in which differential diagnosis depends exclusively on the duration of illness. For example, a diagnosis of schizophreniform disorder requires a duration of less than 6 months but of at least 1 month and can only be given provisionally if assigned before remission has occurred.

Coding and Reporting Procedures

Each disorder is accompanied by an identifying diagnostic and statistical code, which is typically used by institutions and agencies for data collection and billing purposes. There are specific recording protocols for these diagnostic codes (identified as coding notes in the text) that were established by WHO, the U.S. Centers for Medicare and Medicaid Services (CMS), and the Centers for Disease Control and Prevention's National Center for Health Statistics to ensure consistent international recording of prevalence and mortality rates for identified health conditions. For most clinicians, the codes are used to identify the diagnosis or reason for visit for CMS and private insurance service claims. The official coding system in use in the United States as of publication of this manual is ICD-9-CM. Official adoption of ICD-10-CM is scheduled to take place on October 1, 2014, and these codes, which are shown parenthetically in this manual, should not be used until the official implementation occurs. Both ICD-9-CM and ICD-10-CM codes have been listed 1) preceding the name of the disorder in the classification and 2) accompanying the criteria set for each disorder. For some diagnoses (e.g., neurocognitive and substance/medication-induced disorders), the appropriate code depends on further specification and is listed within the criteria set for the disorder, as coding notes, and, in some cases, further clarified in a section on recording procedures. The names of some disorders are followed by alternative terms enclosed in parentheses, which, in most cases, were the DSM-IV names for the disorders.

Looking to the Future: Assessment and Monitoring Tools

The various components of DSM-5 are provided to facilitate patient assessment and to aid in developing a comprehensive case formulation. Whereas the diagnostic criteria in Section II are well-established measures that have undergone extensive review, the assess-

ment tools, a cultural formulation interview, and conditions for further study included in Section III are those for which we determined that the scientific evidence is not yet available to support widespread clinical use. These diagnostic aids and criteria are included to highlight the evolution and direction of scientific advances in these areas and to stimulate further research.

Each of the measures in Section III is provided to aid in a comprehensive assessment of individuals that will contribute to a diagnosis and treatment plan tailored to the individual presentation and clinical context. Where cultural dynamics are particularly important for diagnostic assessment, the cultural formulation interview should be considered as a useful aid to communication with the individual. Cross-cutting symptom and diagnosis-specific severity measures provide quantitative ratings of important clinical areas that are designed to be used at the initial evaluation to establish a baseline for comparison with ratings on subsequent encounters to monitor changes and inform treatment planning.

The use of such measures will undoubtedly be facilitated by digital applications, and the measures are included in Section III to provide for further evaluation and development. As with each DSM edition, the diagnostic criteria and the DSM-5 classification of mental disorders reflect the current consensus on the evolving knowledge in our field.

Cautionary Statement for Forensic Use of DSM-5

Although the DSM-5 diagnostic criteria and text are primarily designed to assist clinicians in conducting clinical assessment, case formulation, and treatment planning, DSM-5 is also used as a reference for the courts and attorneys in assessing the forensic consequences of mental disorders. As a result, it is important to note that the definition of mental disorder included in DSM-5 was developed to meet the needs of clinicians, public health professionals, and research investigators rather than all of the technical needs of the courts and legal professionals. It is also important to note that DSM-5 does not provide treatment guidelines for any given disorder.

When used appropriately, diagnoses and diagnostic information can assist legal decision makers in their determinations. For example, when the presence of a mental disorder is the predicate for a subsequent legal determination (e.g., involuntary civil commitment), the use of an established system of diagnosis enhances the value and reliability of the determination. By providing a compendium based on a review of the pertinent clinical and research literature, DSM-5 may facilitate legal decision makers' understanding of the relevant characteristics of mental disorders. The literature related to diagnoses also serves as a check on ungrounded speculation about mental disorders and about the functioning of a particular individual. Finally, diagnostic information about longitudinal course may improve decision making when the legal issue concerns an individual's mental functioning at a past or future point in time.

However, the use of DSM-5 should be informed by an awareness of the risks and limitations of its use in forensic settings. When DSM-5 categories, criteria, and textual descriptions are employed for forensic purposes, there is a risk that diagnostic information will be misused or misunderstood. These dangers arise because of the imperfect fit between the questions of ultimate concern to the law and the information contained in a clinical diagnosis. In most situations, the clinical diagnosis of a DSM-5 mental disorder such as intellectual disability (intellectual developmental disorder), schizophrenia, major neurocognitive disorder, gambling disorder, or pedophilic disorder does not imply that an individual with such a condition meets legal criteria for the presence of a mental disorder or a specified legal standard (e.g., for competence, criminal responsibility, or disability). For the latter, additional information is usually required beyond that contained in the DSM-5 diagnosis, which might include information about the individual's functional impairments and how these impairments affect the particular abilities in question. It is precisely because impairments, abilities, and disabilities vary widely within each diagnostic category that assignment of a particular diagnosis does not imply a specific level of impairment or disability.

Use of DSM-5 to assess for the presence of a mental disorder by nonclinical, nonmedical, or otherwise insufficiently trained individuals is not advised. Nonclinical decision makers should also be cautioned that a diagnosis does not carry any necessary implications regarding the etiology or causes of the individual's mental disorder or the individual's degree of control over behaviors that may be associated with the disorder. Even when diminished control over one's behavior is a feature of the disorder, having the diagnosis in itself does not demonstrate that a particular individual is (or was) unable to control his or her behavior at a particular time.

SECTION II
Diagnostic Criteria and Codes

This section contains the diagnostic criteria approved for routine clinical use along with the ICD-9-CM codes (ICD-10 codes are shown parenthetically). For each mental disorder, the diagnostic criteria are followed by descriptive text to assist in diagnostic decision making. Where needed, specific recording procedures are presented with the diagnostic criteria to provide guidance in selecting the most appropriate code. In some cases, separate recording procedures for ICD-9-CM and ICD-10-CM are provided. Although not considered as official DSM-5 disorders, medication-induced movement disorders and other adverse effects of medication, as well as other conditions that may be a focus of clinical attention (including additional ICD-9-CM V codes and forthcoming ICD-10-CM Z codes), are provided to indicate other reasons for a clinical visit such as environmental factors and relational problems. These codes are adapted from ICD-9-CM and ICD-10-CM and were neither reviewed nor approved as official DSM-5 diagnoses, but can provide additional context for a clinical formulation and treatment plan. These three components—the criteria and their descriptive text, the medication-induced movement disorders and other adverse effects of medication, and the descriptions of other conditions that may be a focus of clinical attention—represent the key elements of the clinical diagnostic process and thus are presented together.

Neurodevelopmental Disorders

The neurodevelopmental disorders are a group of conditions with onset in the developmental period. The disorders typically manifest early in development, often before the child enters grade school, and are characterized by developmental deficits that produce impairments of personal, social, academic, or occupational functioning. The range of developmental deficits varies from very specific limitations of learning or control of executive functions to global impairments of social skills or intelligence. The neurodevelopmental disorders frequently co-occur; for example, individuals with autism spectrum disorder often have intellectual disability (intellectual developmental disorder), and many children with attention-deficit/hyperactivity disorder (ADHD) also have a specific learning disorder. For some disorders, the clinical presentation includes symptoms of excess as well as deficits and delays in achieving expected milestones. For example, autism spectrum disorder is diagnosed only when the characteristic deficits of social communication are accompanied by excessively repetitive behaviors, restricted interests, and insistence on sameness.

Intellectual disability (intellectual developmental disorder) is characterized by deficits in general mental abilities, such as reasoning, problem solving, planning, abstract thinking, judgment, academic learning, and learning from experience. The deficits result in impairments of adaptive functioning, such that the individual fails to meet standards of personal independence and social responsibility in one or more aspects of daily life, including communication, social participation, academic or occupational functioning, and personal independence at home or in community settings. Global developmental delay, as its name implies, is diagnosed when an individual fails to meet expected developmental milestones in several areas of intellectual functioning. The diagnosis is used for individuals who are unable to undergo systematic assessments of intellectual functioning, including children who are too young to participate in standardized testing. Intellectual disability may result from an acquired insult during the developmental period from, for example, a severe head injury, in which case a neurocognitive disorder also may be diagnosed.

The communication disorders include language disorder, speech sound disorder, social (pragmatic) communication disorder, and childhood-onset fluency disorder (stuttering). The first three disorders are characterized by deficits in the development and use of language, speech, and social communication, respectively. Childhood-onset fluency disorder is characterized by disturbances of the normal fluency and motor production of speech, including repetitive sounds or syllables, prolongation of consonants or vowel sounds, broken words, blocking, or words produced with an excess of physical tension. Like other neurodevelopmental disorders, communication disorders begin early in life and may produce lifelong functional impairments.

Autism spectrum disorder is characterized by persistent deficits in social communication and social interaction across multiple contexts, including deficits in social reciprocity, nonverbal communicative behaviors used for social interaction, and skills in developing, maintaining, and understanding relationships. In addition to the social communication deficits, the diagnosis of autism spectrum disorder requires the presence of restricted, repetitive patterns of behavior, interests, or activities. Because symptoms change with development and may be masked by compensatory mechanisms, the diagnostic criteria may

be met based on historical information, although the current presentation must cause significant impairment.

Within the diagnosis of autism spectrum disorder, individual clinical characteristics are noted through the use of specifiers (with or without accompanying intellectual impairment; with or without accompanying structural language impairment; associated with a known medical/genetic or environmental/acquired condition; associated with another neurodevelopmental, mental, or behavioral disorder), as well as specifiers that describe the autistic symptoms (age at first concern; with or without loss of established skills; severity). These specifiers provide clinicians with an opportunity to individualize the diagnosis and communicate a richer clinical description of the affected individuals. For example, many individuals previously diagnosed with Asperger's disorder would now receive a diagnosis of autism spectrum disorder without language or intellectual impairment.

ADHD is a neurodevelopmental disorder defined by impairing levels of inattention, disorganization, and/or hyperactivity-impulsivity. Inattention and disorganization entail inability to stay on task, seeming not to listen, and losing materials, at levels that are inconsistent with age or developmental level. Hyperactivity-impulsivity entails overactivity, fidgeting, inability to stay seated, intruding into other people's activities, and inability to wait—symptoms that are excessive for age or developmental level. In childhood, ADHD frequently overlaps with disorders that are often considered to be "externalizing disorders," such as oppositional defiant disorder and conduct disorder. ADHD often persists into adulthood, with resultant impairments of social, academic and occupational functioning.

The neurodevelopmental motor disorders include developmental coordination disorder, stereotypic movement disorder, and tic disorders. Developmental coordination disorder is characterized by deficits in the acquisition and execution of coordinated motor skills and is manifested by clumsiness and slowness or inaccuracy of performance of motor skills that cause interference with activities of daily living. Stereotypic movement disorder is diagnosed when an individual has repetitive, seemingly driven, and apparently purposeless motor behaviors, such as hand flapping, body rocking, head banging, self-biting, or hitting. The movements interfere with social, academic, or other activities. If the behaviors cause self-injury, this should be specified as part of the diagnostic description. Tic disorders are characterized by the presence of motor or vocal tics, which are sudden, rapid, recurrent, nonrhythmic, sterotyped motor movements or vocalizations. The duration, presumed etiology, and clinical presentation define the specific tic disorder that is diagnosed: Tourette's disorder, persistent (chronic) motor or vocal tic disorder, provisional tic disorder, other specified tic disorder, and unspecified tic disorder. Tourette's disorder is diagnosed when the individual has multiple motor and vocal tics that have been present for at least 1 year and that have a waxing-waning symptom course.

Specific learning disorder, as the name implies, is diagnosed when there are specific deficits in an individual's ability to perceive or process information efficiently and accurately. This neurodevelopmental disorder first manifests during the years of formal schooling and is characterized by persistent and impairing difficulties with learning foundational academic skills in reading, writing, and/or math. The individual's performance of the affected academic skills is well below average for age, or acceptable performance levels are achieved only with extraordinary effort. Specific learning disorder may occur in individuals identified as intellectually gifted and manifest only when the learning demands or assessment procedures (e.g., timed tests) pose barriers that cannot be overcome by their innate intelligence and compensatory strategies. For all individuals, specific learning disorder can produce lifelong impairments in activities dependent on the skills, including occupational performance.

The use of specifiers for the neurodevelopmental disorder diagnoses enriches the clinical description of the individual's clinical course and current symptomatology. In addition to specifiers that describe the clinical presentation, such as age at onset or severity ratings, the neurodevelopmental disorders may include the specifier "associated with a known medical or genetic condition or environmental factor." This specifier gives clini-

cians an opportunity to document factors that may have played a role in the etiology of the disorder, as well as those that might affect the clinical course. Examples include genetic disorders, such as fragile X syndrome, tuberous sclerosis, and Rett syndrome; medical conditions such as epilepsy; and environmental factors, including very low birth weight and fetal alcohol exposure (even in the absence of stigmata of fetal alcohol syndrome).

Intellectual Disabilities

Intellectual Disability (Intellectual Developmental Disorder)

Diagnostic Criteria

Intellectual disability (intellectual developmental disorder) is a disorder with onset during the developmental period that includes both intellectual and adaptive functioning deficits in conceptual, social, and practical domains. The following three criteria must be met:

A. Deficits in intellectual functions, such as reasoning, problem solving, planning, abstract thinking, judgment, academic learning, and learning from experience, confirmed by both clinical assessment and individualized, standardized intelligence testing.

B. Deficits in adaptive functioning that result in failure to meet developmental and sociocultural standards for personal independence and social responsibility. Without ongoing support, the adaptive deficits limit functioning in one or more activities of daily life, such as communication, social participation, and independent living, across multiple environments, such as home, school, work, and community.

C. Onset of intellectual and adaptive deficits during the developmental period.

Note: The diagnostic term *intellectual disability* is the equivalent term for the ICD-11 diagnosis of *intellectual developmental disorders*. Although the term *intellectual disability* is used throughout this manual, both terms are used in the title to clarify relationships with other classification systems. Moreover, a federal statute in the United States (Public Law 111-256, Rosa's Law) replaces the term *mental retardation* with *intellectual disability*, and research journals use the term *intellectual disability*. Thus, *intellectual disability* is the term in common use by medical, educational, and other professions and by the lay public and advocacy groups.

Coding note: The ICD-9-CM code for intellectual disability (intellectual developmental disorder) is **319**, which is assigned regardless of the severity specifier. The ICD-10-CM code depends on the severity specifier (see below).

Specify current severity (see Table 1):

(F70) Mild
(F71) Moderate
(F72) Severe
(F73) Profound

Specifiers

The various levels of severity are defined on the basis of adaptive functioning, and not IQ scores, because it is adaptive functioning that determines the level of supports required. Moreover, IQ measures are less valid in the lower end of the IQ range.

TABLE 1 Severity levels for intellectual disability (intellectual developmental disorder)

Severity level	Conceptual domain	Social domain	Practical domain
Mild	For preschool children, there may be no obvious conceptual differences. For school-age children and adults, there are difficulties in learning academic skills involving reading, writing, arithmetic, time, or money, with support needed in one or more areas to meet age-related expectations. In adults, abstract thinking, executive function (i.e., planning, strategizing, priority setting, and cognitive flexibility), and short-term memory, as well as functional use of academic skills (e.g., reading, money management), are impaired. There is a somewhat concrete approach to problems and solutions compared with age-mates.	Compared with typically developing age-mates, the individual is immature in social interactions. For example, there may be difficulty in accurately perceiving peers' social cues. Communication, conversation, and language are more concrete or immature than expected for age. There may be difficulties regulating emotion and behavior in age-appropriate fashion; these difficulties are noticed by peers in social situations. There is limited understanding of risk in social situations; social judgment is immature for age, and the person is at risk of being manipulated by others (gullibility).	The individual may function age-appropriately in personal care. Individuals need some support with complex daily living tasks in comparison to peers. In adulthood, supports typically involve grocery shopping, transportation, home and child-care organizing, nutritious food preparation, and banking and money management. Recreational skills resemble those of age-mates, although judgment related to well-being and organization around recreation requires support. In adulthood, competitive employment is often seen in jobs that do not emphasize conceptual skills. Individuals generally need support to make health care decisions and legal decisions, and to learn to perform a skilled vocation competently. Support is typically needed to raise a family.

TABLE 1 Severity levels for intellectual disability (intellectual developmental disorder) *(continued)*

Severity level	Conceptual domain	Social domain	Practical domain
Moderate	All through development, the individual's conceptual skills lag markedly behind those of peers. For preschoolers, language and pre-academic skills develop slowly. For school-age children, progress in reading, writing, mathematics, and understanding of time and money occurs slowly across the school years and is markedly limited compared with that of peers. For adults, academic skill development is typically at an elementary level, and support is required for all use of academic skills in work and personal life. Ongoing assistance on a daily basis is needed to complete conceptual tasks of day-to-day life, and others may take over these responsibilities fully for the individual.	The individual shows marked differences from peers in social and communicative behavior across development. Spoken language is typically a primary tool for social communication but is much less complex than that of peers. Capacity for relationships is evident in ties to family and friends, and the individual may have successful friendships across life and sometimes romantic relations in adulthood. However, individuals may not perceive or interpret social cues accurately. Social judgment and decision-making abilities are limited, and caretakers must assist the person with life decisions. Friendships with typically developing peers are often affected by communication or social limitations. Significant social and communicative support is needed in work settings for success.	The individual can care for personal needs involving eating, dressing, elimination, and hygiene as an adult, although an extended period of teaching and time is needed for the individual to become independent in these areas, and reminders may be needed. Similarly, participation in all household tasks can be achieved by adulthood, although an extended period of teaching is needed, and ongoing supports will typically occur for adult-level performance. Independent employment in jobs that require limited conceptual and communication skills can be achieved, but considerable support from co-workers, supervisors, and others is needed to manage social expectations, job complexities, and ancillary responsibilities such as scheduling, transportation, health benefits, and money management. A variety of recreational skills can be developed. These typically require additional supports and learning opportunities over an extended period of time. Maladaptive behavior is present in a significant minority and causes social problems.

TABLE 1 Severity levels for intellectual disability (intellectual developmental disorder) *(continued)*

Severity level	Conceptual domain	Social domain	Practical domain
Severe	Attainment of conceptual skills is limited. The individual generally has little understanding of written language or of concepts involving numbers, quantity, time, and money. Caretakers provide extensive supports for problem solving throughout life.	Spoken language is quite limited in terms of vocabulary and grammar. Speech may be single words or phrases and may be supplemented through augmentative means. Speech and communication are focused on the here and now within everyday events. Language is used for social communication more than for explication. Individuals understand simple speech and gestural communication. Relationships with family members and familiar others are a source of pleasure and help.	The individual requires support for all activities of daily living, including meals, dressing, bathing, and elimination. The individual requires supervision at all times. The individual cannot make responsible decisions regarding well-being of self or others. In adulthood, participation in tasks at home, recreation, and work requires ongoing support and assistance. Skill acquisition in all domains involves long-term teaching and ongoing support. Maladaptive behavior, including self-injury, is present in a significant minority.
Profound	Conceptual skills generally involve the physical world rather than symbolic processes. The individual may use objects in goal-directed fashion for self-care, work, and recreation. Certain visuospatial skills, such as matching and sorting based on physical characteristics, may be acquired. However, co-occurring motor and sensory impairments may prevent functional use of objects.	The individual has very limited understanding of symbolic communication in speech or gesture. He or she may understand some simple instructions or gestures. The individual expresses his or her own desires and emotions largely through nonverbal, nonsymbolic communication. The individual enjoys relationships with well-known family members, caretakers, and familiar others, and initiates and responds to social interactions through gestural and emotional cues. Co-occurring sensory and physical impairments may prevent many social activities.	The individual is dependent on others for all aspects of daily physical care, health, and safety, although he or she may be able to participate in some of these activities as well. Individuals without severe physical impairments may assist with some daily work tasks at home, like carrying dishes to the table. Simple actions with objects may be the basis of participation in some vocational activities with high levels of ongoing support. Recreational activities may involve, for example, enjoyment in listening to music, watching movies, going out for walks, or participating in water activities, all with the support of others. Co-occurring physical and sensory impairments are frequent barriers to participation (beyond watching) in home, recreational, and vocational activities. Maladaptive behavior is present in a significant minority.

Diagnostic Features

The essential features of intellectual disability (intellectual developmental disorder) are deficits in general mental abilities (Criterion A) and impairment in everyday adaptive functioning, in comparison to an individual's age-, gender-, and socioculturally matched peers (Criterion B). Onset is during the developmental period (Criterion C). The diagnosis of intellectual disability is based on both clinical assessment and standardized testing of intellectual and adaptive functions.

Criterion A refers to intellectual functions that involve reasoning, problem solving, planning, abstract thinking, judgment, learning from instruction and experience, and practical understanding. Critical components include verbal comprehension, working memory, perceptual reasoning, quantitative reasoning, abstract thought, and cognitive efficacy. Intellectual functioning is typically measured with individually administered and psychometrically valid, comprehensive, culturally appropriate, psychometrically sound tests of intelligence. Individuals with intellectual disability have scores of approximately two standard deviations or more below the population mean, including a margin for measurement error (generally +5 points). On tests with a standard deviation of 15 and a mean of 100, this involves a score of 65–75 (70 ± 5). Clinical training and judgment are required to interpret test results and assess intellectual performance.

Factors that may affect test scores include practice effects and the "Flynn effect' (i.e., overly high scores due to out-of-date test norms). Invalid scores may result from the use of brief intelligence screening tests or group tests; highly discrepant individual subtest scores may make an overall IQ score invalid. Instruments must be normed for the individual's sociocultural background and native language. Co-occurring disorders that affect communication, language, and/or motor or sensory function may affect test scores. Individual cognitive profiles based on neuropsychological testing are more useful for understanding intellectual abilities than a single IQ score. Such testing may identify areas of relative strengths and weaknesses, an assessment important for academic and vocational planning.

IQ test scores are approximations of conceptual functioning but may be insufficient to assess reasoning in real-life situations and mastery of practical tasks. For example, a person with an IQ score above 70 may have such severe adaptive behavior problems in social judgment, social understanding, and other areas of adaptive functioning that the person's actual functioning is comparable to that of individuals with a lower IQ score. Thus, clinical judgment is needed in interpreting the results of IQ tests.

Deficits in adaptive functioning (Criterion B) refer to how well a person meets community standards of personal independence and social responsibility, in comparison to others of similar age and sociocultural background. Adaptive functioning involves adaptive reasoning in three domains: conceptual, social, and practical. The *conceptual (academic) domain* involves competence in memory, language, reading, writing, math reasoning, acquisition of practical knowledge, problem solving, and judgment in novel situations, among others. The *social domain* involves awareness of others' thoughts, feelings, and experiences; empathy; interpersonal communication skills; friendship abilities; and social judgment, among others. The *practical domain* involves learning and self-management across life settings, including personal care, job responsibilities, money management, recreation, self-management of behavior, and school and work task organization, among others. Intellectual capacity, education, motivation, socialization, personality features, vocational opportunity, cultural experience, and coexisting general medical conditions or mental disorders influence adaptive functioning.

Adaptive functioning is assessed using both clinical evaluation and individualized, culturally appropriate, psychometrically sound measures. Standardized measures are used with knowledgeable informants (e.g., parent or other family member; teacher; counselor; care provider) and the individual to the extent possible. Additional sources of information include educational, developmental, medical, and mental health evaluations. Scores from standardized measures and interview sources must be interpreted using clinical judgment. When standardized testing is difficult or impossible, because of a variety of

factors (e.g., sensory impairment, severe problem behavior), the individual may be diagnosed with unspecified intellectual disability. Adaptive functioning may be difficult to assess in a controlled setting (e.g., prisons, detention centers); if possible, corroborative information reflecting functioning outside those settings should be obtained.

Criterion B is met when at least one domain of adaptive functioning—conceptual, social, or practical—is sufficiently impaired that ongoing support is needed in order for the person to perform adequately in one or more life settings at school, at work, at home, or in the community. To meet diagnostic criteria for intellectual disability, the deficits in adaptive functioning must be directly related to the intellectual impairments described in Criterion A. Criterion C, onset during the developmental period, refers to recognition that intellectual and adaptive deficits are present during childhood or adolescence.

Associated Features Supporting Diagnosis

Intellectual disability is a heterogeneous condition with multiple causes. There may be associated difficulties with social judgment; assessment of risk; self-management of behavior, emotions, or interpersonal relationships; or motivation in school or work environments. Lack of communication skills may predispose to disruptive and aggressive behaviors. Gullibility is often a feature, involving naiveté in social situations and a tendency for being easily led by others. Gullibility and lack of awareness of risk may result in exploitation by others and possible victimization, fraud, unintentional criminal involvement, false confessions, and risk for physical and sexual abuse. These associated features can be important in criminal cases, including Atkins-type hearings involving the death penalty.

Individuals with a diagnosis of intellectual disability with co-occurring mental disorders are at risk for suicide. They think about suicide, make suicide attempts, and may die from them. Thus, screening for suicidal thoughts is essential in the assessment process. Because of a lack of awareness of risk and danger, accidental injury rates may be increased.

Prevalence

Intellectual disability has an overall general population prevalence of approximately 1%, and prevalence rates vary by age. Prevalence for severe intellectual disability is approximately 6 per 1,000.

Development and Course

Onset of intellectual disability is in the developmental period. The age and characteristic features at onset depend on the etiology and severity of brain dysfunction. Delayed motor, language, and social milestones may be identifiable within the first 2 years of life among those with more severe intellectual disability, while mild levels may not be identifiable until school age when difficulty with academic learning becomes apparent. All criteria (including Criterion C) must be fulfilled by history or current presentation. Some children under age 5 years whose presentation will eventually meet criteria for intellectual disability have deficits that meet criteria for global developmental delay.

When intellectual disability is associated with a genetic syndrome, there may be a characteristic physical appearance (as in, e.g., Down syndrome). Some syndromes have a *behavioral phenotype*, which refers to specific behaviors that are characteristic of particular genetic disorder (e.g., Lesch-Nyhan syndrome). In acquired forms, the onset may be abrupt following an illness such as meningitis or encephalitis or head trauma occurring during the developmental period. When intellectual disability results from a loss of previously acquired cognitive skills, as in severe traumatic brain injury, the diagnoses of intellectual disability and of a neurocognitive disorder may both be assigned.

Although intellectual disability is generally nonprogressive, in certain genetic disorders (e.g., Rett syndrome) there are periods of worsening, followed by stabilization, and in

others (e.g., San Phillippo syndrome) progressive worsening of intellectual function. After early childhood, the disorder is generally lifelong, although severity levels may change over time. The course may be influenced by underlying medical or genetic conditions and co-occurring conditions (e.g., hearing or visual impairments, epilepsy). Early and ongoing interventions may improve adaptive functioning throughout childhood and adulthood. In some cases, these result in significant improvement of intellectual functioning, such that the diagnosis of intellectual disability is no longer appropriate. Thus, it is common practice when assessing infants and young children to delay diagnosis of intellectual disability until after an appropriate course of intervention is provided. For older children and adults, the extent of support provided may allow for full participation in all activities of daily living and improved adaptive function. Diagnostic assessments must determine whether improved adaptive skills are the result of a stable, generalized new skill acquisition (in which case the diagnosis of intellectual disability may no longer be appropriate) or whether the improvement is contingent on the presence of supports and ongoing interventions (in which case the diagnosis of intellectual disability may still be appropriate).

Risk and Prognostic Factors

Genetic and physiological. Prenatal etiologies include genetic syndromes (e.g., sequence variations or copy number variants involving one or more genes; chromosomal disorders), inborn errors of metabolism, brain malformations, maternal disease (including placental disease), and environmental influences (e.g., alcohol, other drugs, toxins, teratogens). Perinatal causes include a variety of labor and delivery-related events leading to neonatal encephalopathy. Postnatal causes include hypoxic ischemic injury, traumatic brain injury, infections, demyelinating disorders, seizure disorders (e.g., infantile spasms), severe and chronic social deprivation, and toxic metabolic syndromes and intoxications (e.g., lead, mercury).

Culture-Related Diagnostic Issues

Intellectual disability occurs in all races and cultures. Cultural sensitivity and knowledge are needed during assessment, and the individual's ethnic, cultural, and linguistic background, available experiences, and adaptive functioning within his or her community and cultural setting must be taken into account.

Gender-Related Diagnostic Issues

Overall, males are more likely than females to be diagnosed with both mild (average male:female ratio 1.6:1) and severe (average male:female ratio 1.2:1) forms of intellectual disability. However, gender ratios vary widely in reported studies. Sex-linked genetic factors and male vulnerability to brain insult may account for some of the gender differences.

Diagnostic Markers

A comprehensive evaluation includes an assessment of intellectual capacity and adaptive functioning; identification of genetic and nongenetic etiologies; evaluation for associated medical conditions (e.g., cerebral palsy, seizure disorder); and evaluation for co-occurring mental, emotional, and behavioral disorders. Components of the evaluation may include basic pre- and perinatal medical history, three-generational family pedigree, physical examination, genetic evaluation (e.g., karyotype or chromosomal microarray analysis and testing for specific genetic syndromes), and metabolic screening and neuroimaging assessment.

Differential Diagnosis

The diagnosis of intellectual disability should be made whenever Criteria A, B, and C are met. A diagnosis of intellectual disability should not be assumed because of a particular

genetic or medical condition. A genetic syndrome linked to intellectual disability should be noted as a concurrent diagnosis with the intellectual disability.

Major and mild neurocognitive disorders. Intellectual disability is categorized as a neurodevelopmental disorder and is distinct from the neurocognitive disorders, which are characterized by a loss of cognitive functioning. Major neurocognitive disorder may co-occur with intellectual disability (e.g., an individual with Down syndrome who develops Alzheimer's disease, or an individual with intellectual disability who loses further cognitive capacity following a head injury). In such cases, the diagnoses of intellectual disability and neurocognitive disorder may both be given.

Communication disorders and specific learning disorder. These neurodevelopmental disorders are specific to the communication and learning domains and do not show deficits in intellectual and adaptive behavior. They may co-occur with intellectual disability. Both diagnoses are made if full criteria are met for intellectual disability and a communication disorder or specific learning disorder.

Autism spectrum disorder. Intellectual disability is common among individuals with autism spectrum disorder. Assessment of intellectual ability may be complicated by social-communication and behavior deficits inherent to autism spectrum disorder, which may interfere with understanding and complying with test procedures. Appropriate assessment of intellectual functioning in autism spectrum disorder is essential, with reassessment across the developmental period, because IQ scores in autism spectrum disorder may be unstable, particularly in early childhood.

Comorbidity

Co-occurring mental, neurodevelopmental, medical, and physical conditions are frequent in intellectual disability, with rates of some conditions (e.g., mental disorders, cerebral palsy, and epilepsy) three to four times higher than in the general population. The prognosis and outcome of co-occurring diagnoses may be influenced by the presence of intellectual disability. Assessment procedures may require modifications because of associated disorders, including communication disorders, autism spectrum disorder, and motor, sensory, or other disorders. Knowledgeable informants are essential for identifying symptoms such as irritability, mood dysregulation, aggression, eating problems, and sleep problems, and for assessing adaptive functioning in various community settings.

The most common co-occurring mental and neurodevelopmental disorders are attention-deficit/hyperactivity disorder; depressive and bipolar disorders; anxiety disorders; autism spectrum disorder; stereotypic movement disorder (with or without self-injurious behavior); impulse-control disorders; and major neurocognitive disorder. Major depressive disorder may occur throughout the range of severity of intellectual disability. Self-injurious behavior requires prompt diagnostic attention and may warrant a separate diagnosis of stereotypic movement disorder. Individuals with intellectual disability, particularly those with more severe intellectual disability, may also exhibit aggression and disruptive behaviors, including harm of others or property destruction.

Relationship to Other Classifications

ICD-11 (in development at the time of this publication) uses the term *intellectual developmental disorders* to indicate that these are disorders that involve impaired brain functioning early in life. These disorders are described in ICD-11 as a metasyndrome occurring in the developmental period analogous to dementia or neurocognitive disorder in later life. There are four subtypes in ICD-11: mild, moderate, severe, and profound.

The American Association on Intellectual and Developmental Disabilities (AAIDD) also uses the term *intellectual disability* with a similar meaning to the term as used in this

manual. The AAIDD's classification is multidimensional rather than categorical and is based on the disability construct. Rather than listing specifiers as is done in DSM-5, the AAIDD emphasizes a profile of supports based on severity.

Global Developmental Delay

315.8 (F88)

This diagnosis is reserved for individuals *under* the age of 5 years when the clinical severity level cannot be reliably assessed during early childhood. This category is diagnosed when an individual fails to meet expected developmental milestones in several areas of intellectual functioning, and applies to individuals who are unable to undergo systematic assessments of intellectual functioning, including children who are too young to participate in standardized testing. This category requires reassessment after a period of time.

Unspecified Intellectual Disability (Intellectual Developmental Disorder)

319 (F79)

This category is reserved for individuals *over* the age of 5 years when assessment of the degree of intellectual disability (intellectual developmental disorder) by means of locally available procedures is rendered difficult or impossible because of associated sensory or physical impairments, as in blindness or prelingual deafness; locomotor disability; or presence of severe problem behaviors or co-occurring mental disorder. This category should only be used in exceptional circumstances and requires reassessment after a period of time.

Communication Disorders

Disorders of communication include deficits in language, speech, and communication. *Speech* is the expressive production of sounds and includes an individual's articulation, fluency, voice, and resonance quality. *Language* includes the form, function, and use of a conventional system of symbols (i.e., spoken words, sign language, written words, pictures) in a rule-governed manner for communication. *Communication* includes any verbal or nonverbal behavior (whether intentional or unintentional) that influences the behavior, ideas, or attitudes of another individual. Assessments of speech, language and communication abilities must take into account the individual's cultural and language context, particularly for individuals growing up in bilingual environments. The standardized measures of language development and of nonverbal intellectual capacity must be relevant for the cultural and linguistic group (i.e., tests developed and standardized for one group may not provide appropriate norms for a different group). The diagnostic category of communication disorders includes the following: language disorder, speech sound disorder, childhood-onset fluency disorder (stuttering), social (pragmatic) communication disorder, and other specified and unspecified communication disorders.

Language Disorder

Diagnostic Criteria **315.39 (F80.9)**

A. Persistent difficulties in the acquisition and use of language across modalities (i.e., spoken, written, sign language, or other) due to deficits in comprehension or production that include the following:
   1. Reduced vocabulary (word knowledge and use).
   2. Limited sentence structure (ability to put words and word endings together to form sentences based on the rules of grammar and morphology).
   3. Impairments in discourse (ability to use vocabulary and connect sentences to explain or describe a topic or series of events or have a conversation).

B. Language abilities are substantially and quantifiably below those expected for age, resulting in functional limitations in effective communication, social participation, academic achievement, or occupational performance, individually or in any combination.

C. Onset of symptoms is in the early developmental period.

D. The difficulties are not attributable to hearing or other sensory impairment, motor dysfunction, or another medical or neurological condition and are not better explained by intellectual disability (intellectual developmental disorder) or global developmental delay.

Diagnostic Features

The core diagnostic features of language disorder are difficulties in the acquisition and use of language due to deficits in the comprehension or production of vocabulary, sentence structure, and discourse. The language deficits are evident in spoken communication, written communication, or sign language. Language learning and use is dependent on both receptive and expressive skills. *Expressive ability* refers to the production of vocal, gestural, or verbal signals, while *receptive ability* refers to the process of receiving and comprehending language messages. Language skills need to be assessed in both expressive and receptive modalities as these may differ in severity. For example, an individual's expressive language may be severely impaired, while his receptive language is hardly impaired at all.

Language disorder usually affects vocabulary and grammar, and these effects then limit the capacity for discourse. The child's first words and phrases are likely to be delayed in onset; vocabulary size is smaller and less varied than expected; and sentences are shorter and less complex with grammatical errors, especially in past tense. Deficits in comprehension of language are frequently underestimated, as children may be good at using context to infer meaning. There may be word-finding problems, impoverished verbal definitions, or poor understanding of synonyms, multiple meanings, or word play appropriate for age and culture. Problems with remembering new words and sentences are manifested by difficulties following instructions of increasing length, difficulties rehearsing strings of verbal information (e.g., remembering a phone number or a shopping list), and difficulties remembering novel sound sequences, a skill that may be important for learning new words. Difficulties with discourse are shown by a reduced ability to provide adequate information about the key events and to narrate a coherent story.

The language difficulty is manifest by abilities substantially and quantifiably below that expected for age and significantly interfering with academic achievement, occupational performance, effective communication, or socialization (Criterion B). A diagnosis of language disorder is made based on the synthesis of the individual's history, direct clinical observation in different contexts (i.e., home, school, or work), and scores from standardized tests of language ability that can be used to guide estimates of severity.

Associated Features Supporting Diagnosis

A positive family history of language disorders is often present. Individuals, even children, can be adept at accommodating to their limited language. They may appear to be shy or reticent to talk. Affected individuals may prefer to communicate only with family members or other familiar individuals. Although these social indicators are not diagnostic of a language disorder, if they are notable and persistent, they warrant referral for a full language assessment. Language disorder, particularly expressive deficits, may co-occur with speech sound disorder.

Deveiopment and Course

Language acquisition is marked by changes from onset in toddlerhood to the adult level of competency that appears during adolescence. Changes appear across the dimensions of language (sounds, words, grammar, narratives/expository texts, and conversational skills) in age-graded increments and synchronies. Language disorder emerges during the early developmental period; however, there is considerable variation in early vocabulary acquisition and early word combinations, and individual differences are not, as single indicators, highly predictive of later outcomes. By age 4 years, individual differences in language ability are more stable, with better measurement accuracy, and are highly predictive of later outcomes. Language disorder diagnosed from 4 years of age is likely to be stable over time and typically persists into adulthood, although the particular profile of language strengths and deficits is likely to change over the course of development.

RIsk and Prognostic Factors

Children with receptive language impairments have a poorer prognosis than those with predominantly expressive impairments. They are more resistant to treatment, and difficulties with reading comprehension are frequently seen.

Genetic and physiological. Language disorders are highly heritable, and family members are more likely to have a history of language impairment.

Differential Diagnosis

Normal variations in language. Language disorder needs to be distinguished from normal developmental variations, and this distinction may be difficult to make before 4 years of age. Regional, social, or cultural/ethnic variations of language (e.g., dialects) must be considered when an individual is being assessed for language impairment.

Hearing or other sensory impairment. Hearing impairment needs to be excluded as the primary cause of language difficulties. Language deficits may be associated with a hearing impairment, other sensory deficit, or a speech-motor deficit. When language deficits are in excess of those usually associated with these problems, a diagnosis of language disorder may be made.

Intellectual disability (intellectual developmental disorder). Language delay is often the presenting feature of intellectual disability, and the definitive diagnosis may not be made until the child is able to complete standardized assessments. A separate diagnosis is not given unless the language deficits are clearly in excess of the intellectual limitations.

Neurological disorders. Language disorder can be acquired in association with neurological disorders, including epilepsy (e.g., acquired aphasia or Landau-Kleffner syndrome).

Language regression. Loss of speech and language in a child younger than 3 years may be a sign of autism spectrum disorder (with developmental regression) or a specific neurological condition, such as Landau-Kleffner syndrome. Among children older than 3 years, language loss may be a symptom of seizures, and a diagnostic assessment is necessary to exclude the presence of epilepsy (e.g., routine and sleep electroencephalogram).

Comorbidity

Language disorder is strongly associated with other neurodevelopmental disorders in terms of specific learning disorder (literacy and numeracy), attention-deficit/hyperactivity disorder, autism spectrum disorder, and developmental coordination disorder. It is also associated with social (pragmatic) communication disorder. A positive family history of speech or language disorders is often present.

Speech Sound Disorder

Diagnostic Criteria	**315.39 (F80.0)**

A. Persistent difficulty with speech sound production that interferes with speech intelligibility or prevents verbal communication of messages.
B. The disturbance causes limitations in effective communication that interfere with social participation, academic achievement, or occupational performance, individually or in any combination.
C. Onset of symptoms is in the early developmental period.
D. The difficulties are not attributable to congenital or acquired conditions, such as cerebral palsy, cleft palate, deafness or hearing loss, traumatic brain injury, or other medical or neurological conditions.

Diagnostic Features

Speech sound production describes the clear articulation of the phonemes (i.e., individual sounds) that in combination make up spoken words. Speech sound production requires both the phonological knowledge of speech sounds and the ability to coordinate the movements of the articulators (i.e., the jaw, tongue, and lips,) with breathing and vocalizing for speech. Children with speech production difficulties may experience difficulty with phonological knowledge of speech sounds or the ability to coordinate movements for speech in varying degrees. Speech sound disorder is thus heterogeneous in its underlying mechanisms and includes phonological disorder and articulation disorder. A speech sound disorder is diagnosed when speech sound production is not what would be expected based on the child's age and developmental stage and when the deficits are not the result of a physical, structural, neurological, or hearing impairment. Among typically developing children at age 4 years, overall speech should be intelligible, whereas at age 2 years, only 50% may be understandable.

Associated Features Supporting Diagnosis

Language disorder, particularly expressive deficits, may be found to co-occur with speech sound disorder. A positive family history of speech or language disorders is often present.

If the ability to rapidly coordinate the articulators is a particular aspect of difficulty, there may be a history of delay or incoordination in acquiring skills that also utilize the articulators and related facial musculature; among others, these skills include chewing, maintaining mouth closure, and blowing the nose. Other areas of motor coordination may be impaired as in developmental coordination disorder. *Verbal dyspraxia* is a term also used for speech production problems.

Speech may be differentially impaired in certain genetic conditions (e.g., Down syndrome, 22q deletion, *FoxP2* gene mutation). If present, these should also be coded.

Development and Course

Learning to produce speech sounds clearly and accurately and learning to produce connected speech fluently are developmental skills. Articulation of speech sounds follows a

developmental pattern, which is reflected in the age norms of standardized tests. It is not unusual for typically developing children to use developmental processes for shortening words and syllables as they are learning to talk, but their progression in mastering speech sound production should result in mostly intelligible speech by age 3 years. Children with speech sound disorder continue to use immature phonological simplification processes past the age when most children can produce words clearly.

Most speech sounds should be produced clearly and most words should be pronounced accurately according to age and community norms by age 7 years. The most frequently misarticulated sounds also tend to be learned later, leading them to be called the "late eight" (*l, r, s, z, th, ch, dzh,* and *zh*). Misarticulation of any of these sounds by itself could be considered within normal limits up to age 8 years. When multiple sounds are involved, it may be appropriate to target some of those sounds as part of a plan to improve intelligibility prior to the age at which almost all children can produce them accurately. Lisping (i.e., misarticulating sibilants) is particularly common and may involve frontal or lateral patterns of airstream direction. It may be associated with an abnormal tongue-thrust swallowing pattern.

Most children with speech sound disorder respond well to treatment, and speech difficulties improve over time, and thus the disorder may not be lifelong. However, when a language disorder is also present, the speech disorder has a poorer prognosis and may be associated with specific learning disorders.

Differential Diagnosis

Normal variations in speech. Regional, social, or cultural/ethnic variations of speech should be considered before making the diagnosis.

Hearing or other sensory impairment. Hearing impairment or deafness may result in abnormalities of speech. Deficits of speech sound production may be associated with a hearing impairment, other sensory deficit, or a speech-motor deficit. When speech deficits are in excess of those usually associated with these problems, a diagnosis of speech sound disorder may be made.

Structural deficits. Speech impairment may be due to structural deficits (e.g., cleft palate).

Dysarthria. Speech impairment may be attributable to a motor disorder, such as cerebral palsy. Neurological signs, as well as distinctive features of voice, differentiate dysarthria from speech sound disorder, although in young children (under 3 years) differentiation may be difficult, particularly when there is no or minimal general body motor involvement (as in, e.g., Worster-Drought syndrome).

Selective mutism. Limited use of speech may be a sign of selective mutism, an anxiety disorder that is characterized by a lack of speech in one or more contexts or settings. Selective mutism may develop in children with a speech disorder because of embarassment about their impairments, but many children with selective mutism exhibit normal speech in "safe" settings, such as at home or with close friends.

Childhood-Onset Fluency Disorder (Stuttering)

Diagnostic Criteria **315.35 (F80.81)**

A. Disturbances in the normal fluency and time patterning of speech that are inappropriate for the individual's age and language skills, persist over time, and are characterized by frequent and marked occurrences of one (or more) of the following:

1. Sound and syllable repetitions.
2. Sound prolongations of consonants as well as vowels.

3. Broken words (e.g., pauses within a word).
4. Audible or silent blocking (filled or unfilled pauses in speech).
5. Circumlocutions (word substitutions to avoid problematic words).
6. Words produced with an excess of physical tension.
7. Monosyllabic whole-word repetitions (e.g., "I-I-I-I see him").

B. The disturbance causes anxiety about speaking or limitations in effective communication, social participation, or academic or occupational performance, individually or in any combination.

C. The onset of symptoms is in the early developmental period. (**Note:** Later-onset cases are diagnosed as 307.0 [F98.5] adult-onset fluency disorder.)

D. The disturbance is not attributable to a speech-motor or sensory deficit, dysfluency associated with neurological insult (e.g., stroke, tumor, trauma), or another medical condition and is not better explained by another mental disorder.

Diagnostic Features

The essential feature of childhood-onset fluency disorder (stuttering) is a disturbance in the normal fluency and time patterning of speech that is inappropriate for the individual's age. This disturbance is characterized by frequent repetitions or prolongations of sounds or syllables and by other types of speech dysfluencies, including broken words (e.g., pauses within a word), audible or silent blocking (i.e., filled or unfilled pauses in speech), circumlocutions (i.e., word substitutions to avoid problematic words), words produced with an excess of physical tension, and monosyllabic whole-word repetitions (e.g., "I-I-I-I see him"). The disturbance in fluency interferes with academic or occupational achievement or with social communication. The extent of the disturbance varies from situation to situation and often is more severe when there is special pressure to communicate (e.g., giving a report at school, interviewing for a job). Dysfluency is often absent during oral reading, singing, or talking to inanimate objects or to pets.

Associated Features Supporting Diagnosis

Fearful anticipation of the problem may develop. The speaker may attempt to avoid dysfluencies by linguistic mechanisms (e.g., altering the rate of speech, avoiding certain words or sounds) or by avoiding certain speech situations, such as telephoning or public speaking. In addition to being features of the condition, stress and anxiety have been shown to exacerbate dysfluency.

Childhood-onset fluency disorder may also be accompanied by motor movements (e.g., eye blinks, tics, tremors of the lips or face, jerking of the head, breathing movements, fist clenching). Children with fluency disorder show a range of language abilities, and the relationship between fluency disorder and language abilities is unclear.

Deveiopment and Course

Childhood-onset fluency disorder, or developmental stuttering, occurs by age 6 for 80%–90% of affected individuals, with age at onset ranging from 2 to 7 years. The onset can be insidious or more sudden. Typically, dysfluencies start gradually, with repetition of initial consonants, first words of a phrase, or long words. The child may not be aware of dysfluencies. As the disorder progresses, the dysfluencies become more frequent and interfering, occurring on the most meaningful words or phrases in the utterance. As the child becomes aware of the speech difficulty, he or she may develop mechanisms for avoiding the dysfluencies and emotional responses, including avoidance of public speaking and use of short and simple utterances. Longitudinal research shows that 65%–85% of children re-

cover from the dysfluency, with severity of fluency disorder at age 8 years predicting recovery or persistence into adolescence and beyond.

Risk and Prognostic Factors

Genetic and physiological. The risk of stuttering among first-degree biological relatives of individuals with childhood-onset fluency disorder is more than three times the risk in the general population.

Functional Consequences of Childhood-Onset Fluency Disorder (Stuttering)

In addition to being features of the condition, stress and anxiety can exacerbate dysfluency. Impairment of social functioning may result from this anxiety.

Differential Diagnosis

Sensory deficits. Dysfluencies of speech may be associated with a hearing impairment or other sensory deficit or a speech-motor deficit. When the speech dysfluencies are in excess of those usually associated with these problems, a diagnosis of childhood-onset fluency disorder may be made.

Normal speech dysfluencies. The disorder must be distinguished from normal dysfluencies that occur frequently in young children, which include whole-word or phrase repetitions (e.g., "I want, I want ice cream"), incomplete phrases, interjections, unfilled pauses, and parenthetical remarks. If these difficulties increase in frequency or complexity as the child grows older, a diagnosis of childhood-onset fluency disorder is appropriate.

Medication side effects. Stuttering may occur as a side effect of medication and may be detected by a temporal relationship with exposure to the medication.

Adult-onset dysfluencies. If onset of dysfluencies is during or after adolescence, it is an "adult-onset dysfluency" rather than a neurodevelopmental disorder. Adult-onset dysfluencies are associated with specific neurological insults and a variety of medical conditions and mental disorders and may be specified with them, but they are not a DSM-5 diagnosis.

Tourette's disorder. Vocal tics and repetitive vocalizations of Tourette's disorder should be distinguishable from the repetitive sounds of childhood-onset fluency disorder by their nature and timing.

Social (Pragmatic) Communication Disorder

Diagnostic Criteria **315.39 (F80.89)**

A. Persistent difficulties in the social use of verbal and nonverbal communication as manifested by all of the following:

1. Deficits in using communication for social purposes, such as greeting and sharing information, in a manner that is appropriate for the social context.
2. Impairment of the ability to change communication to match context or the needs of the listener, such as speaking differently in a classroom than on a playground, talking differently to a child than to an adult, and avoiding use of overly formal language.
3. Difficulties following rules for conversation and storytelling, such as taking turns in conversation, rephrasing when misunderstood, and knowing how to use verbal and nonverbal signals to regulate interaction.

4. Difficulties understanding what is not explicitly stated (e.g., making inferences) and nonliteral or ambiguous meanings of language (e.g., idioms, humor, metaphors, multiple meanings that depend on the context for interpretation).

B. The deficits result in functional limitations in effective communication, social participation, social relationships, academic achievement, or occupational performance, individually or in combination.

C. The onset of the symptoms is in the early developmental period (but deficits may not become fully manifest until social communication demands exceed limited capacities).

D. The symptoms are not attributable to another medical or neurological condition or to low abilities in the domains of word structure and grammar, and are not better explained by autism spectrum disorder, intellectual disability (intellectual developmental disorder), global developmental delay, or another mental disorder.

Diagnostic Features

Social (pragmatic) communication disorder is characterized by a primary difficulty with pragmatics, or the social use of language and communication, as manifested by deficits in understanding and following social rules of verbal and nonverbal communication in naturalistic contexts, changing language according to the needs of the listener or situation, and following rules for conversations and storytelling. The deficits in social communication result in functional limitations in effective communication, social participation, development of social relationships, academic achievement, or occupational performance. The deficits are not better explained by low abilities in the domains of structural language or cognitive ability.

Associated Features Supporting Diagnosis

The most common associated feature of social (pragmatic) communication disorder is language impairment, which is characterized by a history of delay in reaching language milestones, and historical, if not current, structural language problems (see "Language Disorder" earlier in this chapter). Individuals with social communication deficits may avoid social interactions. Attention-deficit/hyperactivity disorder (ADHD), behavioral problems, and specific learning disorders are also more common among affected individuals.

Development and Course

Because social (pragmatic) communication depends on adequate developmental progress in speech and language, diagnosis of social (pragmatic) communication disorder is rare among children younger than 4 years. By age 4 or 5 years, most children should possess adequate speech and language abilities to permit identification of specific deficits in social communication. Milder forms of the disorder may not become apparent until early adolescence, when language and social interactions become more complex.

The outcome of social (pragmatic) communication disorder is variable, with some children improving substantially over time and others continuing to have difficulties persisting into adulthood. Even among those who have significant improvements, the early deficits in pragmatics may cause lasting impairments in social relationships and behavior and also in acquisition of other related skills, such as written expression.

Risk and Prognostic Factors

Genetic and physiological. A family history of autism spectrum disorder, communication disorders, or specific learning disorder appears to increase the risk for social (pragmatic) communication disorder.

Differential Diagnosis

Autism spectrum disorder. Autism spectrum disorder is the primary diagnostic consideration for individuals presenting with social communication deficits. The two disorders can be differentiated by the presence in autism spectrum disorder of restricted/repetitive patterns of behavior, interests, or activities and their absence in social (pragmatic) communication disorder. Individuals with autism spectrum disorder may only display the restricted/repetitive patterns of behavior, interests, and activities during the early developmental period, so a comprehensive history should be obtained. Current absence of symptoms would not preclude a diagnosis of autism spectrum disorder, if the restricted interests and repetitive behaviors were present in the past. A diagnosis of social (pragmatic) communication disorder should be considered only if the developmental history fails to reveal any evidence of restricted/repetitive patterns of behavior, interests, or activities.

Attention-deficit/hyperactivity disorder. Primary deficits of ADHD may cause impairments in social communication and functional limitations of effective communication, social participation, or academic achievement.

Social anxiety disorder (social phobia). The symptoms of social communication disorder overlap with those of social anxiety disorder. The differentiating feature is the timing of the onset of symptoms. In social (pragmatic) communication disorder, the individual has never had effective social communication; in social anxiety disorder, the social communication skills developed appropriately but are not utilized because of anxiety, fear, or distress about social interactions.

Intellectual disability (intellectual developmental disorder) and global developmental delay. Social communication skills may be deficient among individuals with global developmental delay or intellectual disability, but a separate diagnosis is not given unless the social communication deficits are clearly in excess of the intellectual limitations.

Unspecified Communication Disorder

307.9 (F80.9)

This category applies to presentations in which symptoms characteristic of communication disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for communication disorder or for any of the disorders in the neurodevelopmental disorders diagnostic class. The unspecified communication disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for communication disorder or for a specific neurodevelopmental disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis.

Autism Spectrum Disorder

Autism Spectrum Disorder

Diagnostic Criteria **299.00 (F84.0)**

A. Persistent deficits in social communication and social interaction across multiple contexts, as manifested by the following, currently or by history (examples are illustrative, not exhaustive; see text):

1. Deficits in social-emotional reciprocity, ranging, for example, from abnormal social approach and failure of normal back-and-forth conversation; to reduced sharing of interests, emotions, or affect; to failure to initiate or respond to social interactions.
2. Deficits in nonverbal communicative behaviors used for social interaction, ranging, for example, from poorly integrated verbal and nonverbal communication; to abnormalities in eye contact and body language or deficits in understanding and use of gestures; to a total lack of facial expressions and nonverbal communication.
3. Deficits in developing, maintaining, and understanding relationships, ranging, for example, from difficulties adjusting behavior to suit various social contexts; to difficulties in sharing imaginative play or in making friends; to absence of interest in peers.

Specify current severity:

Severity is based on social communication impairments and restricted, repetitive patterns of behavior (seeTable 2).

B. Restricted, repetitive patterns of behavior, interests, or activities, as manifested by at least two of the following, currently or by history (examples are illustrative, not exhaustive; see text):

1. Stereotyped or repetitive motor movements, use of objects, or speech (e.g., simple motor stereotypies, lining up toys or flipping objects, echolalia, idiosyncratic phrases).
2. Insistence on sameness, inflexible adherence to routines, or ritualized patterns of verbal or nonverbal behavior (e.g., extreme distress at small changes, difficulties with transitions, rigid thinking patterns, greeting rituals, need to take same route or eat same food every day).
3. Highly restricted, fixated interests that are abnormal in intensity or focus (e.g., strong attachment to or preoccupation with unusual objects, excessively circumscribed or perseverative interests).
4. Hyper- or hyporeactivity to sensory input or unusual interest in sensory aspects of the environment (e.g., apparent indifference to pain/temperature, adverse response to specific sounds or textures, excessive smelling or touching of objects, visual fascination with lights or movement).

Specify current severity:

Severity is based on social communication impairments and restricted, repetitive patterns of behavior (see Table 2).

C. Symptoms must be present in the early developmental period (but may not become fully manifest until social demands exceed limited capacities, or may be masked by learned strategies in later life).

D. Symptoms cause clinically significant impairment in social, occupational, or other important areas of current functioning.

E. These disturbances are not better explained by intellectual disability (intellectual developmental disorder) or global developmental delay. Intellectual disability and autism spectrum disorder frequently co-occur; to make comorbid diagnoses of autism spectrum disorder and intellectual disability, social communication should be below that expected for general developmental level.

Note: Individuals with a well-established DSM-IV diagnosis of autistic disorder, Asperger's disorder, or pervasive developmental disorder not otherwise specified should be given the diagnosis of autism spectrum disorder. Individuals who have marked deficits in social communication, but whose symptoms do not otherwise meet criteria for autism spectrum disorder, should be evaluated for social (pragmatic) communication disorder.

Specify if:

With or without accompanying intellectual impairment

With or without accompanying language impairment

Associated with a known medical or genetic condition or environmental factor (**Coding note:** Use additional code to identify the associated medical or genetic condition.)

Associated with another neurodevelopmental, mental, or behavioral disorder (**Coding note:** Use additional code[s] to identify the associated neurodevelopmental, mental, or behavioral disorder[s].)

With catatonia (refer to the criteria for catatonia associated with another mental disorder, pp. 119–120, for definition) (**Coding note:** Use additional code 293.89 [F06.1] catatonia associated with autism spectrum disorder to indicate the presence of the comorbid catatonia.)

Recording Procedures

For autism spectrum disorder that is associated with a known medical or genetic condition or environmental factor, or with another neurodevelopmental, mental, or behavioral disorder, record autism spectrum disorder associated with (name of condition, disorder, or factor) (e.g., autism spectrum disorder associated with Rett syndrome). Severity should be recorded as level of support needed for each of the two psychopathological domains in Table 2 (e.g., "requiring very substantial support for deficits in social communication and requiring substantial support for restricted, repetitive behaviors"). Specification of "with accompanying intellectual impairment" or "without accompanying intellectual impairment" should be recorded next. Language impairment specification should be recorded thereafter. If there is accompanying language impairment, the current level of verbal functioning should be recorded (e.g., "with accompanying language impairment—no intelligible speech" or "with accompanying language impairment—phrase speech"). If catatonia is present, record separately "catatonia associated with autism spectrum disorder."

Specifiers

The severity specifiers (see Table 2) may be used to describe succinctly the current symptomatology (which might fall below level 1), with the recognition that severity may vary by context and fluctuate over time. Severity of social communication difficulties and restricted, repetitive behaviors should be separately rated. The descriptive severity categories should not be used to determine eligibility for and provision of services; these can only be developed at an individual level and through discussion of personal priorities and targets.

Regarding the specifier "with or without accompanying intellectual impairment," understanding the (often uneven) intellectual profile of a child or adult with autism spectrum disorder is necessary for interpreting diagnostic features. Separate estimates of verbal and nonverbal skill are necessary (e.g., using untimed nonverbal tests to assess potential strengths in individuals with limited language).

TABLE 2 Severity levels for autism spectrum disorder

Severity level	Social communication	Restricted, repetitive behaviors
Level 3 "Requiring very substantial support"	Severe deficits in verbal and nonverbal social communication skills cause severe impairments in functioning, very limited initiation of social interactions, and minimal response to social overtures from others. For example, a person with few words of intelligible speech who rarely initiates interaction and, when he or she does, makes unusual approaches to meet needs only and responds to only very direct social approaches.	Inflexibility of behavior, extreme difficulty coping with change, or other restricted/repetitive behaviors markedly interfere with functioning in all spheres. Great distress/difficulty changing focus or action.
Level 2 "Requiring substantial support"	Marked deficits in verbal and nonverbal social communication skills; social impairments apparent even with supports in place; limited initiation of social interactions; and reduced or abnormal responses to social overtures from others. For example, a person who speaks simple sentences, whose interaction is limited to narrow special interests, and who has markedly odd nonverbal communication.	Inflexibility of behavior, difficulty coping with change, or other restricted/repetitive behaviors appear frequently enough to be obvious to the casual observer and interfere with functioning in a variety of contexts. Distress and/or difficulty changing focus or action.
Level 1 "Requiring support"	Without supports in place, deficits in social communication cause noticeable impairments. Difficulty initiating social interactions, and clear examples of atypical or unsuccessful responses to social overtures of others. May appear to have decreased interest in social interactions. For example, a person who is able to speak in full sentences and engages in communication but whose to-and-fro conversation with others fails, and whose attempts to make friends are odd and typically unsuccessful.	Inflexibility of behavior causes significant interference with functioning in one or more contexts. Difficulty switching between activities. Problems of organization and planning hamper independence.

To use the specifier "with or without accompanying language impairment," the current level of verbal functioning should be assessed and described. Examples of the specific descriptions for "with accompanying language impairment" might include no intelligible speech (nonverbal), single words only, or phrase speech. Language level in individuals "without accompanying language impairment" might be further described by speaks in full sentences or has fluent speech. Since receptive language may lag behind expressive language development in autism spectrum disorder, receptive and expressive language skills should be considered separately.

The specifier "associated with a known medical or genetic condition or environmental factor" should be used when the individual has a known genetic disorder (e.g., Rett syndrome, Fragile X syndrome, Down syndrome), a medical disorder (e.g. epilepsy), or a history of environmental exposure (e.g., valproate, fetal alcohol syndrome, very low birth weight).

Additional neurodevelopmental, mental or behavioral conditions should also be noted (e.g., attention-deficit/hyperactivity disorder; developmental coordination disorder; disruptive behavior, impulse-control, or conduct disorders; anxiety, depressive, or bipolar disorders; tics or Tourette's disorder; self-injury; feeding, elimination, or sleep disorders).

Diagnostic Features

The essential features of autism spectrum disorder are persistent impairment in reciprocal social communication and social interaction (Criterion A), and restricted, repetitive patterns of behavior, interests, or activities (Criterion B). These symptoms are present from early childhood and limit or impair everyday functioning (Criteria C and D). The stage at which functional impairment becomes obvious will vary according to characteristics of the individual and his or her environment. Core diagnostic features are evident in the developmental period, but intervention, compensation, and current supports may mask difficulties in at least some contexts. Manifestations of the disorder also vary greatly depending on the severity of the autistic condition, developmental level, and chronological age; hence, the term *spectrum*. Autism spectrum disorder encompasses disorders previously referred to as early infantile autism, childhood autism, Kanner's autism, high-functioning autism, atypical autism, pervasive developmental disorder not otherwise specified, childhood disintegrative disorder, and Asperger's disorder.

The impairments in communication and social interaction specified in Criterion A are pervasive and sustained. Diagnoses are most valid and reliable when based on multiple sources of information, including clinician's observations, caregiver history, and, when possible, self-report. Verbal and nonverbal deficits in social communication have varying manifestations, depending on the individual's age, intellectual level, and language ability, as well as other factors such as treatment history and current support. Many individuals have language deficits, ranging from complete lack of speech through language delays, poor comprehension of speech, echoed speech, or stilted and overly literal language. Even when formal language skills (e.g., vocabulary, grammar) are intact, the use of language for reciprocal social communication is impaired in autism spectrum disorder.

Deficits in social-emotional reciprocity (i.e., the ability to engage with others and share thoughts and feelings) are clearly evident in young children with the disorder, who may show little or no initiation of social interaction and no sharing of emotions, along with reduced or absent imitation of others' behavior. What language exists is often one-sided, lacking in social reciprocity, and used to request or label rather than to comment, share feelings, or converse. In adults without intellectual disabilities or language delays, deficits in social-emotional reciprocity may be most apparent in difficulties processing and responding to complex social cues (e.g., when and how to join a conversation, what not to say). Adults who have developed compensation strategies for some social challenges still struggle in novel or unsupported situations and suffer from the effort and anxiety of consciously calculating what is socially intuitive for most individuals.

Deficits in nonverbal communicative behaviors used for social interaction are manifested by absent, reduced, or atypical use of eye contact (relative to cultural norms), gestures, facial expressions, body orientation, or speech intonation. An early feature of autism spectrum disorder is impaired joint attention as manifested by a lack of pointing, showing, or bringing objects to share interest with others, or failure to follow someone's pointing or eye gaze. Individuals may learn a few functional gestures, but their repertoire is smaller than that of others, and they often fail to use expressive gestures spontaneously in communication. Among adults with fluent language, the difficulty in coordinating nonverbal communication with speech may give the impression of odd, wooden, or exaggerated "body language" during interactions. Impairment may be relatively subtle within individual modes (e.g., someone may have relatively good eye contact when speaking) but noticeable in poor integration of eye contact, gesture, body posture, prosody, and facial expression for social communication.

Deficits in developing, maintaining, and understanding relationships should be judged against norms for age, gender, and culture. There may be absent, reduced, or atypical social interest, manifested by rejection of others, passivity, or inappropriate approaches that seem aggressive or disruptive. These difficulties are particularly evident in young children, in whom there is often a lack of shared social play and imagination (e.g., age-appropriate flexible pretend play) and, later, insistence on playing by very fixed rules. Older individuals may struggle to understand what behavior is considered appropriate in one situation but not another (e.g., casual behavior during a job interview), or the different ways that language may be used to communicate (e.g., irony, white lies). There may be an apparent preference for solitary activities or for interacting with much younger or older people. Frequently, there is a desire to establish friendships without a complete or realistic idea of what friendship entails (e.g., one-sided friendships or friendships based solely on shared special interests). Relationships with siblings, co-workers, and caregivers are also important to consider (in terms of reciprocity).

Autism spectrum disorder is also defined by restricted, repetitive patterns of behavior, interests, or activities (as specified in Criterion B), which show a range of manifestations according to age and ability, intervention, and current supports. Stereotyped or repetitive behaviors include simple motor stereotypies (e.g., hand flapping, finger flicking), repetitive use of objects (e.g., spinning coins, lining up toys), and repetitive speech (e.g., echolalia, the delayed or immediate parroting of heard words; use of "you" when referring to self; stereotyped use of words, phrases, or prosodic patterns). Excessive adherence to routines and restricted patterns of behavior may be manifest in resistance to change (e.g., distress at apparently small changes, such as in packaging of a favorite food; insistence on adherence to rules; rigidity of thinking) or ritualized patterns of verbal or nonverbal behavior (e.g., repetitive questioning, pacing a perimeter). Highly restricted, fixated interests in autism spectrum disorder tend to be abnormal in intensity or focus (e.g., a toddler strongly attached to a pan; a child preoccupied with vacuum cleaners; an adult spending hours writing out timetables). Some fascinations and routines may relate to apparent hyper- or hyporeactivity to sensory input, manifested through extreme responses to specific sounds or textures, excessive smelling or touching of objects, fascination with lights or spinning objects, and sometimes apparent indifference to pain, heat, or cold. Extreme reaction to or rituals involving taste, smell, texture, or appearance of food or excessive food restrictions are common and may be a presenting feature of autism spectrum disorder.

Many adults with autism spectrum disorder without intellectual or language disabilities learn to suppress repetitive behavior in public. Special interests may be a source of pleasure and motivation and provide avenues for education and employment later in life. Diagnostic criteria may be met when restricted, repetitive patterns of behavior, interests, or activities were clearly present during childhood or at some time in the past, even if symptoms are no longer present.

Criterion D requires that the features must cause clinically significant impairment in social, occupational, or other important areas of current functioning. Criterion E specifies that the social communication deficits, although sometimes accompanied by intellectual disability (intellectual developmental disorder), are not in line with the individual's developmental level; impairments exceed difficulties expected on the basis of developmental level.

Standardized behavioral diagnostic instruments with good psychometric properties, including caregiver interviews, questionnaires and clinician observation measures, are available and can improve reliability of diagnosis over time and across clinicians.

Associated Features Supporting Diagnosis

Many individuals with autism spectrum disorder also have intellectual impairment and/or language impairment (e.g., slow to talk, language comprehension behind production). Even those with average or high intelligence have an uneven profile of abilities. The gap between intellectual and adaptive functional skills is often large. Motor deficits are often present, including odd gait, clumsiness, and other abnormal motor signs (e.g., walking on tiptoes). Self-injury (e.g., head banging, biting the wrist) may occur, and disruptive/challenging behaviors are more common in children and adolescents with autism spectrum disorder than other disorders, including intellectual disability. Adolescents and adults with autism spectrum disorder are prone to anxiety and depression. Some individuals develop catatonic-like motor behavior (slowing and "freezing" mid-action), but these are typically not of the magnitude of a catatonic episode. However, it is possible for individuals with autism spectrum disorder to experience a marked deterioration in motor symptoms and display a full catatonic episode with symptoms such as mutism, posturing, grimacing and waxy flexibility. The risk period for comorbid catatonia appears to be greatest in the adolescent years.

Prevalence

In recent years, reported frequencies for autism spectrum disorder across U.S. and non-U.S. countries have approached 1% of the population, with similar estimates in child and adult samples. It remains unclear whether higher rates reflect an expansion of the diagnostic criteria of DSM-IV to include subthreshold cases, increased awareness, differences in study methodology, or a true increase in the frequency of autism spectrum disorder.

Development and Course

The age and pattern of onset also should be noted for autism spectrum disorder. Symptoms are typically recognized during the second year of life (12–24 months of age) but may be seen earlier than 12 months if developmental delays are severe, or noted later than 24 months if symptoms are more subtle. The pattern of onset description might include information about early developmental delays or any losses of social or language skills. In cases where skills have been lost, parents or caregivers may give a history of a gradual or relatively rapid deterioration in social behaviors or language skills. Typically, this would occur between 12 and 24 months of age and is distinguished from the rare instances of developmental regression occurring after at least 2 years of normal development (previously described as childhood disintegrative disorder).

The behavioral features of autism spectrum disorder first become evident in early childhood, with some cases presenting a lack of interest in social interaction in the first year of life. Some children with autism spectrum disorder experience developmental plateaus or regression, with a gradual or relatively rapid deterioration in social behaviors or use of language, often during the first 2 years of life. Such losses are rare in other disorders and may be a useful "red flag" for autism spectrum disorder. Much more unusual and warranting more extensive medical investigation are losses of skills beyond social communication (e.g., loss of self-care, toileting, motor skills) or those occurring after the

second birthday (see also Rett syndrome in the section "Differential Diagnosis" for this disorder).

First symptoms of autism spectrum disorder frequently involve delayed language development, often accompanied by lack of social interest or unusual social interactions (e.g., pulling individuals by the hand without any attempt to look at them), odd play patterns (e.g., carrying toys around but never playing with them), and unusual communication patterns (e.g., knowing the alphabet but not responding to own name). Deafness may be suspected but is typically ruled out. During the second year, odd and repetitive behaviors and the absence of typical play become more apparent. Since many typically developing young children have strong preferences and enjoy repetition (e.g., eating the same foods, watching the same video multiple times), distinguishing restricted and repetitive behaviors that are diagnostic of autism spectrum disorder can be difficult in preschoolers. The clinical distinction is based on the type, frequency, and intensity of the behavior (e.g., a child who daily lines up objects for hours and is very distressed if any item is moved).

Autism spectrum disorder is not a degenerative disorder, and it is typical for learning and compensation to continue throughout life. Symptoms are often most marked in early childhood and early school years, with developmental gains typical in later childhood in at least some areas (e.g., increased interest in social interaction). A small proportion of individuals deteriorate behaviorally during adolescence, whereas most others improve. Only a minority of individuals with autism spectrum disorder live and work independently in adulthood; those who do tend to have superior language and intellectual abilities and are able to find a niche that matches their special interests and skills. In general, individuals with lower levels of impairment may be better able to function independently. However, even these individuals may remain socially naive and vulnerable, have difficulties organizing practical demands without aid, and are prone to anxiety and depression. Many adults report using compensation strategies and coping mechanisms to mask their difficulties in public but suffer from the stress and effort of maintaining a socially acceptable facade. Scarcely anything is known about old age in autism spectrum disorder.

Some individuals come for first diagnosis in adulthood, perhaps prompted by the diagnosis of autism in a child in the family or a breakdown of relations at work or home. Obtaining detailed developmental history in such cases may be difficult, and it is important to consider self-reported difficulties. Where clinical observation suggests criteria are currently met, autism spectrum disorder may be diagnosed, provided there is no evidence of good social and communication skills in childhood. For example, the report (by parents or another relative) that the individual had ordinary and sustained reciprocal friendships and good nonverbal communication skills throughout childhood would rule out a diagnosis of autism spectrum disorder; however, the absence of developmental information in itself should not do so.

Manifestations of the social and communication impairments and restricted/repetitive behaviors that define autism spectrum disorder are clear in the developmental period. In later life, intervention or compensation, as well as current supports, may mask these difficulties in at least some contexts. However, symptoms remain sufficient to cause current impairment in social, occupational, or other important areas of functioning.

Risk and Prognostic Factors

The best established prognostic factors for individual outcome within autism spectrum disorder are presence or absence of associated intellectual disability and language impairment (e.g., functional language by age 5 years is a good prognostic sign) and additional mental health problems. Epilepsy, as a comorbid diagnosis, is associated with greater intellectual disability and lower verbal ability.

Environmental. A variety of nonspecific risk factors, such as advanced parental age, low birth weight, or fetal exposure to valproate, may contribute to risk of autism spectrum disorder.

Genetic and physiological. Heritability estimates for autism spectrum disorder have ranged from 37% to higher than 90%, based on twin concordance rates. Currently, as many as 15% of cases of autism spectrum disorder appear to be associated with a known genetic mutation, with different de novo copy number variants or de novo mutations in specific genes associated with the disorder in different families. However, even when an autism spectrum disorder is associated with a known genetic mutation, it does not appear to be fully penetrant. Risk for the remainder of cases appears to be polygenic, with perhaps hundreds of genetic loci making relatively small contributions.

Culture-Related Diagnostic Issues

Cultural differences will exist in norms for social interaction, nonverbal communication, and relationships, but individuals with autism spectrum disorder are markedly impaired against the norms for their cultural context. Cultural and socioeconomic factors may affect age at recognition or diagnosis; for example, in the United States, late or underdiagnosis of autism spectrum disorder among African American children may occur.

Gender-Related Diagnostic Issues

Autism spectrum disorder is diagnosed four times more often in males than in females. In clinic samples, females tend to be more likely to show accompanying intellectual disability, suggesting that girls without accompanying intellectual impairments or language delays may go unrecognized, perhaps because of subtler manifestation of social and communication difficulties.

Functional Consequences of Autism Spectrum Disorder

In young children with autism spectrum disorder, lack of social and communication abilities may hamper learning, especially learning through social interaction or in settings with peers. In the home, insistence on routines and aversion to change, as well as sensory sensitivities, may interfere with eating and sleeping and make routine care (e.g., haircuts, dental work) extremely difficult. Adaptive skills are typically below measured IQ. Extreme difficulties in planning, organization, and coping with change negatively impact academic achievement, even for students with above-average intelligence. During adulthood, these individuals may have difficulties establishing independence because of continued rigidity and difficulty with novelty.

Many individuals with autism spectrum disorder, even without intellectual disability, have poor adult psychosocial functioning as indexed by measures such as independent living and gainful employment. Functional consequences in old age are unknown, but social isolation and communication problems (e.g., reduced help-seeking) are likely to have consequences for health in older adulthood.

Differential Diagnosis

Rett syndrome. Disruption of social interaction may be observed during the regressive phase of Rett syndrome (typically between 1–4 years of age); thus, a substantial proportion of affected young girls may have a presentation that meets diagnostic criteria for autism spectrum disorder. However, after this period, most individuals with Rett syndrome improve their social communication skills, and autistic features are no longer a major area of concern. Consequently, autism spectrum disorder should be considered only when all diagnostic criteria are met.

Selective mutism. In selective mutism, early development is not typically disturbed. The affected child usually exhibits appropriate communication skills in certain contexts and settings. Even in settings where the child is mute, social reciprocity is not impaired, nor are restricted or repetitive patterns of behavior present.

Language disorders and social (pragmatic) communication disorder. In some forms of language disorder, there may be problems of communication and some secondary social difficulties. However, specific language disorder is not usually associated with abnormal nonverbal communication, nor with the presence of restricted, repetitive patterns of behavior, interests, or activities.

When an individual shows impairment in social communication and social interactions but does not show restricted and repetitive behavior or interests, criteria for social (pragmatic) communication disorder, instead of autism spectrum disorder, may be met. The diagnosis of autism spectrum disorder supersedes that of social (pragmatic) communication disorder whenever the criteria for autism spectrum disorder are met, and care should be taken to enquire carefully regarding past or current restricted/repetitive behavior.

Intellectual disability (intellectual developmental disorder) without autism spectrum disorder. Intellectual disability without autism spectrum disorder may be difficult to differentiate from autism spectrum disorder in very young children. Individuals with intellectual disability who have not developed language or symbolic skills also present a challenge for differential diagnosis, since repetitive behavior often occurs in such individuals as well. A diagnosis of autism spectrum disorder in an individual with intellectual disability is appropriate when social communication and interaction are significantly impaired relative to the developmental level of the individual's nonverbal skills (e.g., fine motor skills, nonverbal problem solving). In contrast, intellectual disability is the appropriate diagnosis when there is no apparent discrepancy between the level of social-communicative skills and other intellectual skills.

Stereotypic movement disorder. Motor stereotypies are among the diagnostic characteristics of autism spectrum disorder, so an additional diagnosis of stereotypic movement disorder is not given when such repetitive behaviors are better explained by the presence of autism spectrum disorder. However, when stereotypies cause self-injury and become a focus of treatment, both diagnoses may be appropriate.

Attention-deficit/hyperactivity disorder. Abnormalities of attention (overly focused or easily distracted) are common in individuals with autism spectrum disorder, as is hyperactivity. A diagnosis of attention-deficit/hyperactivity disorder (ADHD) should be considered when attentional difficulties or hyperactivity exceeds that typically seen in individuals of comparable mental age.

Schizophrenia. Schizophrenia with childhood onset usually develops after a period of normal, or near normal, development. A prodromal state has been described in which social impairment and atypical interests and beliefs occur, which could be confused with the social deficits seen in autism spectrum disorder. Hallucinations and delusions, which are defining features of schizophrenia, are not features of autism spectrum disorder. However, clinicians must take into account the potential for individuals with autism spectrum disorder to be concrete in their interpretation of questions regarding the key features of schizophrenia (e.g., "Do you hear voices when no one is there?" "Yes [on the radio]").

Comorbidity

Autism spectrum disorder is frequently associated with intellectual impairment and structural language disorder (i.e., an inability to comprehend and construct sentences with proper grammar), which should be noted under the relevant specifiers when applicable. Many individuals with autism spectrum disorder have psychiatric symptoms that do not form part of the diagnostic criteria for the disorder (about 70% of individuals with autism spectrum disorder may have one comorbid mental disorder, and 40% may have two or more comorbid mental disorders). When criteria for both ADHD and autism spectrum disorder are met, both diagnoses should be given. This same principle applies to concurrent diagnoses of autism spectrum disorder and developmental coordination disorder, anxiety disorders, depressive

disorders, and other comorbid diagnoses. Among individuals who are nonverbal or have language deficits, observable signs such as changes in sleep or eating and increases in challenging behavior should trigger an evaluation for anxiety or depression. Specific learning difficulties (literacy and numeracy) are common, as is developmental coordination disorder. Medical conditions commonly associated with autism spectrum disorder should be noted under the "associated with a known medical/genetic or environmental/acquired condition" specifier. Such medical conditions include epilepsy, sleep problems, and constipation. Avoidant-restrictive food intake disorder is a fairly frequent presenting feature of autism spectrum disorder, and extreme and narrow food preferences may persist.

Attention-Deficit/Hyperactivity Disorder

Attention-Deficit/Hyperactivity Disorder

Diagnostic Criteria

A. A persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development, as characterized by (1) and/or (2):

1. **Inattention:** Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities:

   Note: The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.

   a. Often fails to give close attention to details or makes careless mistakes in schoolwork, at work, or during other activities (e.g., overlooks or misses details, work is inaccurate).
   b. Often has difficulty sustaining attention in tasks or play activities (e.g., has difficulty remaining focused during lectures, conversations, or lengthy reading).
   c. Often does not seem to listen when spoken to directly (e.g., mind seems elsewhere, even in the absence of any obvious distraction).
   d. Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (e.g., starts tasks but quickly loses focus and is easily sidetracked).
   e. Often has difficulty organizing tasks and activities (e.g., difficulty managing sequential tasks; difficulty keeping materials and belongings in order; messy, disorganized work; has poor time management; fails to meet deadlines).
   f. Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (e.g., schoolwork or homework; for older adolescents and adults, preparing reports, completing forms, reviewing lengthy papers).
   g. Often loses things necessary for tasks or activities (e.g., school materials, pencils, books, tools, wallets, keys, paperwork, eyeglasses, mobile telephones).
   h. Is often easily distracted by extraneous stimuli (for older adolescents and adults, may include unrelated thoughts).
   i. Is often forgetful in daily activities (e.g., doing chores, running errands; for older adolescents and adults, returning calls, paying bills, keeping appointments).

2. **Hyperactivity and impulsivity:** Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities: **Note:** The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or a failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.
   a. Often fidgets with or taps hands or feet or squirms in seat.
   b. Often leaves seat in situations when remaining seated is expected (e.g., leaves his or her place in the classroom, in the office or other workplace, or in other situations that require remaining in place).
   c. Often runs about or climbs in situations where it is inappropriate. (**Note:** In adolescents or adults, may be limited to feeling restless.)
   d. Often unable to play or engage in leisure activities quietly.
   e. Is often "on the go," acting as if "driven by a motor" (e.g., is unable to be or uncomfortable being still for extended time, as in restaurants, meetings; may be experienced by others as being restless or difficult to keep up with).
   f. Often talks excessively.
   g. Often blurts out an answer before a question has been completed (e.g., completes people's sentences; cannot wait for turn in conversation).
   h. Often has difficulty waiting his or her turn (e.g., while waiting in line).
   i. Often interrupts or intrudes on others (e.g., butts into conversations, games, or activities; may start using other people's things without asking or receiving permission; for adolescents and adults, may intrude into or take over what others are doing).

B. Several inattentive or hyperactive-impulsive symptoms were present prior to age 12 years.
C. Several inattentive or hyperactive-impulsive symptoms are present in two or more settings (e.g., at home, school, or work; with friends or relatives; in other activities).
D. There is clear evidence that the symptoms interfere with, or reduce the quality of, social, academic, or occupational functioning.
E. The symptoms do not occur exclusively during the course of schizophrenia or another psychotic disorder and are not better explained by another mental disorder (e.g., mood disorder, anxiety disorder, dissociative disorder, personality disorder, substance intoxication or withdrawal).

Specify whether:

314.01 (F90.2) Combined presentation: If both Criterion A1 (inattention) and Criterion A2 (hyperactivity-impulsivity) are met for the past 6 months.

314.00 (F90.0) Predominantly inattentive presentation: If Criterion A1 (inattention) is met but Criterion A2 (hyperactivity-impulsivity) is not met for the past 6 months.

314.01 (F90.1) Predominantly hyperactive/impulsive presentation: If Criterion A2 (hyperactivity-impulsivity) is met and Criterion A1 (inattention) is not met for the past 6 months.

Specify if:

in partial remission: When full criteria were previously met, fewer than the full criteria have been met for the past 6 months, and the symptoms still result in impairment in social, academic, or occupational functioning.

Specify current severity:

Mild: Few, if any, symptoms in excess of those required to make the diagnosis are present, and symptoms result in no more than minor impairments in social or occupational functioning.

Moderate: Symptoms or functional impairment between "mild" and "severe" are present.

Severe: Many symptoms in excess of those required to make the diagnosis, or several symptoms that are particularly severe, are present, or the symptoms result in marked impairment in social or occupational functioning.

Diagnostic Features

The essential feature of attention-deficit/hyperactivity disorder (ADHD) is a persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development. *Inattention* manifests behaviorally in ADHD as wandering off task, lacking persistence, having difficulty sustaining focus, and being disorganized and is not due to defiance or lack of comprehension. *Hyperactivity* refers to excessive motor activity (such as a child running about) when it is not appropriate, or excessive fidgeting, tapping, or talkativeness. In adults, hyperactivity may manifest as extreme restlessness or wearing others out with their activity. *Impulsivity* refers to hasty actions that occur in the moment without forethought and that have high potential for harm to the individual (e.g., darting into the street without looking). Impulsivity may reflect a desire for immediate rewards or an inability to delay gratification. Impulsive behaviors may manifest as social intrusiveness (e.g., interrupting others excessively) and/or as making important decisions without consideration of long-term consequences (e.g., taking a job without adequate information).

ADHD begins in childhood. The requirement that several symptoms be present before age 12 years conveys the importance of a substantial clinical presentation during childhood. At the same time, an earlier age at onset is not specified because of difficulties in establishing precise childhood onset retrospectively. Adult recall of childhood symptoms tends to be unreliable, and it is beneficial to obtain ancillary information.

Manifestations of the disorder must be present in more than one setting (e.g., home and school, work). Confirmation of substantial symptoms across settings typically cannot be done accurately without consulting informants who have seen the individual in those settings. Typically, symptoms vary depending on context within a given setting. Signs of the disorder may be minimal or absent when the individual is receiving frequent rewards for appropriate behavior, is under close supervision, is in a novel setting, is engaged in especially interesting activities, has consistent external stimulation (e.g., via electronic screens), or is interacting in one-on-one situations (e.g., the clinician's office).

Associated Features Supporting Diagnosis

Mild delays in language, motor, or social development are not specific to ADHD but often cooccur. Associated features may include low frustration tolerance, irritability, or mood lability. Even in the absence of a specific learning disorder, academic or work performance is often impaired. Inattentive behavior is associated with various underlying cognitive processes, and individuals with ADHD may exhibit cognitive problems on tests of attention, executive function, or memory, although these tests are not sufficiently sensitive or specific to serve as diagnostic indices. By early adulthood, ADHD is associated with an increased risk of suicide attempt, primarily when comorbid with mood, conduct, or substance use disorders.

No biological marker is diagnostic for ADHD. As a group, compared with peers, children with ADHD display increased slow wave electroencephalograms, reduced total brain volume on magnetic resonance imaging, and possibly a delay in posterior to anterior cortical maturation, but these findings are not diagnostic. In the uncommon cases where there is a known genetic cause (e.g., Fragile X syndrome, 22q11 deletion syndrome), the ADHD presentation should still be diagnosed.

Prevalence

Population surveys suggest that ADHD occurs in most cultures in about 5% of children and about 2.5% of adults.

Development and Course

Many parents first observe excessive motor activity when the child is a toddler, but symptoms are difficult to distinguish from highly variable normative behaviors before age 4 years. ADHD is most often identified during elementary school years, and inattention becomes more prominent and impairing. The disorder is relatively stable through early adolescence, but some individuals have a worsened course with development of antisocial behaviors. In most individuals with ADHD, symptoms of motoric hyperactivity become less obvious in adolescence and adulthood, but difficulties with restlessness, inattention, poor planning, and impulsivity persist. A substantial proportion of children with ADHD remain relatively impaired into adulthood.

In preschool, the main manifestation is hyperactivity. Inattention becomes more prominent during elementary school. During adolescence, signs of hyperactivity (e.g., running and climbing) are less common and may be confined to fidgetiness or an inner feeling of jitteriness, restlessness, or impatience. In adulthood, along with inattention and restlessness, impulsivity may remain problematic even when hyperactivity has diminished.

Risk and Prognostic Factors

Temperamental. ADHD is associated with reduced behavioral inhibition, effortful control, or constraint; negative emotionality; and/or elevated novelty seeking. These traits may predispose some children to ADHD but are not specific to the disorder.

Environmental. Very low birth weight (less than 1,500 grams) conveys a two- to threefold risk for ADHD, but most children with low birth weight do not develop ADHD. Although ADHD is correlated with smoking during pregnancy, some of this association reflects common genetic risk. A minority of cases may be related to reactions to aspects of diet. There may be a history of child abuse, neglect, multiple foster placements, neurotoxin exposure (e.g., lead), infections (e.g., encephalitis), or alcohol exposure in utero. Exposure to environmental toxicants has been correlated with subsequent ADHD, but it is not known whether these associations are causal.

Genetic and physiological. ADHD is elevated in the first-degree biological relatives of individuals with ADHD. The heritability of ADHD is substantial. While specific genes have been correlated with ADHD, they are neither necessary nor sufficient causal factors. Visual and hearing impairments, metabolic abnormalities, sleep disorders, nutritional deficiencies, and epilepsy should be considered as possible influences on ADHD symptoms.

ADHD is not associated with specific physical features, although rates of minor physical anomalies (e.g., hypertelorism, highly arched palate, low-set ears) may be relatively elevated. Subtle motor delays and other neurological soft signs may occur. (Note that marked co-occurring clumsiness and motor delays should be coded separately [e.g., developmental coordination disorder].)

Course modifiers. Family interaction patterns in early childhood are unlikely to cause ADHD but may influence its course or contribute to secondary development of conduct problems.

Culture-Related Diagnostic Issues

Differences in ADHD prevalence rates across regions appear attributable mainly to different diagnostic and methodological practices. However, there also may be cultural variation in attitudes toward or interpretations of children's behaviors. Clinical identification rates in the United States for African American and Latino populations tend to be lower than for Caucasian populations. Informant symptom ratings may be influenced by cultural group of the child and the informant, suggesting that culturally appropriate practices are relevant in assessing ADHD.

Gender-Related Diagnostic Issues

ADHD is more frequent in males than in females in the general population, with a ratio of approximately 2:1 in children and 1.6:1 in adults. Females are more likely than males to present primarily with inattentive features.

Functional Consequences of Attention-Deficit/Hyperactivity Disorder

ADHD is associated with reduced school performance and academic attainment, social rejection, and, in adults, poorer occupational performance, attainment, attendance, and higher probability of unemployment as well as elevated interpersonal conflict. Children with ADHD are significantly more likely than their peers without ADHD to develop conduct disorder in adolescence and antisocial personality disorder in adulthood, consequently increasing the likelihood for substance use disorders and incarceration. The risk of subsequent substance use disorders is elevated, especially when conduct disorder or antisocial personality disorder develops. Individuals with ADHD are more likely than peers to be injured. Traffic accidents and violations are more frequent in drivers with ADHD. There may be an elevated likelihood of obesity among individuals with ADHD.

Inadequate or variable self-application to tasks that require sustained effort is often interpreted by others as laziness, irresponsibility, or failure to cooperate. Family relationships may be characterized by discord and negative interactions. Peer relationships are often disrupted by peer rejection, neglect, or teasing of the individual with ADHD. On average, individuals with ADHD obtain less schooling, have poorer vocational achievement, and have reduced intellectual scores than their peers, although there is great variability. In its severe form, the disorder is markedly impairing, affecting social, familial, and scholastic/occupational adjustment.

Academic deficits, school-related problems, and peer neglect tend to be most associated with elevated symptoms of inattention, whereas peer rejection and, to a lesser extent, accidental injury are most salient with marked symptoms of hyperactivity or impulsivity.

Differential Diagnosis

Oppositional defiant disorder. Individuals with oppositional defiant disorder may resist work or school tasks that require self-application because they resist conforming to others' demands. Their behavior is characterized by negativity, hostility, and defiance. These symptoms must be differentiated from aversion to school or mentally demanding tasks due to difficulty in sustaining mental effort, forgetting instructions, and impulsivity in individuals with ADHD. Complicating the differential diagnosis is the fact that some individuals with ADHD may develop secondary oppositional attitudes toward such tasks and devalue their importance.

Intermittent explosive disorder. ADHD and intermittent explosive disorder share high levels of impulsive behavior. However, individuals with intermittent explosive disorder show serious aggression toward others, which is not characteristic of ADHD, and they do not experience problems with sustaining attention as seen in ADHD. In addition, intermittent explosive disorder is rare in childhood. Intermittent explosive disorder may be diagnosed in the presence of ADHD.

Other neurodevelopmental disorders. The increased motoric activity that may occur in ADHD must be distinguished from the repetitive motor behavior that characterizes stereotypic movement disorder and some cases of autism spectrum disorder. In stereotypic movement disorder, the motoric behavior is generally fixed and repetitive (e.g., body rocking, self-biting), whereas the fidgetiness and restlessness in ADHD are typically generalized and not characterized by repetitive stereotypic movements. In Tourette's disorder,

frequent multiple tics can be mistaken for the generalized fidgetiness of ADHD. Prolonged observation may be needed to differentiate fidgetiness from bouts of multiple tics.

Specific learning disorder. Children with specific learning disorder may appear inattentive because of frustration, lack of interest, or limited ability. However, inattention in individuals with a specific learning disorder who do not have ADHD is not impairing outside of academic work.

Intellectual disability (intellectual developmental disorder). Symptoms of ADHD are common among children placed in academic settings that are inappropriate to their intellectual ability. In such cases, the symptoms are not evident during non-academic tasks. A diagnosis of ADHD in intellectual disability requires that inattention or hyperactivity be excessive for mental age.

Autism spectrum disorder. Individuals with ADHD and those with autism spectrum disorder exhibit inattention, social dysfunction, and difficult-to-manage behavior. The social dysfunction and peer rejection seen in individuals with ADHD must be distinguished from the social disengagement, isolation, and indifference to facial and tonal communication cues seen in individuals with autism spectrum disorder. Children with autism spectrum disorder may display tantrums because of an inability to tolerate a change from their expected course of events. In contrast, children with ADHD may misbehave or have a tantrum during a major transition because of impulsivity or poor self-control.

Reactive attachment disorder. Children with reactive attachment disorder may show social disinhibition, but not the full ADHD symptom cluster, and display other features such as a lack of enduring relationships that are not characteristic of ADHD.

Anxiety disorders. ADHD shares symptoms of inattention with anxiety disorders. Individuals with ADHD are inattentive because of their attraction to external stimuli, new activities, or preoccupation with enjoyable activities. This is distinguished from the inattention due to worry and rumination seen in anxiety disorders. Restlessness might be seen in anxiety disorders. However, in ADHD, the symptom is not associated with worry and rumination.

Depressive disorders. Individuals with depressive disorders may present with inability to concentrate. However, poor concentration in mood disorders becomes prominent only during a depressive episode.

Bipolar disorder. Individuals with bipolar disorder may have increased activity, poor concentration, and increased impulsivity, but these features are episodic, occurring several days at a time. In bipolar disorder, increased impulsivity or inattention is accompanied by elevated mood, grandiosity, and other specific bipolar features. Children with ADHD may show significant changes in mood within the same day; such lability is distinct from a manic episode, which must last 4 or more days to be a clinical indicator of bipolar disorder, even in children. Bipolar disorder is rare in preadolescents, even when severe irritability and anger are prominent, whereas ADHD is common among children and adolescents who display excessive anger and irritability.

Disruptive mood dysregulation disorder. Disruptive mood dysregulation disorder is characterized by pervasive irritability, and intolerance of frustration, but impulsiveness and disorganized attention are not essential features. However, most children and adolescents with the disorder have symptoms that also meet criteria for ADHD, which is diagnosed separately.

Substance use disorders. Differentiating ADHD from substance use disorders may be problematic if the first presentation of ADHD symptoms follows the onset of abuse or frequent use. Clear evidence of ADHD before substance misuse from informants or previous records may be essential for differential diagnosis.

Personality disorders. In adolescents and adults, it may be difficult to distinguish ADHD from borderline, narcissistic, and other personality disorders. All these disorders tend to share the features of disorganization, social intrusiveness, emotional dysregulation, and cognitive dysregulation. However, ADHD is not characterized by fear of abandonment, self-injury, extreme ambivalence, or other features of personality disorder. It may take extended clinical observation, informant interview, or detailed history to distinguish impulsive, socially intrusive, or inappropriate behavior from narcissistic, aggressive, or domineering behavior to make this differential diagnosis.

Psychotic disorders. ADHD is not diagnosed if the symptoms of inattention and hyperactivity occur exclusively during the course of a psychotic disorder.

Medication-induced symptoms of ADHD. Symptoms of inattention, hyperactivity, or impulsivity attributable to the use of medication (e.g., bronchodilators, isoniazid, neuroleptics [resulting in akathisia], thyroid replacement medication) are diagnosed as other specified or unspecified other (or unknown) substance–related disorders.

Neurocognitive disorders. Early major neurocognitive disorder (dementia) and/or mild neurocognitive disorder are not known to be associated with ADHD but may present with similar clinical features. These conditions are distinguished from ADHD by their late onset.

Comorbidity

In clinical settings, comorbid disorders are frequent in individuals whose symptoms meet criteria for ADHD. In the general population, oppositional defiant disorder co-occurs with ADHD in approximately half of children with the combined presentation and about a quarter with the predominantly inattentive presentation. Conduct disorder co-occurs in about a quarter of children or adolescents with the combined presentation, depending on age and setting. Most children and adolescents with disruptive mood dysregulation disorder have symptoms that also meet criteria for ADHD; a lesser percentage of children with ADHD have symptoms that meet criteria for disruptive mood dysregulation disorder. Specific learning disorder commonly co-occurs with ADHD. Anxiety disorders and major depressive disorder occur in a minority of individuals with ADHD but more often than in the general population. Intermittent explosive disorder occurs in a minority of adults with ADHD, but at rates above population levels. Although substance use disorders are relatively more frequent among adults with ADHD in the general population, the disorders are present in only a minority of adults with ADHD. In adults, antisocial and other personality disorders may co-occur with ADHD. Other disorders that may co-occur with ADHD include obsessive-compulsive disorder, tic disorders, and autism spectrum disorder.

Other Specified Attention-Deficit/Hyperactivity Disorder

314.01 (F90.8)

This category applies to presentations in which symptoms characteristic of attention-deficit/hyperactivity disorder that cause clinically significant distress or impairment in social, occupational or other important areas of functioning predominate but do not meet the full criteria for attention-deficit/hyperactivity disorder or any of the disorders in the neurodevelopmental disorders diagnostic class. The other specified attention-deficit/hyperactivity disorder category is used in situations in which the clinician chooses to communicate

the specific reason that the presentation does not meet the criteria for attention-deficit/hyperactivity disorder or any specific neurodevelopmental disorder. This is done by recording "other specified attention-deficit/hyperactivity disorder" followed by the specific reason (e.g., "with insufficient inattention symptoms").

Unspecified Attention-Deficit/Hyperactivity Disorder

314.01 (F90.9)

This category applies to presentations in which symptoms characteristic of attention-deficit/hyperactivity disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for attention-deficit/hyperactivity disorder or any of the disorders in the neurodevelopmental disorders diagnostic class. The unspecified attention-deficit/hyperactivity disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for attention-deficit/hyperactivity disorder or for a specific neurodevelopmental disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis.

Specific Learning Disorder

Specific Learning Disorder

Diagnostic Criteria

A. Difficulties learning and using academic skills, as indicated by the presence of at least one of the following symptoms that have persisted for at least 6 months, despite the provision of interventions that target those difficulties:

1. Inaccurate or slow and effortful word reading (e.g., reads single words aloud incorrectly or slowly and hesitantly, frequently guesses words, has difficulty sounding out words).
2. Difficulty understanding the meaning of what is read (e.g., may read text accurately but not understand the sequence, relationships, inferences, or deeper meanings of what is read).
3. Difficulties with spelling (e.g., may add, omit, or substitute vowels or consonants).
4. Difficulties with written expression (e.g., makes multiple grammatical or punctuation errors within sentences; employs poor paragraph organization; written expression of ideas lacks clarity).
5. Difficulties mastering number sense, number facts, or calculation (e.g., has poor understanding of numbers, their magnitude, and relationships; counts on fingers to add single-digit numbers instead of recalling the math fact as peers do; gets lost in the midst of arithmetic computation and may switch procedures).
6. Difficulties with mathematical reasoning (e.g., has severe difficulty applying mathematical concepts, facts, or procedures to solve quantitative problems).

B. The affected academic skills are substantially and quantifiably below those expected for the individual's chronological age, and cause significant interference with academic or occupational performance, or with activities of daily living, as confirmed by individually administered standardized achievement measures and comprehensive clinical assessment. For individuals age 17 years and older, a documented history of impairing learning difficulties may be substituted for the standardized assessment.
C. The learning difficulties begin during school-age years but may not become fully manifest until the demands for those affected academic skills exceed the individual's limited capacities (e.g., as in timed tests, reading or writing lengthy complex reports for a tight deadline, excessively heavy academic loads).
D. The learning difficulties are not better accounted for by intellectual disabilities, uncorrected visual or auditory acuity, other mental or neurological disorders, psychosocial adversity, lack of proficiency in the language of academic instruction, or inadequate educational instruction.

Note: The four diagnostic criteria are to be met based on a clinical synthesis of the individual's history (developmental, medical, family, educational), school reports, and psychoeducational assessment.

Coding note: Specify all academic domains and subskills that are impaired. When more than one domain is impaired, each one should be coded individually according to the following specifiers.

Specify if:

315.00 (F81.0) With impairment in reading:

Word reading accuracy
Reading rate or fluency
Reading comprehension

Note: *Dyslexia* is an alternative term used to refer to a pattern of learning difficulties characterized by problems with accurate or fluent word recognition, poor decoding, and poor spelling abilities. If dyslexia is used to specify this particular pattern of difficulties, it is important also to specify any additional difficulties that are present, such as difficulties with reading comprehension or math reasoning.

315.2 (F81.81) With impairment in written expression:

Spelling accuracy
Grammar and punctuation accuracy
Clarity or organization of written expression

315.1 (F81.2) With impairment in mathematics:

Number sense
Memorization of arithmetic facts
Accurate or fluent calculation
Accurate math reasoning

Note: *Dyscalculia* is an alternative term used to refer to a pattern of difficulties characterized by problems processing numerical information, learning arithmetic facts, and performing accurate or fluent calculations. If dyscalculia is used to specify this particular pattern of mathematic difficulties, it is important also to specify any additional difficulties that are present, such as difficulties with math reasoning or word reasoning accuracy.

Specify current severity:

Mild: Some difficulties learning skills in one or two academic domains, but of mild enough severity that the individual may be able to compensate or function well when provided with appropriate accommodations or support services, especially during the school years.

Moderate: Marked difficulties learning skills in one or more academic domains, so that the individual is unlikely to become proficient without some intervals of intensive and specialized teaching during the school years. Some accommodations or supportive services at least part of the day at school, in the workplace, or at home may be needed to complete activities accurately and efficiently.

Severe: Severe difficulties learning skills, affecting several academic domains, so that the individual is unlikely to learn those skills without ongoing intensive individualized and specialized teaching for most of the school years. Even with an array of appropriate accommodations or services at home, at school, or in the workplace, the individual may not be able to complete all activities efficiently.

Recording Procedures

Each impaired academic domain and subskill of specific learning disorder should be recorded. Because of ICD coding requirements, impairments in reading, impairments in written expression, and impairments in mathematics, with their corresponding impairments in subskills, must be coded separately. For example, impairments in reading and mathematics and impairments in the subskills of reading rate or fluency, reading comprehension, accurate or fluent calculation, and accurate math reasoning would be coded and recorded as 315.00 (F81.0) specific learning disorder with impairment in reading, with impairment in reading rate or fluency and impairment in reading comprehension; 315.1 (F81.2) specific learning disorder with impairment in mathematics, with impairment in accurate or fluent calculation and impairment in accurate math reasoning.

Diagnostic Features

Specific learning disorder is a neurodevelopmental disorder with a biological origin that is the basis for abnormalities at a cognitive level that are associated with the behavioral signs of the disorder. The biological origin includes an interaction of genetic, epigenetic, and environmental factors, which affect the brain's ability to perceive or process verbal or nonverbal information efficiently and accurately.

One essential feature of specific learning disorder is persistent difficulties learning keystone academic skills (Criterion A), with onset during the years of formal schooling (i.e., the developmental period). Key academic skills include reading of single words accurately and fluently, reading comprehension, written expression and spelling, arithmetic calculation, and mathematical reasoning (solving mathematical problems). In contrast to talking or walking, which are acquired developmental milestones that emerge with brain maturation, academic skills (e.g., reading, spelling, writing, mathematics) have to be taught and learned explicitly. Specific learning disorder disrupts the normal pattern of learning academic skills; it is not simply a consequence of lack of opportunity of learning or inadequate instruction. Difficulties mastering these key academic skills may also impede learning in other academic subjects (e.g., history, science, social studies), but those problems are attributable to difficulties learning the underlying academic skills. Difficulties learning to map letters with the sounds of one's language—to read printed words (often called *dyslexia*)—is one of the most common manifestations of specific learning disorder. The learning difficulties manifest as a range of observable, descriptive behaviors or symptoms (as listed in Criteria A1–A6). These clinical symptoms may be observed, probed by means of the clinical interview, or ascertained from school reports, rating scales, or descriptions in previous educational or psychological assessments. The learning difficulties are persistent, not transitory. In children and adolescents, *persistence* is defined as restricted progress in learning (i.e., no evidence that the individual is catching up with classmates) for at least 6 months despite the provision of extra help at home or school. For example, difficulties learning to read single words that do not fully or rapidly remit with the provision of instruction in phonological skills or word identification strategies may indicate a specific

learning disorder. Evidence of persistent learning difficulties may be derived from cumulative school reports, portfolios of the child's evaluated work, curriculum-based measures, or clinical interview. In adults, persistent difficulty refers to ongoing difficulties in literacy or numeracy skills that manifest during childhood or adolescence, as indicated by cumulative evidence from school reports, evaluated portfolios of work, or previous assessments.

A second key feature is that the individual's performance of the affected academic skills is well below average for age (Criterion B). One robust clinical indicator of difficulties learning academic skills is low academic achievement for age or average achievement that is sustainable only by extraordinarily high levels of effort or support. In children, the low academic skills cause significant interference in school performance (as indicated by school reports and teacher's grades or ratings). Another clinical indicator, particularly in adults, is avoidance of activities that require the academic skills. Also in adulthood, low academic skills interfere with occupational performance or everyday activities requiring those skills (as indicated by self-report or report by others). However, this criterion also requires psychometric evidence from an individually administered, psychometrically sound and culturally appropriate test of academic achievement that is norm-referenced or criterion-referenced. Academic skills are distributed along a continuum, so there is no natural cutpoint that can be used to differentiate individuals with and without specific learning disorder. Thus, any threshold used to specify what constitutes significantly low academic achievement (e.g., academic skills well below age expectation) is to a large extent arbitrary. Low achievement scores on one or more standardized tests or subtests within an academic domain (i.e., at least 1.5 standard deviations [SD] below the population mean for age, which translates to a standard score of 78 or less, which is below the 7th percentile) are needed for the greatest diagnostic certainty. However, precise scores will vary according to the particular standardized tests that are used. On the basis of clinical judgment, a more lenient threshold may be used (e.g., 1.0–2.5 SD below the population mean for age), when learning difficulties are supported by converging evidence from clinical assessment, academic history, school reports, or test scores. Moreover, since standardized tests are not available in all languages, the diagnosis may then be based in part on clinical judgment of scores on available test measures.

A third core feature is that the learning difficulties are readily apparent in the early school years in most individuals (Criterion C). However, in others, the learning difficulties may not manifest fully until later school years, by which time learning demands have increased and exceed the individual's limited capacities.

Another key diagnostic feature is that the learning difficulties are considered "specific," for four reasons. First, they are not attributable to intellectual disabilities (intellectual disability [intellectual developmental disorder]); global developmental delay; hearing or vision disorders, or neurological or motor disorders) (Criterion D). Specific learning disorder affects learning in individuals who otherwise demonstrate normal levels of intellectual functioning (generally estimated by an IQ score of greater than about 70 [±5 points allowing for measurement error]). The phrase "unexpected academic underachievement" is often cited as the defining characteristic of specific learning disorder in that the specific learning disabilities are not part of a more general learning difficulty as manifested in intellectual disability or global developmental delay. Specific learning disorder may also occur in individuals identified as intellectually "gifted." These individuals may be able to sustain apparently adequate academic functioning by using compensatory strategies, extraordinarily high effort, or support, until the learning demands or assessment procedures (e.g., timed tests) pose barriers to their demonstrating their learning or accomplishing required tasks. Second, the learning difficulty cannot be attributed to more general external factors, such as economic or environmental disadvantage, chronic absenteeism, or lack of education as typically provided in the individual's community context. Third, the learning difficulty cannot be attributed to a neurological (e.g., pediatric stroke) or motor disorders or to vision or hearing disorders, which are often associated with problems learning academic skills but are distinguishable by presence of neurological signs.

Finally, the learning difficulty may be restricted to one academic skill or domain (e.g., reading single words, retrieving or calculating number facts).

Comprehensive assessment is required. Specific learning disorder can only be diagnosed after formal education starts but can be diagnosed at any point afterward in children, adolescents, or adults, providing there is evidence of onset during the years of formal schooling (i.e., the developmental period). No single data source is sufficient for a diagnosis of specific learning disorder. Rather, specific learning disorder is a clinical diagnosis based on a synthesis of the individual's medical, developmental, educational, and family history; the history of the learning difficulty, including its previous and current manifestation; the impact of the difficulty on academic, occupational, or social functioning; previous or current school reports; portfolios of work requiring academic skills; curriculum-based assessments; and previous or current scores from individual standardized tests of academic achievement. If an intellectual, sensory, neurological, or motor disorder is suspected, then the clinical assessment for specific learning disorder should also include methods appropriate for these disorders. Thus, comprehensive assessment will involve professionals with expertise in specific learning disorder and psychological/cognitive assessment. Since specific learning disorder typically persists into adulthood, reassessment is rarely necessary, unless indicated by marked changes in the learning difficulties (amelioration or worsening) or requested for specific purposes.

Associated Features Supporting Diagnosis

Specific learning disorder is frequently but not invariably preceded, in preschool years, by delays in attention, language, or motor skills that may persist and co-occur with specific learning disorder. An uneven profile of abilities is common, such as above-average abilities in drawing, design, and other visuospatial abilities, but slow, effortful, and inaccurate reading and poor reading comprehension and written expression. Individuals with specific learning disorder typically (but not invariably) exhibit poor performance on psychological tests of cognitive processing. However, it remains unclear whether these cognitive abnormalities are the cause, correlate, or consequence of the learning difficulties. Also, although cognitive deficits associated with difficulties learning to read words are well documented, those associated with other manifestations of specific learning disorder (e.g., reading comprehension, arithmetic computation, written expression) are underspecified or unknown. Moreover, individuals with similar behavioral symptoms or test scores are found to have a variety of cognitive deficits, and many of these processing deficits are also found in other neurodevelopmental disorders (e.g., attention-deficit/hyperactivity disorder [ADHD], autistic spectrum disorder, communication disorders, developmental coordination disorder). Thus, assessment of cognitive processing deficits is not required for diagnostic assessment. Specific learning disorder is associated with increased risk for suicidal ideation and suicide attempts in children, adolescents, and adults.

There are no known biological markers of specific learning disorder. As a group, individuals with the disorder show circumscribed alterations in cognitive processing and brain structure and function. Genetic differences are also evident at the group level. But cognitive testing, neuroimaging, or genetic testing are not useful for diagnosis at this time.

Prevalence

The prevalence of specific learning disorder across the academic domains of reading, writing, and mathematics is 5%–15% among school-age children across different languages and cultures. Prevalence in adults is unknown but appears to be approximately 4%.

Development and Course

Onset, recognition, and diagnosis of specific learning disorder usually occurs during the elementary school years when children are required to learn to read, spell, write, and learn

mathematics. However, precursors such as language delays or deficits, difficulties in rhyming or counting, or difficulties with fine motor skills required for writing commonly occur in early childhood before the start of formal schooling. Manifestations may be behavioral (e.g., a reluctance to engage in learning; oppositional behavior). Specific learning disorder is lifelong, but the course and clinical expression are variable, in part depending on the interactions among the task demands of the environment, the range and severity of the individual's learning difficulties, the individual's learning abilities, comorbidity, and the available support systems and intervention. Nonetheless, problems with reading fluency and comprehension, spelling, written expression, and numeracy skills in everyday life typically persist into adulthood.

Changes in manifestation of symptoms occur with age, so that an individual may have a persistent or shifting array of learning difficulties across the lifespan.

Examples of symptoms that may be observed among preschool-age children include a lack of interest in playing games with language sounds (e.g., repetition, rhyming), and they may have trouble learning nursery rhymes. Preschool children with specific learning disorder may frequently use baby talk, mispronounce words, and have trouble remembering names of letters, numbers, or days of the week. They may fail to recognize letters in their own names and have trouble learning to count. Kindergarten-age children with specific learning disorder may be unable to recognize and write letters, may be unable to write their own names, or may use invented spelling. They may have trouble breaking down spoken words into syllables (e.g., "cowboy" into "cow" and "boy") and trouble recognizing words that rhyme (e.g., cat, bat, hat). Kindergarten-age children also may have trouble connecting letters with their sounds (e.g., letter b makes the sound /b/) and may be unable to recognize phonemes (e.g., do not know which in a set of words [e.g., dog, man, car] starts with the same sound as "cat").

Specific learning disorder in elementary school–age children typically manifests as marked difficulty learning letter-sound correspondence (particularly in English-speaking children), fluent word decoding, spelling, or math facts; reading aloud is slow, inaccurate, and effortful, and some children struggle to understand the magnitude that a spoken or written number represents. Children in primary grades (grades 1–3) may continue to have problems recognizing and manipulating phonemes, be unable to read common one-syllable words (such as mat or top), and be unable recognize common irregularly spelled words (e.g., said, two). They may commit reading errors that indicate problems in connecting sounds and letters (e.g., "big" for "got") and have difficulty sequencing numbers and letters. Children in grades 1-3 also may have difficulty remembering number facts or arithmetic procedures for adding, subtracting, and so forth, and may complain that reading or arithmetic is hard and avoid doing it. Children with specific learning disorder in the middle grades (grades 4–6) may mispronounce or skip parts of long, multisyllable words (e.g., say "conible" for "convertible," "aminal" for "animal") and confuse words that sound alike (e.g., "tornado" for "volcano"). They may have trouble remembering dates, names, and telephone numbers and may have trouble completing homework or tests on time. Children in the middle grades also may have poor comprehension with or without slow, effortful, and inaccurate reading, and they may have trouble reading small function words (e.g., that, the, an, in). They may have very poor spelling and poor written work. They may get the first part of a word correctly, then guess wildly (e.g., read "clover" as "clock"), and may express fear of reading aloud or refuse to read aloud.

By contrast, adolescents may have mastered word decoding, but reading remains slow and effortful, and they are likely to show marked problems in reading comprehension and written expression (including poor spelling) and poor mastery of math facts or mathematical problem solving. During adolescence and into adulthood, individuals with specific learning disorder may continue to make numerous spelling mistakes and read single words and connected text slowly and with much effort, with trouble pronouncing multisyllable words. They may frequently need to reread material to understand or get the main point and have trouble making inferences from written text. Adolescents and adults may

avoid activities that demand reading or arithmetic (reading for pleasure, reading instructions). Adults with specific learning disorder have ongoing spelling problems, slow and effortful reading, or problems making important inferences from numerical information in work-related written documents. They may avoid both leisure and work-related activities that demand reading or writing or use alternative approaches to access print (e.g., text-to-speech/speech-to-text software, audiobooks, audiovisual media).

An alternative clinical expression is that of circumscribed learning difficulties that persist across the lifespan, such as an inability to master the basic sense of number (e.g., to know which of a pair of numbers or dots represents the larger magnitude), or lack of proficiency in word identification or spelling. Avoidance of or reluctance to engage in activities requiring academic skills is common in children, adolescents, and adults. Episodes of severe anxiety or anxiety disorders, including somatic complaints or panic attacks, are common across the lifespan and accompany both the circumscribed and the broader expression of learning difficulties.

Risk and Prognostic Factors

Environmental. Prematurity or very low birth weight increases the risk for specific learning disorder, as does prenatal exposure to nicotine.

Genetic and physiological. Specific learning disorder appears to aggregate in families, particularly when affecting reading, mathematics, and spelling. The relative risk of specific learning disorder in reading or mathematics is substantially higher (e.g., 4–8 times and 5–10 times higher, respectively) in first-degree relatives of individuals with these learning difficulties compared with those without them. Family history of reading difficulties (dyslexia) and parental literacy skills predict literacy problems or specific learning disorder in offspring, indicating the combined role of genetic and environmental factors.

There is high heritability for both reading ability and reading disability in alphabetic and nonalphabetic languages, including high heritability for most manifestations of learning abilities and disabilities (e.g., heritability estimate values greater than 0.6). Covariation between various manifestations of learning difficulties is high, suggesting that genes related to one presentation are highly correlated with genes related to another manifestation.

Course modifiers. Marked problems with inattentive behavior in preschool years is predictive of later difficulties in reading and mathematics (but not necessarily specific learning disorder) and nonresponse to effective academic interventions. Delay or disorders in speech or language, or impaired cognitive processing (e.g., phonological awareness, working memory, rapid serial naming) in preschool years, predicts later specific learning disorder in reading and written expression. Comorbidity with ADHD is predictive of worse mental health outcome than that associated with specific learning disorder without ADHD. Systematic, intensive, individualized instruction, using evidence-based interventions, may improve or ameliorate the learning difficulties in some individuals or promote the use of compensatory strategies in others, thereby mitigating the otherwise poor outcomes.

Culture-Related Diagnostic Issues

Specific learning disorder occurs across languages, cultures, races, and socioeconomic conditions but may vary in its manifestation according to the nature of the spoken and written symbol systems and cultural and educational practices. For example, the cognitive processing requirements of reading and of working with numbers vary greatly across orthographies. In the English language, the observable hallmark clinical symptom of difficulties learning to read is inaccurate and slow reading of single words; in other alphabetic languages that have more direct mapping between sounds and letters (e.g., Spanish, German) and in non-alphabetic languages (e.g., Chinese, Japanese), the hallmark feature is

slow but accurate reading. In English-language learners, assessment should include consideration of whether the source of reading difficulties is a limited proficiency with English or a specific learning disorder. Risk factors for specific learning disorder in English-language learners include a family history of specific learning disorder or language delay in the native language, as well as learning difficulties in English and failure to catch up with peers. If there is suspicion of cultural or language differences (e.g., as in an English-language learner), the assessment needs to take into account the individual's language proficiency in his or her first or native language as well as in the second language (in this example, English). Also, assessment should consider the linguistic and cultural context in which the individual is living, as well as his or her educational and learning history in the original culture and language.

Gender-Related Diagnostic Issues

Specific learning disorder is more common in males than in females (ratios range from about 2:1 to 3:1) and cannot be attributed to factors such as ascertainment bias, definitional or measurement variation, language, race, or socioeconomic status.

Functional Consequences of Specific Learning Disorder

Specific learning disorder can have negative functional consequences across the lifespan, including lower academic attainment, higher rates of high school dropout, lower rates of postsecondary education, high levels of psychological distress and poorer overall mental health, higher rates of unemployment and under-employment, and lower incomes. School dropout and co-occurring depressive symptoms increase the risk for poor mental health outcomes, including suicidality, whereas high levels of social or emotional support predict better mental health outcomes.

Differential Diagnosis

Normal variations in academic attainment. Specific learning disorder is distinguished from normal variations in academic attainment due to external factors (e.g., lack of educational opportunity, consistently poor instruction, learning in a second language), because the learning difficulties persist in the presence of adequate educational opportunity and exposure to the same instruction as the peer group, and competency in the language of instruction, even when it is different from one's primary spoken language.

Intellectual disability (intellectual developmental disorder). Specific learning disorder differs from general learning difficulties associated with intellectual disability, because the learning difficulties occur in the presence of normal levels of intellectual functioning (i.e., IQ score of at least 70 ± 5). If intellectual disability is present, specific learning disorder can be diagnosed only when the learning difficulties are in excess of those usually associated with the intellectual disability.

Learning difficulties due to neurological or sensory disorders. Specific learning disorder is distinguished from learning difficulties due to neurological or sensory disorders (e.g., pediatric stroke, traumatic brain injury, hearing impairment, vision impairment), because in these cases there are abnormal findings on neurological examination.

Neurocognitive disorders. Specific learning disorder is distinguished from learning problems associated with neurodegenerative cognitive disorders, because in specific learning disorder the clinical expression of specific learning difficulties occurs during the developmental period, and the difficulties do not manifest as a marked decline from a former state.

Attention-deficit/hyperactivity disorder. Specific learning disorder is distinguished from the poor academic performance associated with ADHD, because in the latter condition the problems may not necessarily reflect specific difficulties in learning academic skills but rather may reflect difficulties in performing those skills. However, the co-occurrence of specific learning disorder and ADHD is more frequent than expected by chance. If criteria for both disorders are met, both diagnoses can be given.

Psychotic disorders. Specific learning disorder is distinguished from the academic and cognitive-processing difficulties associated with schizophrenia or psychosis, because with these disorders there is a decline (often rapid) in these functional domains.

Comorbidity

Specific learning disorder commonly co-occurs with neurodevelopmental (e.g., ADHD, communication disorders, developmental coordination disorder, autistic spectrum disorder) or other mental disorders (e.g., anxiety disorders, depressive and bipolar disorders). These comorbidities do not necessarily exclude the diagnosis specific learning disorder but may make testing and differential diagnosis more difficult, because each of the co-occurring disorders independently interferes with the execution of activities of daily living, including learning. Thus, clinical judgment is required to attribute such impairment to learning difficulties. If there is an indication that another diagnosis could account for the difficulties learning keystone academic skills described in Criterion A, specific learning disorder should not be diagnosed.

Motor Disorders

Developmental Coordination Disorder

Diagnostic Criteria **315.4 (F82)**

A. The acquisition and execution of coordinated motor skills is substantially below that expected given the individual's chronological age and opportunity for skill learning and use. Difficulties are manifested as clumsiness (e.g., dropping or bumping into objects) as well as slowness and inaccuracy of performance of motor skills (e.g., catching an object, using scissors or cutlery, handwriting, riding a bike, or participating in sports).

B. The motor skills deficit in Criterion A significantly and persistently interferes with activities of daily living appropriate to chronological age (e.g., self-care and self-maintenance) and impacts academic/school productivity, prevocational and vocational activities, leisure, and play.

C. Onset of symptoms is in the early developmental period.

D. The motor skills deficits are not better explained by intellectual disability (intellectual developmental disorder) or visual impairment and are not attributable to a neurological condition affecting movement (e.g., cerebral palsy, muscular dystrophy, degenerative disorder).

Diagnostic Features

The diagnosis of developmental coordination disorder is made by a clinical synthesis of the history (developmental and medical), physical examination, school or workplace report, and individual assessment using psychometrically sound and culturally appropriate standardized tests. The manifestation of impaired skills requiring motor coordination (Criterion A) varies

with age. Young children may be delayed in achieving motor milestones (i.e., sitting, crawling, walking), although many achieve typical motor milestones. They also may be delayed in developing skills such as negotiating stairs, pedaling, buttoning shirts, completing puzzles, and using zippers. Even when the skill is achieved, movement execution may appear awkward, slow, or less precise than that of peers. Older children and adults may display slow speed or inaccuracy with motor aspects of activities such as assembling puzzles, building models, playing ball games (especially in teams), handwriting, typing, driving, or carrying out self-care skills.

Developmental coordination disorder is diagnosed only if the impairment in motor skills significantly interferes with the performance of, or participation in, daily activities in family, social, school, or community life (Criterion B). Examples of such activities include getting dressed, eating meals with age-appropriate utensils and without mess, engaging in physical games with others, using specific tools in class such as rulers and scissors, and participating in team exercise activities at school. Not only is ability to perform these actions impaired, but also marked slowness in execution is common. Handwriting competence is frequently affected, consequently affecting legibility and/or speed of written output and affecting academic achievement (the impact is distinguished from specific learning difficulty by the emphasis on the motoric component of written output skills). In adults, everyday skills in education and work, especially those in which speed and accuracy are required, are affected by coordination problems.

Criterion C states that the onset of symptoms of developmental coordination disorder must be in the early developmental period. However, developmental coordination disorder is typically not diagnosed before age 5 years because there is considerable variation in the age at acquisition of many motor skills or a lack of stability of measurement in early childhood (e.g., some children catch up) or because other causes of motor delay may not have fully manifested.

Criterion D specifies that the diagnosis of developmental coordination disorder is made if the coordination difficulties are not better explained by visual impairment or attributable to a neurological condition. Thus, visual function examination and neurological examination must be included in the diagnostic evaluation. If intellectual disability (intellectual developmental disorder) is present, the motor difficulties are in excess of those expected for the mental age; however, no IQ cut-off or discrepancy criterion is specified.

Developmental coordination disorder does not have discrete subtypes; however, individuals may be impaired predominantly in gross motor skills or in fine motor skills, including handwriting skills.

Other terms used to describe developmental coordination disorder include *childhood dyspraxia, specific developmental disorder of motor function,* and *clumsy child syndrome.*

Associated Features Supporting Diagnosis

Some children with developmental coordination disorder show additional (usually suppressed) motor activity, such as choreiform movements of unsupported limbs or mirror movements. These "overflow" movements are referred to as *neurodevelopmental immaturities* or *neurological soft signs* rather than neurological abnormalities. In both current literature and clinical practice, their role in diagnosis is still unclear, requiring further evaluation.

Prevalence

The prevalence of developmental coordination disorder in children ages 5–11 years is 5%–6% (in children age 7 years, 1.8% are diagnosed with severe developmental coordination disorder and 3% with probable developmental coordination disorder). Males are more often affected than females, with a male:female ratio between 2:1 and 7:1.

Development and Course

The course of developmental coordination disorder is variable but stable at least to 1 year follow-up. Although there may be improvement in the longer term, problems with coor-

dinated movements continue through adolescence in an estimated 50%–70% of children. Onset is in early childhood. Delayed motor milestones may be the first signs, or the disorder is first recognized when the child attempts tasks such as holding a knife and fork, buttoning clothes, or playing ball games. In middle childhood, there are difficulties with motor aspects of assembling puzzles, building models, playing ball, and handwriting, as well as with organizing belongings, when motor sequencing and coordination are required. In early adulthood, there is continuing difficulty in learning new tasks involving complex/automatic motor skills, including driving and using tools. Inability to take notes and handwrite quickly may affect performance in the workplace. Co-occurrence with other disorders (see the section "Comorbidity" for this disorder) has an additional impact on presentation, course, and outcome.

Risk and Prognostic Factors

Environmental. Developmental coordination disorder is more common following prenatal exposure to alcohol and in preterm and low-birth-weight children.

Genetic and physiological. Impairments in underlying neurodevelopmental processes—particularly in visual-motor skills, both in visual-motor perception and spatial mentalizing—have been found and affect the ability to make rapid motoric adjustments as the complexity of the required movements increases. Cerebellar dysfunction has been proposed, but the neural basis of developmental coordination disorder remains unclear. Because of the co-occurrence of developmental coordination disorder with attention-deficit/hyperactivity disorder (ADHD), specific learning disabilities, and autism spectrum disorder, shared genetic effect has been proposed. However, consistent co-occurrence in twins appears only in severe cases.

Course modifiers. Individuals with ADHD and with developmental coordination disorder demonstrate more impairment than individuals with ADHD without developmental coordination disorder.

Culture-Related Diagnostic Issues

Developmental coordination disorder occurs across cultures, races, and socioeconomic conditions. By definition, "activities of daily living" implies cultural differences necessitating consideration of the context in which the individual child is living as well as whether he or she has had appropriate opportunities to learn and practice such activities.

Functional Consequences of Developmental Coordination Disorder

Developmental coordination disorder leads to impaired functional performance in activities of daily living (Criterion B), and the impairment is increased with co-occurring conditions. Consequences of developmental coordination disorder include reduced participation in team play and sports; poor self-esteem and sense of self-worth; emotional or behavior problems; impaired academic achievement; poor physical fitness; and reduced physical activity and obesity.

Differential Diagnosis

Motor impairments due to another medical condition. Problems in coordination may be associated with visual function impairment and specific neurological disorders (e.g., cerebral palsy, progressive lesions of the cerebellum, neuromuscular disorders). In such cases, there are additional findings on neurological examination.

Intellectual disability (intellectual developmental disorder). If intellectual disability is present, motor competences may be impaired in accordance with the intellectual disabil-

ity. However, if the motor difficulties are in excess of what could be accounted for by the intellectual disability, and criteria for developmental coordination disorder are met, developmental coordination disorder can be diagnosed as well.

Attention-deficit/hyperactivity disorder. Individuals with ADHD may fall, bump into objects, or knock things over. Careful observation across different contexts is required to ascertain if lack of motor competence is attributable to distractibility and impulsiveness rather than to developmental coordination disorder. If criteria for both ADHD and developmental coordination disorder are met, both diagnoses can be given.

Autism spectrum disorder. Individuals with autism spectrum disorder may be uninterested in participating in tasks requiring complex coordination skills, such as ball sports, which will affect test performance and function but not reflect core motor competence. Co-occurrence of developmental coordination disorder and autism spectrum disorder is common. If criteria for both disorders are met, both diagnoses can be given.

Joint hypermobility syndrome. Individuals with syndromes causing hyperextensible joints (found on physical examination; often with a complaint of pain) may present with symptoms similar to those of developmental coordination disorder.

Comorbidity

Disorders that commonly co-occur with developmental coordination disorder include speech and language disorder; specific learning disorder (especially reading and writing); problems of inattention, including ADHD (the most frequent coexisting condition, with about 50% co-occurrence); autism spectrum disorder; disruptive and emotional behavior problems; and joint hypermobility syndrome. Different clusters of co-occurrence may be present (e.g., a cluster with severe reading disorders, fine motor problems, and handwriting problems; another cluster with impaired movement control and motor planning). Presence of other disorders does not exclude developmental coordination disorder but may make testing more difficult and may independently interfere with the execution of activities of daily living, thus requiring examiner judgment in ascribing impairment to motor skills.

Stereotypic Movement Disorder

Diagnostic Criteria **307.3 (F98.4)**

A. Repetitive, seemingly driven, and apparently purposeless motor behavior (e.g., hand shaking or waving, body rocking, head banging, self-biting, hitting own body).

B. The repetitive motor behavior interferes with social, academic, or other activities and may result in self-injury.

C. Onset is in the early developmental period.

D. The repetitive motor behavior is not attributable to the physiological effects of a substance or neurological condition and is not better explained by another neurodevelopmental or mental disorder (e.g., trichotillomania [hair-pulling disorder], obsessive-compulsive disorder).

Specify if:

With self-injurious behavior (or behavior that would result in an injury if preventive measures were not used)

Without self-injurious behavior

Specify if:

Associated with a known medical or genetic condition, neurodevelopmental disorder, or environmental factor (e.g., Lesch-Nyhan syndrome, intellectual disability [intellectual developmental disorder], intrauterine alcohol exposure)

Coding note: Use additional code to identify the associated medical or genetic condition, or neurodevelopmental disorder.

Specify current severity:

Mild: Symptoms are easily suppressed by sensory stimulus or distraction.

Moderate: Symptoms require explicit protective measures and behavioral modification.

Severe: Continuous monitoring and protective measures are required to prevent serious injury.

Recording Procedures

For stereotypic movement disorder that is associated with a known medical or genetic condition, neurodevelopmental disorder, or environmental factor, record stereotypic movement disorder associated with (name of condition, disorder, or factor) (e.g., stereotypic movement disorder associated with Lesch-Nyhan syndrome).

Specifiers

The severity of non-self-injurious stereotypic movements ranges from mild presentations that are easily suppressed by a sensory stimulus or distraction to continuous movements that markedly interfere with all activities of daily living. Self-injurious behaviors range in severity along various dimensions, including the frequency, impact on adaptive functioning, and severity of bodily injury (from mild bruising or erythema from hitting hand against body, to lacerations or amputation of digits, to retinal detachment from head banging).

Diagnostic Features

The essential feature of stereotypic movement disorder is repetitive, seemingly driven, and apparently purposeless motor behavior (Criterion A). These behaviors are often rhythmical movements of the head, hands, or body without obvious adaptive function. The movements may or may not respond to efforts to stop them. Among typically developing children, the repetitive movements may be stopped when attention is directed to them or when the child is distracted from performing them. Among children with neurodevelopmental disorders, the behaviors are typically less responsive to such efforts. In other cases, the individual demonstrates self-restraining behaviors (e.g., sitting on hands, wrapping arms in clothing, finding a protective device).

The repertoire of behaviors is variable; each individual presents with his or her own individually patterned, "signature" behavior. Examples of non-self-injurious stereotypic movements include, but are not limited to, body rocking, bilateral flapping or rotating hand movements, flicking or fluttering fingers in front of the face, arm waving or flapping, and head nodding. Stereotyped self-injurious behaviors include, but are not limited to, repetitive head banging, face slapping, eye poking, and biting of hands, lips, or other body parts. Eye poking is particularly concerning; it occurs more frequently among children with visual impairment. Multiple movements may be combined (e.g., cocking the head, rocking the torso, waving a small string repetitively in front of the face).

Stereotypic movements may occur many times during a day, lasting a few seconds to several minutes or longer. Frequency can vary from many occurrences in a single day to several weeks elapsing between episodes. The behaviors vary in context, occurring when the individual is engrossed in other activities, when excited, stressed, fatigued, or bored. Criterion A requires that the movements be "apparently" purposeless. However, some functions may be served by the movements. For example, stereotypic movements might reduce anxiety in response to external stressors.

Criterion B states that the stereotypic movements interfere with social, academic, or other activities and, in some children, may result in self-injury (or would if protective measures were not used). If self-injury is present, it should be coded using the specifier. Onset

of stereotypic movements is in the early developmental period (Criterion C). Criterion D states that the repetitive, stereotyped behavior in stereotypic movement disorder is not attributable to the physiological effects of a substance or neurological condition and is not better explained by another neurodevelopmental or mental disorder. The presence of stereotypic movements may indicate an undetected neurodevelopmental problem, especially in children ages 1–3 years.

Prevalence

Simple stereotypic movements (e.g., rocking) are common in young typically developing children. Complex stereotypic movements are much less common (occurring in approximately 3%–4%). Between 4% and 16% of individuals with intellectual disability (intellectual developmental disorder) engage in stereotypy and self-injury. The risk is greater in individuals with severe intellectual disability. Among individuals with intellectual disability living in residential facilities, 10%–15% may have stereotypic movement disorder with self-injury.

Development and Course

Stereotypic movements typically begin within the first 3 years of life. Simple stereotypic movements are common in infancy and may be involved in acquisition of motor mastery. In children who develop complex motor stereotypies, approximately 80% exhibit symptoms before 24 months of age, 12% between 24 and 35 months, and 8% at 36 months or older. In most typically developing children, these movements resolve over time or can be suppressed. Onset of complex motor stereotypies may be in infancy or later in the developmental period. Among individuals with intellectual disability, the stereotyped, self-injurious behaviors may persist for years, even though the typography or pattern of self-injury may change.

Risk and Prognostic Factors

Environmental. Social isolation is a risk factor for self-stimulation that may progress to stereotypic movements with repetitive self-injury. Environmental stress may also trigger stereotypic behavior. Fear may alter physiological state, resulting in increased frequency of stereotypic behaviors.

Genetic and physiological. Lower cognitive functioning is linked to greater risk for stereotypic behaviors and poorer response to interventions. Stereotypic movements are more frequent among individuals with moderate-to-severe/profound intellectual disability, who by virtue of a particular syndrome (e.g., Rett syndrome) or environmental factor (e.g., an environment with relatively insufficient stimulation) seem to be at higher risk for stereotypies. Repetitive self-injurious behavior may be a behavioral phenotype in neurogenetic syndromes. For example, in Lesch-Nyhan syndrome, there are both stereotypic dystonic movements and self-mutilation of fingers, lip biting, and other forms of self-injury unless the individual is restrained, and in Rett syndrome and Cornelia de Lange syndrome, self-injury may result from the hand-to-mouth stereotypies. Stereotypic behaviors may result from a painful medical condition (e.g., middle ear infection, dental problems, gastroesophageal reflux).

Culture-Related Diagnostic Issues

Stereotypic movement disorder, with or without self-injury, occurs in all races and cultures. Cultural attitudes toward unusual behaviors may result in delayed diagnosis. Overall cultural tolerance and attitudes toward stereotypic movement vary and must be considered.

Differential Diagnosis

Normal development. Simple stereotypic movements are common in infancy and early childhood. Rocking may occur in the transition from sleep to awake, a behavior that usu-

ally resolves with age. Complex stereotypies are less common in typically developing children and can usually be suppressed by distraction or sensory stimulation. The individual's daily routine is rarely affected, and the movements generally do not cause the child distress. The diagnosis would not be appropriate in these circumstances.

Autism spectrum disorder. Stereotypic movements may be a presenting symptom of autism spectrum disorder and should be considered when repetitive movements and behaviors are being evaluated. Deficits of social communication and reciprocity manifesting in autism spectrum disorder are generally absent in stereotypic movement disorder, and thus social interaction, social communication, and rigid repetitive behaviors and interests are distinguishing features. When autism spectrum disorder is present, stereotypic movement disorder is diagnosed only when there is self-injury or when the stereotypic behaviors are sufficiently severe to become a focus of treatment.

Tic disorders. Typically, stereotypies have an earlier age at onset (before 3 years) than do tics, which have a mean age at onset of 5–7 years. They are consistent and fixed in their pattern or topography compared with tics, which are variable in their presentation. Stereotypies may involve arms, hands, or the entire body, while tics commonly involve eyes, face, head, and shoulders. Stereotypies are more fixed, rhythmic, and prolonged in duration than tics, which, generally, are brief, rapid, random, and fluctuating. Tics and stereotypic movements are both reduced by distraction.

Obsessive-compulsive and related disorders. Stereotypic movement disorder is distinguished from obsessive-compulsive disorder (OCD) by the absence of obsessions, as well as by the nature of the repetitive behaviors. In OCD the individual feels driven to perform repetitive behaviors in response to an obsession or according to rules that must be applied rigidly, whereas in stereotypic movement disorder the behaviors are seemingly driven but apparently purposeless. Trichotillomania (hair-pulling disorder) and excoriation (skin-picking) disorder are characterized by body-focused repetitive behaviors (i.e., hair pulling and skin picking) that may be seemingly driven but that are not apparently purposeless, and that may not be patterned or rhythmical. Furthermore, onset in trichotillomania and excoriation disorder is not typically in the early developmental period, but rather around puberty or later.

Other neurological and medical conditions. The diagnosis of stereotypic movements requires the exclusion of habits, mannerisms, paroxysmal dyskinesias, and benign hereditary chorea. A neurological history and examination are required to assess features suggestive of other disorders, such as myoclonus, dystonia, tics, and chorea. Involuntary movements associated with a neurological condition may be distinguished by their signs and symptoms. For example, repetitive, stereotypic movements in tardive dyskinesia can be distinguished by a history of chronic neuroleptic use and characteristic oral or facial dyskinesia or irregular trunk or limb movements. These types of movements do not result in self-injury. A diagnosis of stereotypic movement disorder is not appropriate for repetitive skin picking or scratching associated with amphetamine intoxication or abuse (e.g., patients are diagnosed with substance/medication-induced obsessive-compulsive and related disorder) and repetitive choreoathetoid movements associated with other neurological disorders.

Comorbidity

Stereotypic movement disorder may occur as a primary diagnosis or secondary to another disorder. For example, stereotypies are a common manifestation of a variety of neurogenetic disorders, such as Lesch-Nyhan syndrome, Rett syndrome, fragile X syndrome, Cornelia de Lange syndrome, and Smith-Magenis syndrome. When stereotypic movement disorder co-occurs with another medical condition, both should be coded.

Tic Disorders

Diagnostic Criteria

Note: A tic is a sudden, rapid, recurrent, nonrhythmic motor movement or vocalization.

Tourette's Disorder 307.23 (F95.2)

A. Both multiple motor and one or more vocal tics have been present at some time during the illness, although not necessarily concurrently.
B. The tics may wax and wane in frequency but have persisted for more than 1 year since first tic onset.
C. Onset is before age 18 years.
D. The disturbance is not attributable to the physiological effects of a substance (e.g., cocaine) or another medical condition (e.g., Huntington's disease, postviral encephalitis).

Persistent (Chronic) Motor or Vocal Tic Disorder 307.22 (F95.1)

A. Single or multiple motor or vocal tics have been present during the illness, but not both motor and vocal.
B. The tics may wax and wane in frequency but have persisted for more than 1 year since first tic onset.
C. Onset is before age 18 years.
D. The disturbance is not attributable to the physiological effects of a substance (e.g., cocaine) or another medical condition (e.g., Huntington's disease, postviral encephalitis).
E. Criteria have never been met for Tourette's disorder.

Specify if:

With motor tics only
With vocal tics only

Provisional Tic Disorder 307.21 (F95.0)

A. Single or multiple motor and/or vocal tics.
B. The tics have been present for less than 1 year since first tic onset.
C. Onset is before age 18 years.
D. The disturbance is not attributable to the physiological effects of a substance (e.g., cocaine) or another medical condition (e.g., Huntington's disease, postviral encephalitis).
E. Criteria have never been met for Tourette's disorder or persistent (chronic) motor or vocal tic disorder.

Specifiers

The "motor tics only" or "vocal tics only" specifier is only required for persistent (chronic) motor or vocal tic disorder.

Diagnostic Features

Tic disorders comprise four diagnostic categories: Tourette's disorder, persistent (chronic) motor or vocal tic disorder, provisional tic disorder, and the other specified and unspecified tic disorders. Diagnosis for any tic disorder is based on the presence of motor and/or vocal tics (Criterion A), duration of tic symptoms (Criterion B), age at onset (Criterion C), and absence of any known cause such as another medical condition or substance use (Criterion D). The tic disorders are hierarchical in order (i.e., Tourette's disorder, followed by persistent [chronic] motor or vocal tic disorder, followed by provisional tic disorder, followed by the

other specified and unspecified tic disorders), such that once a tic disorder at one level of the hierarchy is diagnosed, a lower hierarchy diagnosis cannot be made (Criterion E).

Tics are sudden, rapid, recurrent, nonrhythmic motor movements or vocalizations. An individual may have various tic symptoms over time, but at any point in time, the tic repertoire recurs in a characteristic fashion. Although tics can include almost any muscle group or vocalization, certain tic symptoms, such as eye blinking or throat clearing, are common across patient populations. Tics are generally experienced as involuntary but can be voluntarily suppressed for varying lengths of time.

Tics can be either simple or complex. *Simple motor tics* are of short duration (i.e., milliseconds) and can include eye blinking, shoulder shrugging, and extension of the extremities. Simple vocal tics include throat clearing, sniffing, and grunting often caused by contraction of the diaphragm or muscles of the oropharynx. *Complex motor tics* are of longer duration (i.e., seconds) and often include a combination of simple tics such as simultaneous head turning and shoulder shrugging. Complex tics can appear purposeful, such as a tic-like sexual or obscene gesture (*copropraxia*) or a tic-like imitation of someone else's movements (*echopraxia*). Similarly, complex vocal tics include repeating one's own sounds or words (*palilalia*), repeating the last-heard word or phrase (*echolalia*), or uttering socially unacceptable words, including obscenities, or ethnic, racial, or religious slurs (*coprolalia*). Importantly, coprolalia is an abrupt, sharp bark or grunt utterance and lacks the prosody of similar inappropriate speech observed in human interactions.

The presence of motor and/or vocal tics varies across the four tic disorders (Criterion A). For Tourette's disorder, both motor and vocal tics must be present, whereas for persistent (chronic) motor or vocal tic disorder, only motor or only vocal tics are present. For provisional tic disorder, motor and/or vocal tics may be present. For other specified or unspecified tic disorders, the movement disorder symptoms are best characterized as tics but are atypical in presentation or age at onset, or have a known etiology.

The 1-year minimum duration criterion (Criterion B) assures that individuals diagnosed with either Tourette's disorder or persistent (chronic) motor or vocal tic disorder have had persistent symptoms. Tics wax and wane in severity, and some individuals may have tic-free periods of weeks to months; however, an individual who has had tic symptoms of greater than 1 year's duration since first tic onset would be considered to have persistent symptoms regardless of duration of tic-free periods. For an individual with motor and/or vocal tics of less than 1 year since first tic onset, a provisional tic disorder diagnosis can be considered. There is no duration specification for other specified and unspecified tic disorders. The onset of tics must occur prior to age 18 years (Criterion C). Tic disorders typically begin in the prepubertal period, with an average age at onset between 4 and 6 years, and with the incidence of new-onset tic disorders decreasing in the teen years. New onset of tic symptoms in adulthood is exceedingly rare and is often associated with exposures to drugs (e.g., excessive cocaine use) or is a result of a central nervous system insult (e.g., postviral encephalitis). Although tic onset is uncommon in teenagers and adults, it is not uncommon for adolescents and adults to present for an initial diagnostic assessment and, when carefully evaluated, provide a history of milder symptoms dating back to childhood. New-onset abnormal movements suggestive of tics outside of the usual age range should result in evaluation for other movement disorders or for specific etiologies.

Tic symptoms cannot be attributable to the physiological effects of a substance or another medical condition (Criterion D). When there is strong evidence from the history, physical examination, and/or laboratory results to suggest a plausible, proximal, and probable cause for a tic disorder, a diagnosis of other specified tic disorder should be used.

Having previously met diagnostic criteria for Tourette's disorder negates a possible diagnosis of persistent (chronic) motor or vocal tic disorder (Criterion E). Similarly, a previous diagnosis of persistent (chronic) motor or vocal tic disorder negates a diagnosis of provisional tic disorder or other specified or unspecified tic disorder (Criterion E).

Prevalence

Tics are common in childhood but transient in most cases. The estimated prevalence of Tourette's disorder ranges from 3 to 8 per 1,000 in school-age children. Males are more commonly affected than females, with the ratio varying from 2:1 to 4:1. A national survey in the United States estimated 3 per 1,000 for the prevalence of clinically identified cases. The frequency of identified cases was lower among African Americans and Hispanic Americans, which may be related to differences in access to care.

Development and Course

Onset of tics is typically between ages 4 and 6 years. Peak severity occurs between ages 10 and 12 years, with a decline in severity during adolescence. Many adults with tic disorders experience diminished symptoms. A small percentage of individuals will have persistently severe or worsening symptoms in adulthood.

Tic symptoms manifest similarly in all age groups and across the lifespan. Tics wax and wane in severity and change in affected muscle groups and vocalizations over time. As children get older, they begin to report their tics being associated with a premonitory urge—a somatic sensation that precedes the tic—and a feeling of tension reduction following the expression of the tic. Tics associated with a premonitory urge may be experienced as not completely "involuntary" in that the urge and the tic can be resisted. An individual may also feel the need to perform a tic in a specific way or repeat it until he or she achieves the feeling that the tic has been done "just right."

The vulnerability toward developing co-occurring conditions changes as individuals pass through the age of risk for various co-occurring conditions. For example, prepubertal children with tic disorders are more likely to experience attention-deficit/hyperactivity disorder (ADHD), obsessive-compulsive disorder (OCD), and separation anxiety disorder than are teenagers and adults, who are more likely to experience the new onset of major depressive disorder, substance use disorder, or bipolar disorder.

Risk and Prognostic Factors

Temperamental. Tics are worsened by anxiety, excitement, and exhaustion and are better during calm, focused activities. Individuals may have fewer tics when engaged in schoolwork or tasks at work than when relaxing at home after school or in the evening. Stressful/exciting events (e.g., taking a test, participating in exciting activities) often make tics worse.

Environmental. Observing a gesture or sound in another person may result in an individual with a tic disorder making a similar gesture or sound, which may be incorrectly perceived by others as purposeful. This can be a particular problem when the individual is interacting with authority figures (e.g., teachers, supervisors, police).

Genetic and physiological. Genetic and environmental factors influence tic symptom expression and severity. Important risk alleles for Tourette's disorder and rare genetic variants in families with tic disorders have been identified. Obstetrical complications, older paternal age, lower birth weight, and maternal smoking during pregnancy are associated with worse tic severity.

Culture-Related Diagnostic Issues

Tic disorders do not appear to vary in clinical characteristics, course, or etiology by race, ethnicity, and culture. However, race, ethnicity, and culture may impact how tic disorders are perceived and managed in the family and community, as well as influencing patterns of help seeking, and choices of treatment.

Gender-Related Diagnostic Issues

Males are more commonly affected than females, but there are no gender differences in the kinds of tics, age at onset, or course. Women with persistent tic disorders may be more likely to experience anxiety and depression.

Functional Consequences of Tic Disorders

Many individuals with mild to moderate tic severity experience no distress or impairment in functioning and may even be unaware of their tics. Individuals with more severe symptoms generally have more impairment in daily living, but even individuals with moderate or even severe tic disorders may function well. The presence of a co-occurring condition, such as ADHD or OCD, can have greater impact on functioning. Less commonly, tics disrupt functioning in daily activities and result in social isolation, interpersonal conflict, peer victimization, inability to work or to go to school, and lower quality of life. The individual also may experience substantial psychological distress. Rare complications of Tourette's disorder include physical injury, such as eye injury (from hitting oneself in the face), and orthopedic and neurological injury (e.g., disc disease related to forceful head and neck movements).

Differential Diagnosis

Abnormal movements that may accompany other medical conditions and stereotypic movement disorder. *Motor stereotypies* are defined as involuntary rhythmic, repetitive, predictable movements that appear purposeful but serve no obvious adaptive function or purpose and stop with distraction. Examples include repetitive hand waving/rotating, arm flapping, and finger wiggling. Motor stereotypies can be differentiated from tics based on the former's earlier age at onset (younger than 3 years), prolonged duration (seconds to minutes), constant repetitive fixed form and location, exacerbation when engrossed in activities, lack of a premonitory urge, and cessation with distraction (e.g., name called or touched). *Chorea* represents rapid, random, continual, abrupt, irregular, unpredictable, nonstereotyped actions that are usually bilateral and affect all parts of the body (i.e., face, trunk, and limbs). The timing, direction, and distribution of movements vary from moment to moment, and movements usually worsen during attempted voluntary action. *Dystonia* is the simultaneous sustained contracture of both agonist and antagonist muscles, resulting in a distorted posture or movement of parts of the body. Dystonic postures are often triggered by attempts at voluntary movements and are not seen during sleep.

Substance-induced and paroxysmal dyskinesias. Paroxysmal dyskinesias usually occur as dystonic or choreoathetoid movements that are precipitated by voluntary movement or exertion and less commonly arise from normal background activity.

Myoclonus. Myoclonus is characterized by a sudden unidirectional movement that is often nonrhythmic. It may be worsened by movement and occur during sleep. Myoclonus is differentiated from tics by its rapidity, lack of suppressibility, and absence of a premonitory urge.

Obsessive-compulsive and related disorders. Differentiating obsessive-compulsive behaviors from tics may be difficult. Clues favoring an obsessive-compulsive behavior include a cognitive-based drive (e.g., fear of contamination) and the need to perform the action in a particular fashion a certain number of times, equally on both sides of the body, or until a "just right" feeling is achieved. Impulse-control problems and other repetitive behaviors, including persistent hair pulling, skin picking, and nail biting, appear more goal directed and complex than tics.

Comorbidity

Many medical and psychiatric conditions have been described as co-occurring with tic disorders, with ADHD and obsessive-compulsive and related disorders being particularly common. The obsessive-compulsive symptoms observed in tic disorder tend to be characterized by more aggressive symmetry and order symptoms and poorer response to pharmacotherapy with selective serotonin reuptake inhibitors. Children with ADHD may demonstrate disruptive behavior, social immaturity, and learning difficulties that may interfere with academic progress and interpersonal relationships and lead to greater impairment than that caused by a tic disorder. Individuals with tic disorders can also have other movement disorders and other mental disorders, such as depressive, bipolar, or substance use disorders.

Other Specified Tic Disorder

307.20 (F95.8)

This category applies to presentations in which symptoms characteristic of a tic disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for a tic disorder or any of the disorders in the neurodevelopmental disorders diagnostic class. The other specified tic disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for a tic disorder or any specific neurodevelopmental disorder. This is done by recording "other specified tic disorder" followed by the specific reason (e.g., "with onset after age 18 years").

Unspecified Tic Disorder

307.20 (F95.9)

This category applies to presentations in which symptoms characteristic of a tic disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for a tic disorder or for any of the disorders in the neurodevelopmental disorders diagnostic class. The unspecified tic disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a tic disorder or for a specific neurodevelopmental disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis.

Other Neurodevelopmental Disorders

Other Specified Neurodevelopmental Disorder

315.8 (F88)

This category applies to presentations in which symptoms characteristic of a neurodevelopmental disorder that cause impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the neurodevelopmental disorders diagnostic class. The other specified neurodevelopmental disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific neurodevelopmental disorder. This is done by recording "other specified neurodevelopmental disorder" followed by the specific reason (e.g., "neurodevelopmental disorder associated with prenatal alcohol exposure").

An example of a presentation that can be specified using the "other specified" designation is the following:

Neurodevelopmental disorder associated with prenatal alcohol exposure: Neurodevelopmental disorder associated with prenatal alcohol exposure is characterized by a range of developmental disabilities following exposure to alcohol in utero.

Unspecified Neurodevelopmental Disorder

315.9 (F89)

This category applies to presentations in which symptoms characteristic of a neurodevelopmental disorder that cause impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the neurodevelopmental disorders diagnostic class. The unspecified neurodevelopmental disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific neurodevelopmental disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Schizophrenia Spectrum and Other Psychotic Disorders

Schizophrenia spectrum and other psychotic disorders include schizophrenia, other psychotic disorders, and schizotypal (personality) disorder. They are defined by abnormalities in one or more of the following five domains: delusions, hallucinations, disorganized thinking (speech), grossly disorganized or abnormal motor behavior (including catatonia), and negative symptoms.

Key Features That Define the Psychotic Disorders

Delusions

Delusions are fixed beliefs that are not amenable to change in light of conflicting evidence. Their content may include a variety of themes (e.g., persecutory, referential, somatic, religious, grandiose). *Persecutory delusions* (i.e., belief that one is going to be harmed, harassed, and so forth by an individual, organization, or other group) are most common. *Referential delusions* (i.e., belief that certain gestures, comments, environmental cues, and so forth are directed at oneself) are also common. *Grandiose delusions* (i.e., when an individual believes that he or she has exceptional abilities, wealth, or fame) and *erotomanic delusions* (i.e., when an individual believes falsely that another person is in love with him or her) are also seen. *Nihilistic delusions* involve the conviction that a major catastrophe will occur, and *somatic delusions* focus on preoccupations regarding health and organ function.

Delusions are deemed *bizarre* if they are clearly implausible and not understandable to same-culture peers and do not derive from ordinary life experiences. An example of a bizarre delusion is the belief that an outside force has removed his or her internal organs and replaced them with someone else's organs without leaving any wounds or scars. An example of a nonbizarre delusion is the belief that one is under surveillance by the police, despite a lack of convincing evidence. Delusions that express a loss of control over mind or body are generally considered to be bizarre; these include the belief that one's thoughts have been "removed" by some outside force (*thought withdrawal*), that alien thoughts have been put into one's mind (*thought insertion*), or that one's body or actions are being acted on or manipulated by some outside force (*delusions of control*). The distinction between a delusion and a strongly held idea is sometimes difficult to make and depends in part on the degree of conviction with which the belief is held despite clear or reasonable contradictory evidence regarding its veracity.

Hallucinations

Hallucinations are perception-like experiences that occur without an external stimulus. They are vivid and clear, with the full force and impact of normal perceptions, and not under voluntary control. They may occur in any sensory modality, but auditory hallucinations are the most common in schizophrenia and related disorders. Auditory hallucinations are usually experienced as voices, whether familiar or unfamiliar, that are perceived as distinct from the individual's own thoughts. The hallucinations must occur in the context of a clear sensorium; those that occur while falling asleep (*hypnagogic*) or waking up

(*hypnopompic*) are considered to be within the range of normal experience. Hallucinations may be a normal part of religious experience in certain cultural contexts.

Disorganized Thinking (Speech)

Disorganized thinking (*formal thought disorder*) is typically inferred from the individual's speech. The individual may switch from one topic to another (*derailment or loose associations*). Answers to questions may be obliquely related or completely unrelated (*tangentiality*). Rarely, speech may be so severely disorganized that it is nearly incomprehensible and resembles receptive aphasia in its linguistic disorganization (*incoherence* or "word salad"). Because mildly disorganized speech is common and nonspecific, the symptom must be severe enough to substantially impair effective communication. The severity of the impairment may be difficult to evaluate if the person making the diagnosis comes from a different linguistic background than that of the person being examined. Less severe disorganized thinking or speech may occur during the prodromal and residual periods of schizophrenia.

Grossly Disorganized or Abnormai Motor Behavior (Inciuding Catatonia)

Grossly disorganized or abnormal motor behavior may manifest itself in a variety of ways, ranging from childlike "silliness" to unpredictable agitation. Problems may be noted in any form of goal-directed behavior, leading to difficulties in performing activities of daily living.

Catatonic behavior is a marked decrease in reactivity to the environment. This ranges from resistance to instructions (*negativism*); to maintaining a rigid, inappropriate or bizarre posture; to a complete lack of verbal and motor responses (*mutism* and *stupor*). It can also include purposeless and excessive motor activity without obvious cause (*catatonic excitement*). Other features are repeated stereotyped movements, staring, grimacing, mutism, and the echoing of speech. Although catatonia has historically been associated with schizophrenia, catatonic symptoms are nonspecific and may occur in other mental disorders (e.g., bipolar or depressive disorders with catatonia) and in medical conditions (catatonic disorder due to another medical condition).

Negative Symptoms

Negative symptoms account for a substantial portion of the morbidity associated with schizophrenia but are less prominent in other psychotic disorders. Two negative symptoms are particularly prominent in schizophrenia: diminished emotional expression and avolition. *Diminished emotional expression* includes reductions in the expression of emotions in the face, eye contact, intonation of speech (prosody), and movements of the hand, head, and face that normally give an emotional emphasis to speech. *Avolition* is a decrease in motivated self-initiated purposeful activities. The individual may sit for long periods of time and show little interest in participating in work or social activities. Other negative symptoms include alogia, anhedonia, and asociality. *Alogia* is manifested by diminished speech output. *Anhedonia* is the decreased ability to experience pleasure from positive stimuli or a degradation in the recollection of pleasure previously experienced. *Asociality* refers to the apparent lack of interest in social interactions and may be associated with avolition, but it can also be a manifestation of limited opportunities for social interactions.

Disorders in This Chapter

This chapter is organized along a gradient of psychopathology. Clinicians should first consider conditions that do not reach full criteria for a psychotic disorder or are limited to one

domain of psychopathology. Then they should consider time-limited conditions. Finally, the diagnosis of a schizophrenia spectrum disorder requires the exclusion of another condition that may give rise to psychosis.

Schizotypal personality disorder is noted within this chapter as it is considered within the schizophrenia spectrum, although its full description is found in the chapter "Personality Disorders." The diagnosis schizotypal personality disorder captures a pervasive pattern of social and interpersonal deficits, including reduced capacity for close relationships; cognitive or perceptual distortions; and eccentricities of behavior, usually beginning by early adulthood but in some cases first becoming apparent in childhood and adolescence. Abnormalities of beliefs, thinking, and perception are below the threshold for the diagnosis of a psychotic disorder.

Two conditions are defined by abnormalities limited to one domain of psychosis: delusions or catatonia. Delusional disorder is characterized by at least 1 month of delusions but no other psychotic symptoms. Catatonia is described later in the chapter and further in this discussion.

Brief psychotic disorder lasts more than 1 day and remits by 1 month. Schizophreniform disorder is characterized by a symptomatic presentation equivalent to that of schizophrenia except for its duration (less than 6 months) and the absence of a requirement for a decline in functioning.

Schizophrenia lasts for at least 6 months and includes at least 1 month of active-phase symptoms. In schizoaffective disorder, a mood episode and the active-phase symptoms of schizophrenia occur together and were preceded or are followed by at least 2 weeks of delusions or hallucinations without prominent mood symptoms.

Psychotic disorders may be induced by another condition. In substance/medication-induced psychotic disorder, the psychotic symptoms are judged to be a physiological consequence of a drug of abuse, a medication, or toxin exposure and cease after removal of the agent. In psychotic disorder due to another medical condition, the psychotic symptoms are judged to be a direct physiological consequence of another medical condition.

Catatonia can occur in several disorders, including neurodevelopmental, psychotic, bipolar, depressive, and other mental disorders. This chapter also includes the diagnoses catatonia associated with another mental disorder (catatonia specifier), catatonic disorder due to another medical condition, and unspecified catatonia, and the diagnostic criteria for all three conditions are described together.

Other specified and unspecified schizophrenia spectrum and other psychotic disorders are included for classifying psychotic presentations that do not meet the criteria for any of the specific psychotic disorders, or psychotic symptomatology about which there is inadequate or contradictory information.

Clinician-Rated Assessment of Symptoms and Related Clinical Phenomena in Psychosis

Psychotic disorders are heterogeneous, and the severity of symptoms can predict important aspects of the illness, such as the degree of cognitive or neurobiological deficits. To move the field forward, a detailed framework for the assessment of severity is included in Section III "Assessment Measures," which may help with treatment planning, prognostic decision making, and research on pathophysiological mechanisms. Section III "Assessment Measures" also contains dimensional assessments of the primary symptoms of psychosis, including hallucinations, delusions, disorganized speech (except for substance/medication-induced psychotic disorder and psychotic disorder due to another medical condition), abnormal psychomotor behavior, and negative symptoms, as well as dimensional assessments of depression and mania. The severity of mood symptoms in psychosis has prognostic value and guides treatment. There is growing evidence that schizoaffective

disorder is not a distinct nosological category. Thus, dimensional assessments of depression and mania for all psychotic disorders alert clinicians to mood pathology and the need to treat where appropriate. The Section III scale also includes a dimensional assessment of cognitive impairment. Many individuals with psychotic disorders have impairments in a range of cognitive domains that predict functional status. Clinical neuropsychological assessment can help guide diagnosis and treatment, but brief assessments without formal neuropsychological assessment can provide useful information that can be sufficient for diagnostic purposes. Formal neuropsychological testing, when conducted, should be administered and scored by personnel trained in the use of testing instruments. If a formal neuropsychological assessment is not conducted, the clinician should use the best available information to make a judgment. Further research on these assessments is necessary in order to determine their clinical utility; thus, the assessments available in Section III should serve as a prototype to stimulate such research.

Schizotypal (Personality) Disorder

Criteria and text for schizotypal personality disorder can be found in the chapter "Personality Disorders." Because this disorder is considered part of the schizophrenia spectrum of disorders, and is labeled in this section of ICD-9 and ICD-10 as schizotypal disorder, it is listed in this chapter and discussed in detail in the DSM-5 chapter "Personality Disorders."

Delusional Disorder

Diagnostic Criteria **297.1 (F22)**

A. The presence of one (or more) delusions with a duration of 1 month or longer.

B. Criterion A for schizophrenia has never been met.

Note: Hallucinations, if present, are not prominent and are related to the delusional theme (e.g., the sensation of being infested with insects associated with delusions of infestation).

C. Apart from the impact of the delusion(s) or its ramifications, functioning is not markedly impaired, and behavior is not obviously bizarre or odd.

D. If manic or major depressive episodes have occurred, these have been brief relative to the duration of the delusional periods.

E. The disturbance is not attributable to the physiological effects of a substance or another medical condition and is not better explained by another mental disorder, such as body dysmorphic disorder or obsessive-compulsive disorder.

Specify whether:

Erotomanic type: This subtype applies when the central theme of the delusion is that another person is in love with the individual.

Grandiose type: This subtype applies when the central theme of the delusion is the conviction of having some great (but unrecognized) talent or insight or having made some important discovery.

Jealous type: This subtype applies when the central theme of the individual's delusion is that his or her spouse or lover is unfaithful.

Persecutory type: This subtype applies when the central theme of the delusion involves the individual's belief that he or she is being conspired against, cheated, spied on, followed, poisoned or drugged, maliciously maligned, harassed, or obstructed in the pursuit of long-term goals.

Somatic type: This subtype applies when the central theme of the delusion involves bodily functions or sensations.

Mixed type: This subtype applies when no one delusional theme predominates.

Unspecified type: This subtype applies when the dominant delusional belief cannot be clearly determined or is not described in the specific types (e.g., referential delusions without a prominent persecutory or grandiose component).

Specify if:

With bizarre content: Delusions are deemed bizarre if they are clearly implausible, not understandable, and not derived from ordinary life experiences (e.g., an individual's belief that a stranger has removed his or her internal organs and replaced them with someone else's organs without leaving any wounds or scars).

Specify if:

The following course specifiers are only to be used after a 1-year duration of the disorder:

First episode, currently in acute episode: First manifestation of the disorder meeting the defining diagnostic symptom and time criteria. An *acute episode* is a time period in which the symptom criteria are fulfilled.

First episode, currently in partial remission: *Partial remission* is a time period during which an improvement after a previous episode is maintained and in which the defining criteria of the disorder are only partially fulfilled.

First episode, currently in full remission: *Full remission* is a period of time after a previous episode during which no disorder-specific symptoms are present.

Multiple episodes, currently in acute episode

Multiple episodes, currently in partial remission

Multiple episodes, currently in full remission

Continuous: Symptoms fulfilling the diagnostic symptom criteria of the disorder are remaining for the majority of the illness course, with subthreshold symptom periods being very brief relative to the overall course.

Unspecified

Specify current severity:

Severity is rated by a quantitative assessment of the primary symptoms of psychosis, including delusions, hallucinations, disorganized speech, abnormal psychomotor behavior, and negative symptoms. Each of these symptoms may be rated for its current severity (most severe in the last 7 days) on a 5-point scale ranging from 0 (not present) to 4 (present and severe). (See Clinician-Rated Dimensions of Psychosis Symptom Severity in the chapter "Assessment Measures.")

Note: Diagnosis of delusional disorder can be made without using this severity specifier.

Subtypes

In *erotomanic type,* the central theme of the delusion is that another person is in love with the individual. The person about whom this conviction is held is usually of higher status (e.g., a famous individual or a superior at work) but can be a complete stranger. Efforts to contact the object of the delusion are common. In *grandiose type,* the central theme of the delusion is the conviction of having some great talent or insight or of having made some important discovery. Less commonly, the individual may have the delusion of having a special relationship with a prominent individual or of being a prominent person (in which case the actual individual may be regarded as an impostor). Grandiose delusions may have a religious content. In *jealous type,* the central theme of the delusion is that of an unfaithful partner. This belief is arrived at without due cause and is based on incorrect inferences supported by small bits of "evidence" (e.g., disarrayed clothing). The individual with the delusion usually confronts the spouse or lover and attempts to intervene in the imagined infidelity. In *persecutory type,* the central theme of the delusion involves the in-

dividual's belief of being conspired against, cheated, spied on, followed, poisoned, maliciously maligned, harassed, or obstructed in the pursuit of long-term goals. Small slights may be exaggerated and become the focus of a delusional system. The affected individual may engage in repeated attempts to obtain satisfaction by legal or legislative action. Individuals with persecutory delusions are often resentful and angry and may resort to violence against those they believe are hurting them. In *somatic type*, the central theme of the delusion involves bodily functions or sensations. Somatic delusions can occur in several forms. Most common is the belief that the individual emits a foul odor; that there is an infestation of insects on or in the skin; that there is an internal parasite; that certain parts of the body are misshapen or ugly; or that parts of the body are not functioning.

Diagnostic Features

The essential feature of delusional disorder is the presence of one or more delusions that persist for at least 1 month (Criterion A). A diagnosis of delusional disorder is not given if the individual has ever had a symptom presentation that met Criterion A for schizophrenia (Criterion B). Apart from the direct impact of the delusions, impairments in psychosocial functioning may be more circumscribed than those seen in other psychotic disorders such as schizophrenia, and behavior is not obviously bizarre or odd (Criterion C). If mood episodes occur concurrently with the delusions, the total duration of these mood episodes is brief relative to the total duration of the delusional periods (Criterion D). The delusions are not attributable to the physiological effects of a substance (e.g., cocaine) or another medical condition (e.g., Alzheimer's disease) and are not better explained by another mental disorder, such as body dysmorphic disorder or obsessive-compulsive disorder (Criterion E).

In addition to the five symptom domain areas identified in the diagnostic criteria, the assessment of cognition, depression, and mania symptom domains is vital for making critically important distinctions between the various schizophrenia spectrum and other psychotic disorders.

Associated Features Supporting Diagnosis

Social, marital, or work problems can result from the delusional beliefs of delusional disorder. Individuals with delusional disorder may be able to factually describe that others view their beliefs as irrational but are unable to accept this themselves (i.e., there may be "factual insight" but no true insight). Many individuals develop irritable or dysphoric mood, which can usually be understood as a reaction to their delusional beliefs. Anger and violent behavior can occur with persecutory, jealous, and erotomanic types. The individual may engage in litigious or antagonistic behavior (e.g., sending hundreds of letters of protest to the government). Legal difficulties can occur, particularly in jealous and erotomanic types.

Prevalence

The lifetime prevalence of delusional disorder has been estimated at around 0.2%, and the most frequent subtype is persecutory. Delusional disorder, jealous type, is probably more common in males than in females, but there are no major gender differences in the overall frequency of delusional disorder.

Development and Course

On average, global function is generally better than that observed in schizophrenia. Although the diagnosis is generally stable, a proportion of individuals go on to develop

schizophrenia. Delusional disorder has a significant familial relationship with both schizophrenia and schizotypal personality disorder. Although it can occur in younger age groups, the condition may be more prevalent in older individuals.

Culture-Related Diagnostic Issues

An individual's cultural and religious background must be taken into account in evaluating the possible presence of delusional disorder. The content of delusions also varies across cultural contexts.

Functional Consequences of Delusional Disorder

The functional impairment is usually more circumscribed than that seen with other psychotic disorders, although in some cases, the impairment may be substantial and include poor occupational functioning and social isolation. When poor psychosocial functioning is present, delusional beliefs themselves often play a significant role. A common characteristic of individuals with delusional disorder is the apparent normality of their behavior and appearance when their delusional ideas are not being discussed or acted on.

Differential Diagnosis

Obsessive-compulsive and related disorders. If an individual with obsessive-compulsive disorder is completely convinced that his or her obsessive-compulsive disorder beliefs are true, then the diagnosis of obsessive-compulsive disorder, with absent insight/delusional beliefs specifier, should be given rather than a diagnosis of delusional disorder. Similarly, if an individual with body dysmorphic disorder is completely convinced that his or her body dysmorphic disorder beliefs are true, then the diagnosis of body dysmorphic disorder, with absent insight/delusional beliefs specifier, should be given rather than a diagnosis of delusional disorder.

Delirium, major neurocognitive disorder, psychotic disorder due to another medical condition, and substance/medication-induced psychotic disorder. Individuals with these disorders may present with symptoms that suggest delusional disorder. For example, simple persecutory delusions in the context of major neurocognitive disorder would be diagnosed as major neurocognitive disorder, with behavioral disturbance. A substance/medication-induced psychotic disorder cross-sectionally may be identical in symptomatology to delusional disorder but can be distinguished by the chronological relationship of substance use to the onset and remission of the delusional beliefs.

Schizophrenia and schizophreniform disorder. Delusional disorder can be distinguished from schizophrenia and schizophreniform disorder by the absence of the other characteristic symptoms of the active phase of schizophrenia.

Depressive and bipolar disorders and schizoaffective disorder. These disorders may be distinguished from delusional disorder by the temporal relationship between the mood disturbance and the delusions and by the severity of the mood symptoms. If delusions occur exclusively during mood episodes, the diagnosis is depressive or bipolar disorder with psychotic features. Mood symptoms that meet full criteria for a mood episode can be superimposed on delusional disorder. Delusional disorder can be diagnosed only if the total duration of all mood episodes remains brief relative to the total duration of the delusional disturbance. If not, then a diagnosis of other specified or unspecified schizophrenia spectrum and other psychotic disorder accompanied by other specified depressive disorder, unspecified depressive disorder, other specified bipolar and related disorder, or unspecified bipolar and related disorder is appropriate.

Brief Psychotic Disorder

Diagnostic Criteria **298.8 (F23)**

A. Presence of one (or more) of the following symptoms. At least one of these must be (1), (2), or (3):

1. Delusions.
2. Hallucinations.
3. Disorganized speech (e.g., frequent derailment or incoherence).
4. Grossly disorganized or catatonic behavior.

Note: Do not include a symptom if it is a culturally sanctioned response.

B. Duration of an episode of the disturbance is at least 1 day but less than 1 month, with eventual full return to premorbid level of functioning.

C. The disturbance is not better explained by major depressive or bipolar disorder with psychotic features or another psychotic disorder such as schizophrenia or catatonia, and is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.

Specify if:

With marked stressor(s) (brief reactive psychosis): If symptoms occur in response to events that, singly or together, would be markedly stressful to almost anyone in similar circumstances in the individual's culture.

Without marked stressor(s): If symptoms do not occur in response to events that, singly or together, would be markedly stressful to almost anyone in similar circumstances in the individual's culture.

With postpartum onset: If onset is during pregnancy or within 4 weeks postpartum.

Specify if:

With catatonia (refer to the criteria for catatonia associated with another mental disorder, pp. 119–120, for definition)

Coding note: Use additional code 293.89 (F06.1) catatonia associated with brief psychotic disorder to indicate the presence of the comorbid catatonia.

Specify current severity:

Severity is rated by a quantitative assessment of the primary symptoms of psychosis, including delusions, hallucinations, disorganized speech, abnormal psychomotor behavior, and negative symptoms. Each of these symptoms may be rated for its current severity (most severe in the last 7 days) on a 5-point scale ranging from 0 (not present) to 4 (present and severe). (See Clinician-Rated Dimensions of Psychosis Symptom Severity in the chapter "Assessment Measures.")

Note: Diagnosis of brief psychotic disorder can be made without using this severity specifier.

Diagnostic Features

The essential feature of brief psychotic disorder is a disturbance that involves the sudden onset of at least one of the following positive psychotic symptoms: delusions, hallucinations, disorganized speech (e.g., frequent derailment or incoherence), or grossly abnormal psychomotor behavior, including catatonia (Criterion A). *Sudden onset* is defined as change from a nonpsychotic state to a clearly psychotic state within 2 weeks, usually without a prodrome. An episode of the disturbance lasts at least 1 day but less than 1 month, and the individual eventually has a full return to the premorbid level of functioning (Cri-

terion B). The disturbance is not better explained by a depressive or bipolar disorder with psychotic features, by schizoaffective disorder, or by schizophrenia and is not attributable to the physiological effects of a substance (e.g., a hallucinogen) or another medical condition (e.g., subdural hematoma) (Criterion C).

In addition to the five symptom domain areas identified in the diagnostic criteria, the assessment of cognition, depression, and mania symptom domains is vital for making critically important distinctions between the various schizophrenia spectrum and other psychotic disorders.

Associated Features Supporting Diagnosis

Individuals with brief psychotic disorder typically experience emotional turmoil or overwhelming confusion. They may have rapid shifts from one intense affect to another. Although the disturbance is brief, the level of impairment may be severe, and supervision may be required to ensure that nutritional and hygienic needs are met and that the individual is protected from the consequences of poor judgment, cognitive impairment, or acting on the basis of delusions. There appears to be an increased risk of suicidal behavior, particularly during the acute episode.

Prevaience

In the United States, brief psychotic disorder may account for 9% of cases of first-onset psychosis. Psychotic disturbances that meet Criteria A and C, but not Criterion B, for brief psychotic disorder (i.e., duration of active symptoms is 1–6 months as opposed to remission within 1 month) are more common in developing countries than in developed countries. Brief psychotic disorder is twofold more common in females than in males.

Deveiopment and Course

Brief psychotic disorder may appear in adolescence or early adulthood, and onset can occur across the lifespan, with the average age at onset being the mid 30s. By definition, a diagnosis of brief psychotic disorder requires a full remission of all symptoms and an eventual full return to the premorbid level of functioning within 1 month of the onset of the disturbance. In some individuals, the duration of psychotic symptoms may be quite brief (e.g., a few days).

Risk and Prognostic Factors

Temperamental. Preexisting personality disorders and traits (e.g., schizotypal personality disorder; borderline personality disorder; or traits in the psychoticism domain, such as perceptual dysregulation, and the negative affectivity domain, such as suspiciousness) may predispose the individual to the development of the disorder.

Culture-Reiated Diagnostic Issues

It is important to distinguish symptoms of brief psychotic disorder from culturally sanctioned response patterns. For example, in some religious ceremonies, an individual may report hearing voices, but these do not generally persist and are not perceived as abnormal by most members of the individual's community. In addition, cultural and religious background must be taken into account when considering whether beliefs are delusional.

Functionai Consequences of Brief Psychotic Disorder

Despite high rates of relapse, for most individuals, outcome is excellent in terms of social functioning and symptomatology.

Differential Diagnosis

Other medical conditions. A variety of medical disorders can manifest with psychotic symptoms of short duration. Psychotic disorder due to another medical condition or a delirium is diagnosed when there is evidence from the history, physical examination, or laboratory tests that the delusions or hallucinations are the direct physiological consequence of a specific medical condition (e.g., Cushing's syndrome, brain tumor) (see "Psychotic Disorder Due to Another Medical Condition" later in this chapter).

Substance-related disorders. Substance/medication-induced psychotic disorder, substance-induced delirium, and substance intoxication are distinguished from brief psychotic disorder by the fact that a substance (e.g., a drug of abuse, a medication, exposure to a toxin) is judged to be etiologically related to the psychotic symptoms (see "Substance/Medication-Induced Psychotic Disorder" later in this chapter). Laboratory tests, such as a urine drug screen or a blood alcohol level, may be helpful in making this determination, as may a careful history of substance use with attention to temporal relationships between substance intake and onset of the symptoms and to the nature of the substance being used.

Depressive and bipolar disorders. The diagnosis of brief psychotic disorder cannot be made if the psychotic symptoms are better explained by a mood episode (i.e., the psychotic symptoms occur exclusively during a full major depressive, manic, or mixed episode).

Other psychotic disorders. If the psychotic symptoms persist for 1 month or longer, the diagnosis is either schizophreniform disorder, delusional disorder, depressive disorder with psychotic features, bipolar disorder with psychotic features, or other specified or unspecified schizophrenia spectrum and other psychotic disorder, depending on the other symptoms in the presentation. The differential diagnosis between brief psychotic disorder and schizophreniform disorder is difficult when the psychotic symptoms have remitted before 1 month in response to successful treatment with medication. Careful attention should be given to the possibility that a recurrent disorder (e.g., bipolar disorder, recurrent acute exacerbations of schizophrenia) may be responsible for any recurring psychotic episodes.

Malingering and factitious disorders. An episode of factitious disorder, with predominantly psychological signs and symptoms, may have the appearance of brief psychotic disorder, but in such cases there is evidence that the symptoms are intentionally produced. When malingering involves apparently psychotic symptoms, there is usually evidence that the illness is being feigned for an understandable goal.

Personality disorders. In certain individuals with personality disorders, psychosocial stressors may precipitate brief periods of psychotic symptoms. These symptoms are usually transient and do not warrant a separate diagnosis. If psychotic symptoms persist for at least 1 day, an additional diagnosis of brief psychotic disorder may be appropriate.

Schizophreniform Disorder

Diagnostic Criteria **295.40 (F20.81)**

A. Two (or more) of the following, each present for a significant portion of time during a 1-month period (or less if successfully treated). At least one of these must be (1), (2), or (3):

1. Delusions.
2. Hallucinations.
3. Disorganized speech (e.g., frequent derailment or incoherence).
4. Grossly disorganized or catatonic behavior.
5. Negative symptoms (i.e., diminished emotional expression or avolition).

B. An episode of the disorder lasts at least 1 month but less than 6 months. When the diagnosis must be made without waiting for recovery, it should be qualified as "provisional."
C. Schizoaffective disorder and depressive or bipolar disorder with psychotic features have been ruled out because either 1) no major depressive or manic episodes have occurred concurrently with the active-phase symptoms, or 2) if mood episodes have occurred during active-phase symptoms, they have been present for a minority of the total duration of the active and residual periods of the illness.
D. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.

Specify if:

With good prognostic features: This specifier requires the presence of at least two of the following features: onset of prominent psychotic symptoms within 4 weeks of the first noticeable change in usual behavior or functioning; confusion or perplexity; good premorbid social and occupational functioning; and absence of blunted or flat affect.

Without good prognostic features: This specifier is applied if two or more of the above features have not been present.

Specify if:

With catatonia (refer to the criteria for catatonia associated with another mental disorder, pp. 119–120, for definition).

Coding note: Use additional code 293.89 (F06.1) catatonia associated with schizophreniform disorder to indicate the presence of the comorbid catatonia.

Specify current severity:

Severity is rated by a quantitative assessment of the primary symptoms of psychosis, including delusions, hallucinations, disorganized speech, abnormal psychomotor behavior, and negative symptoms. Each of these symptoms may be rated for its current severity (most severe in the last 7 days) on a 5-point scale ranging from 0 (not present) to 4 (present and severe). (See Clinician-Rated Dimensions of Psychosis Symptom Severity in the chapter "Assessment Measures.")

Note: Diagnosis of schizophreniform disorder can be made without using this severity specifier.

Note: For additional information on Associated Features Supporting Diagnosis, Development and Course (age-related factors), Culture-Related Diagnostic Issues, Gender-Related Diagnostic Issues, Differential Diagnosis, and Comorbidity, see the corresponding sections in schizophrenia.

Diagnostic Features

The characteristic symptoms of schizophreniform disorder are identical to those of schizophrenia (Criterion A). Schizophreniform disorder is distinguished by its difference in duration: the total duration of the illness, including prodromal, active, and residual phases, is at least 1 month but less than 6 months (Criterion B). The duration requirement for schizophreniform disorder is intermediate between that for brief psychotic disorder, which lasts more than 1 day and remits by 1 month, and schizophrenia, which lasts for at least 6 months. The diagnosis of schizophreniform disorder is made under two conditions. 1) when an episode of illness lasts between 1 and 6 months and the individual has already recovered, and 2) when an individual is symptomatic for less than the 6 months' duration required for the diagnosis of schizophrenia but has not yet recovered. In this case, the diagnosis should be noted as "schizophreniform disorder (provisional)" because it is uncertain if the individual will recover from the disturbance within the 6-month period. If the disturbance persists beyond 6 months, the diagnosis should be changed to schizophrenia.

Another distinguishing feature of schizophreniform disorder is the lack of a criterion requiring impaired social and occupational functioning. While such impairments may potentially be present, they are not necessary for a diagnosis of schizophreniform disorder.

In addition to the five symptom domain areas identified in the diagnostic criteria, the assessment of cognition, depression, and mania symptom domains is vital for making critically important distinctions between the various schizophrenia spectrum and other psychotic disorders.

Associated Features Supporting Diagnosis

As with schizophrenia, currently there are no laboratory or psychometric tests for schizophreniform disorder. There are multiple brain regions where neuroimaging, neuropathological, and neurophysiological research has indicated abnormalities, but none are diagnostic.

Prevalence

Incidence of schizophreniform disorder across sociocultural settings is likely similar to that observed in schizophrenia. In the United States and other developed countries, the incidence is low, possibly fivefold less than that of schizophrenia. In developing countries, the incidence may be higher, especially for the specifier "with good prognostic features"; in some of these settings schizophreniform disorder may be as common as schizophrenia.

Development and Course

The development of schizophreniform disorder is similar to that of schizophrenia. About one-third of individuals with an initial diagnosis of schizophreniform disorder (provisional) recover within the 6-month period and schizophreniform disorder is their final diagnosis. The majority of the remaining two-thirds of individuals will eventually receive a diagnosis of schizophrenia or schizoaffective disorder.

Risk and Prognostic Factors

Genetic and physiological. Relatives of individuals with schizophreniform disorder have an increased risk for schizophrenia.

Functional Consequences of Schizophreniform Disorder

For the majority of individuals with schizophreniform disorder who eventually receive a diagnosis of schizophrenia or schizoaffective disorder, the functional consequences are similar to the consequences of those disorders. Most individuals experience dysfunction in several areas of daily functioning, such as school or work, interpersonal relationships, and self-care. Individuals who recover from schizophreniform disorder have better functional outcomes.

Differential Diagnosis

Other mental disorders and medical conditions. A wide variety of mental and medical conditions can manifest with psychotic symptoms that must be considered in the differential diagnosis of schizophreniform disorder. These include psychotic disorder due to another medical condition or its treatment; delirium or major neurocognitive disorder; substance/medication-induced psychotic disorder or delirium; depressive or bipolar disorder with psychotic features; schizoaffective disorder; other specified or unspecified bipolar and related disorder; depressive or bipolar disorder with catatonic features; schizophre-

nia; brief psychotic disorder; delusional disorder; other specified or unspecified schizophrenia spectrum and other psychotic disorder; schizotypal, schizoid, or paranoid personality disorders; autism spectrum disorder; disorders presenting in childhood with disorganized speech; attention-deficit/hyperactivity disorder; obsessive-compulsive disorder; posttraumatic stress disorder; and traumatic brain injury.

Since the diagnostic criteria for schizophreniform disorder and schizophrenia differ primarily in duration of illness, the discussion of the differential diagnosis of schizophrenia also applies to schizophreniform disorder.

Brief psychotic disorder. Schizophreniform disorder differs in duration from brief psychotic disorder, which has a duration of less than 1 month.

Schizophrenia

Diagnostic Criteria **295.90 (F20.9)**

A. Two (or more) of the following, each present for a significant portion of time during a 1-month period (or less if successfully treated). At least one of these must be (1), (2), or (3):
   1. Delusions.
   2. Hallucinations.
   3. Disorganized speech (e.g., frequent derailment or incoherence).
   4. Grossly disorganized or catatonic behavior.
   5. Negative symptoms (i.e., diminished emotional expression or avolition).

B. For a significant portion of the time since the onset of the disturbance, level of functioning in one or more major areas, such as work, interpersonal relations, or self-care, is markedly below the level achieved prior to the onset (or when the onset is in childhood or adolescence, there is failure to achieve expected level of interpersonal, academic, or occupational functioning).

C. Continuous signs of the disturbance persist for at least 6 months. This 6-month period must include at least 1 month of symptoms (or less if successfully treated) that meet Criterion A (i.e., active-phase symptoms) and may include periods of prodromal or residual symptoms. During these prodromal or residual periods, the signs of the disturbance may be manifested by only negative symptoms or by two or more symptoms listed in Criterion A present in an attenuated form (e.g., odd beliefs, unusual perceptual experiences).

D. Schizoaffective disorder and depressive or bipolar disorder with psychotic features have been ruled out because either 1) no major depressive or manic episodes have occurred concurrently with the active-phase symptoms, or 2) if mood episodes have occurred during active-phase symptoms, they have been present for a minority of the total duration of the active and residual periods of the illness.

E. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.

F. If there is a history of autism spectrum disorder or a communication disorder of childhood onset, the additional diagnosis of schizophrenia is made only if prominent delusions or hallucinations, in addition to the other required symptoms of schizophrenia, are also present for at least 1 month (or less if successfully treated).

Specify if:

The following course specifiers are only to be used after a 1-year duration of the disorder and if they are not in contradiction to the diagnostic course criteria.

First episode, currently in acute episode: First manifestation of the disorder meeting the defining diagnostic symptom and time criteria. An *acute episode* is a time period in which the symptom criteria are fulfilled.

First episode, currently in partial remission: *Partial remission* is a period of time during which an improvement after a previous episode is maintained and in which the defining criteria of the disorder are only partially fulfilled.

First episode, currently in full remission: *Full remission* is a period of time after a previous episode during which no disorder-specific symptoms are present.

Multiple episodes, currently in acute episode: Multiple episodes may be determined after a minimum of two episodes (i.e., after a first episode, a remission and a minimum of one relapse).

Multiple episodes, currently in partial remission

Multiple episodes, currently in full remission

Continuous: Symptoms fulfilling the diagnostic symptom criteria of the disorder are remaining for the majority of the illness course, with subthreshold symptom periods being very brief relative to the overall course.

Unspecified

Specify if:

With catatonia (refer to the criteria for catatonia associated with another mental disorder, pp. 119–120, for definition).

Coding note: Use additional code 293.89 (F06.1) catatonia associated with schizophrenia to indicate the presence of the comorbid catatonia.

Specify current severity:

Severity is rated by a quantitative assessment of the primary symptoms of psychosis, including delusions, hallucinations, disorganized speech, abnormal psychomotor behavior, and negative symptoms. Each of these symptoms may be rated for its current severity (most severe in the last 7 days) on a 5-point scale ranging from 0 (not present) to 4 (present and severe). (See Clinician-Rated Dimensions of Psychosis Symptom Severity in the chapter "Assessment Measures.")

Note: Diagnosis of schizophrenia can be made without using this severity specifier.

Diagnostic Features

The characteristic symptoms of schizophrenia involve a range of cognitive, behavioral, and emotional dysfunctions, but no single symptom is pathognomonic of the disorder. The diagnosis involves the recognition of a constellation of signs and symptoms associated with impaired occupational or social functioning. Individuals with the disorder will vary substantially on most features, as schizophrenia is a heterogeneous clinical syndrome.

At least two Criterion A symptoms must be present for a significant portion of time during a 1-month period or longer. At least one of these symptoms must be the clear presence of delusions (Criterion A1), hallucinations (Criterion A2), or disorganized speech (Criterion A3). Grossly disorganized or catatonic behavior (Criterion A4) and negative symptoms (Criterion A5) may also be present. In those situations in which the active-phase symptoms remit within a month in response to treatment, Criterion A is still met if the clinician estimates that they would have persisted in the absence of treatment.

Schizophrenia involves impairment in one or more major areas of functioning (Criterion B). If the disturbance begins in childhood or adolescence, the expected level of function is not attained. Comparing the individual with unaffected siblings may be helpful. The dysfunction persists for a substantial period during the course of the disorder and does not appear to be a direct result of any single feature. Avolition (i.e., reduced drive to pursue goal-directed behavior; Criterion A5) is linked to the social dysfunction described under Criterion B. There is also strong evidence for a relationship between cognitive impairment (see the section "Associated Features Supporting Diagnosis" for this disorder) and functional impairment in individuals with schizophrenia.

Some signs of the disturbance must persist for a continuous period of at least 6 months (Criterion C). Prodromal symptoms often precede the active phase, and residual symptoms may follow it, characterized by mild or subthreshold forms of hallucinations or delusions. Individuals may express a variety of unusual or odd beliefs that are not of delusional proportions (e.g., ideas of reference or magical thinking); they may have unusual perceptual experiences (e.g., sensing the presence of an unseen person); their speech may be generally understandable but vague; and their behavior may be unusual but not grossly disorganized (e.g., mumbling in public). Negative symptoms are common in the prodromal and residual phases and can be severe. Individuals who had been socially active may become withdrawn from previous routines. Such behaviors are often the first sign of a disorder.

Mood symptoms and full mood episodes are common in schizophrenia and may be concurrent with active-phase symptomatology. However, as distinct from a psychotic mood disorder, a schizophrenia diagnosis requires the presence of delusions or hallucinations in the absence of mood episodes. In addition, mood episodes, taken in total, should be present for only a minority of the total duration of the active and residual periods of the illness.

In addition to the five symptom domain areas identified in the diagnostic criteria, the assessment of cognition, depression, and mania symptom domains is vital for making critically important distinctions between the various schizophrenia spectrum and other psychotic disorders.

Associated Features Supporting Diagnosis

Individuals with schizophrenia may display inappropriate affect (e.g., laughing in the absence of an appropriate stimulus); a dysphoric mood that can take the form of depression, anxiety, or anger; a disturbed sleep pattern (e.g., daytime sleeping and nighttime activity); and a lack of interest in eating or food refusal. Depersonalization, derealization, and somatic concerns may occur and sometimes reach delusional proportions. Anxiety and phobias are common. Cognitive deficits in schizophrenia are common and are strongly linked to vocational and functional impairments. These deficits can include decrements in declarative memory, working memory, language function, and other executive functions, as well as slower processing speed. Abnormalities in sensory processing and inhibitory capacity, as well as reductions in attention, are also found. Some individuals with schizophrenia show social cognition deficits, including deficits in the ability to infer the intentions of other people (theory of mind), and may attend to and then interpret irrelevant events or stimuli as meaningful, perhaps leading to the generation of explanatory delusions. These impairments frequently persist during symptomatic remission.

Some individuals with psychosis may lack insight or awareness of their disorder (i.e., anosognosia). This lack of "insight" includes unawareness of symptoms of schizophrenia and may be present throughout the entire course of the illness. Unawareness of illness is typically a symptom of schizophrenia itself rather than a coping strategy. It is comparable to the lack of awareness of neurological deficits following brain damage, termed *anosognosia*. This symptom is the most common predictor of non-adherence to treatment, and it predicts higher relapse rates, increased number of involuntary treatments, poorer psychosocial functioning, aggression, and a poorer course of illness.

Hostility and aggression can be associated with schizophrenia, although spontaneous or random assault is uncommon. Aggression is more frequent for younger males and for individuals with a past history of violence, non-adherence with treatment, substance abuse, and impulsivity. It should be noted that the vast majority of persons with schizophrenia are not aggressive and are more frequently victimized than are individuals in the general population.

Currently, there are no radiological, laboratory, or psychometric tests for the disorder. Differences are evident in multiple brain regions between groups of healthy individuals

and persons with schizophrenia, including evidence from neuroimaging, neuropathological, and neurophysiological studies. Differences are also evident in cellular architecture, white matter connectivity, and gray matter volume in a variety of regions such as the prefrontal and temporal cortices. Reduced overall brain volume has been observed, as well as increased brain volume reduction with age. Brain volume reductions with age are more pronounced in individuals with schizophrenia than in healthy individuals. Finally, individuals with schizophrenia appear to differ from individuals without the disorder in eye-tracking and electrophysiological indices.

Neurological soft signs common in individuals with schizophrenia include impairments in motor coordination, sensory integration, and motor sequencing of complex movements; left-right confusion; and disinhibition of associated movements. In addition, minor physical anomalies of the face and limbs may occur.

Prevalence

The lifetime prevalence of schizophrenia appears to be approximately 0.3%–0.7%, although there is reported variation by race/ethnicity, across countries, and by geographic origin for immigrants and children of immigrants. The sex ratio differs across samples and populations: for example, an emphasis on negative symptoms and longer duration of disorder (associated with poorer outcome) shows higher incidence rates for males, whereas definitions allowing for the inclusion of more mood symptoms and brief presentations (associated with better outcome) show equivalent risks for both sexes.

Development and Course

The psychotic features of schizophrenia typically emerge between the late teens and the mid-30s; onset prior to adolescence is rare. The peak age at onset for the first psychotic episode is in the early- to mid-20s for males and in the late-20s for females. The onset may be abrupt or insidious, but the majority of individuals manifest a slow and gradual development of a variety of clinically significant signs and symptoms. Half of these individuals complain of depressive symptoms. Earlier age at onset has traditionally been seen as a predictor of worse prognosis. However, the effect of age at onset is likely related to gender, with males having worse premorbid adjustment, lower educational achievement, more prominent negative symptoms and cognitive impairment, and in general a worse outcome. Impaired cognition is common, and alterations in cognition are present during development and precede the emergence of psychosis, taking the form of stable cognitive impairments during adulthood. Cognitive impairments may persist when other symptoms are in remission and contribute to the disability of the disease.

The predictors of course and outcome are largely unexplained, and course and outcome may not be reliably predicted. The course appears to be favorable in about 20% of those with schizophrenia, and a small number of individuals are reported to recover completely. However, most individuals with schizophrenia still require formal or informal daily living supports, and many remain chronically ill, with exacerbations and remissions of active symptoms, while others have a course of progressive deterioration.

Psychotic symptoms tend to diminish over the life course, perhaps in association with normal age-related declines in dopamine activity. Negative symptoms are more closely related to prognosis than are positive symptoms and tend to be the most persistent. Furthermore, cognitive deficits associated with the illness may not improve over the course of the illness.

The essential features of schizophrenia are the same in childhood, but it is more difficult to make the diagnosis. In children, delusions and hallucinations may be less elaborate than in adults, and visual hallucinations are more common and should be distinguished from normal fantasy play. Disorganized speech occurs in many disorders with childhood onset (e.g., autism spectrum disorder), as does disorganized behavior (e.g., attention-deficit/

hyperactivity disorder). These symptoms should not be attributed to schizophrenia without due consideration of the more common disorders of childhood. Childhood-onset cases tend to resemble poor-outcome adult cases, with gradual onset and prominent negative symptoms. Children who later receive the diagnosis of schizophrenia are more likely to have experienced nonspecific emotional-behavioral disturbances and psychopathology, intellectual and language alterations, and subtle motor delays.

Late-onset cases (i.e., onset after age 40 years) are overrepresented by females, who may have married. Often, the course is characterized by a predominance of psychotic symptoms with preservation of affect and social functioning. Such late-onset cases can still meet the diagnostic criteria for schizophrenia, but it is not yet clear whether this is the same condition as schizophrenia diagnosed prior to mid-life (e.g., prior to age 55 years).

Risk and Prognostic Factors

Environmental. Season of birth has been linked to the incidence of schizophrenia, including late winter/early spring in some locations and summer for the deficit form of the disease. The incidence of schizophrenia and related disorders is higher for children growing up in an urban environment and for some minority ethnic groups.

Genetic and physiological. There is a strong contribution for genetic factors in determining risk for schizophrenia, although most individuals who have been diagnosed with schizophrenia have no family history of psychosis. Liability is conferred by a spectrum of risk alleles, common and rare, with each allele contributing only a small fraction to the total population variance. The risk alleles identified to date are also associated with other mental disorders, including bipolar disorder, depression, and autism spectrum disorder.

Pregnancy and birth complications with hypoxia and greater paternal age are associated with a higher risk of schizophrenia for the developing fetus. In addition, other prenatal and perinatal adversities, including stress, infection, malnutrition, maternal diabetes, and other medical conditions, have been linked with schizophrenia. However, the vast majority of offspring with these risk factors do not develop schizophrenia.

Culture-Related Diagnostic Issues

Cultural and socioeconomic factors must be considered, particularly when the individual and the clinician do not share the same cultural and socioeconomic background. Ideas that appear to be delusional in one culture (e.g., witchcraft) may be commonly held in another. In some cultures, visual or auditory hallucinations with a religious content (e.g., hearing God's voice) are a normal part of religious experience. In addition, the assessment of disorganized speech may be made difficult by linguistic variation in narrative styles across cultures. The assessment of affect requires sensitivity to differences in styles of emotional expression, eye contact, and body language, which vary across cultures. If the assessment is conducted in a language that is different from the individual's primary language, care must be taken to ensure that alogia is not related to linguistic barriers. In certain cultures, distress may take the form of hallucinations or pseudo-hallucinations and overvalued ideas that may present clinically similar to true psychosis but are normative to the patient's subgroup.

Gender-Related Diagnostic Issues

A number of features distinguish the clinical expression of schizophrenia in females and males. The general incidence of schizophrenia tends to be slightly lower in females, particularly among treated cases. The age at onset is later in females, with a second mid-life peak as described earlier (see the section "Development and Course" for this disorder). Symptoms tend to be more affect-laden among females, and there are more psychotic symptoms, as well as a greater propensity for psychotic symptoms to worsen in later life.

Other symptom differences include less frequent negative symptoms and disorganization. Finally, social functioning tends to remain better preserved in females. There are, however, frequent exceptions to these general caveats.

Suicide Risk

Approximately 5%–6% of individuals with schizophrenia die by suicide, about 20% attempt suicide on one or more occasions, and many more have significant suicidal ideation. Suicidal behavior is sometimes in response to command hallucinations to harm oneself or others. Suicide risk remains high over the whole lifespan for males and females, although it may be especially high for younger males with comorbid substance use. Other risk factors include having depressive symptoms or feelings of hopelessness and being unemployed, and the risk is higher, also, in the period after a psychotic episode or hospital discharge.

Functional Consequences of Schizophrenia

Schizophrenia is associated with significant social and occupational dysfunction. Making educational progress and maintaining employment are frequently impaired by avolition or other disorder manifestations, even when the cognitive skills are sufficient for the tasks at hand. Most individuals are employed at a lower level than their parents, and most, particularly men, do not marry or have limited social contacts outside of their family.

Differential Diagnosis

Major depressive or bipolar disorder with psychotic or catatonic features. The distinction between schizophrenia and major depressive or bipolar disorder with psychotic features or with catatonia depends on the temporal relationship between the mood disturbance and the psychosis, and on the severity of the depressive or manic symptoms. If delusions or hallucinations occur exclusively during a major depressive or manic episode, the diagnosis is depressive or bipolar disorder with psychotic features.

Schizoaffective disorder. A diagnosis of schizoaffective disorder requires that a major depressive or manic episode occur concurrently with the active-phase symptoms and that the mood symptoms be present for a majority of the total duration of the active periods.

Schizophreniform disorder and brief psychotic disorder. These disorders are of shorter duration than schizophrenia as specified in Criterion C, which requires 6 months of symptoms. In schizophreniform disorder, the disturbance is present less than 6 months, and in brief psychotic disorder, symptoms are present at least 1 day but less than 1 month.

Delusional disorder. Delusional disorder can be distinguished from schizophrenia by the absence of the other symptoms characteristic of schizophrenia (e.g., delusions, prominent auditory or visual hallucinations, disorganized speech, grossly disorganized or catatonic behavior, negative symptoms).

Schizotypal personality disorder. Schizotypal personality disorder may be distinguished from schizophrenia by subthreshold symptoms that are associated with persistent personality features.

Obsessive-compulsive disorder and body dysmorphic disorder. Individuals with obsessive-compulsive disorder and body dysmorphic disorder may present with poor or absent insight, and the preoccupations may reach delusional proportions. But these disorders are distinguished from schizophrenia by their prominent obsessions, compulsions, preoccupations with appearance or body odor, hoarding, or body-focused repetitive behaviors.

Posttraumatic stress disorder. Posttraumatic stress disorder may include flashbacks that have a hallucinatory quality, and hypervigilance may reach paranoid proportions. But a trau-

matic event and characteristic symptom features relating to reliving or reacting to the event are required to make the diagnosis.

Autism spectrum disorder or communication disorders. These disorders may also have symptoms resembling a psychotic episode but are distinguished by their respective deficits in social interaction with repetitive and restricted behaviors and other cognitive and communication deficits. An individual with autism spectrum disorder or communication disorder must have symptoms that meet full criteria for schizophrenia, with prominent hallucinations or delusions for at least 1 month, in order to be diagnosed with schizophrenia as a comorbid condition.

Other mental disorders associated with a psychotic episode. The diagnosis of schizophrenia is made only when the psychotic episode is persistent and not attributable to the physiological effects of a substance or another medical condition. Individuals with a delirium or major or minor neurocognitive disorder may present with psychotic symptoms, but these would have a temporal relationship to the onset of cognitive changes consistent with those disorders. Individuals with substance/medication-induced psychotic disorder may present with symptoms characteristic of Criterion A for schizophrenia, but the substance/medication-induced psychotic disorder can usually be distinguished by the chronological relationship of substance use to the onset and remission of the psychosis in the absence of substance use.

Comorbidity

Rates of comorbidity with substance-related disorders are high in schizophrenia. Over half of individuals with schizophrenia have tobacco use disorder and smoke cigarettes regularly. Comorbidity with anxiety disorders is increasingly recognized in schizophrenia. Rates of obsessive-compulsive disorder and panic disorder are elevated in individuals with schizophrenia compared with the general population. Schizotypal or paranoid personality disorder may sometimes precede the onset of schizophrenia.

Life expectancy is reduced in individuals with schizophrenia because of associated medical conditions. Weight gain, diabetes, metabolic syndrome, and cardiovascular and pulmonary disease are more common in schizophrenia than in the general population. Poor engagement in health maintenance behaviors (e.g., cancer screening, exercise) increases the risk of chronic disease, but other disorder factors, including medications, lifestyle, cigarette smoking, and diet, may also play a role. A shared vulnerability for psychosis and medical disorders may explain some of the medical comorbidity of schizophrenia.

Schizoaffective Disorder

Diagnostic Criteria

A. An uninterrupted period of illness during which there is a major mood episode (major depressive or manic) concurrent with Criterion A of schizophrenia.

   Note: The major depressive episode must include Criterion A1: Depressed mood.

B. Delusions or hallucinations for 2 or more weeks in the absence of a major mood episode (depressive or manic) during the lifetime duration of the illness.

C. Symptoms that meet criteria for a major mood episode are present for the majority of the total duration of the active and residual portions of the illness.

D. The disturbance is not attributable to the effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.

Specify whether:

295.70 (F25.0) Bipolar type: This subtype applies if a manic episode is part of the presentation. Major depressive episodes may also occur.

295.70 (F25.1) Depressive type: This subtype applies if only major depressive episodes are part of the presentation.

Specify if:

With catatonia (refer to the criteria for catatonia associated with another mental disorder, pp. 119–120, for definition).

Coding note: Use additional code 293.89 (F06.1) catatonia associated with schizoaffective disorder to indicate the presence of the comorbid catatonia.

Specify if:

The following course specifiers are only to be used after a 1-year duration of the disorder and if they are not in contradiction to the diagnostic course criteria.

First episode, currently in acute episode: First manifestation of the disorder meeting the defining diagnostic symptom and time criteria. An *acute episode* is a time period in which the symptom criteria are fulfilled.

First episode, currently in partial remission: *Partial remission* is a time period during which an improvement after a previous episode is maintained and in which the defining criteria of the disorder are only partially fulfilled.

First episode, currently in full remission: *Full remission* is a period of time after a previous episode during which no disorder-specific symptoms are present.

Multiple episodes, currently in acute episode: Multiple episodes may be determined after a minimum of two episodes (i.e., after a first episode, a remission and a minimum of one relapse).

Multiple episodes, currently in partial remission

Multiple episodes, currently in full remission

Continuous: Symptoms fulfilling the diagnostic symptom criteria of the disorder are remaining for the majority of the illness course, with subthreshold symptom periods being very brief relative to the overall course.

Unspecified

Specify current severity:

Severity is rated by a quantitative assessment of the primary symptoms of psychosis, including delusions, hallucinations, disorganized speech, abnormal psychomotor behavior, and negative symptoms. Each of these symptoms may be rated for its current severity (most severe in the last 7 days) on a 5-point scale ranging from 0 (not present) to 4 (present and severe). (See Clinician-Rated Dimensions of Psychosis Symptom Severity in the chapter "Assessment Measures.")

Note: Diagnosis of schizoaffective disorder can be made without using this severity specifier.

Note: For additional information on Development and Course (age-related factors), Risk and Prognostic Factors (environmental risk factors), Culture-Related Diagnostic Issues, and Gender-Related Diagnostic Issues, see the corresponding sections in schizophrenia, bipolar I and II disorders, and major depressive disorder in their respective chapters.

Diagnostic Features

The diagnosis of schizoaffective disorder is based on the assessment of an uninterrupted period of illness during which the individual continues to display active or residual symptoms of psychotic illness. The diagnosis is usually, but not necessarily, made during the period of psychotic illness. At some time during the period, Criterion A for schizophrenia

has to be met. Criteria B (social dysfunction) and F (exclusion of autism spectrum disorder or other communication disorder of childhood onset) for schizophrenia do not have to be met. In addition to meeting Criterion A for schizophrenia, there is a major mood episode (major depressive or manic) (Criterion A for schizoaffective disorder). Because loss of interest or pleasure is common in schizophrenia, to meet Criterion A for schizoaffective disorder, the major depressive episode must include pervasive depressed mood (i.e., the presence of markedly diminished interest or pleasure is not sufficient). Episodes of depression or mania are present for the majority of the total duration of the illness (i.e., after Criterion A has been met) (Criterion C for schizoaffective disorder). To separate schizoaffective disorder from a depressive or bipolar disorder with psychotic features, delusions or hallucinations must be present for at least 2 weeks in the absence of a major mood episode (depressive or manic) at some point during the lifetime duration of the illness (Criterion B for schizoaffective disorder). The symptoms must not be attributable to the effects of a substance or another medical condition (Criterion D for schizoaffective disorder).

Criterion C for schizoaffective disorder specifies that mood symptoms meeting criteria for a major mood episode must be present for the majority of the total duration of the active and residual portion of the illness. Criterion C requires the assessment of mood symptoms for the entire course of a psychotic illness, which differs from the criterion in DSM-IV, which required only an assessment of the current period of illness. If the mood symptoms are present for only a relatively brief period, the diagnosis is schizophrenia, not schizoaffective disorder. When deciding whether an individual's presentation meets Criterion C, the clinician should review the total duration of psychotic illness (i.e., both active and residual symptoms) and determine when significant mood symptoms (untreated or in need of treatment with antidepressant and/or mood-stabilizing medication) accompanied the psychotic symptoms. This determination requires sufficient historical information and clinical judgment. For example, an individual with a 4-year history of active and residual symptoms of schizophrenia develops depressive and manic episodes that, taken together, do not occupy more than 1 year during the 4-year history of psychotic illness. This presentation would not meet Criterion C.

In addition to the five symptom domain areas identified in the diagnostic criteria, the assessment of cognition, depression, and mania symptom domains is vital for making critically important distinctions between the various schizophrenia spectrum and other psychotic disorders.

Associated Features Supporting Diagnosis

Occupational functioning is frequently impaired, but this is not a defining criterion (in contrast to schizophrenia). Restricted social contact and difficulties with self-care are associated with schizoaffective disorder, but negative symptoms may be less severe and less persistent than those seen in schizophrenia. Anosognosia (i.e., poor insight) is also common in schizoaffective disorder, but the deficits in insight may be less severe and pervasive than those in schizophrenia. Individuals with schizoaffective disorder may be at increased risk for later developing episodes of major depressive disorder or bipolar disorder if mood symptoms continue following the remission of symptoms meeting Criterion A for schizophrenia. There may be associated alcohol and other substance-related disorders.

There are no tests or biological measures that can assist in making the diagnosis of schizoaffective disorder. Whether schizoaffective disorder differs from schizophrenia with regard to associated features such as structural or functional brain abnormalities, cognitive deficits, or genetic risk factors is not clear.

Prevalence

Schizoaffective disorder appears to be about one-third as common as schizophrenia. Lifetime prevalence of schizoaffective disorder is estimated to be 0.3%. The incidence of

schizoaffective disorder is higher in females than in males, mainly due to an increased incidence of the depressive type among females.

Development and Course

The typical age at onset of schizoaffective disorder is early adulthood, although onset can occur anywhere from adolescence to late in life. A significant number of individuals diagnosed with another psychotic illness initially will receive the diagnosis schizoaffective disorder later when the pattern of mood episodes has become more apparent. With the current diagnostic Criterion C, it is expected that the diagnosis for some individuals will convert from schizoaffective disorder to another disorder as mood symptoms become less prominent. The prognosis for schizoaffective disorder is somewhat better than the prognosis for schizophrenia but worse than the prognosis for mood disorders.

Schizoaffective disorder may occur in a variety of temporal patterns. The following is a typical pattern: An individual may have pronounced auditory hallucinations and persecutory delusions for 2 months before the onset of a prominent major depressive episode. The psychotic symptoms and the full major depressive episode are then present for 3 months. Then, the individual recovers completely from the major depressive episode, but the psychotic symptoms persist for another month before they too disappear. During this period of illness, the individual's symptoms concurrently met criteria for a major depressive episode and Criterion A for schizophrenia, and during this same period of illness, auditory hallucinations and delusions were present both before and after the depressive phase. The total period of illness lasted for about 6 months, with psychotic symptoms alone present during the initial 2 months, both depressive and psychotic symptoms present during the next 3 months, and psychotic symptoms alone present during the last month. In this instance, the duration of the depressive episode was not brief relative to the total duration of the psychotic disturbance, and thus the presentation qualifies for a diagnosis of schizoaffective disorder.

The expression of psychotic symptoms across the lifespan is variable. Depressive or manic symptoms can occur before the onset of psychosis, during acute psychotic episodes, during residual periods, and after cessation of psychosis. For example, an individual might present with prominent mood symptoms during the prodromal stage of schizophrenia. This pattern is not necessarily indicative of schizoaffective disorder, since it is the co-occurrence of psychotic and mood symptoms that is diagnostic. For an individual with symptoms that clearly meet the criteria for schizoaffective disorder but who on further follow-up only presents with residual psychotic symptoms (such as subthreshold psychosis and/or prominent negative symptoms), the diagnosis may be changed to schizophrenia, as the total proportion of psychotic illness compared with mood symptoms becomes more prominent. Schizoaffective disorder, bipolar type, may be more common in young adults, whereas schizoaffective disorder, depressive type, may be more common in older adults.

Risk and Prognostic Factors

Genetic and physiological. Among individuals with schizophrenia, there may be an increased risk for schizoaffective disorder in first-degree relatives. The risk for schizoaffective disorder may be increased among individuals who have a first-degree relative with schizophrenia, bipolar disorder, or schizoaffective disorder.

Culture-Related Diagnostic Issues

Cultural and socioeconomic factors must be considered, particularly when the individual and the clinician do not share the same cultural and economic background. Ideas that appear to be delusional in one culture (e.g., witchcraft) may be commonly held in another. There is also some evidence in the literature for the overdiagnosis of schizophrenia com-

pared with schizoaffective disorder in African American and Hispanic populations, so care must be taken to ensure a culturally appropriate evaluation that includes both psychotic and affective symptoms.

Suicide Risk

The lifetime risk of suicide for schizophrenia and schizoaffective disorder is 5%, and the presence of depressive symptoms is correlated with a higher risk for suicide. There is evidence that suicide rates are higher in North American populations than in European, Eastern European, South American, and Indian populations of individuals with schizophrenia or schizoaffective disorder.

Functional Consequences of Schizoaffective Disorder

Schizoaffective disorder is associated with social and occupational dysfunction, but dysfunction is not a diagnostic criterion (as it is for schizophrenia), and there is substantial variability between individuals diagnosed with schizoaffective disorder.

Differential Diagnosis

Other mental disorders and medical conditions. A wide variety of psychiatric and medical conditions can manifest with psychotic and mood symptoms that must be considered in the differential diagnosis of schizoaffective disorder. These include psychotic disorder due to another medical condition; delirium; major neurocognitive disorder; substance/medication-induced psychotic disorder or neurocognitive disorder; bipolar disorders with psychotic features; major depressive disorder with psychotic features; depressive or bipolar disorders with catatonic features; schizotypal, schizoid, or paranoid personality disorder; brief psychotic disorder; schizophreniform disorder; schizophrenia; delusional disorder; and other specified and unspecified schizophrenia spectrum and other psychotic disorders. Medical conditions and substance use can present with a combination of psychotic and mood symptoms, and thus psychotic disorder due to another medical condition needs to be excluded. Distinguishing schizoaffective disorder from schizophrenia and from depressive and bipolar disorders with psychotic features is often difficult. Criterion C is designed to separate schizoaffective disorder from schizophrenia, and Criterion B is designed to distinguish schizoaffective disorder from a depressive or bipolar disorder with psychotic features. More specifically, schizoaffective disorder can be distinguished from a depressive or bipolar disorder with psychotic features due to the presence of prominent delusions and/or hallucinations for at least 2 weeks in the absence of a major mood episode. In contrast, in depressive or bipolar disorders with psychotic features, the psychotic features primarily occur during the mood episode(s). Because the relative proportion of mood to psychotic symptoms may change over time, the appropriate diagnosis may change from and to schizoaffective disorder (e.g., a diagnosis of schizoaffective disorder for a severe and prominent major depressive episode lasting 3 months during the first 6 months of a persistent psychotic illness would be changed to schizophrenia if active psychotic or prominent residual symptoms persist over several years without a recurrence of another mood episode).

Psychotic disorder due to another medical condition. Other medical conditions and substance use can manifest with a combination of psychotic and mood symptoms, and thus psychotic disorder due to another medical condition needs to be excluded.

Schizophrenia, bipolar, and depressive disorders. Distinguishing schizoaffective disorder from schizophrenia and from depressive and bipolar disorders with psychotic features is often difficult. Criterion C is designed to separate schizoaffective disorder from schizophrenia, and Criterion B is designed to distinguish schizoaffective disorder from a

depressive or bipolar disorder with psychotic features. More specifically, schizoaffective disorder can be distinguished from a depressive or bipolar disorder with psychotic features based on the presence of prominent delusions and/or hallucinations for at least 2 weeks in the absence of a major mood episode. In contrast, in depressive or bipolar disorder with psychotic features, the psychotic features primarily occur during the mood episode(s). Because the relative proportion of mood to psychotic symptoms may change over time, the appropriate diagnosis may change from and to schizoaffective disorder. (For example, a diagnosis of schizoaffective disorder for a severe and prominent major depressive episode lasting 3 months during the first 6 months of a chronic psychotic illness would be changed to schizophrenia if active psychotic or prominent residual symptoms persist over several years without a recurrence of another mood episode.)

Comorbidity

Many individuals diagnosed with schizoaffective disorder are also diagnosed with other mental disorders, especially substance use disorders and anxiety disorders. Similarly, the incidence of medical conditions is increased above base rate for the general population and leads to decreased life expectancy.

Substance/Medication-Induced Psychotic Disorder

Diagnostic Criteria

A. Presence of one or both of the following symptoms:
   1. Delusions.
   2. Hallucinations.

B. There is evidence from the history, physical examination, or laboratory findings of both (1) and (2):
   1. The symptoms in Criterion A developed during or soon after substance intoxication or withdrawal or after exposure to a medication.
   2. The involved substance/medication is capable of producing the symptoms in Criterion A.

C. The disturbance is not better explained by a psychotic disorder that is not substance/medication-induced. Such evidence of an independent psychotic disorder could include the following:

   The symptoms preceded the onset of the substance/medication use; the symptoms persist for a substantial period of time (e.g., about 1 month) after the cessation of acute withdrawal or severe intoxication; or there is other evidence of an independent non-substance/medication-induced psychotic disorder (e.g., a history of recurrent non-substance/medication-related episodes).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Note: This diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and when they are sufficiently severe to warrant clinical attention.

Coding note: The ICD-9-CM and ICD-10-CM codes for the [specific substance/medication]-induced psychotic disorders are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. If a mild substance use disorder is comorbid with the substance-induced psychotic disorder, the 4th position character is "1," and the clinician should record "mild [substance] use disorder" before the substance-induced psychotic disorder (e.g., "mild cocaine use disorder with cocaine-induced psychotic disorder"). If a moderate or severe substance use disorder is comorbid with the substance-induced psychotic disorder, the 4th position character is "2," and the clinician should record "moderate [substance] use disorder" or "severe [substance] use disorder," depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder (e.g., after a one-time heavy use of the substance), then the 4th position character is "9," and the clinician should record only the substance-induced psychotic disorder.

		ICD-10-CM		
	ICD-9-CM	With use disorder, mild	With use disorder, moderate or severe	Without use disorder
Alcohol	291.9	F10.159	F10.259	F10.959
Cannabis	292.9	F12.159	F12.259	F12.959
Phencyclidine	292.9	F16.159	F16.259	F16.959
Other hallucinogen	292.9	F16.159	F16.259	F16.959
Inhalant	292.9	F18.159	F18.259	F18.959
Sedative, hypnotic, or anxiolytic	292.9	F13.159	F13.259	F13.959
Amphetamine (or other stimulant)	292.9	F15.159	F15.259	F15.959
Cocaine	292.9	F14.159	F14.259	F14.959
Other (or unknown) substance	292.9	F19.159	F19.259	F19.959

Specify if (see Table 1 in the chapter "Substance-Related and Addictive Disorders" for diagnoses associated with substance class):

With onset during intoxication: If the criteria are met for intoxication with the substance and the symptoms develop during intoxication.

With onset during withdrawal: If the criteria are met for withdrawal from the substance and the symptoms develop during, or shortly after, withdrawal.

Specify current severity:

Severity is rated by a quantitative assessment of the primary symptoms of psychosis, including delusions, hallucinations, abnormal psychomotor behavior, and negative symptoms. Each of these symptoms may be rated for its current severity (most severe in the last 7 days) on a 5-point scale ranging from 0 (not present) to 4 (present and severe). (See Clinician-Rated Dimensions of Psychosis Symptom Severity in the chapter "Assessment Measures.")

Note: Diagnosis of substance/medication-induced psychotic disorder can be made without using this severity specifier.

Recording Procedures

ICD-9-CM. The name of the substance/medication-induced psychotic disorder begins with the specific substance (e.g., cocaine, dexamethasone) that is presumed to be causing the delusions or hallucinations. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class. For substances that do not fit into any of the classes (e.g., dexamethasone), the code for "other substance" should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category "unknown substance" should be used.

The name of the disorder is followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal). Unlike the recording procedures for ICD-10-CM, which combine the substance-induced disorder and substance use disorder into a single code, for ICD-9-CM a separate diagnostic code is given for the substance use disorder. For example, in the case of delusions occurring during intoxication in a man with a severe cocaine use disorder, the diagnosis is 292.9 cocaine-induced psychotic disorder, with onset during intoxication. An additional diagnosis of 304.20 severe cocaine use disorder is also given. When more than one substance is judged to play a significant role in the development of psychotic symptoms, each should be listed separately (e.g., 292.9 cannabis-induced psychotic disorder with onset during intoxication, with severe cannabis use disorder; 292.9 phencyclidine-induced psychotic disorder, with onset during intoxication, with mild phencyclidine use disorder).

ICD-10-CM. The name of the substance/medication-induced psychotic disorder begins with the specific substance (e.g., cocaine, dexamethasone) that is presumed to be causing the delusions or hallucinations. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class and presence or absence of a comorbid substance use disorder. For substances that do not fit into any of the classes (e.g., dexamethasone), the code for "other substance" with no comorbid substance use should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category "unknown substance" with no comorbid substance use should be used.

When recording the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by the word "with," followed by the name of the substance-induced psychotic disorder, followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal). For example, in the case of delusions occurring during intoxication in a man with a severe cocaine use disorder, the diagnosis is F14.259 severe cocaine use disorder with cocaine-induced psychotic disorder, with onset during intoxication. A separate diagnosis of the comorbid severe cocaine use disorder is not given. If the substance-induced psychotic disorder occurs without a comorbid substance use disorder (e.g., after a one-time heavy use of the substance), no accompanying substance use disorder is noted (e.g., F16.959 phencyclidine-induced psychotic disorder, with onset during intoxication). When more than one substance is judged to play a significant role in the development of psychotic symptoms, each should be listed separately (e.g., F12.259 severe cannabis use disorder with cannabis-induced psychotic disorder, with onset during intoxication; F16.159 mild phencyclidine use disorder with phencyclidine-induced psychotic disorder, with onset during intoxication).

Diagnostic Features

The essential features of substance/medication-induced psychotic disorder are prominent delusions and/or hallucinations (Criterion A) that are judged to be due to the physiological effects of a substance/medication (i.e., a drug of abuse, a medication, or a toxin exposure) (Criterion B). Hallucinations that the individual realizes are substance/medication-induced are not included here and instead would be diagnosed as substance intoxication

or substance withdrawal with the accompanying specifier "with perceptual disturbances" (applies to alcohol withdrawal; cannabis intoxication; sedative, hypnotic, or anxiolytic withdrawal; and stimulant intoxication).

A substance/medication-induced psychotic disorder is distinguished from a primary psychotic disorder by considering the onset, course, and other factors. For drugs of abuse, there must be evidence from the history, physical examination, or laboratory findings of substance use, intoxication, or withdrawal. Substance/medication-induced psychotic disorders arise during or soon after exposure to a medication or after substance intoxication or withdrawal but can persist for weeks, whereas primary psychotic disorders may precede the onset of substance/medication use or may occur during times of sustained abstinence. Once initiated, the psychotic symptoms may continue as long as the substance/medication use continues. Another consideration is the presence of features that are atypical of a primary psychotic disorder (e.g., atypical age at onset or course). For example, the appearance of delusions de novo in a person older than 35 years without a known history of a primary psychotic disorder should suggest the possibility of a substance/medication-induced psychotic disorder. Even a prior history of a primary psychotic disorder does not rule out the possibility of a substance/medication-induced psychotic disorder. In contrast, factors that suggest that the psychotic symptoms are better accounted for by a primary psychotic disorder include persistence of psychotic symptoms for a substantial period of time (i.e., a month or more) after the end of substance intoxication or acute substance withdrawal or after cessation of medication use; or a history of prior recurrent primary psychotic disorders. Other causes of psychotic symptoms must be considered even in an individual with substance intoxication or withdrawal, because substance use problems are not uncommon among individuals with non-substance/medication-induced psychotic disorders.

In addition to the four symptom domain areas identified in the diagnostic criteria, the assessment of cognition, depression, and mania symptom domains is vital for making critically important distinctions between the various schizophrenia spectrum and other psychotic disorders.

Associated Features Supporting Diagnosis

Psychotic disorders can occur in association with intoxication with the following classes of substances: alcohol; cannabis; hallucinogens, including phencyclidine and related substances; inhalants; sedatives, hypnotics, and anxiolytics; stimulants (including cocaine); and other (or unknown) substances. Psychotic disorders can occur in association with withdrawal from the following classes of substances: alcohol; sedatives, hypnotics, and anxiolytics; and other (or unknown) substances.

Some of the medications reported to evoke psychotic symptoms include anesthetics and analgesics, anticholinergic agents, anticonvulsants, antihistamines, antihypertensive and cardiovascular medications, antimicrobial medications, antiparkinsonian medications, chemotherapeutic agents (e.g., cyclosporine, procarbazine), corticosteroids, gastrointestinal medications, muscle relaxants, nonsteroidal anti-inflammatory medications, other over-the-counter medications (e.g., phenylephrine, pseudoephedrine), antidepressant medication, and disulfiram. Toxins reported to induce psychotic symptoms include anticholinesterase, organophosphate insecticides, sarin and other nerve gases, carbon monoxide, carbon dioxide, and volatile substances such as fuel or paint.

Prevalence

Prevalence of substance/medication-induced psychotic disorder in the general population is unknown. Between 7% and 25% of individuals presenting with a first episode of psychosis in different settings are reported to have substance/medication-induced psychotic disorder.

Development and Course

The initiation of the disorder may vary considerably with the substance. For example, smoking a high dose of cocaine may produce psychosis within minutes, whereas days or weeks of high-dose alcohol or sedative use may be required to produce psychosis. Alcohol-induced psychotic disorder, with hallucinations, usually occurs only after prolonged, heavy ingestion of alcohol in individuals who have moderate to severe alcohol use disorder, and the hallucinations are generally auditory in nature.

Psychotic disorders induced by amphetamine and cocaine share similar clinical features. Persecutory delusions may rapidly develop shortly after use of amphetamine or a similarly acting sympathomimetic. The hallucination of bugs or vermin crawling in or under the skin (formication) can lead to scratching and extensive skin excoriations. Cannabis-induced psychotic disorder may develop shortly after high-dose cannabis use and usually involves persecutory delusions, marked anxiety, emotional lability, and depersonalization. The disorder usually remits within a day but in some cases may persist for a few days.

Substance/medication-induced psychotic disorder may at times persist when the offending agent is removed, such that it may be difficult initially to distinguish it from an independent psychotic disorder. Agents such as amphetamines, phencyclidine, and cocaine have been reported to evoke temporary psychotic states that can sometimes persist for weeks or longer despite removal of the agent and treatment with neuroleptic medication. In later life, polypharmacy for medical conditions and exposure to medications for parkinsonism, cardiovascular disease, and other medical disorders may be associated with a greater likelihood of psychosis induced by prescription medications as opposed to substances of abuse.

Diagnostic Markers

With substances for which relevant blood levels are available (e.g., blood alcohol level, other quantifiable blood levels such as digoxin), the presence of a level consistent with toxicity may increase diagnostic certainty.

Functional Consequences of Substance/Medication-Induced Psychotic Disorder

Substance/medication-induced psychotic disorder is typically severely disabling and consequently is observed most frequently in emergency rooms, as individuals are often brought to the acute-care setting when it occurs. However, the disability is typically self-limited and resolves upon removal of the offending agent.

Differential Diagnosis

Substance intoxication or substance withdrawal. Individuals intoxicated with stimulants, cannabis, the opioid meperidine, or phencyclidine, or those withdrawing from alcohol or sedatives, may experience altered perceptions that they recognize as drug effects. If reality testing for these experiences remains intact (i.e., the individual recognizes that the perception is substance induced and neither believes in nor acts on it), the diagnosis is not substance/medication-induced psychotic disorder. Instead, substance intoxication or substance withdrawal, with perceptual disturbances, is diagnosed (e.g., cocaine intoxication, with perceptual disturbances). "Flashback" hallucinations that can occur long after the use of hallucinogens has stopped are diagnosed as hallucinogen persisting perception disorder. If substance/medication-induced psychotic symptoms occur exclusively during the course of a delirium, as in severe forms of alcohol withdrawal, the psychotic symptoms are considered to be an associated feature of the delirium and are not diagnosed separately. Delusions in the context of a major or mild neurocognitive disorder would be diagnosed as major or mild neurocognitive disorder, with behavioral disturbance.

Primary psychotic disorder. A substance/medication-induced psychotic disorder is distinguished from a primary psychotic disorder, such as schizophrenia, schizoaffective disorder, delusional disorder, brief psychotic disorder, other specified schizophrenia spectrum and other psychotic disorder, or unspecified schizophrenia spectrum and other psychotic disorder, by the fact that a substance is judged to be etiologically related to the symptoms.

Psychotic disorder due to another medical condition. A substance/medication-induced psychotic disorder due to a prescribed treatment for a mental or medical condition must have its onset while the individual is receiving the medication (or during withdrawal, if there is a withdrawal syndrome associated with the medication). Because individuals with medical conditions often take medications for those conditions, the clinician must consider the possibility that the psychotic symptoms are caused by the physiological consequences of the medical condition rather than the medication, in which case psychotic disorder due to another medical condition is diagnosed. The history often provides the primary basis for such a judgment. At times, a change in the treatment for the medical condition (e.g., medication substitution or discontinuation) may be needed to determine empirically for that individual whether the medication is the causative agent. If the clinician has ascertained that the disturbance is attributable to both a medical condition and substance/medication use, both diagnoses (i.e., psychotic disorder due to another medical condition and substance/medication-induced psychotic disorder) may be given.

Psychotic Disorder Due to Another Medical Condition

Diagnostic Criteria

A. Prominent hallucinations or delusions.
B. There is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct pathophysiological consequence of another medical condition.
C. The disturbance is not better explained by another mental disorder.
D. The disturbance does not occur exclusively during the course of a delirium.
E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify whether:

Code based on predominant symptom:

293.81 (F06.2) With delusions: If delusions are the predominant symptom.

293.82 (F06.0) With hallucinations: If hallucinations are the predominant symptom.

Coding note: Include the name of the other medical condition in the name of the mental disorder (e.g., 293.81 [F06.2] psychotic disorder due to malignant lung neoplasm, with delusions). The other medical condition should be coded and listed separately immediately before the psychotic disorder due to the medical condition (e.g., 162.9 [C34.90] malignant lung neoplasm; 293.81 [F06.2] psychotic disorder due to malignant lung neoplasm, with delusions).

Specify current severity:

Severity is rated by a quantitative assessment of the primary symptoms of psychosis, including delusions, hallucinations, abnormal psychomotor behavior, and negative symptoms. Each of these symptoms may be rated for its current severity (most severe in the last 7 days) on a 5-point scale ranging from 0 (not present) to 4 (present and

severe). (See Clinician-Rated Dimensions of Psychosis Symptom Severity in the chapter "Assessment Measures.")

Note: Diagnosis of psychotic disorder due to another medical condition can be made without using this severity specifier.

Specifiers

In addition to the symptom domain areas identified in the diagnostic criteria, the assessment of cognition, depression, and mania symptom domains is vital for making critically important distinctions between the various schizophrenia spectrum and other psychotic disorders.

Diagnostic Features

The essential features of psychotic disorder due to another medical condition are prominent delusions or hallucinations that are judged to be attributable to the physiological effects of another medical condition and are not better explained by another mental disorder (e.g., the symptoms are not a psychologically mediated response to a severe medical condition, in which case a diagnosis of brief psychotic disorder, with marked stressor, would be appropriate).

Hallucinations can occur in any sensory modality (i.e., visual, olfactory, gustatory, tactile, or auditory), but certain etiological factors are likely to evoke specific hallucinatory phenomena. Olfactory hallucinations are suggestive of temporal lobe epilepsy. Hallucinations may vary from simple and unformed to highly complex and organized, depending on etiological and environmental factors. Psychotic disorder due to another medical condition is generally not diagnosed if the individual maintains reality testing for the hallucinations and appreciates that they result from the medical condition. Delusions may have a variety of themes, including somatic, grandiose, religious, and, most commonly, persecutory. On the whole, however, associations between delusions and particular medical conditions appear to be less specific than is the case for hallucinations.

In determining whether the psychotic disturbance is attributable to another medical condition, the presence of a medical condition must be identified and considered to be the etiology of the psychosis through a physiological mechanism. Although there are no infallible guidelines for determining whether the relationship between the psychotic disturbance and the medical condition is etiological, several considerations provide some guidance. One consideration is the presence of a temporal association between the onset, exacerbation, or remission of the medical condition and that of the psychotic disturbance. A second consideration is the presence of features that are atypical for a psychotic disorder (e.g., atypical age at onset or presence of visual or olfactory hallucinations). The disturbance must also be distinguished from a substance/medication-induced psychotic disorder or another mental disorder (e.g., an adjustment disorder).

Associated Features Supporting Diagnosis

The temporal association of the onset or exacerbation of the medical condition offers the greatest diagnostic certainty that the delusions or hallucinations are attributable to a medical condition. Additional factors may include concomitant treatments for the underlying medical condition that confer a risk for psychosis independently, such as steroid treatment for autoimmune disorders.

Prevalence

Prevalence rates for psychotic disorder due to another medical condition are difficult to estimate given the wide variety of underlying medical etiologies. Lifetime prevalence has

been estimated to range from 0.21% to 0.54%. When the prevalence findings are stratified by age group, individuals older than 65 years have a significantly greater prevalence of 0.74% compared with those in younger age groups. Rates of psychosis also vary according to the underlying medical condition; conditions most commonly associated with psychosis include untreated endocrine and metabolic disorders, autoimmune disorders (e.g., systemic lupus erythematosus, *N*-methyl-D-aspartate (NMDA) receptor autoimmune encephalitis), or temporal lobe epilepsy. Psychosis due to epilepsy has been further differentiated into ictal, postictal, and interictal psychosis. The most common of these is postictal psychosis, observed in 2%–7.8% of epilepsy patients. Among older individuals, there may be a higher prevalence of the disorder in females, although additional gender-related features are not clear and vary considerably with the gender distributions of the underlying medical conditions.

Development and Course

Psychotic disorder due to another medical condition may be a single transient state or it may be recurrent, cycling with exacerbations and remissions of the underlying medical condition. Although treatment of the underlying medical condition often results in a resolution of the psychosis, this is not always the case, and psychotic symptoms may persist long after the medical event (e.g., psychotic disorder due to focal brain injury). In the context of chronic conditions such as multiple sclerosis or chronic interictal psychosis of epilepsy, the psychosis may assume a long-term course.

The expression of psychotic disorder due to another medical condition does not differ substantially in phenomenology depending on age at occurrence. However, older age groups have a higher prevalence of the disorder, which is most likely due to the increasing medical burden associated with advanced age and the cumulative effects of deleterious exposures and age-related processes (e.g., atherosclerosis). The nature of the underlying medical conditions is likely to change across the lifespan, with younger age groups more affected by epilepsy, head trauma, autoimmune, and neoplastic diseases of early to midlife, and older age groups more affected by stroke disease, anoxic events, and multiple system comorbidities. Underlying factors with increasing age, such as preexisting cognitive impairment as well as vision and hearing impairments, may incur a greater risk for psychosis, possibly by serving to lower the threshold for experiencing psychosis.

Risk and Prognostic Factors

Course modifiers. Identification and treatment of the underlying medical condition has the greatest impact on course, although preexisting central nervous system injury may confer a worse course outcome (e.g., head trauma, cerebrovascular disease).

Diagnostic Markers

The diagnosis of psychotic disorder due to another medical condition depends on the clinical condition of each individual, and the diagnostic tests will vary according to that condition. A variety of medical conditions may cause psychotic symptoms. These include neurological conditions (e.g., neoplasms, cerebrovascular disease, Huntington's disease, multiple sclerosis, epilepsy, auditory or visual nerve injury or impairment, deafness, migraine, central nervous system infections), endocrine conditions (e.g., hyper- and hypothyroidism, hyper- and hypoparathyroidism, hyper- and hypoadrenocorticism), metabolic conditions (e.g., hypoxia, hypercarbia, hypoglycemia), fluid or electrolyte imbalances, hepatic or renal diseases, and autoimmune disorders with central nervous system involvement (e.g., systemic lupus erythematosus). The associated physical examination findings, laboratory findings, and patterns of prevalence or onset reflect the etiological medical condition.

Suicide Risk

Suicide risk in the context of psychotic disorder due to another medical condition is not clearly delineated, although certain conditions such as epilepsy and multiple sclerosis are associated with increased rates of suicide, which may be further increased in the presence of psychosis.

Functional Consequences of Psychotic Disorder Due to Another Medical Condition

Functional disability is typically severe in the context of psychotic disorder due to another medical condition but will vary considerably by the type of condition and likely improve with successful resolution of the condition.

Differential Diagnosis

Delirium. Hallucinations and delusions commonly occur in the context of a delirium; however, a separate diagnosis of psychotic disorder due to another medical condition is not given if the disturbance occurs exclusively during the course of a delirium. Delusions in the context of a major or mild neurocognitive disorder would be diagnosed as major or mild neurocognitive disorder, with behavioral disturbance.

Substance/medication-induced psychotic disorder. If there is evidence of recent or prolonged substance use (including medications with psychoactive effects), withdrawal from a substance, or exposure to a toxin (e.g., LSD [lysergic acid diethylamide] intoxication, alcohol withdrawal), a substance/medication-induced psychotic disorder should be considered. Symptoms that occur during or shortly after (i.e., within 4 weeks) of substance intoxication or withdrawal or after medication use may be especially indicative of a substance-induced psychotic disorder, depending on the character, duration, or amount of the substance used. If the clinician has ascertained that the disturbance is due to both a medical condition and substance use, both diagnoses (i.e., psychotic disorder due to another medical condition and substance/medication-induced psychotic disorder) can be given.

Psychotic disorder. Psychotic disorder due to another medical condition must be distinguished from a psychotic disorder (e.g., schizophrenia, delusional disorder, schizoaffective disorder) or a depressive or bipolar disorder, with psychotic features. In psychotic disorders and in depressive or bipolar disorders, with psychotic features, no specific and direct causative physiological mechanisms associated with a medical condition can be demonstrated. Late age at onset and the absence of a personal or family history of schizophrenia or delusional disorder suggest the need for a thorough assessment to rule out the diagnosis of psychotic disorder due to another medical condition. Auditory hallucinations that involve voices speaking complex sentences are more characteristic of schizophrenia than of psychotic disorder due to a medical condition. Other types of hallucinations (e.g., visual, olfactory) commonly signal a psychotic disorder due to another medical condition or a substance/medication-induced psychotic disorder.

Comorbidity

Psychotic disorder due to another medical condition in individuals older than 80 years is associated with concurrent major neurocognitive disorder (dementia).

Catatonia

Catatonia can occur in the context of several disorders, including neurodevelopmental, psychotic, bipolar, depressive disorders, and other medical conditions (e.g., cerebral folate deficiency, rare autoimmune and paraneoplastic disorders. The manual does not treat catatonia as an independent class but recognizes a) catatonia associated with another mental disorder (i.e., a neurodevelopmental, psychotic disorder, a bipolar disorder, a depressive disorder, or other mental disorder), b) catatonic disorder due to another medical condition, and c) unspecified catatonia.

Catatonia is defined by the presence of three or more of 12 psychomotor features in the diagnostic criteria for catatonia associated with another mental disorder and catatonic disorder due to another medical condition. The essential feature of catatonia is a marked psychomotor disturbance that may involve decreased motor activity, decreased engagement during interview or physical examination, or excessive and peculiar motor activity. The clinical presentation of catatonia can be puzzling, as the psychomotor disturbance may range from marked unresponsiveness to marked agitation. Motoric immobility may be severe (stupor) or moderate (catalepsy and waxy flexibility). Similarly, decreased engagement may be severe (mutism) or moderate (negativism). Excessive and peculiar motor behaviors can be complex (e.g., stereotypy) or simple (agitation) and may include echolalia and echopraxia. In extreme cases, the same individual may wax and wane between decreased and excessive motor activity. The seemingly opposing clinical features and variable manifestations of the diagnosis contribute to a lack of awareness and decreased recognition of catatonia. During severe stages of catatonia, the individual may need careful supervision to avoid self-harm or harming others. There are potential risks from malnutrition, exhaustion, hyperpyrexia and self-inflicted injury.

Catatonia Associated With Another Mental Disorder (Catatonia Specifier)

293.89 (F06.1)

A. The clinical picture is dominated by three (or more) of the following symptoms:

1. Stupor (i.e., no psychomotor activity; not actively relating to environment).
2. Catalepsy (i.e., passive induction of a posture held against gravity).
3. Waxy flexibility (i.e., slight, even resistance to positioning by examiner).
4. Mutism (i.e., no, or very little, verbal response [exclude if known aphasia]).
5. Negativism (i.e., opposition or no response to instructions or external stimuli).
6. Posturing (i.e., spontaneous and active maintenance of a posture against gravity).
7. Mannerism (i.e., odd, circumstantial caricature of normal actions).
8. Stereotypy (i.e., repetitive, abnormally frequent, non-goal-directed movements).
9. Agitation, not influenced by external stimuli.
10. Grimacing.
11. Echolalia (i.e., mimicking another's speech).
12. Echopraxia (i.e., mimicking another's movements).

Coding note: Indicate the name of the associated mental disorder when recording the name of the condition (i.e., 293.89 [F06.1] catatonia associated with major depressive disorder). Code first the associated mental disorder (e.g., neurodevelopmental disorder, brief

psychotic disorder, schizophreniform disorder, schizophrenia, schizoaffective disorder, bipolar disorder, major depressive disorder, or other mental disorder) (e.g., 295.70 [F25.1] schizoaffective disorder, depressive type; 293.89 [F06.1] catatonia associated with schizoaffective disorder).

Diagnostic Features

Catatonia associated with another mental disorder (catatonia specifier) may be used when criteria are met for catatonia during the course of a neurodevelopmental, psychotic, bipolar, depressive, or other mental disorder. The catatonia specifier is appropriate when the clinical picture is characterized by marked psychomotor disturbance and involves at least three of the 12 diagnostic features listed in Criterion A. Catatonia is typically diagnosed in an inpatient setting and occurs in up to 35% of individuals with schizophrenia, but the majority of catatonia cases involve individuals with depressive or bipolar disorders. Before the catatonia specifier is used in neurodevelopmental, psychotic, bipolar, depressive, or other mental disorders, a wide variety of other medical conditions need to be ruled out; these conditions include, but are not limited to, medical conditions due to infectious, metabolic, or neurological conditions (see "Catatonic Disorder Due to Another Medical Condition"). Catatonia can also be a side effect of a medication (see the chapter "Medication-Induced Movement Disorders and Other Adverse Effects of Medication"). Because of the seriousness of the complications, particular attention should be paid to the possibility that the catatonia is attributable to 333.92 (G21.0) neuroleptic malignant syndrome.

Catatonic Disorder Due to Another Medical Condition

Diagnostic Criteria — 293.89 (F06.1)

A. The clinical picture is dominated by three (or more) of the following symptoms:
   1. Stupor (i.e., no psychomotor activity; not actively relating to environment).
   2. Catalepsy (i.e., passive induction of a posture held against gravity).
   3. Waxy flexibility (i.e., slight, even resistance to positioning by examiner).
   4. Mutism (i.e., no, or very little, verbal response [**Note:** not applicable if there is an established aphasia]).
   5. Negativism (i.e., opposition or no response to instructions or external stimuli).
   6. Posturing (i.e., spontaneous and active maintenance of a posture against gravity).
   7. Mannerism (i.e., odd, circumstantial caricature of normal actions).
   8. Stereotypy (i.e., repetitive, abnormally frequent, non-goal-directed movements).
   9. Agitation, not influenced by external stimuli.
   10. Grimacing.
   11. Echolalia (i.e., mimicking another's speech).
   12. Echopraxia (i.e., mimicking another's movements).

B. There is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct pathophysiological consequence of another medical condition.

C. The disturbance is not better explained by another mental disorder (e.g., a manic episode).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Coding note: Include the name of the medical condition in the name of the mental disorder (e.g., 293.89 [F06.1]) catatonic disorder due to hepatic encephalopathy). The other medical condition should be coded and listed separately immediately before the catatonic disorder due to the medical condition (e.g., 572.2 [K71.90] hepatic encephalopathy; 293.89 [F06.1] catatonic disorder due to hepatic encephalopathy).

Diagnostic Features

The essential feature of catatonic disorder due to another medical condition is the presence of catatonia that is judged to be attributed to the physiological effects of another medical condition. Catatonia can be diagnosed by the presence of at least three of the 12 clinical features in Criterion A. There must be evidence from the history, physical examination, or laboratory findings that the catatonia is attributable to another medical condition (Criterion B). The diagnosis is not given if the catatonia is better explained by another mental disorder (e.g., manic episode) (Criterion C) or if it occurs exclusively during the course of a delirium (Criterion D).

Associated Features Supporting Diagnosis

A variety of medical conditions may cause catatonia, especially neurological conditions (e.g., neoplasms, head trauma, cerebrovascular disease, encephalitis) and metabolic conditions (e.g., hypercalcemia, hepatic encephalopathy, homocystinuria, diabetic ketoacidosis). The associated physical examination findings, laboratory findings, and patterns of prevalence and onset reflect those of the etiological medical condition.

Differential Diagnosis

A separate diagnosis of catatonic disorder due to another medical condition is not given if the catatonia occurs exclusively during the course of a delirium or neuroleptic malignant syndrome. If the individual is currently taking neuroleptic medication, consideration should be given to medication-induced movement disorders (e.g., abnormal positioning may be due to neuroleptic-induced acute dystonia) or neuroleptic malignant syndrome (e.g., catatonic-like features may be present, along with associated vital sign and/or laboratory abnormalities). Catatonic symptoms may be present in any of the following five psychotic disorders: brief psychotic disorder, schizophreniform disorder, schizophrenia, schizoaffective disorder, and substance/medication-induced psychotic disorder. It may also be present in some of the neurodevelopmental disorders, in all of the bipolar and depressive disorders, and in other mental disorders.

Unspecified Catatonia

This category applies to presentations in which symptoms characteristic of catatonia cause clinically significant distress or impairment in social, occupational, or other important areas of functioning but either the nature of the underlying mental disorder or other medical condition is unclear, full criteria for catatonia are not met, or there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Coding note: Code first **781.99 (R29.818)** other symptoms involving nervous and musculoskeletal systems, followed by **293.89 (F06.1)** unspecified catatonia.

Other Specified Schizophrenia Spectrum and Other Psychotic Disorder

298.8 (F28)

This category applies to presentations in which symptoms characteristic of a schizophrenia spectrum and other psychotic disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the schizophrenia spectrum and other psychotic disorders diagnostic class. The other specified schizophrenia spectrum and other psychotic disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific schizophrenia spectrum and other psychotic disorder. This is done by recording "other specified schizophrenia spectrum and other psychotic disorder" followed by the specific reason (e.g., "persistent auditory hallucinations").

Examples of presentations that can be specified using the "other specified" designation include the following:

1. **Persistent auditory hallucinations** occurring in the absence of any other features.
2. **Delusions with significant overlapping mood episodes:** This includes persistent delusions with periods of overlapping mood episodes that are present for a substantial portion of the delusional disturbance (such that the criterion stipulating only brief mood disturbance in delusional disorder is not met).
3. **Attenuated psychosis syndrome:** This syndrome is characterized by psychotic-like symptoms that are below a threshold for full psychosis (e.g., the symptoms are less severe and more transient, and insight is relatively maintained).
4. **Delusional symptoms in partner of individual with delusional disorder:** In the context of a relationship, the delusional material from the dominant partner provides content for delusional belief by the individual who may not otherwise entirely meet criteria for delusional disorder.

Unspecified Schizophrenia Spectrum and Other Psychotic Disorder

298.9 (F29)

This category applies to presentations in which symptoms characteristic of a schizophrenia spectrum and other psychotic disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the schizophrenia spectrum and other psychotic disorders diagnostic class. The unspecified schizophrenia spectrum and other psychotic disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific schizophrenia spectrum and other psychotic disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Bipolar and Related Disorders

Bipolar and related disorders are separated from the depressive disorders in DSM-5 and placed between the chapters on schizophrenia spectrum and other psychotic disorders and depressive disorders in recognition of their place as a bridge between the two diagnostic classes in terms of symptomatology, family history, and genetics. The diagnoses included in this chapter are bipolar I disorder, bipolar II disorder, cyclothymic disorder, substance/medication-induced bipolar and related disorder, bipolar and related disorder due to another medical condition, other specified bipolar and related disorder, and unspecified bipolar and related disorder.

The bipolar I disorder criteria represent the modern understanding of the classic manic-depressive disorder or affective psychosis described in the nineteenth century, differing from that classic description only to the extent that neither psychosis nor the lifetime experience of a major depressive episode is a requirement. However, the vast majority of individuals whose symptoms meet the criteria for a fully syndromal manic episode also experience major depressive episodes during the course of their lives.

Bipolar II disorder, requiring the lifetime experience of at least one episode of major depression and at least one hypomanic episode, is no longer thought to be a "milder" condition than bipolar I disorder, largely because of the amount of time individuals with this condition spend in depression and because the instability of mood experienced by individuals with bipolar II disorder is typically accompanied by serious impairment in work and social functioning.

The diagnosis of cyclothymic disorder is given to adults who experience at least 2 years (for children, a full year) of both hypomanic and depressive periods without ever fulfilling the criteria for an episode of mania, hypomania, or major depression.

A large number of substances of abuse, some prescribed medications, and several medical conditions can be associated with manic-like phenomena. This fact is recognized in the diagnoses of substance/medication-induced bipolar and related disorder and bipolar and related disorder due to another medical condition.

The recognition that many individuals, particularly children and, to a lesser extent, adolescents, experience bipolar-like phenomena that do not meet the criteria for bipolar I, bipolar II, or cyclothymic disorder is reflected in the availability of the other specified bipolar and related disorder category. Indeed, specific criteria for a disorder involving short-duration hypomania are provided in Section III in the hope of encouraging further study of this disorder.

Bipolar I Disorder

Diagnostic Criteria

For a diagnosis of bipolar I disorder, it is necessary to meet the following criteria for a manic episode. The manic episode may have been preceded by and may be followed by hypomanic or major depressive episodes.

Manic Episode

A. A distinct period of abnormally and persistently elevated, expansive, or irritable mood and abnormally and persistently increased goal-directed activity or energy, lasting at least 1 week and present most of the day, nearly every day (or any duration if hospitalization is necessary).

B. During the period of mood disturbance and increased energy or activity, three (or more) of the following symptoms (four if the mood is only irritable) are present to a significant degree and represent a noticeable change from usual behavior:

1. Inflated self-esteem or grandiosity.
2. Decreased need for sleep (e.g., feels rested after only 3 hours of sleep).
3. More talkative than usual or pressure to keep talking.
4. Flight of ideas or subjective experience that thoughts are racing.
5. Distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli), as reported or observed.
6. Increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation (i.e., purposeless non-goal-directed activity).
7. Excessive involvement in activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments).

C. The mood disturbance is sufficiently severe to cause marked impairment in social or occupational functioning or to necessitate hospitalization to prevent harm to self or others, or there are psychotic features.

D. The episode is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication, other treatment) or to another medical condition.

Note: A full manic episode that emerges during antidepressant treatment (e.g., medication, electroconvulsive therapy) but persists at a fully syndromal level beyond the physiological effect of that treatment is sufficient evidence for a manic episode and, therefore, a bipolar I diagnosis.

Note: Criteria A–D constitute a manic episode. At least one lifetime manic episode is required for the diagnosis of bipolar I disorder.

Hypomanic Episode

A. A distinct period of abnormally and persistently elevated, expansive, or irritable mood and abnormally and persistently increased activity or energy, lasting at least 4 consecutive days and present most of the day, nearly every day.

B. During the period of mood disturbance and increased energy and activity, three (or more) of the following symptoms (four if the mood is only irritable) have persisted, represent a noticeable change from usual behavior, and have been present to a significant degree:

1. Inflated self-esteem or grandiosity.
2. Decreased need for sleep (e.g., feels rested after only 3 hours of sleep).
3. More talkative than usual or pressure to keep talking.
4. Flight of ideas or subjective experience that thoughts are racing.
5. Distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli), as reported or observed.
6. Increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation.
7. Excessive involvement in activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments).

C. The episode is associated with an unequivocal change in functioning that is uncharacteristic of the individual when not symptomatic.
D. The disturbance in mood and the change in functioning are observable by others.
E. The episode is not severe enough to cause marked impairment in social or occupational functioning or to necessitate hospitalization. If there are psychotic features, the episode is, by definition, manic.
F. The episode is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication, other treatment).

Note: A full hypomanic episode that emerges during antidepressant treatment (e.g., medication, electroconvulsive therapy) but persists at a fully syndromal level beyond the physiological effect of that treatment is sufficient evidence for a hypomanic episode diagnosis. However, caution is indicated so that one or two symptoms (particularly increased irritability, edginess, or agitation following antidepressant use) are not taken as sufficient for diagnosis of a hypomanic episode, nor necessarily indicative of a bipolar diathesis.

Note: Criteria A–F constitute a hypomanic episode. Hypomanic episodes are common in bipolar I disorder but are not required for the diagnosis of bipolar I disorder.

Major Depressive Episode

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

   Note: Do not include symptoms that are clearly attributable to another medical condition.

   1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad, empty, or hopeless) or observation made by others (e.g., appears tearful). (**Note:** In children and adolescents, can be irritable mood.)
   2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation).
   3. Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. (**Note:** In children, consider failure to make expected weight gain.)
   4. Insomnia or hypersomnia nearly every day.
   5. Psychomotor agitation or retardation nearly every day (observable by others; not merely subjective feelings of restlessness or being slowed down).
   6. Fatigue or loss of energy nearly every day.
   7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
   8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
   9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
C. The episode is not attributable to the physiological effects of a substance or another medical condition.

Note: Criteria A–C constitute a major depressive episode. Major depressive episodes are common in bipolar I disorder but are not required for the diagnosis of bipolar I disorder.

Note: Responses to a significant loss (e.g., bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability) may include the feelings of intense

sadness, rumination about the loss, insomnia, poor appetite, and weight loss noted in Criterion A, which may resemble a depressive episode. Although such symptoms may be understandable or considered appropriate to the loss, the presence of a major depressive episode in addition to the normal response to a significant loss should also be carefully considered. This decision inevitably requires the exercise of clinical judgment based on the individual's history and the cultural norms for the expression of distress in the context of loss.[1]

Bipolar I Disorder

A. Criteria have been met for at least one manic episode (Criteria A–D under "Manic Episode" above).
B. The occurrence of the manic and major depressive episode(s) is not better explained by schizoaffective disorder, schizophrenia, schizophreniform disorder, delusional disorder, or other specified or unspecified schizophrenia spectrum and other psychotic disorder.

Coding and Recording Procedures

The diagnostic code for bipolar I disorder is based on type of current or most recent episode and its status with respect to current severity, presence of psychotic features, and remission status. Current severity and psychotic features are only indicated if full criteria are currently met for a manic or major depressive episode. Remission specifiers are only indicated if the full criteria are not currently met for a manic, hypomanic, or major depressive episode. Codes are as follows:

Bipolar I disorder	Current or most recent episode manic	Current or most recent episode hypomanic*	Current or most recent episode depressed	Current or most recent episode unspecified**
Mild (p. 154)	296.41 (F31.11)	NA	296.51 (F31.31)	NA
Moderate (p. 154)	296.42 (F31.12)	NA	296.52 (F31.32)	NA
Severe (p. 154)	296.43 (F31.13)	NA	296.53 (F31.4)	NA

[1] In distinguishing grief from a major depressive episode (MDE), it is useful to consider that in grief the predominant affect is feelings of emptiness and loss, while in MDE it is persistent depressed mood and the inability to anticipate happiness or pleasure. The dysphoria in grief is likely to decrease in intensity over days to weeks and occurs in waves, the so-called pangs of grief. These waves tend to be associated with thoughts or reminders of the deceased. The depressed mood of a MDE is more persistent and not tied to specific thoughts or preoccupations. The pain of grief may be accompanied by positive emotions and humor that are uncharacteristic of the pervasive unhappiness and misery characteristic of a major depressive episode. The thought content associated with grief generally features a preoccupation with thoughts and memories of the deceased, rather than the self-critical or pessimistic ruminations seen in a MDE. In grief, self-esteem is generally preserved, whereas in a MDE, feelings of worthlessness and self-loathing are common. If self-derogatory ideation is present in grief, it typically involves perceived failings vis-à-vis the deceased (e.g., not visiting frequently enough, not telling the deceased how much he or she was loved). If a bereaved individual thinks about death and dying, such thoughts are generally focused on the deceased and possibly about "joining" the deceased, whereas in a major depressive episode such thoughts are focused on ending one's own life because of feeling worthless, undeserving of life, or unable to cope with the pain of depression.

Bipolar I disorder	Current or most recent episode manic	Current or most recent episode hypomanic*	Current or most recent episode depressed	Current or most recent episode unspecified**
With psychotic features*** (p. 152)	296.44 (F31.2)	NA	296.54 (F31.5)	NA
In partial remission (p. 154)	296.45 (F31.73)	296.45 (F31.73)	296.55 (F31.75)	NA
In full remission (p. 154)	296.46 (F31.74)	296.46 (F31.74)	296.56 (F31.76)	NA
Unspecified	296.40 (F31.9)	296.40 (F31.9)	296.50 (F31.9)	NA

*Severity and psychotic specifiers do not apply; code 296.40 (F31.0) for cases not in remission.
**Severity, psychotic, and remission specifiers do not apply. Code 296.7 (F31.9).
***If psychotic features are present, code the "with psychotic features" specifier irrespective of episode severity.

In recording the name of a diagnosis, terms should be listed in the following order: bipolar I disorder, type of current or most recent episode, severity/psychotic/remission specifiers, followed by as many specifiers without codes as apply to the current or most recent episode.

Specify:

With anxious distress (p. 149)
With mixed features (pp. 149–150)
With rapid cycling (pp. 150–151)
With melancholic features (p. 151)
With atypical features (pp. 151–152)
With mood-congruent psychotic features (p. 152)
With mood-incongruent psychotic features (p. 152)
With catatonia (p. 152). **Coding note:** Use additional code 293.89 (F06.1).
With peripartum onset (pp. 152–153)
With seasonal pattern (pp. 153–154)

Diagnostic Features

The essential feature of a manic episode is a distinct period during which there is an abnormally, persistently elevated, expansive, or irritable mood and persistently increased activity or energy that is present for most of the day, nearly every day, for a period of at least 1 week (or any duration if hospitalization is necessary), accompanied by at least three additional symptoms from Criterion B. If the mood is irritable rather than elevated or expansive, at least four Criterion B symptoms must be present.

Mood in a manic episode is often described as euphoric, excessively cheerful, high, or "feeling on top of the world." In some cases, the mood is of such a highly infectious quality that it is easily recognized as excessive and may be characterized by unlimited and haphazard enthusiasm for interpersonal, sexual, or occupational interactions. For example, the individual may spontaneously start extensive conversations with strangers in public. Often the predominant mood is irritable rather than elevated, particularly when the individual's wishes are denied or if the individual has been using substances. Rapid shifts in mood over brief periods of time may occur and are referred to as lability (i.e., the alterna-

tion among euphoria, dysphoria, and irritability). In children, happiness, silliness and "goofiness" are normal in the context of special occasions; however, if these symptoms are recurrent, inappropriate to the context, and beyond what is expected for the developmental level of the child, they may meet Criterion A. If the happiness is unusual for a child (i.e., distinct from baseline), and the mood change occurs at the same time as symptoms that meet Criterion B for mania, diagnostic certainty is increased; however, the mood change must be accompanied by persistently increased activity or energy levels that are obvious to those who know the child well.

During the manic episode, the individual may engage in multiple overlapping new projects. The projects are often initiated with little knowledge of the topic, and nothing seems out of the individual's reach. The increased activity levels may manifest at unusual hours of the day.

Inflated self-esteem is typically present, ranging from uncritical self-confidence to marked grandiosity, and may reach delusional proportions (Criterion B1). Despite lack of any particular experience or talent, the individual may embark on complex tasks such as writing a novel or seeking publicity for some impractical invention. Grandiose delusions (e.g., of having a special relationship to a famous person) are common. In children, overestimation of abilities and belief that, for example, they are the best at a sport or the smartest in the class is normal; however, when such beliefs are present despite clear evidence to the contrary or the child attempts feats that are clearly dangerous and, most important, represent a change from the child's normal behavior, the grandiosity criterion should be considered satisfied.

One of the most common features is a decreased need for sleep (Criterion B2) and is distinct from insomnia in which the individual wants to sleep or feels the need to sleep but is unable. The individual may sleep little, if at all, or may awaken several hours earlier than usual, feeling rested and full of energy. When the sleep disturbance is severe, the individual may go for days without sleep, yet not feel tired. Often a decreased need for sleep heralds the onset of a manic episode.

Speech can be rapid, pressured, loud, and difficult to interrupt (Criterion B3). Individuals may talk continuously and without regard for others' wishes to communicate, often in an intrusive manner or without concern for the relevance of what is said. Speech is sometimes characterized by jokes, puns, amusing irrelevancies, and theatricality, with dramatic mannerisms, singing, and excessive gesturing. Loudness and forcefulness of speech often become more important than what is conveyed. If the individual's mood is more irritable than expansive, speech may be marked by complaints, hostile comments, or angry tirades, particularly if attempts are made to interrupt the individual. Both Criterion A and Criterion B symptoms may be accompanied by symptoms of the opposite (i.e., depressive) pole (see "with mixed features" specifier, pp. 149–150).

Often the individual's thoughts race at a rate faster than they can be expressed through speech (Criterion B4). Frequently there is flight of ideas evidenced by a nearly continuous flow of accelerated speech, with abrupt shifts from one topic to another. When flight of ideas is severe, speech may become disorganized, incoherent, and particularly distressful to the individual. Sometimes thoughts are experienced as so crowded that it is very difficult to speak.

Distractibility (Criterion B5) is evidenced by an inability to censor immaterial external stimuli (e.g., the interviewer's attire, background noises or conversations, furnishings in the room) and often prevents individuals experiencing mania from holding a rational conversation or attending to instructions.

The increase in goal-directed activity often consists of excessive planning and participation in multiple activities, including sexual, occupational, political, or religious activities. Increased sexual drive, fantasies, and behavior are often present. Individuals in a manic episode usually show increased sociability (e.g., renewing old acquaintances or calling or contacting friends or even strangers), without regard to the intrusive, domineering, and demanding nature of these interactions. They often display psychomotor agitation or restlessness (i.e., purposeless activity) by pacing or by holding multiple conversations simulta-

neously. Some individuals write excessive letters, e-mails, text messages, and so forth, on many different topics to friends, public figures, or the media.

The increased activity criterion can be difficult to ascertain in children; however, when the child takes on many tasks simultaneously, starts devising elaborate and unrealistic plans for projects, develops previously absent and developmentally inappropriate sexual preoccupations (not accounted for by sexual abuse or exposure to sexually explicit material), then Criterion B might be met based on clinical judgment. It is essential to determine whether the behavior represents a change from the child's baseline behavior; occurs most of the day, nearly every day for the requisite time period; and occurs in temporal association with other symptoms of mania.

The expansive mood, excessive optimism, grandiosity, and poor judgment often lead to reckless involvement in activities such as spending sprees, giving away possessions, reckless driving, foolish business investments, and sexual promiscuity that is unusual for the individual, even though these activities are likely to have catastrophic consequences (Criterion B7). The individual may purchase many unneeded items without the money to pay for them and, in some cases, give them away. Sexual behavior may include infidelity or indiscriminate sexual encounters with strangers, often disregarding the risk of sexually transmitted diseases or interpersonal consequences.

The manic episode must result in marked impairment in social or occupational functioning or require hospitalization to prevent harm to self or others (e.g., financial losses, illegal activities, loss of employment, self-injurious behavior). By definition, the presence of psychotic features during a manic episode also satisfies Criterion C.

Manic symptoms or syndromes that are attributable to the physiological effects of a drug of abuse (e.g., in the context of cocaine or amphetamine intoxication), the side effects of medications or treatments (e.g., steroids, L-dopa, antidepressants, stimulants), or another medical condition do not count toward the diagnosis of bipolar I disorder. However, a fully syndromal manic episode that arises during treatment (e.g., with medications, electroconvulsive therapy, light therapy) or drug use and persists beyond the physiological effect of the inducing agent (i.e., after a medication is fully out of the individual's system or the effects of electroconvulsive therapy would be expected to have dissipated completely) is sufficient evidence for a manic episode diagnosis (Criterion D). Caution is indicated so that one or two symptoms (particularly increased irritability, edginess, or agitation following antidepressant use) are not taken as sufficient for diagnosis of a manic or hypomanic episode, nor necessarily an indication of a bipolar disorder diathesis. It is necessary to meet criteria for a manic episode to make a diagnosis of bipolar I disorder, but it is not required to have hypomanic or major depressive episodes. However, they may precede or follow a manic episode. Full descriptions of the diagnostic features of a hypomanic episode may be found within the text for bipolar II disorder, and the features of a major depressive episode are described within the text for major depressive disorder.

Associated Features Supporting Diagnosis

During a manic episode, individuals often do not perceive that they are ill or in need of treatment and vehemently resist efforts to be treated. Individuals may change their dress, makeup, or personal appearance to a more sexually suggestive or flamboyant style. Some perceive a sharper sense of smell, hearing, or vision. Gambling and antisocial behaviors may accompany the manic episode. Some individuals may become hostile and physically threatening to others and, when delusional, may become physically assaultive or suicidal. Catastrophic consequences of a manic episode (e.g., involuntary hospitalization, difficulties with the law, serious financial difficulties) often result from poor judgment, loss of insight, and hyperactivity.

Mood may shift very rapidly to anger or depression. Depressive symptoms may occur during a manic episode and, if present, may last moments, hours, or, more rarely, days (see "with mixed features" specifier, pp. 149–150).

Prevalence

The 12-month prevalence estimate in the continental United States was 0.6% for bipolar I disorder as defined in DSM-IV. Twelve-month prevalence of bipolar I disorder across 11 countries ranged from 0.0% to 0.6%. The lifetime male-to-female prevalence ratio is approximately 1.1:1.

Development and Course

Mean age at onset of the first manic, hypomanic, or major depressive episode is approximately 18 years for bipolar I disorder. Special considerations are necessary to detect the diagnosis in children. Since children of the same chronological age may be at different developmental stages, it is difficult to define with precision what is "normal" or "expected" at any given point. Therefore, each child should be judged according to his or her own baseline. Onset occurs throughout the life cycle, including first onsets in the 60s or 70s. Onset of manic symptoms (e.g., sexual or social disinhibition) in late mid-life or late-life should prompt consideration of medical conditions (e.g., frontotemporal neurocognitive disorder) and of substance ingestion or withdrawal.

More than 90% of individuals who have a single manic episode go on to have recurrent mood episodes. Approximately 60% of manic episodes occur immediately before a major depressive episode. Individuals with bipolar I disorder who have multiple (four or more) mood episodes (major depressive, manic, or hypomanic) within 1 year receive the specifier "with rapid cycling."

Risk and Prognostic Factors

Environmental. Bipolar disorder is more common in high-income than in low-income countries (1.4 vs. 0.7%). Separated, divorced, or widowed individuals have higher rates of bipolar I disorder than do individuals who are married or have never been married, but the direction of the association is unclear.

Genetic and physiological. A family history of bipolar disorder is one of the strongest and most consistent risk factors for bipolar disorders. There is an average 10-fold increased risk among adult relatives of individuals with bipolar I and bipolar II disorders. Magnitude of risk increases with degree of kinship. Schizophrenia and bipolar disorder likely share a genetic origin, reflected in familial co-aggregation of schizophrenia and bipolar disorder.

Course modifiers. After an individual has a manic episode with psychotic features, subsequent manic episodes are more likely to include psychotic features. Incomplete interepisode recovery is more common when the current episode is accompanied by mood-incongruent psychotic features.

Culture-Related Diagnostic Issues

Little information exists on specific cultural differences in the expression of bipolar I disorder. One possible explanation for this may be that diagnostic instruments are often translated and applied in different cultures with no transcultural validation. In one U.S. study, 12-month prevalence of bipolar I disorder was significantly lower for Afro-Caribbeans than for African Americans or whites.

Gender-Related Diagnostic Issues

Females are more likely to experience rapid cycling and mixed states, and to have patterns of comorbidity that differ from those of males, including higher rates of lifetime eating disorders. Females with bipolar I or II disorder are more likely to experience depressive symptoms than males. They also have a higher lifetime risk of alcohol use disorder than are males and a much greater likelihood of alcohol use disorder than do females in the general population.

Suicide Risk

The lifetime risk of suicide in individuals with bipolar disorder is estimated to be at least 15 times that of the general population. In fact, bipolar disorder may account for one-quarter of all completed suicides. A past history of suicide attempt and percent days spent depressed in the past year are associated with greater risk of suicide attempts or completions.

Functional Consequences of Bipoiar I Disorder

Although many individuals with bipolar disorder return to a fully functional level between episodes, approximately 30% show severe impairment in work role function. Functional recovery lags substantially behind recovery from symptoms, especially with respect to occupational recovery, resulting in lower socioeconomic status despite equivalent levels of education when compared with the general population. Individuals with bipolar I disorder perform more poorly than healthy individuals on cognitive tests. Cognitive impairments may contribute to vocational and interpersonal difficulties and persist through the lifespan, even during euthymic periods.

Differential Diagnosis

Major depressive disorder. Major depressive disorder may also be accompanied by hypomanic or manic symptoms (i.e., fewer symptoms or for a shorter duration than required for mania or hypomania). When the individual presents in an episode of major depression, one must depend on corroborating history regarding past episodes of mania or hypomania. Symptoms of irritability may be associated with either major depressive disorder or bipolar disorder, adding to diagnostic complexity.

Other bipolar disorders. Diagnosis of bipolar I disorder is differentiated from bipolar II disorder by determining whether there have been any past episodes of mania. Other specified and unspecified bipolar and related disorders should be differentiated from bipolar I and II disorders by considering whether either the episodes involving manic or hypomanic symptoms or the episodes of depressive symptoms fail to meet the full criteria for those conditions.

Bipolar disorder due to another medical condition may be distinguished from bipolar I and II disorders by identifying, based on best clinical evidence, a causally related medical condition.

Generalized anxiety disorder, panic disorder, posttraumatic stress disorder, or other anxiety disorders. These disorders need to be considered in the differential diagnosis as either the primary disorder or, in some cases, a comorbid disorder. A careful history of symptoms is needed to differentiate generalized anxiety disorder from bipolar disorder, as anxious ruminations may be mistaken for racing thoughts, and efforts to minimize anxious feelings may be taken as impulsive behavior. Similarly, symptoms of posttraumatic stress disorder need to be differentiated from bipolar disorder. It is helpful to assess the episodic nature of the symptoms described, as well as to consider symptom triggers, in making this differential diagnosis.

Substance/medication-induced bipolar disorder. Substance use disorders may manifest with substance.medication-induced manic symptoms that must be distinguished from bipolar I disorder; response to mood stabilizers during a substance/medication-induced mania may not necessarily be diagnostic for bipolar disorder. There may be substantial overlap in view of the tendency for individuals with bipolar I disorder to overuse substances during an episode. A primary diagnosis of bipolar disorder must be established based on symptoms that remain once substances are no longer being used.

Attention-deficit/hyperactivity disorder. This disorder may be misdiagnosed as bipolar disorder, especially in adolescents and children. Many symptoms overlap with the symp-

toms of mania, such as rapid speech, racing thoughts, distractibility, and less need for sleep. The "double counting" of symptoms toward both ADHD and bipolar disorder can be avoided if the clinician clarifies whether the symptom(s) represents a distinct episode.

Personality disorders. Personality disorders such as borderline personality disorder may have substantial symptomatic overlap with bipolar disorders, since mood lability and impulsivity are common in both conditions. Symptoms must represent a distinct episode, and the noticeable increase over baseline required for the diagnosis of bipolar disorder must be present. A diagnosis of a personality disorder should not be made during an untreated mood episode.

Disorders with prominent irritability. In individuals with severe irritability, particularly children and adolescents, care must be taken to apply the diagnosis of bipolar disorder only to those who have had a clear episode of mania or hypomania—that is, a distinct time period, of the required duration, during which the irritability was clearly different from the individual's baseline and was accompanied by the onset of Criterion B symptoms. When a child's irritability is persistent and particularly severe, the diagnosis of disruptive mood dysregulation disorder would be more appropriate. Indeed, when any child is being assessed for mania, it is essential that the symptoms represent a clear change from the child's typical behavior.

Comorbidity

Co-occurring mental disorders are common, with the most frequent disorders being any anxiety disorder (e.g., panic attacks, social anxiety disorder [social phobia], specific phobia), occurring in approximately three-fourths of individuals; ADHD, any disruptive, impulse-control, or conduct disorder (e.g., intermittent explosive disorder, oppositional defiant disorder, conduct disorder), and any substance use disorder (e.g., alcohol use disorder) occur in over half of individuals with bipolar I disorder. Adults with bipolar I disorder have high rates of serious and/or untreated co-occurring medical conditions. Metabolic syndrome and migraine are more common among individuals with bipolar disorder than in the general population. More than half of individuals whose symptoms meet criteria for bipolar disorder have an alcohol use disorder, and those with both disorders are at greater risk for suicide attempt.

Bipolar II Disorder

Diagnostic Criteria **296.89 (F31.81)**

For a diagnosis of bipolar II disorder, it is necessary to meet the following criteria for a current or past hypomanic episode *and* the following criteria for a current or past major depressive episode:

Hypomanic Episode

A. A distinct period of abnormally and persistently elevated, expansive, or irritable mood and abnormally and persistently increased activity or energy, lasting at least 4 consecutive days and present most of the day, nearly every day.

B. During the period of mood disturbance and increased energy and activity, three (or more) of the following symptoms have persisted (four if the mood is only irritable), represent a noticeable change from usual behavior, and have been present to a significant degree:

1. Inflated self-esteem or grandiosity.
2. Decreased need for sleep (e.g., feels rested after only 3 hours of sleep).
3. More talkative than usual or pressure to keep talking.

4. Flight of ideas or subjective experience that thoughts are racing.
5. Distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli), as reported or observed.
6. Increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation.
7. Excessive involvement in activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments).

C. The episode is associated with an unequivocal change in functioning that is uncharacteristic of the individual when not symptomatic.
D. The disturbance in mood and the change in functioning are observable by others.
E. The episode is not severe enough to cause marked impairment in social or occupational functioning or to necessitate hospitalization. If there are psychotic features, the episode is, by definition, manic.
F. The episode is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication or other treatment).

Note: A full hypomanic episode that emerges during antidepressant treatment (e.g., medication, electroconvulsive therapy) but persists at a fully syndromal level beyond the physiological effect of that treatment is sufficient evidence for a hypomanic episode diagnosis. However, caution is indicated so that one or two symptoms (particularly increased irritability, edginess, or agitation following antidepressant use) are not taken as sufficient for diagnosis of a hypomanic episode, nor necessarily indicative of a bipolar diathesis.

Major Depressive Episode

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

Note: Do not include symptoms that are clearly attributable to a medical condition.

1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad, empty, or hopeless) or observation made by others (e.g., appears tearful). (**Note:** In children and adolescents, can be irritable mood.)
2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation).
3. Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. (**Note:** In children, consider failure to make expected weight gain.)
4. Insomnia or hypersomnia nearly every day.
5. Psychomotor agitation or retardation nearly every day (observable by others; not merely subjective feelings of restlessness or being slowed down).
6. Fatigue or loss of energy nearly every day.
7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, a suicide attempt, or a specific plan for committing suicide.

B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
C. The episode is not attributable to the physiological effects of a substance or another medical condition.

Note: Criteria A–C above constitute a major depressive episode.

Note: Responses to a significant loss (e.g., bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability) may include the feelings of intense sadness, rumination about the loss, insomnia, poor appetite, and weight loss noted in Criterion A, which may resemble a depressive episode. Although such symptoms may be understandable or considered appropriate to the loss, the presence of a major depressive episode in addition to the normal response to a significant loss should be carefully considered. This decision inevitably requires the exercise of clinical judgment based on the individual's history and the cultural norms for the expression of distress in the context of loss.[1]

Bipolar II Disorder

A. Criteria have been met for at least one hypomanic episode (Criteria A–F under "Hypomanic Episode" above) and at least one major depressive episode (Criteria A–C under "Major Depressive Episode" above).

B. There has never been a manic episode.

C. The occurrence of the hypomanic episode(s) and major depressive episode(s) is not better explained by schizoaffective disorder, schizophrenia, schizophreniform disorder, delusional disorder, or other specified or unspecified schizophrenia spectrum and other psychotic disorder.

D. The symptoms of depression or the unpredictability caused by frequent alternation between periods of depression and hypomania causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Coding and Recording Procedures

Bipolar II disorder has one diagnostic code: 296.89 (F31.81). Its status with respect to current severity, presence of psychotic features, course, and other specifiers cannot be coded but should be indicated in writing (e.g., 296.89 [F31.81] bipolar II disorder, current episode depressed, moderate severity, with mixed features; 296.89 [F31.81] bipolar II disorder, most recent episode depressed, in partial remission).

Specify current or most recent episode:

Hypomanic
Depressed

Specify if:

With anxious distress (p. 149)
With mixed features (pp. 149–150)

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[1] In distinguishing grief from a major depressive episode (MDE), it is useful to consider that in grief the predominant affect is feelings of emptiness and loss, while in a MDE it is persistent depressed mood and the inability to anticipate happiness or pleasure. The dysphoria in grief is likely to decrease in intensity over days to weeks and occurs in waves, the so-called pangs of grief. These waves tend to be associated with thoughts or reminders of the deceased. The depressed mood of a MDE is more persistent and not tied to specific thoughts or preoccupations. The pain of grief may be accompanied by positive emotions and humor that are uncharacteristic of the pervasive unhappiness and misery characteristic of a MDE. The thought content associated with grief generally features a preoccupation with thoughts and memories of the deceased, rather than the self-critical or pessimistic ruminations seen in a MDE. In grief, self-esteem is generally preserved, whereas in a MDE feelings of worthlessness and self-loathing are common. If self-derogatory ideation is present in grief, it typically involves perceived failings vis-à-vis the deceased (e.g., not visiting frequently enough, not telling the deceased how much he or she was loved). If a bereaved individual thinks about death and dying, such thoughts are generally focused on the deceased and possibly about "joining" the deceased, whereas in a MDE such thoughts are focused on ending one's own life because of feeling worthless, undeserving of life, or unable to cope with the pain of depression.

With rapid cycling (pp. 150–151)
With mood-congruent psychotic features (p. 152)
With mood-incongruent psychotic features (p. 152)
With catatonia (p. 152). **Coding note:** Use additional code 293.89 (F06.1).
With peripartum onset (pp. 152–153)
With seasonal pattern (pp. 153–154): Applies only to the pattern of major depressive episodes.

Specify course if full criteria for a mood episode are not currently met:
In partial remission (p. 154)
In full remission (p. 154)

Specify severity if full criteria for a mood episode are currently met:
Mild (p. 154)
Moderate (p. 154)
Severe (p. 154)

Diagnostic Features

Bipolar II disorder is characterized by a clinical course of recurring mood episodes consisting of one or more major depressive episodes (Criteria A–C under "Major Depressive Episode") and at least one hypomanic episode (Criteria A–F under "Hypomanic Episode"). The major depressive episode must last at least 2 weeks, and the hypomanic episode must last at least 4 days, to meet the diagnostic criteria. During the mood episode(s), the requisite number of symptoms must be present most of the day, nearly every day, and represent a noticeable change from usual behavior and functioning. The presence of a manic episode during the course of illness precludes the diagnosis of bipolar II disorder (Criterion B under "Bipolar II Disorder"). Episodes of substance/medication-induced depressive disorder or substance/medication-induced bipolar and related disorder (representing the physiological effects of a medication, other somatic treatments for depression, drugs of abuse, or toxin exposure) or of depressive and related disorder due to another medical condition or bipolar and related disorder due to another medical condition do not count toward a diagnosis of bipolar II disorder unless they persist beyond the physiological effects of the treatment or substance and then meet duration criteria for an episode. In addition, the episodes must not be better accounted for by schizoaffective disorder and are not superimposed on schizophrenia, schizophreniform disorder, delusional disorder, or other specified or unspecified schizophrenia spectrum or other psychotic disorders (Criterion C under "Bipolar II Disorder"). The depressive episodes or hypomanic fluctuations must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion D under "Bipolar II Disorder"); however, for hypomanic episodes, this requirement does not have to be met. A hypomanic episode that causes significant impairment would likely qualify for the diagnosis of manic episode and, therefore, for a lifetime diagnosis of bipolar I disorder. The recurrent major depressive episodes are often more frequent and lengthier than those occurring in bipolar I disorder.

Individuals with bipolar II disorder typically present to a clinician during a major depressive episode and are unlikely to complain initially of hypomania. Typically, the hypomanic episodes themselves do not cause impairment. Instead, the impairment results from the major depressive episodes or from a persistent pattern of unpredictable mood changes and fluctuating, unreliable interpersonal or occupational functioning. Individuals with bipolar II disorder may not view the hypomanic episodes as pathological or disadvantageous, although others may be troubled by the individual's erratic behavior. Clinical information from other informants, such as close friends or relatives, is often useful in establishing the diagnosis of bipolar II disorder.

A hypomanic episode should not be confused with the several days of euthymia and restored energy or activity that may follow remission of a major depressive episode. Despite the substantial differences in duration and severity between a manic and hypomanic episode, bipolar II disorder is not a "milder form" of bipolar I disorder. Compared with individuals with bipolar I disorder, individuals with bipolar II disorder have greater chronicity of illness and spend, on average, more time in the depressive phase of their illness, which can be severe and/or disabling. Depressive symptoms co-occurring with a hypomanic episode or hypomanic symptoms co-occurring with a depressive episode are common in individuals with bipolar II disorder and are overrepresented in females, particularly hypomania with mixed features. Individuals experiencing hypomania with mixed features may not label their symptoms as hypomania, but instead experience them as depression with increased energy or irritability.

Associated Features Supporting Diagnosis

A common feature of bipolar II disorder is impulsivity, which can contribute to suicide attempts and substance use disorders. Impulsivity may also stem from a concurrent personality disorder, substance use disorder, anxiety disorder, another mental disorder, or a medical condition. There may be heightened levels of creativity in some individuals with a bipolar disorder. However, that relationship may be nonlinear; that is, greater lifetime creative accomplishments have been associated with milder forms of bipolar disorder, and higher creativity has been found in unaffected family members. The individual's attachment to heightened creativity during hypomanic episodes may contribute to ambivalence about seeking treatment or undermine adherence to treatment.

Prevaience

The 12-month prevalence of bipolar II disorder, internationally, is 0.3%. In the United States, 12-month prevalence is 0.8%. The prevalence rate of pediatric bipolar II disorder is difficult to establish. DSM-IV bipolar I, bipolar II, and bipolar disorder not otherwise specified yield a combined prevalence rate of 1.8% in U.S. and non-U.S. community samples, with higher rates (2.7% inclusive) in youths age 12 years or older.

Deveiopment and Course

Although bipolar II disorder can begin in late adolescence and throughout adulthood, average age at onset is the mid-20s, which is slightly later than for bipolar I disorder but earlier than for major depressive disorder. The illness most often begins with a depressive episode and is not recognized as bipolar II disorder until a hypomanic episode occurs; this happens in about 12% of individuals with the initial diagnosis of major depressive disorder. Anxiety, substance use, or eating disorders may also precede the diagnosis, complicating its detection. Many individuals experience several episodes of major depression prior to the first recognized hypomanic episode.

The number of lifetime episodes (both hypomanic and major depressive episodes) tends to be higher for bipolar II disorder than for major depressive disorder or bipolar I disorder. However, individuals with bipolar I disorder are actually more likely to experience hypomanic symptoms than are individuals with bipolar II disorder.The interval between mood episodes in the course of bipolar II disorder tends to decrease as the individual ages. While the hypomanic episode is the feature that defines bipolar II disorder, depressive episodes are more enduring and disabling over time. Despite the predominance of depression, once a hypomanic episode has occurred, the diagnosis becomes bipolar II disorder and never reverts to major depressive disorder.

Approximately 5%–15% of individuals with bipolar II disorder have multiple (four or more) mood episodes (hypomanic or major depressive) within the previous 12 months. If

this pattern is present, it is noted by the specifier "with rapid cycling." By definition, psychotic symptoms do not occur in hypomanic episodes, and they appear to be less frequent in the major depressive episodes in bipolar II disorder than in those of bipolar I disorder.

Switching from a depressive episode to a manic or hypomanic episode (with or without mixed features) may occur, both spontaneously and during treatment for depression. About 5%–15% of individuals with bipolar II disorder will ultimately develop a manic episode, which changes the diagnosis to bipolar I disorder, regardless of subsequent course.

Making the diagnosis in children is often a challenge, especially in those with irritability and hyperarousal that is *nonepisodic* (i.e., lacks the well-demarcated periods of altered mood). Nonepisodic irritability in youth is associated with an elevated risk for anxiety disorders and major depressive disorder, but not bipolar disorder, in adulthood. Persistently irritable youths have lower familial rates of bipolar disorder than do youths who have bipolar disorder. For a hypomanic episode to be diagnosed, the child's symptoms must exceed what is expected in a given environment and culture for the child's developmental stage. Compared with adult onset of bipolar II disorder, childhood or adolescent onset of the disorder may be associated with a more severe lifetime course. The 3-year incidence rate of first-onset bipolar II disorder in adults older than 60 years is 0.34%. However, distinguishing individuals older than 60 years with bipolar II disorder by late versus early age at onset does not appear to have any clinical utility.

Risk and Prognostic Factors

Genetic and physiological. The risk of bipolar II disorder tends to be highest among relatives of individuals with bipolar II disorder, as opposed to individuals with bipolar I disorder or major depressive disorder. There may be genetic factors influencing the age at onset for bipolar disorders.

Course modifiers. A rapid-cycling pattern is associated with a poorer prognosis. Return to previous level of social function for individuals with bipolar II disorder is more likely for individuals of younger age and with less severe depression, suggesting adverse effects of prolonged illness on recovery. More education, fewer years of illness, and being married are independently associated with functional recovery in individuals with bipolar disorder, even after diagnostic type (I vs. II), current depressive symptoms, and presence of psychiatric comorbidity are taken into account.

Gender-Related Diagnostic Issues

Whereas the gender ratio for bipolar I disorder is equal, findings on gender differences in bipolar II disorder are mixed, differing by type of sample (i.e., registry, community, or clinical) and country of origin. There is little to no evidence of bipolar gender differences, whereas some, but not all, clinical samples suggest that bipolar II disorder is more common in females than in males, which may reflect gender differences in treatment seeking or other factors.

Patterns of illness and comorbidity, however, seem to differ by gender, with females being more likely than males to report hypomania with mixed depressive features and a rapid-cycling course. Childbirth may be a specific trigger for a hypomanic episode, which can occur in 10%–20% of females in nonclinical populations and most typically in the early postpartum period. Distinguishing hypomania from the elated mood and reduced sleep that normally accompany the birth of a child may be challenging. Postpartum hypomania may foreshadow the onset of a depression that occurs in about half of females who experience postpartum "highs." Accurate detection of bipolar II disorder may help in establishing appropriate treatment of the depression, which may reduce the risk of suicide and infanticide.

Suicide Risk

Suicide risk is high in bipolar II disorder. Approximately one-third of individuals with bipolar II disorder report a lifetime history of suicide attempt. The prevalence rates of lifetime attempted suicide in bipolar II and bipolar I disorder appear to be similar (32.4% and 36.3%, respectively). However, the lethality of attempts, as defined by a lower ratio of attempts to completed suicides, may be higher in individuals with bipolar II disorder compared with individuals with bipolar I disorder. There may be an association between genetic markers and increased risk for suicidal behavior in individuals with bipolar disorder, including a 6.5-fold higher risk of suicide among first-degree relatives of bipolar II probands compared with those with bipolar I disorder.

Functional Consequences of Bipolar II Disorder

Although many individuals with bipolar II disorder return to a fully functional level between mood episodes, at least 15% continue to have some inter-episode dysfunction, and 20% transition directly into another mood episode without inter-episode recovery. Functional recovery lags substantially behind recovery from symptoms of bipolar II disorder, especially in regard to occupational recovery, resulting in lower socioeconomic status despite equivalent levels of education with the general population. Individuals with bipolar II disorder perform more poorly than healthy individuals on cognitive tests and, with the exception of memory and semantic fluency, have similar cognitive impairment as do individuals with bipolar I disorder. Cognitive impairments associated with bipolar II disorder may contribute to vocational difficulties. Prolonged unemployment in individuals with bipolar disorder is associated with more episodes of depression, older age, increased rates of current panic disorder, and lifetime history of alcohol use disorder.

Differential Diagnosis

Major depressive disorder. Perhaps the most challenging differential diagnosis to consider is major depressive disorder, which may be accompanied by hypomanic or manic symptoms that do not meet full criteria (i.e., either fewer symptoms or a shorter duration than required for a hypomanic episode). This is especially true in evaluating individuals with symptoms of irritability, which may be associated with either major depressive disorder or bipolar II disorder.

Cyclothymic disorder. In cyclothymic disorder, there are numerous periods of hypomanic symptoms and numerous periods of depressive symptoms that do not meet symptom or duration criteria for a major depressive episode. Bipolar II disorder is distinguished from cyclothymic disorder by the presence of one or more major depressive episodes. If a major depressive episode occurs after the first 2 years of cyclothymic disorder, the additional diagnosis of bipolar II disorder is given.

Schizophrenia spectrum and other related psychotic disorders. Bipolar II disorder must be distinguished from psychotic disorders (e.g., schizoaffective disorder, schizophrenia, and delusional disorder). Schizophrenia, schizoaffective disorder, and delusional disorder are all characterized by periods of psychotic symptoms that occur in the absence of prominent mood symptoms. Other helpful considerations include the accompanying symptoms, previous course, and family history.

Panic disorder or other anxiety disorders. Anxiety disorders need to be considered in the differential diagnosis and may frequently be present as co-occurring disorders.

Substance use disorders. Substance use disorders are included in the differential diagnosis.

Attention-deficit/hyperactivity disorder. Attention-deficit/hyperactivity disorder (ADHD) may be misdiagnosed as bipolar II disorder, especially in adolescents and children. Many

symptoms of ADHD, such as rapid speech, racing thoughts, distractibility, and less need for sleep, overlap with the symptoms of hypomania. The double counting of symptoms toward both ADHD and bipolar II disorder can be avoided if the clinician clarifies whether the symptoms represent a distinct episode and if the noticeable increase over baseline required for the diagnosis of bipolar II disorder is present.

Personality disorders. The same convention as applies for ADHD also applies when evaluating an individual for a personality disorder such as borderline personality disorder, since mood lability and impulsivity are common in both personality disorders and bipolar II disorder. Symptoms must represent a distinct episode, and the noticeable increase over baseline required for the diagnosis of bipolar II disorder must be present. A diagnosis of a personality disorder should not be made during an untreated mood episode unless the lifetime history supports the presence of a personality disorder.

Other bipolar disorders. Diagnosis of bipolar II disorder should be differentiated from bipolar I disorder by carefully considering whether there have been any past episodes of mania and from other specified and unspecified bipolar and related disorders by confirming the presence of fully syndromal hypomania and depression.

Comorbidity

Bipolar II disorder is more often than not associated with one or more co-occurring mental disorders, with anxiety disorders being the most common. Approximately 60% of individuals with bipolar II disorder have three or more co-occurring mental disorders; 75% have an anxiety disorder; and 37% have a substance use disorder. Children and adolescents with bipolar II disorder have a higher rate of co-occurring anxiety disorders compared with those with bipolar I disorder, and the anxiety disorder most often predates the bipolar disorder. Anxiety and substance use disorders occur in individuals with bipolar II disorder at a higher rate than in the general population. Approximately 14% of individuals with bipolar II disorder have at least one lifetime eating disorder, with binge-eating disorder being more common than bulimia nervosa and anorexia nervosa.

These commonly co-occurring disorders do not seem to follow a course of illness that is truly independent from that of the bipolar disorder, but rather have strong associations with mood states. For example, anxiety and eating disorders tend to associate most with depressive symptoms, and substance use disorders are moderately associated with manic symptoms.

Cyclothymic Disorder

Diagnostic Criteria **301.13 (F34.0)**

A. For at least 2 years (at least 1 year in children and adolescents) there have been numerous periods with hypomanic symptoms that do not meet criteria for a hypomanic episode and numerous periods with depressive symptoms that do not meet criteria for a major depressive episode.

B. During the above 2-year period (1 year in children and adolescents), the hypomanic and depressive periods have been present for at least half the time and the individual has not been without the symptoms for more than 2 months at a time.

C. Criteria for a major depressive, manic, or hypomanic episode have never been met.

D. The symptoms in Criterion A are not better explained by schizoaffective disorder, schizophrenia, schizophreniform disorder, delusional disorder, or other specified or unspecified schizophrenia spectrum and other psychotic disorder.

E. The symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g., hyperthyroidism).

F. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if:

With anxious distress (see p. 149)

Diagnostic Features

The essential feature of cyclothymic disorder is a chronic, fluctuating mood disturbance involving numerous periods of hypomanic symptoms and periods of depressive symptoms that are distinct from each other (Criterion A). The hypomanic symptoms are of insufficient number, severity, pervasiveness, or duration to meet full criteria for a hypomanic episode, and the depressive symptoms are of insufficient number, severity, pervasiveness, or duration to meet full criteria for a major depressive episode. During the initial 2-year period (1 year for children or adolescents), the symptoms must be persistent (present more days than not), and any symptom-free intervals last no longer than 2 months (Criterion B). The diagnosis of cyclothymic disorder is made only if the criteria for a major depressive, manic, or hypomanic episode have never been met (Criterion C).

If an individual with cyclothymic disorder subsequently (i.e., after the initial 2 years in adults or 1 year in children or adolescents) experiences a major depressive, manic, or hypomanic episode, the diagnosis changes to major depressive disorder, bipolar I disorder, or other specified or unspecified bipolar and related disorder (subclassified as hypomanic episode without prior major depressive episode), respectively, and the cyclothymic disorder diagnosis is dropped.

The cyclothymic disorder diagnosis is not made if the pattern of mood swings is better explained by schizoaffective disorder, schizophrenia, schizophreniform disorder, delusional disorder, or other specified and unspecified schizophrenia spectrum and other psychotic disorders (Criterion D), in which case the mood symptoms are considered associated features of the psychotic disorder. The mood disturbance must also not be attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g., hyperthyroidism) (Criterion E). Although some individuals may function particularly well during some of the periods of hypomania, over the prolonged course of the disorder, there must be clinically significant distress or impairment in social, occupational, or other important areas of functioning as a result of the mood disturbance (Criterion F). The impairment may develop as a result of prolonged periods of cyclical, often unpredictable mood changes (e.g., the individual may be regarded as temperamental, moody, unpredictable, inconsistent, or unreliable).

Prevaience

The lifetime prevalence of cyclothymic disorder is approximately 0.4%–1%. Prevalence in mood disorders clinics may range from 3% to 5%. In the general population, cyclothymic disorder is apparently equally common in males and females. In clinical settings, females with cyclothymic disorder may be more likely to present for treatment than males.

Development and Course

Cyclothymic disorder usually begins in adolescence or early adult life and is sometimes considered to reflect a temperamental predisposition to other disorders in this chapter. Cyclothymic disorder usually has an insidious onset and a persistent course. There is a 15%–50% risk that an individual with cyclothymic disorder will subsequently develop bipolar I disorder or bipolar II disorder. Onset of persistent, fluctuating hypomanic and depressive symptoms late in adult life needs to be clearly differentiated from bipolar and

related disorder due to another medical condition and depressive disorder due to another medical condition (e.g., multiple sclerosis) before the cyclothymic disorder diagnosis is assigned. Among children with cyclothymic disorder, the mean age at onset of symptoms is 6.5 years of age.

Risk and Prognostic Factors

Genetic and physiological. Major depressive disorder, bipolar I disorder, and bipolar II disorder are more common among first-degree biological relatives of individuals with cyclothymic disorder than in the general population. There may also be an increased familial risk of substance-related disorders. Cyclothymic disorder may be more common in the first-degree biological relatives of individuals with bipolar I disorder than in the general population.

Differentiai Diagnosis

Bipolar and related disorder due to another medical condition and depressive disorder due to another medical condition. The diagnosis of bipolar and related disorder due to another medical condition or depressive disorder due to another medical condition is made when the mood disturbance is judged to be attributable to the physiological effect of a specific, usually chronic medical condition (e.g., hyperthyroidism). This determination is based on the history, physical examination, or laboratory findings. If it is judged that the hypomanic and depressive symptoms are not the physiological consequence of the medical condition, then the primary mental disorder (i.e., cyclothymic disorder) and the medical condition are coded. For example, this would be the case if the mood symptoms are considered to be the psychological (not the physiological) consequence of having a chronic medical condition, or if there is no etiological relationship between the hypomanic and depressive symptoms and the medical condition.

Substance/medication-induced bipolar and related disorder and substance/medication-induced depressive disorder. Substance/medication-induced bipolar and related disorder and substance/medication-induced depressive disorder are distinguished from cyclothymic disorder by the judgment that a substance/medication (especially stimulants) is etiologically related to the mood disturbance. The frequent mood swings in these disorders that are suggestive of cyclothymic disorder usually resolve following cessation of substance/medication use.

Bipolar I disorder, with rapid cycling, and bipolar II disorder, with rapid cycling. Both disorders may resemble cyclothymic disorder by virtue of the frequent marked shifts in mood. By definition, in cyclothymic disorder the criteria for a major depressive, manic, or hypomanic episode has never been met, whereas the bipolar I disorder and bipolar II disorder specifier "with rapid cycling" requires that full mood episodes be present.

Borderline personality disorder. Borderline personality disorder is associated with marked shifts in mood that may suggest cyclothymic disorder. If the criteria are met for both disorders, both borderline personality disorder and cyclothymic disorder may be diagnosed.

Comorbidity

Substance-related disorders and sleep disorders (i.e., difficulties in initiating and maintaining sleep) may be present in individuals with cyclothymic disorder. Most children with cyclothymic disorder treated in outpatient psychiatric settings have comorbid mental conditions; they are more likely than other pediatric patients with mental disorders to have comorbid attention-deficit/hyperactivity disorder.

Substance/Medication-Induced Bipolar and Related Disorder

Diagnostic Criteria

A. A prominent and persistent disturbance in mood that predominates in the clinical picture and is characterized by elevated, expansive, or irritable mood, with or without depressed mood, or markedly diminished interest or pleasure in all, or almost all, activities.

B. There is evidence from the history, physical examination, or laboratory findings of both (1) and (2):

   1. The symptoms in Criterion A developed during or soon after substance intoxication or withdrawal or after exposure to a medication.
   2. The involved substance/medication is capable of producing the symptoms in Criterion A.

C. The disturbance is not better explained by a bipolar or related disorder that is not substance/medication-induced. Such evidence of an independent bipolar or related disorder could include the following:

   The symptoms precede the onset of the substance/medication use; the symptoms persist for a substantial period of time (e.g., about 1 month) after the cessation of acute withdrawal or severe intoxication; or there is other evidence suggesting the existence of an independent non-substance/medication-induced bipolar and related disorder (e.g., a history of recurrent non-substance/medication-related episodes).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Coding note: The ICD-9-CM and ICD-10-CM codes for the [specific substance/medication]-induced bipolar and related disorders are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. If a mild substance use disorder is comorbid with the substance-induced bipolar and related disorder, the 4th position character is "1," and the clinician should record "mild [substance] use disorder" before the substance-induced bipolar and related disorder (e.g., "mild cocaine use disorder with cocaine-induced bipolar and related disorder"). If a moderate or severe substance use disorder is comorbid with the substance-induced bipolar and related disorder, the 4th position character is "2," and the clinician should record "moderate [substance] use disorder" or "severe [substance] use disorder," depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder (e.g., after a one-time heavy use of the substance), then the 4th position character is "9," and the clinician should record only the substance-induced bipolar and related disorder.

		ICD-10-CM		
	ICD-9-CM	With use disorder, mild	With use disorder, moderate or severe	Without use disorder
Alcohol	291.89	F10.14	F10.24	F10.94
Phencyclidine	292.84	F16.14	F16.24	F16.94
Other hallucinogen	292.84	F16.14	F16.24	F16.94

		ICD-10-CM		
	ICD-9-CM	With use disorder, mild	With use disorder, moderate or severe	Without use disorder
Sedative, hypnotic, or anxiolytic	292.84	F13.14	F13.24	F13.94
Amphetamine (or other stimulant)	292.84	F15.14	F15.24	F15.94
Cocaine	292.84	F14.14	F14.24	F14.94
Other (or unknown) substance	292.84	F19.14	F19.24	F19.94

Specify if (see Table 1 in the chapter "Substance-Related and Addictive Disorders" for diagnoses associated with substance class):

With onset during intoxication: If the criteria are met for intoxication with the substance and the symptoms develop during intoxication.

With onset during withdrawal: If criteria are met for withdrawal from the substance and the symptoms develop during, or shortly after, withdrawal.

Recording Procedures

ICD-9-CM. The name of the substance/medication-induced bipolar and related disorder begins with the specific substance (e.g., cocaine, dexamethasone) that is presumed to be causing the bipolar mood symptoms. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class. For substances that do not fit into any of the classes (e.g., dexamethasone), the code for "other substance" should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category "unknown substance" should be used.

The name of the disorder is followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal). Unlike the recording procedures for ICD-10-CM, which combine the substance-induced disorder and substance use disorder into a single code, for ICD-9-CM a separate diagnostic code is given for the substance use disorder. For example, in the case of irritable symptoms occurring during intoxication in a man with a severe cocaine use disorder, the diagnosis is 292.84 cocaine-induced bipolar and related disorder, with onset during intoxication. An additional diagnosis of 304.20 severe cocaine use disorder is also given. When more than one substance is judged to play a significant role in the development of bipolar mood symptoms, each should be listed separately (e.g., 292.84 methylphenidate-induced bipolar and related disorder, with onset during intoxication; 292.84 dexamethasone-induced bipolar and related disorder, with onset during intoxication).

ICD-10-CM. The name of the substance/medication-induced bipolar and related disorder begins with the specific substance (e.g., cocaine, dexamethasone) that is presumed to be causing the bipolar mood symptoms. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class and presence or absence of a comorbid substance use disorder. For substances that do not fit into any of the classes (e.g., dexamethasone), the code for "other substance" should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category "unknown substance" should be used.

When recording the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by the word "with," followed by the name of the substance-induced

bipolar and related disorder, followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal). For example, in the case of irritable symptoms occurring during intoxication in a man with a severe cocaine use disorder, the diagnosis is F14.24 severe cocaine use disorder with cocaine-induced bipolar and related disorder, with onset during intoxication. A separate diagnosis of the comorbid severe cocaine use disorder is not given. If the substance-induced bipolar and related disorder occurs without a comorbid substance use disorder (e.g., after a one-time heavy use of the substance), no accompanying substance use disorder is noted (e.g., F15.94 amphetamine-induced bipolar and related disorder, with onset during intoxication). When more than one substance is judged to play a significant role in the development of bipolar mood symptoms, each should be listed separately (e.g., F15.24 severe methylphenidate use disorder with methylphenidate-induced bipolar and related disorder, with onset during intoxication; F19.94 dexamethasone-induced bipolar and related disorder, with onset during intoxication).

Diagnostic Features

The diagnostic features of substance/medication-induced bipolar and related disorder are essentially the same as those for mania, hypomania, or depression. A key exception to the diagnosis of substance/medication-induced bipolar and related disorder is the case of hypomania or mania that occurs after antidepressant medication use or other treatments and persists beyond the physiological effects of the medication. This condition is considered an indicator of true bipolar disorder, not substance/medication-induced bipolar and related disorder. Similarly, individuals with apparent electroconvulsive therapy–induced manic or hypomanic episodes that persist beyond the physiological effects of the treatment are diagnosed with bipolar disorder, not substance/medication-induced bipolar and related disorder.

Side effects of some antidepressants and other psychotropic drugs (e.g., edginess, agitation) may resemble the primary symptoms of a manic syndrome, but they are fundamentally distinct from bipolar symptoms and are insufficient for the diagnosis. That is, the criterion symptoms of mania/hypomania have specificity (simple agitation is not the same as excess involvement in purposeful activities), and a sufficient number of symptoms must be present (not just one or two symptoms) to make these diagnoses. In particular, the appearance of one or two nonspecific symptoms—irritability, edginess, or agitation during antidepressant treatment—in the absence of a full manic or hypomanic syndrome should not be taken to support a diagnosis of a bipolar disorder.

Associated Features Supporting Diagnosis

Etiology (causally related to the use of psychotropic medications or substances of abuse based on best clinical evidence) is the key variable in this etiologically specified form of bipolar disorder. Substances/medications that are typically considered to be associated with substance/medication-induced bipolar and related disorder include the stimulant class of drugs, as well as phencyclidine and steroids; however, a number of potential substances continue to emerge as new compounds are synthesized (e.g., so-called bath salts). A history of such substance use may help increase diagnostic certainty.

Prevalence

There are no epidemiological studies of substance/medication-induced mania or bipolar disorder. Each etiological substance may have its own individual risk of inducing a bipolar (manic/hypomanic) disorder.

Development and Course

In phencyclidine-induced mania, the initial presentation may be one of a delirium with affective features, which then becomes an atypically appearing manic or mixed manic state.

This condition follows the ingestion or inhalation quickly, usually within hours or, at the most, a few days. In stimulant-induced manic or hypomanic states, the response is in minutes to 1 hour after one or several ingestions or injections. The episode is very brief and typically resolves over 1–2 days. With corticosteroids and some immunosuppressant medications, the mania (or mixed or depressed state) usually follows several days of ingestion, and the higher doses appear to have a much greater likelihood of producing bipolar symptoms.

Diagnostic Markers

Determination of the substance of use can be made through markers in the blood or urine to corroborate diagnosis.

Differential Diagnosis

Substance/medication-induced bipolar and related disorder should be differentiated from other bipolar disorders, substance intoxication or substance-induced delirium, and medication side effects (as noted earlier). A full manic episode that emerges during antidepressant treatment (e.g., medication, electroconvulsive therapy) but persists at a fully syndromal level beyond the physiological effect of that treatment is sufficient evidence for a bipolar I diagnosis. A full hypomanic episode that emerges during antidepressant treatment (e.g., medication, electroconvulsive therapy) but persists at a fully syndromal level beyond the physiological effect of that treatment is sufficient evidence for a bipolar II diagnosis only if preceded by a major depressive episode.

Comorbidity

Comorbidities are those associated with the use of illicit substances (in the case of illegal stimulants or phencyclidine) or diversion of prescribed stimulants. Comorbidities related to steroid or immunosuppressant medications are those medical indications for these preparations. Delirium can occur before or along with manic symptoms in individuals ingesting phencyclidine or those who are prescribed steroid medications or other immunosuppressant medications.

Bipolar and Related Disorder Due to Another Medical Condition

Diagnostic Criteria

A. A prominent and persistent period of abnormally elevated, expansive, or irritable mood and abnormally increased activity or energy that predominates in the clinical picture.
B. There is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct pathophysiological consequence of another medical condition.
C. The disturbance is not better explained by another mental disorder.
D. The disturbance does not occur exclusively during the course of a delirium.
E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning, or necessitates hospitalization to prevent harm to self or others, or there are psychotic features.

Coding note: The ICD-9-CM code for bipolar and related disorder due to another medical condition is **293.83**, which is assigned regardless of the specifier. The ICD-10-CM code depends on the specifier (see below).

Specify if:

(F06.33) With manic features: Full criteria are not met for a manic or hypomanic episode.

(F06.33) With manic- or hypomanic-like episode: Full criteria are met except Criterion D for a manic episode or except Criterion F for a hypomanic episode.

(F06.34) With mixed features: Symptoms of depression are also present but do not predominate in the clinical picture.

Coding note: Include the name of the other medical condition in the name of the mental disorder (e.g., 293.83 [F06.33] bipolar disorder due to hyperthyroidism, with manic features). The other medical condition should also be coded and listed separately immediately before the bipolar and related disorder due to the medical condition (e.g., 242.90 [E05.90] hyperthyroidism; 293.83 [F06.33] bipolar disorder due to hyperthyroidism, with manic features).

Diagnostic Features

The essential features of bipolar and related disorder due to another medical condition are presence of a prominent and persistent period of abnormally elevated, expansive, or irritable mood and abnormally increased activity or energy predominating in the clinical picture that is attributable to another medical condition (Criterion B). In most cases the manic or hypomanic picture may appear during the initial presentation of the medical condition (i.e., within 1 month); however, there are exceptions, especially in chronic medical conditions that might worsen or relapse and herald the appearance of the manic or hypomanic picture. Bipolar and related disorder due to another medical condition would not be diagnosed when the manic or hypomanic episodes definitely preceded the medical condition, since the proper diagnosis would be bipolar disorder (except in the unusual circumstance in which all preceding manic or hypomanic episodes—or, when only one such episode has occurred, the preceding manic or hypomanic episode—were associated with ingestion of a substance/medication). The diagnosis of bipolar and related disorder due to another medical condition should not be made during the course of a delirium (Criterion D). The manic or hypomanic episode in bipolar and related disorder due to another medical condition must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning to qualify for this diagnosis (Criterion E).

Associated Features Supporting Diagnosis

Etiology (i.e., a causal relationship to another medical condition based on best clinical evidence) is the key variable in this etiologically specified form of bipolar disorder. The listing of medical conditions that are said to be able to induce mania is never complete, and the clinician's best judgment is the essence of this diagnosis. Among the best known of the medical conditions that can cause a bipolar manic or hypomanic condition are Cushing's disease and multiple sclerosis, as well as stroke and traumatic brain injuries.

Development and Course

Bipolar and related disorder due to another medical condition usually has its onset acutely or subacutely within the first weeks or month of the onset of the associated medical condition. However, this is not always the case, as a worsening or later relapse of the associated medical condition may precede the onset of the manic or hypomanic syndrome. The clinician must make a clinical judgment in these situations about whether the medical condition is causative, based on temporal sequence as well as plausibility of a causal relation-

ship. Finally, the condition may remit before or just after the medical condition remits, particularly when treatment of the manic/hypomanic symptoms is effective.

Culture-Related Diagnostic Issues

Culture-related differences, to the extent that there is any evidence, pertain to those associated with the medical condition (e.g., rates of multiple sclerosis and stroke vary around the world based on dietary, genetic factors, and other environmental factors).

Gender-Related Diagnostic Issues

Gender differences pertain to those associated with the medical condition (e.g., systemic lupus erythematosus is more common in females; stroke is somewhat more common in middle-age males compared with females).

Diagnostic Markers

Diagnostic markers pertain to those associated with the medical condition (e.g., steroid levels in blood or urine to help corroborate the diagnosis of Cushing's disease, which can be associated with manic or depressive syndromes; laboratory tests confirming the diagnosis of multiple sclerosis).

Functional Consequences of Bipolar and Related Disorder Due to Another Medical Condition

Functional consequences of the bipolar symptoms may exacerbate impairments associated with the medical condition and may incur worse outcomes due to interference with medical treatment. In general, it is believed, but not established, that the illness, when induced by Cushing's disease, will not recur if the Cushing's disease is cured or arrested. However, it is also suggested, but not established, that mood syndromes, including depressive and manic/hypomanic ones, may be episodic (i.e., recurring) with static brain injuries and other central nervous system diseases.

Differential Diagnosis

Symptoms of delirium, catatonia, and acute anxiety. It is important to differentiate symptoms of mania from excited or hypervigilant delirious symptoms; from excited catatonic symptoms; and from agitation related to acute anxiety states.

Medication-induced depressive or manic symptoms. An important differential diagnostic observation is that the other medical condition may be treated with medications (e.g., steroids or alpha-interferon) that can induce depressive or manic symptoms. In these cases, clinical judgment using all of the evidence in hand is the best way to try to separate the most likely and/or the most important of two etiological factors (i.e., association with the medical condition vs. a substance/medication-induced syndrome). The differential diagnosis of the associated medical conditions is relevant but largely beyond the scope of the present manual.

Comorbidity

Conditions comorbid with bipolar and related disorder due to another medical condition are those associated with the medical conditions of etiological relevance. Delirium can occur before or along with manic symptoms in individuals with Cushing's disease.

Other Specified Bipolar and Related Disorder

296.89 (F31.89)

This category applies to presentations in which symptoms characteristic of a bipolar and related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the bipolar and related disorders diagnostic class. The other specified bipolar and related disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific bipolar and related disorder. This is done by recording "other specified bipolar and related disorder" followed by the specific reason (e.g., "short-duration cyclothymia").

Examples of presentations that can be specified using the "other specified" designation include the following:

1. **Short-duration hypomanic episodes (2–3 days) and major depressive episodes:** A lifetime history of one or more major depressive episodes in individuals whose presentation has never met full criteria for a manic or hypomanic episode but who have experienced two or more episodes of short-duration hypomania that meet the full symptomatic criteria for a hypomanic episode but that only last for 2–3 days. The episodes of hypomanic symptoms do not overlap in time with the major depressive episodes, so the disturbance does not meet criteria for major depressive episode, with mixed features.
2. **Hypomanic episodes with insufficient symptoms and major depressive episodes:** A lifetime history of one or more major depressive episodes in individuals whose presentation has never met full criteria for a manic or hypomanic episode but who have experienced one or more episodes of hypomania that do not meet full symptomatic criteria (i.e., at least 4 consecutive days of elevated mood and one or two of the other symptoms of a hypomanic episode, or irritable mood and two or three of the other symptoms of a hypomanic episode). The episodes of hypomanic symptoms do not overlap in time with the major depressive episodes, so the disturbance does not meet criteria for major depressive episode, with mixed features.
3. **Hypomanic episode without prior major depressive episode:** One or more hypomanic episodes in an individual whose presentation has never met full criteria for a major depressive episode or a manic episode. If this occurs in an individual with an established diagnosis of persistent depressive disorder (dysthymia), both diagnoses can be concurrently applied during the periods when the full criteria for a hypomanic episode are met.
4. **Short-duration cyclothymia (less than 24 months):** Multiple episodes of hypomanic symptoms that do not meet criteria for a hypomanic episode and multiple episodes of depressive symptoms that do not meet criteria for a major depressive episode that persist over a period of less than 24 months (less than 12 months for children or adolescents) in an individual whose presentation has never met full criteria for a major depressive, manic, or hypomanic episode and does not meet criteria for any psychotic disorder. During the course of the disorder, the hypomanic or depressive symptoms are present for more days than not, the individual has not been without symptoms for more than 2 months at a time, and the symptoms cause clinically significant distress or impairment.

Unspecified Bipolar and Related Disorder

296.80 (F31.9)

This category applies to presentations in which symptoms characteristic of a bipolar and related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the bipolar and related disorders diagnostic class. The unspecified bipolar and related disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific bipolar and related disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Specifiers for Bipolar and Related Disorders

Specify if:

With anxious distress: The presence of at least two of the following symptoms during the majority of days of the current or most recent episode of mania, hypomania, or depression:

1. Feeling keyed up or tense.
2. Feeling unusually restless.
3. Difficulty concentrating because of worry.
4. Fear that something awful may happen.
5. Feeling that the individual might lose control of himself or herself.

Specify current severity:

Mild: Two symptoms.

Moderate: Three symptoms.

Moderate-severe: Four or five symptoms.

Severe: Four or five symptoms with motor agitation.

Note: Anxious distress has been noted as a prominent feature of both bipolar and major depressive disorder in both primary care and specialty mental health settings. High levels of anxiety have been associated with higher suicide risk, longer duration of illness, and greater likelihood of treatment nonresponse. As a result, it is clinically useful to specify accurately the presence and severity levels of anxious distress for treatment planning and monitoring of response to treatment.

With mixed features: The mixed features specifier can apply to the current manic, hypomanic, or depressive episode in bipolar I or bipolar II disorder:

Manic or hypomanic episode, with mixed features:

A. Full criteria are met for a manic episode or hypomanic episode, and at least three of the following symptoms are present during the majority of days of the current or most recent episode of mania or hypomania:

1. Prominent dysphoria or depressed mood as indicated by either subjective report (e.g., feels sad or empty) or observation made by others (e.g., appears tearful).
2. Diminished interest or pleasure in all, or almost all, activities (as indicated by either subjective account or observation made by others).
3. Psychomotor retardation nearly every day (observable by others; not merely subjective feelings of being slowed down).

4. Fatigue or loss of energy.
5. Feelings of worthlessness or excessive or inappropriate guilt (not merely self-reproach or guilt about being sick).
6. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

B. Mixed symptoms are observable by others and represent a change from the person's usual behavior.

C. For individuals whose symptoms meet full episode criteria for both mania and depression simultaneously, the diagnosis should be manic episode, with mixed features, due to the marked impairment and clinical severity of full mania.

D. The mixed symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication, other treatment).

Depressive episode, with mixed features:

A. Full criteria are met for a major depressive episode, and at least three of the following manic/hypomanic symptoms are present during the majority of days of the current or most recent episode of depression:

1. Elevated, expansive mood.
2. Inflated self-esteem or grandiosity.
3. More talkative than usual or pressure to keep talking.
4. Flight of ideas or subjective experience that thoughts are racing.
5. Increase in energy or goal-directed activity (either socially, at work or school, or sexually).
6. Increased or excessive involvement in activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments).
7. Decreased need for sleep (feeling rested despite sleeping less than usual; to be contrasted with insomnia).

B. Mixed symptoms are observable by others and represent a change from the person's usual behavior.

C. For individuals whose symptoms meet full episode criteria for both mania and depression simultaneously, the diagnosis should be manic episode, with mixed features.

D. The mixed symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication, or other treatment).

Note: Mixed features associated with a major depressive episode have been found to be a significant risk factor for the development of bipolar I or bipolar II disorder. As a result, it is clinically useful to note the presence of this specifier for treatment planning and monitoring of response to treatment.

With rapid cycling (can be applied to bipolar I or bipolar II disorder): Presence of at least four mood episodes in the previous 12 months that meet the criteria for manic, hypomanic, or major depressive episode.

Note: Episodes are demarcated by either partial or full remissions of at least 2 months or a switch to an episode of the opposite polarity (e.g., major depressive episode to manic episode).

Note: The essential feature of a rapid-cycling bipolar disorder is the occurrence of at least four mood episodes during the previous 12 months. These episodes can occur in any combination and order. The episodes must meet both the duration and

symptom number criteria for a major depressive, manic, or hypomanic episode and must be demarcated by either a period of full remission or a switch to an episode of the opposite polarity. Manic and hypomanic episodes are counted as being on the same pole. Except for the fact that they occur more frequently, the episodes that occur in a rapid-cycling pattern are no different from those that occur in a non-rapid-cycling pattern. Mood episodes that count toward defining a rapid-cycling pattern exclude those episodes directly caused by a substance (e.g., cocaine, corticosteroids) or another medical condition.

With melancholic features:

A. One of the following is present during the most severe period of the current episode:
   1. Loss of pleasure in all, or almost all, activities.
   2. Lack of reactivity to usually pleasurable stimuli (does not feel much better, even temporarily, when something good happens).

B. Three (or more) of the following:
   1. A distinct quality of depressed mood characterized by profound despondency, despair, and/or moroseness or by so-called empty mood.
   2. Depression that is regularly worse in the morning.
   3. Early-morning awakening (i.e., at least 2 hours before usual awakening).
   4. Marked psychomotor agitation or retardation.
   5. Significant anorexia or weight loss.
   6. Excessive or inappropriate guilt.

   Note: The specifier "with melancholic features" is applied if these features are present at the most severe stage of the episode. There is a near-complete absence of the capacity for pleasure, not merely a diminution. A guideline for evaluating the lack of reactivity of mood is that even highly desired events are not associated with marked brightening of mood. Either mood does not brighten at all, or it brightens only partially (e.g., up to 20%–40% of normal for only minutes at a time). The "distinct quality" of mood that is characteristic of the "with melancholic features" specifier is experienced as qualitatively different from that during a nonmelancholic depressive episode. A depressed mood that is described as merely more severe, longer lasting, or present without a reason is not considered distinct in quality. Psychomotor changes are nearly always present and are observable by others.

   Melancholic features exhibit only a modest tendency to repeat across episodes in the same individual. They are more frequent in inpatients, as opposed to outpatients; are less likely to occur in milder than in more severe major depressive episodes; and are more likely to occur in those with psychotic features.

With atypical features: This specifier can be applied when these features predominate during the majority of days of the current or most recent major depressive episode.

A. Mood reactivity (i.e., mood brightens in response to actual or potential positive events).

B. Two (or more) of the following features:
   1. Significant weight gain or increase in appetite.
   2. Hypersomnia.
   3. Leaden paralysis (i.e., heavy, leaden feelings in arms or legs).
   4. A long-standing pattern of interpersonal rejection sensitivity (not limited to episodes of mood disturbance) that results in significant social or occupational impairment.

C. Criteria are not met for "with melancholic features" or "with catatonia" during the same episode.

Note: "Atypical depression" has historical significance (i.e., atypical in contradistinction to the more classical agitated, "endogenous" presentations of depression that were the norm when depression was rarely diagnosed in outpatients and almost never in adolescents or younger adults) and today does not connote an uncommon or unusual clinical presentation as the term might imply.

Mood reactivity is the capacity to be cheered up when presented with positive events (e.g., a visit from children, compliments from others). Mood may become euthymic (not sad) even for extended periods of time if the external circumstances remain favorable. Increased appetite may be manifested by an obvious increase in food intake or by weight gain. Hypersomnia may include either an extended period of nighttime sleep or daytime napping that totals at least 10 hours of sleep per day (or at least 2 hours more than when not depressed). Leaden paralysis is defined as feeling heavy, leaden, or weighted down, usually in the arms or legs. This sensation is generally present for at least an hour a day but often lasts for many hours at a time. Unlike the other atypical features, pathological sensitivity to perceived interpersonal rejection is a trait that has an early onset and persists throughout most of adult life. Rejection sensitivity occurs both when the person is and is not depressed, though it may be exacerbated during depressive periods.

With psychotic features: Delusions or hallucinations are present at any time in the episode. If psychotic features are present, specify if mood-congruent or mood-incongruent:

With mood-congruent psychotic features: During manic episodes, the content of all delusions and hallucinations is consistent with the typical manic themes of grandiosity, invulnerability, etc., but may also include themes of suspiciousness or paranoia, especially with respect to others' doubts about the individual's capacities, accomplishments, and so forth.

With mood-incongruent psychotic features: The content of delusions and hallucinations is inconsistent with the episode polarity themes as described above, or the content is a mixture of mood-incongruent and mood-congruent themes.

With catatonia: This specifier can apply to an episode of mania or depression if catatonic features are present during most of the episode. See criteria for catatonia associated with a mental disorder in the chapter "Schizophrenia Spectrum and Other Psychotic Disorders."

With peripartum onset: This specifier can be applied to the current or, if the full criteria are not currently met for a mood episode, most recent episode of mania, hypomania, or major depression in bipolar I or bipolar II disorder if onset of mood symptoms occurs during pregnancy or in the 4 weeks following delivery.

Note: Mood episodes can have their onset either during pregnancy or postpartum. Although the estimates differ according to the period of follow-up after delivery, between 3% and 6% of women will experience the onset of a major depressive episode during pregnancy or in the weeks or months following delivery. Fifty percent of "postpartum" major depressive episodes actually begin prior to delivery. Thus, these episodes are referred to collectively as *peripartum* episodes. Women with peripartum major depressive episodes often have severe anxiety and even panic attacks. Prospective studies have demonstrated that mood and anxiety symptoms during pregnancy, as well as the "baby blues," increase the risk for a postpartum major depressive episode.

Peripartum-onset mood episodes can present either with or without psychotic features. Infanticide is most often associated with postpartum psychotic episodes that are characterized by command hallucinations to kill the infant or delusions that the infant is possessed, but psychotic symptoms can also occur in severe postpartum mood episodes without such specific delusions or hallucinations.

Postpartum mood (major depressive or manic) episodes with psychotic features appear to occur in from 1 in 500 to 1 in 1,000 deliveries and may be more common in primiparous women. The risk of postpartum episodes with psychotic features is particularly increased for women with prior postpartum mood episodes but is also elevated for those with a prior history of a depressive or bipolar disorder (especially bipolar I disorder) and those with a family history of bipolar disorders.

Once a woman has had a postpartum episode with psychotic features, the risk of recurrence with each subsequent delivery is between 30% and 50%. Postpartum episodes must be differentiated from delirium occurring in the postpartum period, which is distinguished by a fluctuating level of awareness or attention. The postpartum period is unique with respect to the degree of neuroendocrine alterations and psychosocial adjustments, the potential impact of breast-feeding on treatment planning, and the long-term implications of a history of postpartum mood disorder on subsequent family planning.

With seasonal pattern: This specifier applies to the lifetime pattern of mood episodes. The essential feature is a regular seasonal pattern of at least one type of episode (i.e., mania, hypomania, or depression). The other types of episodes may not follow this pattern. For example, an individual may have seasonal manias, but his or her depressions do not regularly occur at a specific time of year.

A. There has been a regular temporal relationship between the onset of manic, hypomanic, or major depressive episodes and a particular time of the year (e.g., in the fall or winter) in bipolar I or bipolar II disorder.

   Note: Do not include cases in which there is an obvious effect of seasonally related psychosocial stressors (e.g., regularly being unemployed every winter).

B. Full remissions (or a change from major depression to mania or hypomania or vice versa) also occur at a characteristic time of the year (e.g., depression disappears in the spring).

C. In the last 2 years, the individual's manic, hypomanic, or major depressive episodes have demonstrated a temporal seasonal relationship, as defined above, and no non-seasonal episodes of that polarity have occurred during that 2-year period.

D. Seasonal manias, hypomanias, or depressions (as described above) substantially outnumber any nonseasonal manias, hypomanias, or depressions that may have occurred over the individual's lifetime.

   Note: This specifier can be applied to the pattern of major depressive episodes in bipolar I disorder, bipolar II disorder, or major depressive disorder, recurrent. The essential feature is the onset and remission of major depressive episodes at characteristic times of the year. In most cases, the episodes begin in fall or winter and remit in spring. Less commonly, there may be recurrent summer depressive episodes. This pattern of onset and remission of episodes must have occurred during at least a 2-year period, without any nonseasonal episodes occurring during this period. In addition, the seasonal depressive episodes must substantially outnumber any nonseasonal depressive episodes over the individual's lifetime.

   This specifier does not apply to those situations in which the pattern is better explained by seasonally linked psychosocial stressors (e.g., seasonal unemployment or school schedule). Major depressive episodes that occur in a seasonal pattern

are often characterized by prominent energy, hypersomnia, overeating, weight gain, and a craving for carbohydrates. It is unclear whether a seasonal pattern is more likely in recurrent major depressive disorder or in bipolar disorders. However, within the bipolar disorders group, a seasonal pattern appears to be more likely in bipolar II disorder than in bipolar I disorder. In some individuals, the onset of manic or hypomanic episodes may also be linked to a particular season.

The prevalence of winter-type seasonal pattern appears to vary with latitude, age, and sex. Prevalence increases with higher latitudes. Age is also a strong predictor of seasonality, with younger persons at higher risk for winter depressive episodes.

Specify if:

In partial remission: Symptoms of the immediately previous manic, hypomanic, or depressive episode are present, but full criteria are not met, or there is a period lasting less than 2 months without any significant symptoms of a manic, hypomanic, or major depressive episode following the end of such an episode.

In full remission: During the past 2 months, no significant signs or symptoms of the disturbance were present.

Specify current severity:

Severity is based on the number of criterion symptoms, the severity of those symptoms, and the degree of functional disability.

Mild: Few, if any, symptoms in excess of those required to meet the diagnostic criteria are present, the intensity of the symptoms is distressing but manageable, and the symptoms result in minor impairment in social or occupational functioning.

Moderate: The number of symptoms, intensity of symptoms, and/or functional impairment are between those specified for "mild" and "severe."

Severe: The number of symptoms is substantially in excess of those required to make the diagnosis, the intensity of the symptoms is seriously distressing and unmanageable, and the symptoms markedly interfere with social and occupational functioning.

Depressive Disorders

Depressive disorders include disruptive mood dysregulation disorder, major depressive disorder (including major depressive episode), persistent depressive disorder (dysthymia), premenstrual dysphoric disorder, substance/medication-induced depressive disorder, depressive disorder due to another medical condition, other specified depressive disorder, and unspecified depressive disorder. Unlike in DSM-IV, this chapter "Depressive Disorders" has been separated from the previous chapter "Bipolar and Related Disorders." The common feature of all of these disorders is the presence of sad, empty, or irritable mood, accompanied by somatic and cognitive changes that significantly affect the individual's capacity to function. What differs among them are issues of duration, timing, or presumed etiology.

In order to address concerns about the potential for the overdiagnosis of and treatment for bipolar disorder in children, a new diagnosis, disruptive mood dysregulation disorder, referring to the presentation of children with persistent irritability and frequent episodes of extreme behavioral dyscontrol, is added to the depressive disorders for children up to 12 years of age. Its placement in this chapter reflects the finding that children with this symptom pattern typically develop unipolar depressive disorders or anxiety disorders, rather than bipolar disorders, as they mature into adolescence and adulthood.

Major depressive disorder represents the classic condition in this group of disorders. It is characterized by discrete episodes of at least 2 weeks' duration (although most episodes last considerably longer) involving clear-cut changes in affect, cognition, and neurovegetative functions and inter-episode remissions. A diagnosis based on a single episode is possible, although the disorder is a recurrent one in the majority of cases. Careful consideration is given to the delineation of normal sadness and grief from a major depressive episode. Bereavement may induce great suffering, but it does not typically induce an episode of major depressive disorder. When they do occur together, the depressive symptoms and functional impairment tend to be more severe and the prognosis is worse compared with bereavement that is not accompanied by major depressive disorder. Bereavement-related depression tends to occur in persons with other vulnerabilities to depressive disorders, and recovery may be facilitated by antidepressant treatment.

A more chronic form of depression, persistent depressive disorder (dysthymia), can be diagnosed when the mood disturbance continues for at least 2 years in adults or 1 year in children. This diagnosis, new in DSM-5, includes both the DSM-IV diagnostic categories of chronic major depression and dysthymia.

After careful scientific review of the evidence, premenstrual dysphoric disorder has been moved from an appendix of DSM-IV ("Criteria Sets and Axes Provided for Further Study") to Section II of DSM-5. Almost 20 years of additional of research on this condition has confirmed a specific and treatment-responsive form of depressive disorder that begins sometime following ovulation and remits within a few days of menses and has a marked impact on functioning.

A large number of substances of abuse, some prescribed medications, and several medical conditions can be associated with depression-like phenomena. This fact is recognized in the diagnoses of substance/medication-induced depressive disorder and depressive disorder due to another medical condition.

Disruptive Mood Dysregulation Disorder

Diagnostic Criteria **296.99 (F34.8)**

A. Severe recurrent temper outbursts manifested verbally (e.g., verbal rages) and/or behaviorally (e.g., physical aggression toward people or property) that are grossly out of proportion in intensity or duration to the situation or provocation.
B. The temper outbursts are inconsistent with developmental level.
C. The temper outbursts occur, on average, three or more times per week.
D. The mood between temper outbursts is persistently irritable or angry most of the day, nearly every day, and is observable by others (e.g., parents, teachers, peers).
E. Criteria A–D have been present for 12 or more months. Throughout that time, the individual has not had a period lasting 3 or more consecutive months without all of the symptoms in Criteria A–D.
F. Criteria A and D are present in at least two of three settings (i.e., at home, at school, with peers) and are severe in at least one of these.
G. The diagnosis should not be made for the first time before age 6 years or after age 18 years.
H. By history or observation, the age at onset of Criteria A–E is before 10 years.
I. There has never been a distinct period lasting more than 1 day during which the full symptom criteria, except duration, for a manic or hypomanic episode have been met.
Note: Developmentally appropriate mood elevation, such as occurs in the context of a highly positive event or its anticipation, should not be considered as a symptom of mania or hypomania.
J. The behaviors do not occur exclusively during an episode of major depressive disorder and are not better explained by another mental disorder (e.g., autism spectrum disorder, posttraumatic stress disorder, separation anxiety disorder, persistent depressive disorder [dysthymia]).
Note: This diagnosis cannot coexist with oppositional defiant disorder, intermittent explosive disorder, or bipolar disorder, though it can coexist with others, including major depressive disorder, attention-deficit/hyperactivity disorder, conduct disorder, and substance use disorders. Individuals whose symptoms meet criteria for both disruptive mood dysregulation disorder and oppositional defiant disorder should only be given the diagnosis of disruptive mood dysregulation disorder. If an individual has ever experienced a manic or hypomanic episode, the diagnosis of disruptive mood dysregulation disorder should not be assigned.
K. The symptoms are not attributable to the physiological effects of a substance or to another medical or neurological condition.

Diagnostic Features

The core feature of disruptive mood dysregulation disorder is chronic, severe persistent irritability. This severe irritability has two prominent clinical manifestations, the first of which is frequent temper outbursts. These outbursts typically occur in response to frustration and can be verbal or behavioral (the latter in the form of aggression against property, self, or others). They must occur frequently (i.e., on average, three or more times per week) (Criterion C) over at least 1 year in at least two settings (Criteria E and F), such as in the home and at school, and they must be developmentally inappropriate (Criterion B). The second manifestation of severe irritability consists of chronic, persistently irritable or angry mood that is present between the severe temper outbursts. This irritable or angry mood must be characteristic of the child, being present most of the day, nearly every day, and noticeable by others in the child's environment (Criterion D).

The clinical presentation of disruptive mood dysregulation disorder must be carefully distinguished from presentations of other, related conditions, particularly pediatric bipolar disorder. In fact, disruptive mood dysregulation disorder was added to DSM-5 to address the considerable concern about the appropriate classification and treatment of children who present with chronic, persistent irritability relative to children who present with classic (i.e., episodic) bipolar disorder.

Some researchers view severe, non-episodic irritability as characteristic of bipolar disorder in children, although both DSM-IV and DSM-5 require that both children and adults have distinct episodes of mania or hypomania to qualify for the diagnosis of bipolar I disorder. During the latter decades of the 20th century, this contention by researchers that severe, nonepisodic irritability is a manifestation of pediatric mania coincided with an upsurge in the rates at which clinicians assigned the diagnosis of bipolar disorder to their pediatric patients. This sharp increase in rates appears to be attributable to clinicians combining at least two clinical presentations into a single category. That is, both classic, episodic presentations of mania and non-episodic presentations of severe irritability have been labeled as bipolar disorder in children. In DSM-5, the term *bipolar disorder* is explicitly reserved for episodic presentations of bipolar symptoms. DSM-IV did not include a diagnosis designed to capture youths whose hallmark symptoms consisted of very severe, nonepisodic irritability, whereas DSM-5, with the inclusion of disruptive mood dysregulation disorder, provides a distinct category for such presentations.

Prevalence

Disruptive mood dysregulation disorder is common among children presenting to pediatric mental health clinics. Prevalence estimates of the disorder in the community are unclear. Based on rates of chronic and severe persistent irritability, which is the core feature of the disorder, the overall 6-month to 1-year period-prevalence of disruptive mood dysregulation disorder among children and adolescents probably falls in the 2%–5% range. However, rates are expected to be higher in males and school-age children than in females and adolescents.

Development and Course

The onset of disruptive mood dysregulation disorder must be before age 10 years, and the diagnosis should not be applied to children with a developmental age of less than 6 years. It is unknown whether the condition presents only in this age-delimited fashion. Because the symptoms of disruptive mood dysregulation disorder are likely to change as children mature, use of the diagnosis should be restricted to age groups similar to those in which validity has been established (7–18 years). Approximately half of children with severe, chronic irritability will have a presentation that continues to meet criteria for the condition 1 year later. Rates of conversion from severe, nonepisodic irritability to bipolar disorder are very low. Instead, children with chronic irritability are at risk to develop unipolar depressive and/or anxiety disorders in adulthood.

Age-related variations also differentiate classic bipolar disorder and disruptive mood dysregulation disorder. Rates of bipolar disorder generally are very low prior to adolescence (<1%), with a steady increase into early adulthood (1%–2% prevalence). Disruptive mood dysregulation disorder is more common than bipolar disorder prior to adolescence, and symptoms of the condition generally become less common as children transition into adulthood.

Risk and Prognostic Factors

Temperamental. Children with chronic irritability typically exhibit complicated psychiatric histories. In such children, a relatively extensive history of chronic irritability is

common, typically manifesting before full criteria for the syndrome are met. Such prediagnostic presentations may have qualified for a diagnosis of oppositional defiant disorder. Many children with disruptive mood dysregulation disorder have symptoms that also meet criteria for attention-deficit/hyperactivity disorder (ADHD) and for an anxiety disorder, with such diagnoses often being present from a relatively early age. For some children, the criteria for major depressive disorder may also be met.

Genetic and physiological. In terms of familial aggregation and genetics, it has been suggested that children presenting with chronic, non-episodic irritability can be differentiated from children with bipolar disorder in their family-based risk. However, these two groups do not differ in familial rates of anxiety disorders, unipolar depressive disorders, or substance abuse. Compared with children with pediatric bipolar disorder or other mental illnesses, those with disruptive mood dysregulation disorder exhibit both commonalities and differences in information-processing deficits. For example, face-emotion labeling deficits, as well as perturbed decision making and cognitive control, are present in children with bipolar disorder and chronically irritable children, as well as in children with some other psychiatric conditions. There is also evidence for disorder-specific dysfunction, such as during tasks assessing attention deployment in response to emotional stimuli, which has demonstrated unique signs of dysfunction in children with chronic irritability.

Gender-Related Diagnostic Issues

Children presenting to clinics with features of disruptive mood dysregulation disorder are predominantly male. Among community samples, a male preponderance appears to be supported. This difference in prevalence between males and females differentiates disruptive mood dysregulation disorder from bipolar disorder, in which there is an equal gender prevalence.

Suicide Risk

In general, evidence documenting suicidal behavior and aggression, as well as other severe functional consequences, in disruptive mood dysregulation disorder should be noted when evaluating children with chronic irritability.

Functional Consequences of Disruptive Mood Dysregulation Disorder

Chronic, severe irritability, such as is seen in disruptive mood dysregulation disorder, is associated with marked disruption in a child's family and peer relationships, as well as in school performance. Because of their extremely low frustration tolerance, such children generally have difficulty succeeding in school; they are often unable to participate in the activities typically enjoyed by healthy children; their family life is severely disrupted by their outbursts and irritability; and they have trouble initiating or sustaining friendships. Levels of dysfunction in children with bipolar disorder and disruptive mood dysregulation disorder are generally comparable. Both conditions cause severe disruption in the lives of the affected individual and their families. In both disruptive mood dysregulation disorder and pediatric bipolar disorder, dangerous behavior, suicidal ideation or suicide attempts, severe aggression, and psychiatric hospitalization are common.

Differential Diagnosis

Because chronically irritable children and adolescents typically present with complex histories, the diagnosis of disruptive mood dysregulation disorder must be made while considering the presence or absence of multiple other conditions. Despite the need to consider

many other syndromes, differentiation of disruptive mood dysregulation disorder from bipolar disorder and oppositional defiant disorder requires particularly careful assessment.

Bipolar disorders. The central feature differentiating disruptive mood dysregulation disorder and bipolar disorders in children involves the longitudinal course of the core symptoms. In children, as in adults, bipolar I disorder and bipolar II disorder manifest as an episodic illness with discrete episodes of mood perturbation that can be differentiated from the child's typical presentation. The mood perturbation that occurs during a manic episode is distinctly different from the child's usual mood. In addition, during a manic episode, the change in mood must be accompanied by the onset, or worsening, of associated cognitive, behavioral, and physical symptoms (e.g., distractibility, increased goal-directed activity), which are also present to a degree that is distinctly different from the child's usual baseline. Thus, in the case of a manic episode, parents (and, depending on developmental level, children) should be able to identify a distinct time period during which the child's mood and behavior were markedly different from usual. In contrast, the irritability of disruptive mood dysregulation disorder is persistent and is present over many months; while it may wax and wane to a certain degree, severe irritability is characteristic of the child with disruptive mood dysregulation disorder. Thus, while bipolar disorders are episodic conditions, disruptive mood dysregulation disorder is not. In fact, the diagnosis of disruptive mood dysregulation disorder cannot be assigned to a child who has ever experienced a full-duration hypomanic or manic episode (irritable or euphoric) or who has ever had a manic or hypomanic episode lasting more than 1 day. Another central differentiating feature between bipolar disorders and disruptive mood dysregulation disorder is the presence of elevated or expansive mood and grandiosity. These symptoms are common features of mania but are not characteristic of disruptive mood dysregulation disorder.

Oppositional defiant disorder. While symptoms of oppositional defiant disorder typically do occur in children with disruptive mood dysregulation disorder, mood symptoms of disruptive mood dysregulation disorder are relatively rare in children with oppositional defiant disorder. The key features that warrant the diagnosis of disruptive mood dysregulation disorder in children whose symptoms also meet criteria for oppositional defiant disorder are the presence of severe and frequently recurrent outbursts and a persistent disruption in mood between outbursts. In addition, the diagnosis of disruptive mood dysregulation disorder requires severe impairment in at least one setting (i.e., home, school, or among peers) and mild to moderate impairment in a second setting. For this reason, while most children whose symptoms meet criteria for disruptive mood dysregulation disorder will also have a presentation that meets criteria for oppositional defiant disorder, the reverse is not the case. That is, in only approximately 15% of individuals with oppositional defiant disorder would criteria for disruptive mood dysregulation disorder be met. Moreover, even for children in whom criteria for both disorders are met, only the diagnosis of disruptive mood dysregulation disorder should be made. Finally, both the prominent mood symptoms in disruptive mood dysregulation disorder and the high risk for depressive and anxiety disorders in follow-up studies justify placement of disruptive mood dysregulation disorder among the depressive disorders in DSM-5. (Oppositional defiant disorder is included in the chapter "Disruptive, Impulse-Control, and Conduct Disorders.") This reflects the more prominent mood component among individuals with disruptive mood dysregulation disorder, as compared with individuals with oppositional defiant disorder. Nevertheless, it also should be noted that disruptive mood dysregulation disorder appears to carry a high risk for behavioral problems as well as mood problems.

Attention-deficit/hyperactivity disorder, major depressive disorder, anxiety disorders, and autism spectrum disorder. Unlike children diagnosed with bipolar disorder or oppositional defiant disorder, a child whose symptoms meet criteria for disruptive mood dysregulation disorder also can receive a comorbid diagnosis of ADHD, major depressive disorder, and/or anxiety disorder. However, children whose irritability is present only in the context of a major depressive episode or persistent depressive disorder (dysthymia)

should receive one of those diagnoses rather than disruptive mood dysregulation disorder. Children with disruptive mood dysregulation disorder may have symptoms that also meet criteria for an anxiety disorder and can receive both diagnoses, but children whose irritability is manifest only in the context of exacerbation of an anxiety disorder should receive the relevant anxiety disorder diagnosis rather than disruptive mood dysregulation disorder. In addition, children with autism spectrum disorders frequently present with temper outbursts when, for example, their routines are disturbed. In that instance, the temper outbursts would be considered secondary to the autism spectrum disorder, and the child should not receive the diagnosis of disruptive mood dysregulation disorder.

Intermittent explosive disorder. Children with symptoms suggestive of intermittent explosive disorder present with instances of severe temper outbursts, much like children with disruptive mood dysregulation disorder. However, unlike disruptive mood dysregulation disorder, intermittent explosive disorder does not require persistent disruption in mood between outbursts. In addition, intermittent explosive disorder requires only 3 months of active symptoms, in contrast to the 12-month requirement for disruptive mood dysregulation disorder. Thus, these two diagnoses should not be made in the same child. For children with outbursts and intercurrent, persistent irritability, only the diagnosis of disruptive mood dysregulation disorder should be made.

Comorbidity

Rates of comorbidity in disruptive mood dysregulation disorder are extremely high. It is rare to find individuals whose symptoms meet criteria for disruptive mood dysregulation disorder alone. Comorbidity between disruptive mood dysregulation disorder and other DSM-defined syndromes appears higher than for many other pediatric mental illnesses; the strongest overlap is with oppositional defiant disorder. Not only is the overall rate of comorbidity high in disruptive mood dysregulation disorder, but also the range of comorbid illnesses appears particularly diverse. These children typically present to the clinic with a wide range of disruptive behavior, mood, anxiety, and even autism spectrum symptoms and diagnoses. However, children with disruptive mood dysregulation disorder should not have symptoms that meet criteria for bipolar disorder, as in that context, only the bipolar disorder diagnosis should be made. If children have symptoms that meet criteria for oppositional defiant disorder or intermittent explosive disorder *and* disruptive mood dysregulation disorder, only the diagnosis of disruptive mood dysregulation disorder should be assigned. Also, as noted earlier, the diagnosis of disruptive mood dysregulation disorder should not be assigned if the symptoms occur only in an anxiety-provoking context, when the routines of a child with autism spectrum disorder or obsessive-compulsive disorder are disturbed, or in the context of a major depressive episode.

Major Depressive Disorder

Diagnostic Criteria

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

Note: Do not include symptoms that are clearly attributable to another medical condition.

1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad, empty, hopeless) or observation made by others (e.g., appears tearful). (**Note:** In children and adolescents, can be irritable mood.)
2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation).

3. Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. (**Note:** In children, consider failure to make expected weight gain.)
4. Insomnia or hypersomnia nearly every day.
5. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
6. Fatigue or loss of energy nearly every day.
7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The episode is not attributable to the physiological effects of a substance or to another medical condition.

Note: Criteria A–C represent a major depressive episode.

Note: Responses to a significant loss (e.g., bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability) may include the feelings of intense sadness, rumination about the loss, insomnia, poor appetite, and weight loss noted in Criterion A, which may resemble a depressive episode. Although such symptoms may be understandable or considered appropriate to the loss, the presence of a major depressive episode in addition to the normal response to a significant loss should also be carefully considered. This decision inevitably requires the exercise of clinical judgment based on the individual's history and the cultural norms for the expression of distress in the context of loss.[1]

D. The occurrence of the major depressive episode is not better explained by schizoaffective disorder, schizophrenia, schizophreniform disorder, delusional disorder, or other specified and unspecified schizophrenia spectrum and other psychotic disorders.

E. There has never been a manic episode or a hypomanic episode.

Note: This exclusion does not apply if all of the manic-like or hypomanic-like episodes are substance-induced or are attributable to the physiological effects of another medical condition.

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[1] In distinguishing grief from a major depressive episode (MDE), it is useful to consider that in grief the predominant affect is feelings of emptiness and loss, while in MDE it is persistent depressed mood and the inability to anticipate happiness or pleasure. The dysphoria in grief is likely to decrease in intensity over days to weeks and occurs in waves, the so-called pangs of grief. These waves tend to be associated with thoughts or reminders of the deceased. The depressed mood of MDE is more persistent and not tied to specific thoughts or preoccupations. The pain of grief may be accompanied by positive emotions and humor that are uncharacteristic of the pervasive unhappiness and misery characteristic of MDE. The thought content associated with grief generally features a preoccupation with thoughts and memories of the deceased, rather than the self-critical or pessimistic ruminations seen in MDE. In grief, self-esteem is generally preserved, whereas in MDE feelings of worthlessness and self-loathing are common. If self-derogatory ideation is present in grief, it typically involves perceived failings vis-à-vis the deceased (e.g., not visiting frequently enough, not telling the deceased how much he or she was loved). If a bereaved individual thinks about death and dying, such thoughts are generally focused on the deceased and possibly about "joining" the deceased, whereas in MDE such thoughts are focused on ending one's own life because of feeling worthless, undeserving of life, or unable to cope with the pain of depression.

Coding and Recording Procedures

The diagnostic code for major depressive disorder is based on whether this is a single or recurrent episode, current severity, presence of psychotic features, and remission status. Current severity and psychotic features are only indicated if full criteria are currently met for a major depressive episode. Remission specifiers are only indicated if the full criteria are not currently met for a major depressive episode. Codes are as follows:

Severity/course specifier	Single episode	Recurrent episode*
Mild (p. 188)	296.21 (F32.0)	296.31 (F33.0)
Moderate (p. 188)	296.22 (F32.1)	296.32 (F33.1)
Severe (p. 188)	296.23 (F32.2)	296.33 (F33.2)
With psychotic features** (p. 186)	296.24 (F32.3)	296.34 (F33.3)
In partial remission (p. 188)	296.25 (F32.4)	296.35 (F33.41)
In full remission (p. 188)	296.26 (F32.5)	296.36 (F33.42)
Unspecified	296.20 (F32.9)	296.30 (F33.9)

*For an episode to be considered recurrent, there must be an interval of at least 2 consecutive months between separate episodes in which criteria are not met for a major depressive episode. The definitions of specifiers are found on the indicated pages.

**If psychotic features are present, code the "with psychotic features" specifier irrespective of episode severity.

In recording the name of a diagnosis, terms should be listed in the following order: major depressive disorder, single or recurrent episode, severity/psychotic/remission specifiers, followed by as many of the following specifiers without codes that apply to the current episode.

Specify:

With anxious distress (p. 184)
With mixed features (pp. 184–185)
With melancholic features (p. 185)
With atypical features (pp. 185–186)
With mood-congruent psychotic features (p. 186)
With mood-incongruent psychotic features (p. 186)
With catatonia (p. 186). **Coding note:** Use additional code 293.89 (F06.1).
With peripartum onset (pp. 186–187)
With seasonal pattern (recurrent episode only) (pp. 187–188)

Diagnostic Features

The criterion symptoms for major depressive disorder must be present nearly every day to be considered present, with the exception of weight change and suicidal ideation. Depressed mood must be present for most of the day, in addition to being present nearly every day. Often insomnia or fatigue is the presenting complaint, and failure to probe for accompanying depressive symptoms will result in underdiagnosis. Sadness may be denied at first but may be elicited through interview or inferred from facial expression and demeanor. With individuals who focus on a somatic complaint, clinicians should determine whether the distress from that complaint is associated with specific depressive symptoms. Fatigue and sleep disturbance are present in a high proportion of cases; psychomotor disturbances are much less common but are indicative of greater overall severity, as is the presence of delusional or near-delusional guilt.

The essential feature of a major depressive episode is a period of at least 2 weeks during which there is either depressed mood or the loss of interest or pleasure in nearly all activities (Criterion A). In children and adolescents, the mood may be irritable rather than sad. The individual must also experience at least four additional symptoms drawn from a list that includes changes in appetite or weight, sleep, and psychomotor activity; decreased energy; feelings of worthlessness or guilt; difficulty thinking, concentrating, or making decisions; or recurrent thoughts of death or suicidal ideation or suicide plans or attempts. To count toward a major depressive episode, a symptom must either be newly present or must have clearly worsened compared with the person's pre-episode status. The symptoms must persist for most of the day, nearly every day, for at least 2 consecutive weeks. The episode must be accompanied by clinically significant distress or impairment in social, occupational, or other important areas of functioning. For some individuals with milder episodes, functioning may appear to be normal but requires markedly increased effort.

The mood in a major depressive episode is often described by the person as depressed, sad, hopeless, discouraged, or "down in the dumps" (Criterion A1). In some cases, sadness may be denied at first but may subsequently be elicited by interview (e.g., by pointing out that the individual looks as if he or she is about to cry). In some individuals who complain of feeling "blah," having no feelings, or feeling anxious, the presence of a depressed mood can be inferred from the person's facial expression and demeanor. Some individuals emphasize somatic complaints (e.g., bodily aches and pains) rather than reporting feelings of sadness. Many individuals report or exhibit increased irritability (e.g., persistent anger, a tendency to respond to events with angry outbursts or blaming others, an exaggerated sense of frustration over minor matters). In children and adolescents, an irritable or cranky mood may develop rather than a sad or dejected mood. This presentation should be differentiated from a pattern of irritability when frustrated.

Loss of interest or pleasure is nearly always present, at least to some degree. Individuals may report feeling less interested in hobbies, "not caring anymore," or not feeling any enjoyment in activities that were previously considered pleasurable (Criterion A2). Family members often notice social withdrawal or neglect of pleasurable avocations (e.g., a formerly avid golfer no longer plays, a child who used to enjoy soccer finds excuses not to practice). In some individuals, there is a significant reduction from previous levels of sexual interest or desire.

Appetite change may involve either a reduction or increase. Some depressed individuals report that they have to force themselves to eat. Others may eat more and may crave specific foods (e.g., sweets or other carbohydrates). When appetite changes are severe (in either direction), there may be a significant loss or gain in weight, or, in children, a failure to make expected weight gains may be noted (Criterion A3).

Sleep disturbance may take the form of either difficulty sleeping or sleeping excessively (Criterion A4). When insomnia is present, it typically takes the form of middle insomnia (i.e., waking up during the night and then having difficulty returning to sleep) or terminal insomnia (i.e., waking too early and being unable to return to sleep). Initial insomnia (i.e., difficulty falling asleep) may also occur. Individuals who present with oversleeping (hypersomnia) may experience prolonged sleep episodes at night or increased daytime sleep. Sometimes the reason that the individual seeks treatment is for the disturbed sleep.

Psychomotor changes include agitation (e.g., the inability to sit still, pacing, handwringing; or pulling or rubbing of the skin, clothing, or other objects) or retardation (e.g., slowed speech, thinking, and body movements; increased pauses before answering; speech that is decreased in volume, inflection, amount, or variety of content, or muteness) (Criterion A5). The psychomotor agitation or retardation must be severe enough to be observable by others and not represent merely subjective feelings.

Decreased energy, tiredness, and fatigue are common (Criterion A6). A person may report sustained fatigue without physical exertion. Even the smallest tasks seem to require

substantial effort. The efficiency with which tasks are accomplished may be reduced. For example, an individual may complain that washing and dressing in the morning are exhausting and take twice as long as usual.

The sense of worthlessness or guilt associated with a major depressive episode may include unrealistic negative evaluations of one's worth or guilty preoccupations or ruminations over minor past failings (Criterion A7). Such individuals often misinterpret neutral or trivial day-to-day events as evidence of personal defects and have an exaggerated sense of responsibility for untoward events. The sense of worthlessness or guilt may be of delusional proportions (e.g., an individual who is convinced that he or she is personally responsible for world poverty). Blaming oneself for being sick and for failing to meet occupational or interpersonal responsibilities as a result of the depression is very common and, unless delusional, is not considered sufficient to meet this criterion.

Many individuals report impaired ability to think, concentrate, or make even minor decisions (Criterion A8). They may appear easily distracted or complain of memory difficulties. Those engaged in cognitively demanding pursuits are often unable to function. In children, a precipitous drop in grades may reflect poor concentration. In elderly individuals, memory difficulties may be the chief complaint and may be mistaken for early signs of a dementia ("pseudodementia"). When the major depressive episode is successfully treated, the memory problems often fully abate. However, in some individuals, particularly elderly persons, a major depressive episode may sometimes be the initial presentation of an irreversible dementia.

Thoughts of death, suicidal ideation, or suicide attempts (Criterion A9) are common. They may range from a passive wish not to awaken in the morning or a belief that others would be better off if the individual were dead, to transient but recurrent thoughts of committing suicide, to a specific suicide plan. More severely suicidal individuals may have put their affairs in order (e.g., updated wills, settled debts), acquired needed materials (e.g., a rope or a gun), and chosen a location and time to accomplish the suicide. Motivations for suicide may include a desire to give up in the face of perceived insurmountable obstacles, an intense wish to end what is perceived as an unending and excruciatingly painful emotional state, an inability to foresee any enjoyment in life, or the wish to not be a burden to others. The resolution of such thinking may be a more meaningful measure of diminished suicide risk than denial of further plans for suicide.

The evaluation of the symptoms of a major depressive episode is especially difficult when they occur in an individual who also has a general medical condition (e.g., cancer, stroke, myocardial infarction, diabetes, pregnancy). Some of the criterion signs and symptoms of a major depressive episode are identical to those of general medical conditions (e.g., weight loss with untreated diabetes; fatigue with cancer; hypersomnia early in pregnancy; insomnia later in pregnancy or the postpartum). Such symptoms count toward a major depressive diagnosis except when they are clearly and fully attributable to a general medical condition. Nonvegetative symptoms of dysphoria, anhedonia, guilt or worthlessness, impaired concentration or indecision, and suicidal thoughts should be assessed with particular care in such cases. Definitions of major depressive episodes that have been modified to include only these nonvegetative symptoms appear to identify nearly the same individuals as do the full criteria.

Associated Features Supporting Diagnosis

Major depressive disorder is associated with high mortality, much of which is accounted for by suicide; however, it is not the only cause. For example, depressed individuals admitted to nursing homes have a markedly increased likelihood of death in the first year. Individuals frequently present with tearfulness, irritability, brooding, obsessive rumination, anxiety, phobias, excessive worry over physical health, and complaints of pain (e.g., headaches; joint, abdominal, or other pains). In children, separation anxiety may occur.

Although an extensive literature exists describing neuroanatomical, neuroendocrinological, and neurophysiological correlates of major depressive disorder, no laboratory test has yielded results of sufficient sensitivity and specificity to be used as a diagnostic tool for this disorder. Until recently, hypothalamic-pituitary-adrenal axis hyperactivity had been the most extensively investigated abnormality associated with major depressive episodes, and it appears to be associated with melancholia, psychotic features, and risks for eventual suicide. Molecular studies have also implicated peripheral factors, including genetic variants in neurotrophic factors and pro-inflammatory cytokines. Additionally, functional magnetic resonance imaging studies provide evidence for functional abnormalities in specific neural systems supporting emotion processing, reward seeking, and emotion regulation in adults with major depression.

Prevalence

Twelve-month prevalence of major depressive disorder in the United States is approximately 7%, with marked differences by age group such that the prevalence in 18- to 29-year-old individuals is threefold higher than the prevalence in individuals age 60 years or older. Females experience 1.5- to 3-fold higher rates than males beginning in early adolescence.

Development and Course

Major depressive disorder may first appear at any age, but the likelihood of onset increases markedly with puberty. In the United States, incidence appears to peak in the 20s; however, first onset in late life is not uncommon.

The course of major depressive disorder is quite variable, such that some individuals rarely, if ever, experience remission (a period of 2 or more months with no symptoms, or only one or two symptoms to no more than a mild degree), while others experience many years with few or no symptoms between discrete episodes. It is important to distinguish individuals who present for treatment during an exacerbation of a chronic depressive illness from those whose symptoms developed recently. Chronicity of depressive symptoms substantially increases the likelihood of underlying personality, anxiety, and substance use disorders and decreases the likelihood that treatment will be followed by full symptom resolution. It is therefore useful to ask individuals presenting with depressive symptoms to identify the last period of at least 2 months during which they were entirely free of depressive symptoms.

Recovery typically begins within 3 months of onset for two in five individuals with major depression and within 1 year for four in five individuals. Recency of onset is a strong determinant of the likelihood of near-term recovery, and many individuals who have been depressed only for several months can be expected to recover spontaneously. Features associated with lower recovery rates, other than current episode duration, include psychotic features, prominent anxiety, personality disorders, and symptom severity.

The risk of recurrence becomes progessively lower over time as the duration of remission increases. The risk is higher in individuals whose preceding episode was severe, in younger individuals, and in individuals who have already experienced multiple episodes. The persistence of even mild depressive symptoms during remission is a powerful predictor of recurrence.

Many bipolar illnesses begin with one or more depressive episodes, and a substantial proportion of individuals who initially appear to have major depressive disorder will prove, in time, to instead have a bipolar disorder. This is more likely in individuals with onset of the illness in adolescence, those with psychotic features, and those with a family history of bipolar illness. The presence of a "with mixed features" specifier also increases the risk for future manic or hypomanic diagnosis. Major depressive disorder, particularly with psychotic features, may also transition into schizophrenia, a change that is much more frequent than the reverse.

Despite consistent differences between genders in prevalence rates for depressive disorders, there appear to be no clear differences by gender in phenomenology, course, or treatment response. Similarly, there are no clear effects of current age on the course or treatment response of major depressive disorder. Some symptom differences exist, though, such that hypersomnia and hyperphagia are more likely in younger individuals, and melancholic symptoms, particularly psychomotor disturbances, are more common in older individuals. The likelihood of suicide attempts lessens in middle and late life, although the risk of completed suicide does not. Depressions with earlier ages at onset are more familial and more likely to involve personality disturbances. The course of major depressive disorder within individuals does not generally change with aging. Mean times to recovery appear to be stable over long periods, and the likelihood of being in an episode does not generally increase or decrease with time.

Risk and Prognostic Factors

Temperamental. Neuroticism (negative affectivity) is a well-established risk factor for the onset of major depressive disorder, and high levels appear to render individuals more likely to develop depressive episodes in response to stressful life events.

Environmental. Adverse childhood experiences, particularly when there are multiple experiences of diverse types, constitute a set of potent risk factors for major depressive disorder. Stressful life events are well recognized as precipitants of major depressive episodes, but the presence or absence of adverse life events near the onset of episodes does not appear to provide a useful guide to prognosis or treatment selection.

Genetic and physiological. First-degree family members of individuals with major depressive disorder have a risk for major depressive disorder two- to fourfold higher than that of the general population. Relative risks appear to be higher for early-onset and recurrent forms. Heritability is approximately 40%, and the personality trait neuroticism accounts for a substantial portion of this genetic liability.

Course modifiers. Essentially all major nonmood disorders increase the risk of an individual developing depression. Major depressive episodes that develop against the background of another disorder often follow a more refractory course. Substance use, anxiety, and borderline personality disorders are among the most common of these, and the presenting depressive symptoms may obscure and delay their recognition. However, sustained clinical improvement in depressive symptoms may depend on the appropriate treatment of underlying illnesses. Chronic or disabling medical conditions also increase risks for major depressive episodes. Such prevalent illnesses as diabetes, morbid obesity, and cardiovascular disease are often complicated by depressive episodes, and these episodes are more likely to become chronic than are depressive episodes in medically healthy individuals.

Culture-Related Diagnostic Issues

Surveys of major depressive disorder across diverse cultures have shown sevenfold differences in 12-month prevalence rates but much more consistency in female-to-male ratio, mean ages at onset, and the degree to which presence of the disorder raises the likelihood of comorbid substance abuse. While these findings suggest substantial cultural differences in the expression of major depressive disorder, they do not permit simple linkages between particular cultures and the likelihood of specific symptoms. Rather, clinicians should be aware that in most countries the majority of cases of depression go unrecognized in primary care settings and that in many cultures, somatic symptoms are very likely to constitute the presenting complaint. Among the Criterion A symptoms, insomnia and loss of energy are the most uniformly reported.

Gender-Related Diagnostic Issues

Although the most reproducible finding in the epidemiology of major depressive disorder has been a higher prevalence in females, there are no clear differences between genders in symptoms, course, treatment response, or functional consequences. In women, the risk for suicide attempts is higher, and the risk for suicide completion is lower. The disparity in suicide rate by gender is not as great among those with depressive disorders as it is in the population as a whole.

Suicide Risk

The possibility of suicidal behavior exists at all times during major depressive episodes. The most consistently described risk factor is a past history of suicide attempts or threats, but it should be remembered that most completed suicides are not preceded by unsuccessful attempts. Other features associated with an increased risk for completed suicide include male sex, being single or living alone, and having prominent feelings of hopelessness. The presence of borderline personality disorder markedly increases risk for future suicide attempts.

Functional Consequences of Major Depressive Disorder

Many of the functional consequences of major depressive disorder derive from individual symptoms. Impairment can be very mild, such that many of those who interact with the affected individual are unaware of depressive symptoms. Impairment may, however, range to complete incapacity such that the depressed individual is unable to attend to basic self-care needs or is mute or catatonic. Among individuals seen in general medical settings, those with major depressive disorder have more pain and physical illness and greater decreases in physical, social, and role functioning.

Differential Diagnosis

Manic episodes with irritable mood or mixed episodes. Major depressive episodes with prominent irritable mood may be difficult to distinguish from manic episodes with irritable mood or from mixed episodes. This distinction requires a careful clinical evaluation of the presence of manic symptoms.

Mood disorder due to another medical condition. A major depressive episode is the appropriate diagnosis if the mood disturbance is not judged, based on individual history, physical examination, and laboratory findings, to be the direct pathophysiological consequence of a specific medical condition (e.g., multiple sclerosis, stroke, hypothyroidism).

Substance/medication-induced depressive or bipolar disorder. This disorder is distinguished from major depressive disorder by the fact that a substance (e.g., a drug of abuse, a medication, a toxin) appears to be etiologically related to the mood disturbance. For example, depressed mood that occurs only in the context of withdrawal from cocaine would be diagnosed as cocaine-induced depressive disorder.

Attention-deficit/hyperactivity disorder. Distractibility and low frustration tolerance can occur in both attention-deficit/ hyperactivity disorder and a major depressive episode; if the criteria are met for both, attention-deficit/hyperactivity disorder may be diagnosed in addition to the mood disorder. However, the clinician must be cautious not to overdiagnose a major depressive episode in children with attention-deficit/hyperactivity disorder whose disturbance in mood is characterized by irritability rather than by sadness or loss of interest.

Adjustment disorder with depressed mood. A major depressive episode that occurs in response to a psychosocial stressor is distinguished from adjustment disorder with depressed mood by the fact that the full criteria for a major depressive episode are not met in adjustment disorder.

Sadness. Finally, periods of sadness are inherent aspects of the human experience. These periods should not be diagnosed as a major depressive episode unless criteria are met for severity (i.e., five out of nine symptoms), duration (i.e., most of the day, nearly every day for at least 2 weeks), and clinically significant distress or impairment. The diagnosis other specified depressive disorder may be appropriate for presentations of depressed mood with clinically significant impairment that do not meet criteria for duration or severity.

Comorbidity

Other disorders with which major depressive disorder frequently co-occurs are substance-related disorders, panic disorder, obsessive-compulsive disorder, anorexia nervosa, bulimia nervosa, and borderline personality disorder.

Persistent Depressive Disorder (Dysthymia)

Diagnostic Criteria **300.4 (F34.1)**

This disorder represents a consolidation of DSM-IV-defined chronic major depressive disorder and dysthymic disorder.

A. Depressed mood for most of the day, for more days than not, as indicated by either subjective account or observation by others, for at least 2 years.

   Note: In children and adolescents, mood can be irritable and duration must be at least 1 year.

B. Presence, while depressed, of two (or more) of the following:

   1. Poor appetite or overeating.
   2. Insomnia or hypersomnia.
   3. Low energy or fatigue.
   4. Low self-esteem.
   5. Poor concentration or difficulty making decisions.
   6. Feelings of hopelessness.

C. During the 2-year period (1 year for children or adolescents) of the disturbance, the individual has never been without the symptoms in Criteria A and B for more than 2 months at a time.

D. Criteria for a major depressive disorder may be continuously present for 2 years.

E. There has never been a manic episode or a hypomanic episode, and criteria have never been met for cyclothymic disorder.

F. The disturbance is not better explained by a persistent schizoaffective disorder, schizophrenia, delusional disorder, or other specified or unspecified schizophrenia spectrum and other psychotic disorder.

G. The symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g. hypothyroidism).

H. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Note: Because the criteria for a major depressive episode include four symptoms that are absent from the symptom list for persistent depressive disorder (dysthymia), a very limited

number of individuals will have depressive symptoms that have persisted longer than 2 years but will not meet criteria for persistent depressive disorder. If full criteria for a major depressive episode have been met at some point during the current episode of illness, they should be given a diagnosis of major depressive disorder. Otherwise, a diagnosis of other specified depressive disorder or unspecified depressive disorder is warranted.

Specify if:

With anxious distress (p. 184)
With mixed features (pp. 184–185)
With melancholic features (p. 185)
With atypical features (pp. 185–186)
With mood-congruent psychotic features (p. 186)
With mood-incongruent psychotic features (p. 186)
With peripartum onset (pp. 186–187)

Specify if:

In partial remission (p. 188)
In full remission (p. 188)

Specify if:

Early onset: If onset is before age 21 years.
Late onset: If onset is at age 21 years or older.

Specify if (for most recent 2 years of persistent depressive disorder):

With pure dysthymic syndrome: Full criteria for a major depressive episode have not been met in at least the preceding 2 years.
With persistent major depressive episode: Full criteria for a major depressive episode have been met throughout the preceding 2-year period.
With intermittent major depressive episodes, with current episode: Full criteria for a major depressive episode are currently met, but there have been periods of at least 8 weeks in at least the preceding 2 years with symptoms below the threshold for a full major depressive episode.
With intermittent major depressive episodes, without current episode: Full criteria for a major depressive episode are not currently met, but there has been one or more major depressive episodes in at least the preceding 2 years.

Specify current severity:

Mild (p. 188)
Moderate (p. 188)
Severe (p. 188)

Diagnostic Features

The essential feature of persistent depressive disorder (dysthymia) is a depressed mood that occurs for most of the day, for more days than not, for at least 2 years, or at least 1 year for children and adolescents (Criterion A). This disorder represents a consolidation of DSM-IV-defined chronic major depressive disorder and dysthymic disorder. Major depression may precede persistent depressive disorder, and major depressive episodes may occur during persistent depressive disorder. Individuals whose symptoms meet major depressive disorder criteria for 2 years should be given a diagnosis of persistent depressive disorder as well as major depressive disorder.

Individuals with persistent depressive disorder describe their mood as sad or "down in the dumps." During periods of depressed mood, at least two of the six symptoms from Criterion B are present. Because these symptoms have become a part of the individual's day-to-day experience, particularly in the case of early onset (e.g., "I've always been this

way"), they may not be reported unless the individual is directly prompted. During the 2-year period (1 year for children or adolescents), any symptom-free intervals last no longer than 2 months (Criterion C).

Prevalence

Persistent depressive disorder is effectively an amalgam of DSM-IV dysthymic disorder and chronic major depressive episode. The 12-month prevalence in the United States is approximately 0.5% for persistent depressive disorder and 1.5% for chronic major depressive disorder.

Development and Course

Persistent depressive disorder often has an early and insidious onset (i.e., in childhood, adolescence, or early adult life) and, by definition, a chronic course. Among individuals with both persistent depressive disorder and borderline personality disorder, the covariance of the corresponding features over time suggests the operation of a common mechanism. Early onset (i.e., before age 21 years) is associated with a higher likelihood of comorbid personality disorders and substance use disorders.

When symptoms rise to the level of a major depressive episode, they are likely to subsequently revert to a lower level. However, depressive symptoms are much less likely to resolve in a given period of time in the context of persistent depressive disorder than they are in a major depressive episode.

Risk and Prognostic Factors

Temperamental. Factors predictive of poorer long-term outcome include higher levels of neuroticism (negative affectivity), greater symptom severity, poorer global functioning, and presence of anxiety disorders or conduct disorder.

Environmental. Childhood risk factors include parental loss or separation.

Genetic and physiological. There are no clear differences in illness development, course, or family history between DSM-IV dysthymic disorder and chronic major depressive disorder. Earlier findings pertaining to either disorder are therefore likely to apply to persistent depressive disorder. It is thus likely that individuals with persistent depressive disorder will have a higher proportion of first-degree relatives with persistent depressive disorder than do individuals with major depressive disorder, and more depressive disorders in general.

A number of brain regions (e.g., prefrontal cortex, anterior cingulate, amygdala, hippocampus) have been implicated in persistent depressive disorder. Possible polysomnographic abnormalities exist as well.

Functional Consequences of Persistent Depressive Disorder

The degree to which persistent depressive disorder impacts social and occupational functioning is likely to vary widely, but effects can be as great as or greater than those of major depressive disorder.

Differential Diagnosis

Major depressive disorder. If there is a depressed mood plus two or more symptoms meeting criteria for a persistent depressive episode for 2 years or more, then the diagnosis of persistent depressive disorder is made. The diagnosis depends on the 2-year duration, which distinguishes it from episodes of depression that do not last 2 years. If the symptom

criteria are sufficient for a diagnosis of a major depressive episode at any time during this period, then the diagnosis of major depression should be noted, but it is coded not as a separate diagnosis but rather as a specifier with the diagnosis of persistent depressive disorder. If the individual's symptoms currently meet full criteria for a major depressive episode, then the specifier of "with intermittent major depressive episodes, with current episode" would be made. If the major depressive episode has persisted for at least a 2-year duration and remains present, then the specifier "with persistent major depressive episode" is used. When full major depressive episode criteria are not currently met but there has been at least one previous episode of major depression in the context of at least 2 years of persistent depressive symptoms, then the specifier of "with intermittent major depressive episodes, without current episode" is used. If the individual has not experienced an episode of major depression in the last 2 years, then the specifier "with pure dysthymic syndrome" is used.

Psychotic disorders. Depressive symptoms are a common associated feature of chronic psychotic disorders (e.g., schizoaffective disorder, schizophrenia, delusional disorder). A separate diagnosis of persistent depressive disorder is not made if the symptoms occur only during the course of the psychotic disorder (including residual phases).

Depressive or bipolar and related disorder due to another medical condition. Persistent depressive disorder must be distinguished from a depressive or bipolar and related disorder due to another medical condition. The diagnosis is depressive or bipolar and related disorder due to another medical condition if the mood disturbance is judged, based on history, physical examination, or laboratory findings, to be attributable to the direct pathophysiological effects of a specific, usually chronic, medical condition (e.g., multiple sclerosis). If it is judged that the depressive symptoms are not attributable to the physiological effects of another medical condition, then the primary mental disorder (e.g., persistent depressive disorder) is recorded, and the medical condition is noted as a concomitant medical condition (e.g., diabetes mellitus).

Substance/medication-induced depressive or bipolar disorder. A substance/medication-induced depressive or bipolar and related disorder is distinguished from persistent depressive disorder when a substance (e.g., a drug of abuse, a medication, a toxin) is judged to be etiologically related to the mood disturbance.

Personality disorders. Often, there is evidence of a coexisting personality disturbance. When an individual's presentation meets the criteria for both persistent depressive disorder and a personality disorder, both diagnoses are given.

Comorbidity

In comparison to individuals with major depressive disorder, those with persistent depressive disorder are at higher risk for psychiatric comorbidity in general, and for anxiety disorders and substance use disorders in particular. Early-onset persistent depressive disorder is strongly associated with DSM-IV Cluster B and C personality disorders.

Premenstrual Dysphoric Disorder

Diagnostic Criteria **625.4 (N94.3)**

A. In the majority of menstrual cycles, at least five symptoms must be present in the final week before the onset of menses, start to *improve* within a few days after the onset of menses, and become *minimal* or absent in the week postmenses.

B. One (or more) of the following symptoms must be present:

   1. Marked affective lability (e.g., mood swings; feeling suddenly sad or tearful, or increased sensitivity to rejection).

2. Marked irritability or anger or increased interpersonal conflicts.
3. Marked depressed mood, feelings of hopelessness, or self-deprecating thoughts.
4. Marked anxiety, tension, and/or feelings of being keyed up or on edge.

C. One (or more) of the following symptoms must additionally be present, to reach a total of *five* symptoms when combined with symptoms from Criterion B above.

1. Decreased interest in usual activities (e.g., work, school, friends, hobbies).
2. Subjective difficulty in concentration.
3. Lethargy, easy fatigability, or marked lack of energy.
4. Marked change in appetite; overeating; or specific food cravings.
5. Hypersomnia or insomnia.
6. A sense of being overwhelmed or out of control.
7. Physical symptoms such as breast tenderness or swelling, joint or muscle pain, a sensation of "bloating," or weight gain.

Note: The symptoms in Criteria A–C must have been met for most menstrual cycles that occurred in the preceding year.

D. The symptoms are associated with clinically significant distress or interference with work, school, usual social activities, or relationships with others (e.g., avoidance of social activities; decreased productivity and efficiency at work, school, or home).

E. The disturbance is not merely an exacerbation of the symptoms of another disorder, such as major depressive disorder, panic disorder, persistent depressive disorder (dysthymia), or a personality disorder (although it may co-occur with any of these disorders).

F. Criterion A should be confirmed by prospective daily ratings during at least two symptomatic cycles. (**Note:** The diagnosis may be made provisionally prior to this confirmation.)

G. The symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication, other treatment) or another medical condition (e.g., hyperthyroidism).

Recording Procedures

If symptoms have not been confirmed by prospective daily ratings of at least two symptomatic cycles, "provisional" should be noted after the name of the diagnosis (i.e., "premenstrual dysphoric disorder, provisional").

Diagnostic Features

The essential features of premenstrual dysphoric disorder are the expression of mood lability, irritability, dysphoria, and anxiety symptoms that occur repeatedly during the premenstrual phase of the cycle and remit around the onset of menses or shortly thereafter. These symptoms may be accompanied by behavioral and physical symptoms. Symptoms must have occurred in most of the menstrual cycles during the past year and must have an adverse effect on work or social functioning. The intensity and/or expressivity of the accompanying symptoms may be closely related to social and cultural background characteristics of the affected female, family perspectives, and more specific factors such as religious beliefs, social tolerance, and female gender role issues.

Typically, symptoms peak around the time of the onset of menses. Although it is not uncommon for symptoms to linger into the first few days of menses, the individual must have a symptom-free period in the follicular phase after the menstrual period begins. While the core symptoms include mood and anxiety symptoms, behavioral and somatic symptoms commonly also occur. However, the presence of physical and/or behavioral symptoms in the absence of mood and/or anxious symptoms is not sufficient for a diag-

nosis. Symptoms are of comparable severity (but not duration) to those of another mental disorder, such as a major depressive episode or generalized anxiety disorder. In order to confirm a provisional diagnosis, daily prospective symptom ratings are required for at least two symptomatic cycles.

Associated Features Supporting Diagnosis

Delusions and hallucinations have been described in the late luteal phase of the menstrual cycle but are rare. The premenstrual phase has been considered by some to be a risk period for suicide.

Prevalence

Twelve-month prevalence of premenstrual dysphoric disorder is between 1.8% and 5.8% of menstruating women. Estimates are substantially inflated if they are based on retrospective reports rather than prospective daily ratings. However, estimated prevalence based on a daily record of symptoms for 1–2 months may be less representative, as individuals with the most severe symptoms may be unable to sustain the rating process. The most rigorous estimate of premenstrual dysphoric disorder is 1.8% for women whose symptoms meet the full criteria without functional impairment and 1.3% for women whose symptoms meet the current criteria with functional impairment and without co-occurring symptoms from another mental disorder.

Development and Course

Onset of premenstrual dysphoric disorder can occur at any point after menarche. Incidence of new cases over a 40-month follow-up period is 2.5% (95% confidence interval = 1.7–3.7). Anecdotally, many individuals, as they approach menopause, report that symptoms worsen. Symptoms cease after menopause, although cyclical hormone replacement can trigger the re-expression of symptoms.

Risk and Prognostic Factors

Environmental. Environmental factors associated with the expression of premenstrual dysphoric disorder include stress, history of interpersonal trauma, seasonal changes, and sociocultural aspects of female sexual behavior in general, and female gender role in particular.

Genetic and physiological. Heritability of premenstrual dysphoric disorder is unknown. However, for premenstrual symptoms, estimates for heritability range between 30% and 80%, with the most stable component of premenstrual symptoms estimated to be about 50% heritable.

Course modifiers. Women who use oral contraceptives may have fewer premenstrual complaints than do women who do not use oral contraceptives.

Culture-Related Diagnostic Issues

Premenstrual dysphoric disorder is not a culture-bound syndrome and has been observed in individuals in the United States, Europe, India, and Asia. It is unclear as to whether rates differ by race. Nevertheless, frequency, intensity, and expressivity of symptoms and help-seeking patterns may be significantly influenced by cultural factors.

Diagnostic Markers

As indicated earlier, the diagnosis of premenstrual dysphoric disorder is appropriately confirmed by 2 months of prospective symptom ratings. A number of scales, including the

Daily Rating of Severity of Problems and the Visual Analogue Scales for Premenstrual Mood Symptoms, have undergone validation and are commonly used in clinical trials for premenstrual dysphoric disorder. The Premenstrual Tension Syndrome Rating Scale has a self-report and an observer version, both of which have been validated and used widely to measure illness severity in women who have premenstrual dysphoric disorder.

Functional Consequences of Premenstrual Dysphoric Disorder

Symptoms must be associated with clinically meaningful distress and/or an obvious and marked impairment in the ability to function socially or occupationally in the week prior to menses. Impairment in social functioning may be manifested by marital discord and problems with children, other family members, or friends. Chronic marital or job problems should not be confused with dysfunction that occurs only in association with premenstrual dysphoric disorder.

Differential Diagnosis

Premenstrual syndrome. Premenstrual syndrome differs from premenstrual dysphoric disorder in that a minimum of five symptoms is not required, and there is no stipulation of affective symptoms for individuals who have premenstrual syndrome. This condition may be more common than premenstrual dysphoric disorder, although the estimated prevalence of premenstrual syndrome varies. While premenstrual syndrome shares the feature of symptom expression during the premenstrual phase of the menstrual cycle, it is generally considered to be less severe than premenstrual dysphoric disorder. The presence of physical or behavioral symptoms in the premenstruum, without the required affective symptoms, likely meets criteria for premenstrual syndrome and not for premenstrual dysphoric disorder.

Dysmenorrhea. Dysmenorrhea is a syndrome of painful menses, but this is distinct from a syndrome characterized by affective changes. Moreover, symptoms of dysmenorrhea begin with the onset of menses, whereas symptoms of premenstrual dysphoric disorder, by definition, begin before the onset of menses, even if they linger into the first few days of menses.

Bipolar disorder, major depressive disorder, and persistent depressive disorder (dysthymia). Many women with (either naturally occurring or substance/medication-induced) bipolar or major depressive disorder or persistent depressive disorder believe that they have premenstrual dysphoric disorder. However, when they chart symptoms, they realize that the symptoms do not follow a premenstrual pattern. Women with another mental disorder may experience chronic symptoms or intermittent symptoms that are unrelated to menstrual cycle phase. However, because the onset of menses constitutes a memorable event, they may report that symptoms occur only during the premenstruum or that symptoms worsen premenstrually. This is one of the rationales for the requirement that symptoms be confirmed by daily prospective ratings. The process of differential diagnosis, particularly if the clinician relies on retrospective symptoms only, is made more difficult because of the overlap between symptoms of premenstrual dysphoric disorder and some other diagnoses. The overlap of symptoms is particularly salient for differentiating premenstrual dysphoric disorder from major depressive episodes, persistent depressive disorder, bipolar disorders, and borderline personality disorder. However, the rate of personality disorders is no higher in individuals with premenstrual dysphoric disorder than in those without the disorder.

Use of hormonal treatments. Some women who present with moderate to severe premenstrual symptoms may be using hormonal treatments, including hormonal contraceptives. If such symptoms occur after initiation of exogenous hormone use, the symptoms

may be due to the use of hormones rather than to the underlying condition of premenstrual dysphoric disorder. If the woman stops hormones and the symptoms disappear, this is consistent with substance/medication-induced depressive disorder.

Comorbidity

A major depressive episode is the most frequently reported previous disorder in individuals presenting with premenstrual dysphoric disorder. A wide range of medical (e.g., migraine, asthma, allergies, seizure disorders) or other mental disorders (e.g., depressive and bipolar disorders, anxiety disorders, bulimia nervosa, substance use disorders) may worsen in the premenstrual phase; however, the absence of a symptom-free period during the postmenstrual interval obviates a diagnosis of premenstrual dysphoric disorder. These conditions are better considered premenstrual exacerbation of a current mental or medical disorder. Although the diagnosis of premenstrual dysphoric disorder should not be assigned in situations in which an individual only experiences a premenstrual exacerbation of another mental or physical disorder, it can be considered in addition to the diagnosis of another mental or physical disorder if the individual experiences symptoms and changes in level of functioning that are characteristic of premenstrual dysphoric disorder and markedly different from the symptoms experienced as part of the ongoing disorder.

Substance/Medication-Induced Depressive Disorder

Diagnostic Criteria

A. A prominent and persistent disturbance in mood that predominates in the clinical picture and is characterized by depressed mood or markedly diminished interest or pleasure in all, or almost all, activities.

B. There is evidence from the history, physical examination, or laboratory findings of both (1) and (2):

   1. The symptoms in Criterion A developed during or soon after substance intoxication or withdrawal or after exposure to a medication.
   2. The involved substance/medication is capable of producing the symptoms in Criterion A.

C. The disturbance is not better explained by a depressive disorder that is not substance/medication-induced. Such evidence of an independent depressive disorder could include the following:

   The symptoms preceded the onset of the substance/medication use; the symptoms persist for a substantial period of time (e.g., about 1 month) after the cessation of acute withdrawal or severe intoxication; or there is other evidence suggesting the existence of an independent non-substance/medication-induced depressive disorder (e.g., a history of recurrent non-substance/medication-related episodes).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Note: This diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and when they are sufficiently severe to warrant clinical attention.

Coding note: The ICD-9-CM and ICD-10-CM codes for the [specific substance/medication]-induced depressive disorders are indicated in the table below. Note that the ICD-10-

CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. If a mild substance use disorder is comorbid with the substance-induced depressive disorder, the 4th position character is "1," and the clinician should record "mild [substance] use disorder" before the substance-induced depressive disorder (e.g., "mild cocaine use disorder with cocaine-induced depressive disorder"). If a moderate or severe substance use disorder is comorbid with the substance-induced depressive disorder, the 4th position character is "2," and the clinician should record "moderate [substance] use disorder" or "severe [substance] use disorder," depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder (e.g., after a one-time heavy use of the substance), then the 4th position character is "9," and the clinician should record only the substance-induced depressive disorder.

		ICD-10-CM		
	ICD-9-CM	With use disorder, mild	With use disorder, moderate or severe	Without use disorder
Alcohol	291.89	F10.14	F10.24	F10.94
Phencyclidine	292.84	F16.14	F16.24	F16.94
Other hallucinogen	292.84	F16.14	F16.24	F16.94
Inhalant	292.84	F18.14	F18.24	F18.94
Opioid	292.84	F11.14	F11.24	F11.94
Sedative, hypnotic, or anxiolytic	292.84	F13.14	F13.24	F13.94
Amphetamine (or other stimulant)	292.84	F15.14	F15.24	F15.94
Cocaine	292.84	F14.14	F14.24	F14.94
Other (or unknown) substance	292.84	F19.14	F19.24	F19.94

Specify if (see Table 1 in the chapter "Substance-Related and Addictive Disorders" for diagnoses associated with substance class):

With onset during intoxication: If criteria are met for intoxication with the substance and the symptoms develop during intoxication.

With onset during withdrawal: If criteria are met for withdrawal from the substance and the symptoms develop during, or shortly after, withdrawal.

Recording Procedures

ICD-9-CM. The name of the substance/medication-induced depressive disorder begins with the specific substance (e.g., cocaine, dexamethasone) that is presumed to be causing the depressive symptoms. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class. For substances that do not fit into any of the classes (e.g., dexamethasone), the code for "other substance" should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category "unknown substance" should be used.

The name of the disorder is followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal). Unlike the recording procedures for ICD-10-CM, which combine the substance-induced disorder and substance use disorder into a single

code, for ICD-9-CM a separate diagnostic code is given for the substance use disorder. For example, in the case of depressive symptoms occurring during withdrawal in a man with a severe cocaine use disorder, the diagnosis is 292.84 cocaine-induced depressive disorder, with onset during withdrawal. An additional diagnosis of 304.20 severe cocaine use disorder is also given. When more than one substance is judged to play a significant role in the development of depressive mood symptoms, each should be listed separately (e.g., 292.84 methylphenidate-induced depressive disorder, with onset during withdrawal; 292.84 dexamethasone-induced depressive disorder, with onset during intoxication).

ICD-10-CM. The name of the substance/medication-induced depressive disorder begins with the specific substance (e.g., cocaine, dexamethasone) that is presumed to be causing the depressive symptoms. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class and presence or absence of a comorbid substance use disorder. For substances that do not fit into any of the classes (e.g., dexamethasone), the code for "other substance" should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category "unknown substance" should be used.

When recording the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by the word "with," followed by the name of the substance-induced depressive disorder, followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal). For example, in the case of depressive symptoms occurring during withdrawal in a man with a severe cocaine use disorder, the diagnosis is F14.24 severe cocaine use disorder with cocaine-induced depressive disorder, with onset during withdrawal. A separate diagnosis of the comorbid severe cocaine use disorder is not given. If the substance-induced depressive disorder occurs without a comorbid substance use disorder (e.g., after a one-time heavy use of the substance), no accompanying substance use disorder is noted (e.g., F16.94 phencyclidine-induced depressive disorder, with onset during intoxication). When more than one substance is judged to play a significant role in the development of depressive mood symptoms, each should be listed separately (e.g., F15.24 severe methylphenidate use disorder with methylphenidate-induced depressive disorder, with onset during withdrawal; F19.94 dexamethasone-induced depressive disorder, with onset during intoxication).

Diagnostic Features

The diagnostic features of substance/medication-induced depressive disorder include the symptoms of a depressive disorder, such as major depressive disorder; however, the depressive symptoms are associated with the ingestion, injection, or inhalation of a substance (e.g., drug of abuse, toxin, psychotropic medication, other medication), and the depressive symptoms persist beyond the expected length of physiological effects, intoxication, or withdrawal period. As evidenced by clinical history, physical examination, or laboratory findings, the relevant depressive disorder should have developed during or within 1 month after use of a substance that is capable of producing the depressive disorder (Criterion B1). In addition, the diagnosis is not better explained by an independent depressive disorder. Evidence of an independent depressive disorder includes the depressive disorder preceded the onset of ingestion or withdrawal from the substance; the depressive disorder persists beyond a substantial period of time after the cessation of substance use; or other evidence suggests the existence of an independent non-substance/medication-induced depressive disorder (Criterion C). This diagnosis should not be made when symptoms occur exclusively during the course of a delirium (Criterion D). The depressive disorder associated with the substance use, intoxication, or withdrawal must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning to qualify for this diagnosis (Criterion E).

Some medications (e.g., stimulants, steroids, L-dopa, antibiotics, central nervous system drugs, dermatological agents, chemotherapeutic drugs, immunological agents)

can induce depressive mood disturbances. Clinical judgment is essential to determine whether the medication is truly associated with inducing the depressive disorder or whether a primary depressive disorder happened to have its onset while the person was receiving the treatment. For example, a depressive episode that developed within the first several weeks of beginning alpha-methyldopa (an antihypertensive agent) in an individual with no history of major depressive disorder would qualify for the diagnosis of medication-induced depressive disorder. In some cases, a previously established condition (e.g., major depressive disorder, recurrent) can recur while the individual is coincidentally taking a medication that has the capacity to cause depressive symptoms (e.g., L-dopa, oral contraceptives). In such cases, the clinician must make a judgment as to whether the medication is causative in this particular situation.

A substance/medication-induced depressive disorder is distinguished from a primary depressive disorder by considering the onset, course, and other factors associated with the substance use. There must be evidence from the history, physical examination, or laboratory findings of substance use, abuse, intoxication, or withdrawal prior to the onset of the depressive disorder. The withdrawal state for some substances can be relatively protracted, and thus intense depressive symptoms can last for a long period after the cessation of substance use.

Prevalence

In a nationally representative U.S. adult population, the lifetime prevalence of substance/medication-induced depressive disorder is 0.26%.

Development and Course

A depressive disorder associated with the use of substance (i.e., alcohol, illicit drugs, or a prescribed treatment for a mental disorder or another medical condition) must have its onset while the individual is using the substance or during withdrawal, if there is a withdrawal syndrome associated with the substance. Most often, the depressive disorder has its onset within the first few weeks or 1 month of use of the substance. Once the substance is discontinued, the depressive symptoms usually remit within days to several weeks, depending on the half-life of the substance/medication and the presence of a withdrawal syndrome. If symptoms persist 4 weeks beyond the expected time course of withdrawal of a particular substance/medication, other causes for the depressive mood symptoms should be considered.

Although there are a few prospective controlled trials examining the association of depressive symptoms with use of a medication, most reports are from postmarketing surveillance studies, retrospective observational studies, or case reports, making evidence of causality difficult to determine. Substances implicated in medication-induced depressive disorder, with varying degrees of evidence, include antiviral agents (efavirenz), cardiovascular agents (clonidine, guanethidine, methyldopa, reserpine), retinoic acid derivatives (isotretinoin), antidepressants, anticonvulsants, anti-migraine agents (triptans), antipsychotics, hormonal agents (corticosteroids, oral contraceptives, gonadotropin-releasing hormone agonists, tamoxifen), smoking cessation agents (varenicline), and immunological agents (interferon). However, other potential substances continue to emerge as new compounds are synthesized. A history of such substance use may help increase diagnostic certainty.

Risk and Prognostic Factors

Temperamental. Factors that appear to increase the risk of substance/medication-induced depressive disorder can be conceptualized as pertaining to the specific type of drug or to a group of individuals with underlying alcohol or drug use disorders. Risk fac-

tors common to all drugs include history of major depressive disorder, history of drug-induced depression, and psychosocial stressors.

Environmental. There are also risks factors pertaining to a specific type of medication (e.g., increased immune activation prior to treatment for hepatitis C associated with interferon-alfa-induced depression); high doses (greater than 80 mg/day prednisone-equivalents) of corticosteroids or high plasma concentrations of efavirenz; and high estrogen/progesterone content in oral contraceptives.

Course modifiers. In a representative U.S. adult population, compared with individuals with major depressive disorder who did not have a substance use disorder, individuals with substance-induced depressive disorder were more likely to be male, to be black, to have at most a high school diploma, to lack insurance, and to have lower family income. They were also more likely to report higher family history of substance use disorders and antisocial behavior, higher 12-month history of stressful life events, and a greater number of DSM-IV major depressive disorder criteria. They were more likely to report feelings of worthlessness, insomnia/hypersomnia, and thoughts of death and suicide attempts, but less likely to report depressed mood and parental loss by death before age 18 years.

Diagnostic Markers

Determination of the substance of use can sometimes be made through laboratory assays of the suspected substance in the blood or urine to corroborate the diagnosis.

Suicide Risk

Drug-induced or treatment-emergent suicidality represents a marked change in thoughts and behavior from the person's baseline, is usually temporally associated with initiation of a substance, and must be distinguished from the underlying primary mental disorders.

In regard to the treatment-emergent suicidality associated with antidepressants, a U.S. Food and Drug Administration (FDA) advisory committee considered meta-analyses of 99,839 participants enrolled in 372 randomized clinical trials of antidepressants in trials for mental disorders. The analyses showed that when the data were pooled across all adult age groups, there was no perceptible increased risk of suicidal behavior or ideation. However, in age-stratified analyses, the risk for patients ages 18–24 years was elevated, albeit not significantly (odds ratio [OR] = 1.55; 95% confidence interval [CI] = 0.91–2.70). The FDA meta-analyses reveal an absolute risk of suicide in patients taking investigational antidepressants of 0.01%. In conclusion, suicide is clearly an extremely rare treatment-emergent phenomenon, but the outcome of suicide was serious enough to prompt the FDA to issue an expanded black-box warning in 2007 regarding the importance of careful monitoring of treatment-emergent suicidal ideation in patients receiving antidepressants.

Differential Diagnosis

Substance intoxication and withdrawal. Depressive symptoms occur commonly in substance intoxication and substance withdrawal, and the diagnosis of the substance-specific intoxication or withdrawal will usually suffice to categorize the symptom presentation. A diagnosis of substance-induced depressive disorder should be made instead of a diagnosis of substance intoxication or substance withdrawal when the mood symptoms are sufficiently severe to warrant independent clinical attention. For example, dysphoric mood is a characteristic feature of cocaine withdrawal. Substance/medication-induced depressive disorder should be diagnosed instead of cocaine withdrawal only if the mood disturbance is substantially more intense or longer lasting than what is usually encountered with cocaine withdrawal and is sufficiently severe to be a separate focus of attention and treatment.

Primary depressive disorder. A substance/medication-induced depressive disorder is distinguished from a primary depressive disorder by the fact that a substance is judged to be etiologically related to the symptoms, as described earlier (see section "Development and Course" for this disorder).

Depressive disorder due to another medical condition. Because individuals with other medical conditions often take medications for those conditions, the clinician must consider the possibility that the mood symptoms are caused by the physiological consequences of the medical condition rather than the medication, in which case depressive disorder due to another medical condition is diagnosed. The history often provides the primary basis for such a judgment. At times, a change in the treatment for the other medical condition (e.g., medication substitution or discontinuation) may be needed to determine empirically whether the medication is the causative agent. If the clinician has ascertained that the disturbance is a function of both another medical condition and substance use or withdrawal, both diagnoses (i.e., depressive disorder due to another medical condition and substance/medication-induced depressive disorder) may be given. When there is insufficient evidence to determine whether the depressive symptoms are associated with substance (including a medication) ingestion or withdrawal or with another medical condition or are primary (i.e., not a function of either a substance or another medical condition), a diagnosis of other specified depressive disorder or unspecified depressive disorder would be indicated.

Comorbidity

Compared with individuals with major depressive disorder and no comorbid substance use disorder, those with substance/medication-induced depressive disorder have higher rates of comorbidity with any DSM-IV mental disorder; are more likely to have specific DSM-IV disorders of pathological gambling and paranoid, histrionic, and antisocial personality disorders; and are less likely to have persistent depressive disorder (dysthymia). Compared with individuals with major depressive disorder and a comorbid substance use disorder, individuals with substance/medication-induced depressive disorder are more likely to have alcohol use disorder, any other substance use disorder, and histrionic personality disorder; however, they are less likely to have persistent depressive disorder.

Depressive Disorder Due to Another Medical Condition

Diagnostic Criteria

A. A prominent and persistent period of depressed mood or markedly diminished interest or pleasure in all, or almost all, activities that predominates in the clinical picture.
B. There is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct pathophysiological consequence of another medical condition.
C. The disturbance is not better explained by another mental disorder (e.g., adjustment disorder, with depressed mood, in which the stressor is a serious medical condition).
D. The disturbance does not occur exclusively during the course of a delirium.
E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Coding note: The ICD-9-CM code for depressive disorder due to another medical condition is **293.83,** which is assigned regardless of the specifier. The ICD-10-CM code depends on the specifier (see below).

Specify if:

(F06.31) With depressive features: Full criteria are not met for a major depressive episode.

(F06.32) With major depressive–like episode: Full criteria are met (except Criterion C) for a major depressive episode.

(F06.34) With mixed features: Symptoms of mania or hypomania are also present but do not predominate in the clinical picture.

Coding note: Include the name of the other medical condition in the name of the mental disorder (e.g., 293.83 [F06.31] depressive disorder due to hypothyroidism, with depressive features). The other medical condition should also be coded and listed separately immediately before the depressive disorder due to the medical condition (e.g., 244.9 [E03.9] hypothyroidism; 293.83 [F06.31] depressive disorder due to hypothyroidism, with depressive features).

Diagnostic Features

The essential feature of depressive disorder due to another medical condition is a prominent and persistent period of depressed mood or markedly diminished interest or pleasure in all, or almost all, activities that predominates in the clinical picture (Criterion A) and that is thought to be related to the direct physiological effects of another medical condition (Criterion B). In determining whether the mood disturbance is due to a general medical condition, the clinician must first establish the presence of a general medical condition. Further, the clinician must establish that the mood disturbance is etiologically related to the general medical condition through a physiological mechanism. A careful and comprehensive assessment of multiple factors is necessary to make this judgment. Although there are no infallible guidelines for determining whether the relationship between the mood disturbance and the general medical condition is etiological, several considerations provide some guidance in this area. One consideration is the presence of a temporal association between the onset, exacerbation, or remission of the general medical condition and that of the mood disturbance. A second consideration is the presence of features that are atypical of primary Mood Disorders (e.g., atypical age at onset or course or absence of family history). Evidence from the literature that suggests that there can be a direct association between the general medical condition in question and the development of mood symptoms can provide a useful context in the assessment of a particular situation.

Associated Features Supporting Diagnosis

Etiology (i.e., a causal relationship to another medical condition based on best clinical evidence) is the key variable in depressive disorder due to another medical condition. The listing of the medical conditions that are said to be able to induce major depression is never complete, and the clinician's best judgment is the essence of this diagnosis.

There are clear associations, as well as some neuroanatomical correlates, of depression with stroke, Huntington's disease, Parkinson's disease, and traumatic brain injury. Among the neuroendocrine conditions most closely associated with depression are Cushing's disease and hypothyroidism. There are numerous other conditions thought to be associated with depression, such as multiple sclerosis. However, the literature's support for a causal association is greater with some conditions, such as Parkinson's disease and Huntington's disease, than with others, for which the differential diagnosis may be adjustment disorder, with depressed mood.

Development and Course

Following stroke, the onset of depression appears to be very acute, occurring within 1 day or a few days of the cerebrovascular accident (CVA) in the largest case series. However, in

some cases, onset of the depression is weeks to months following the CVA. In the largest series, the duration of the major depressive episode following stroke was 9–11 months on average. Similarly, in Huntington's disease the depressive state comes quite early in the course of the illness. With Parkinson's disease and Huntington's disease, it often precedes the major motor impairments and cognitive impairments associated with each condition. This is more prominently the case for Huntington's disease, in which depression is considered to be the first neuropsychiatric symptom. There is some observational evidence that depression is less common as the dementia of Huntington's disease progresses.

Risk and Prognostic Factors

The risk of acute onset of a major depressive disorder following a CVA (within 1 day to a week of the event) appears to be strongly correlated with lesion location, with greatest risk associated with left frontal strokes and least risk apparently associated with right frontal lesions in those individuals who present within days of the stroke. The association with frontal regions and laterality is not observed in depressive states that occur in the 2–6 months following stroke.

Gender-Related Diagnostic Issues

Gender differences pertain to those associated with the medical condition (e.g., systemic lupus erythematosus is more common in females; stroke is somewhat more common in middle-age males compared with females).

Diagnostic Markers

Diagnostic markers pertain to those associated with the medical condition (e.g., steroid levels in blood or urine to help corroborate the diagnosis of Cushing's disease, which can be associated with manic or depressive syndromes).

Suicide Risk

There are no epidemiological studies that provide evidence to differentiate the risk of suicide from a major depressive episode due to another medical condition compared with the risk from a major depressive episode in general. There are case reports of suicides in association with major depressive episodes associated with another medical condition. There is a clear association between serious medical illnesses and suicide, particularly shortly after onset or diagnosis of the illness. Thus, it would be prudent to assume that the risk of suicide for major depressive episodes associated with medical conditions is not less than that for other forms of major depressive episode, and might even be greater.

Functional Consequences of Depressive Disorder Due to Another Medical Condition

Functional consequences pertain to those associated with the medical condition. In general, it is believed, but not established, that a major depressive episode induced by Cushing's disease will not recur if the Cushing's disease is cured or arrested. However, it is also suggested, but not established, that mood syndromes, including depressive and manic/hypomanic ones, may be episodic (i.e., recurring) in some individuals with static brain injuries and other central nervous system diseases.

Differential Diagnosis

Depressive disorders not due to another medical condition. Determination of whether a medical condition accompanying a depressive disorder is causing the disorder depends on a) the absence of an episode(s) of depressive episodes prior to the onset of the medical

condition, b) the probability that the associated medical condition has a potential to promote or cause a depressive disorder, and c) a course of the depressive symptoms shortly after the onset or worsening of the medical condition, especially if the depressive symptoms remit near the time that the medical disorder is effectively treated or remits.

Medication-induced depressive disorder. An important caveat is that some medical conditions are treated with medications (e.g., steroids or alpha-interferon) that can induce depressive or manic symptoms. In these cases, clinical judgment, based on all the evidence in hand, is the best way to try to separate the most likely and/or the most important of two etiological factors (i.e., association with the medical condition vs. a substance-induced syndrome).

Adjustment disorders. It is important to differentiate a depressive episode from an adjustment disorder, as the onset of the medical condition is in itself a life stressor that could bring on either an adjustment disorder or an episode of major depression. The major differentiating elements are the pervasiveness the depressive picture and the number and quality of the depressive symptoms that the patient reports or demonstrates on the mental status examination. The differential diagnosis of the associated medical conditions is relevant but largely beyond the scope of the present manual.

Comorbidity

Conditions comorbid with depressive disorder due to another medical condition are those associated with the medical conditions of etiological relevance. It has been noted that delirium can occur before or along with depressive symptoms in individuals with a variety of medical conditions, such as Cushing's disease. The association of anxiety symptoms, usually generalized symptoms, is common in depressive disorders, regardless of cause.

Other Specified Depressive Disorder

311 (F32.8)

This category applies to presentations in which symptoms characteristic of a depressive disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the depressive disorders diagnostic class. The other specified depressive disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific depressive disorder. This is done by recording "other specified depressive disorder" followed by the specific reason (e.g., "short-duration depressive episode").

Examples of presentations that can be specified using the "other specified" designation include the following:

1. **Recurrent brief depression:** Concurrent presence of depressed mood and at least four other symptoms of depression for 2–13 days at least once per month (not associated with the menstrual cycle) for at least 12 consecutive months in an individual whose presentation has never met criteria for any other depressive or bipolar disorder and does not currently meet active or residual criteria for any psychotic disorder.
2. **Short-duration depressive episode (4–13 days):** Depressed affect and at least four of the other eight symptoms of a major depressive episode associated with clinically significant distress or impairment that persists for more than 4 days, but less than 14 days, in an individual whose presentation has never met criteria for any other depressive or bipolar disorder, does not currently meet active or residual criteria for any psychotic disorder, and does not meet criteria for recurrent brief depression.
3. **Depressive episode with insufficient symptoms:** Depressed affect and at least one of the other eight symptoms of a major depressive episode associated with clinically

significant distress or impairment that persist for at least 2 weeks in an individual whose presentation has never met criteria for any other depressive or bipolar disorder, does not currently meet active or residual criteria for any psychotic disorder, and does not meet criteria for mixed anxiety and depressive disorder symptoms.

Unspecified Depressive Disorder

311 (F32.9)

This category applies to presentations in which symptoms characteristic of a depressive disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the depressive disorders diagnostic class. The unspecified depressive disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific depressive disorder, and includes presentations for which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Specifiers for Depressive Disorders

Specify if:

With anxious distress: Anxious distress is defined as the presence of at least two of the following symptoms during the majority of days of a major depressive episode or persistent depressive disorder (dysthymia):

1. Feeling keyed up or tense.
2. Feeling unusually restless.
3. Difficulty concentrating because of worry.
4. Fear that something awful may happen.
5. Feeling that the individual might lose control of himself or herself.

Specify current severity:

Mild: Two symptoms.
Moderate: Three symptoms.
Moderate-severe: Four or five symptoms.
Severe: Four or five symptoms and with motor agitation.

Note: Anxious distress has been noted as a prominent feature of both bipolar and major depressive disorder in both primary care and specialty mental health settings. High levels of anxiety have been associated with higher suicide risk, longer duration of illness, and greater likelihood of treatment nonresponse. As a result, it is clinically useful to specify accurately the presence and severity levels of anxious distress for treatment planning and monitoring of response to treatment.

With mixed features:

A. At least three of the following manic/hypomanic symptoms are present nearly every day during the majority of days of a major depressive episode:

1. Elevated, expansive mood.
2. Inflated self-esteem or grandiosity.
3. More talkative than usual or pressure to keep talking.
4. Flight of ideas or subjective experience that thoughts are racing.
5. Increase in energy or goal-directed activity (either socially, at work or school, or sexually).

6. Increased or excessive involvement in activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, foolish business investments).
7. Decreased need for sleep (feeling rested despite sleeping less than usual; to be contrasted with insomnia).

B. Mixed symptoms are observable by others and represent a change from the person's usual behavior.

C. For individuals whose symptoms meet full criteria for either mania or hypomania, the diagnosis should be bipolar I or bipolar II disorder.

D. The mixed symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication or other treatment).

Note: Mixed features associated with a major depressive episode have been found to be a significant risk factor for the development of bipolar I or bipolar II disorder. As a result, it is clinically useful to note the presence of this specifier for treatment planning and monitoring of response to treatment.

With melancholic features:

A. One of the following is present during the most severe period of the current episode:

1. Loss of pleasure in all, or almost all, activities.
2. Lack of reactivity to usually pleasurable stimuli (does not feel much better, even temporarily, when something good happens).

B. Three (or more) of the following:

1. A distinct quality of depressed mood characterized by profound despondency, despair, and/or moroseness or by so-called empty mood.
2. Depression that is regularly worse in the morning.
3. Early-morning awakening (i.e., at least 2 hours before usual awakening).
4. Marked psychomotor agitation or retardation.
5. Significant anorexia or weight loss.
6. Excessive or inappropriate guilt.

Note: The specifier "with melancholic features" is applied if these features are present at the most severe stage of the episode. There is a near-complete absence of the capacity for pleasure, not merely a diminution. A guideline for evaluating the lack of reactivity of mood is that even highly desired events are not associated with marked brightening of mood. Either mood does not brighten at all, or it brightens only partially (e.g., up to 20%–40% of normal for only minutes at a time). The "distinct quality" of mood that is characteristic of the "with melancholic features" specifier is experienced as qualitatively different from that during a nonmelancholic depressive episode. A depressed mood that is described as merely more severe, longer lasting, or present without a reason is not considered distinct in quality. Psychomotor changes are nearly always present and are observable by others.

Melancholic features exhibit only a modest tendency to repeat across episodes in the same individual. They are more frequent in inpatients, as opposed to outpatients; are less likely to occur in milder than in more severe major depressive episodes; and are more likely to occur in those with psychotic features.

With atypical features: This specifier can be applied when these features predominate during the majority of days of the current or most recent major depressive episode or persistent depressive disorder.

A. Mood reactivity (i.e., mood brightens in response to actual or potential positive events).

B. Two (or more) of the following:
   1. Significant weight gain or increase in appetite.
   2. Hypersomnia.
   3. Leaden paralysis (i.e., heavy, leaden feelings in arms or legs).
   4. A long-standing pattern of interpersonal rejection sensitivity (not limited to episodes of mood disturbance) that results in significant social or occupational impairment.

C. Criteria are not met for "with melancholic features" or "with catatonia" during the same episode.

Note: "Atypical depression" has historical significance (i.e., atypical in contradistinction to the more classical agitated, "endogenous" presentations of depression that were the norm when depression was rarely diagnosed in outpatients and almost never in adolescents or younger adults) and today does not connote an uncommon or unusual clinical presentation as the term might imply.

Mood reactivity is the capacity to be cheered up when presented with positive events (e.g., a visit from children, compliments from others). Mood may become euthymic (not sad) even for extended periods of time if the external circumstances remain favorable. Increased appetite may be manifested by an obvious increase in food intake or by weight gain. Hypersomnia may include either an extended period of nighttime sleep or daytime napping that totals at least 10 hours of sleep per day (or at least 2 hours more than when not depressed). Leaden paralysis is defined as feeling heavy, leaden, or weighted down, usually in the arms or legs. This sensation is generally present for at least an hour a day but often lasts for many hours at a time. Unlike the other atypical features, pathological sensitivity to perceived interpersonal rejection is a trait that has an early onset and persists throughout most of adult life. Rejection sensitivity occurs both when the person is and is not depressed, though it may be exacerbated during depressive periods.

With psychotic features: Delusions and/or hallucinations are present.

With mood-congruent psychotic features: The content of all delusions and hallucinations is consistent with the typical depressive themes of personal inadequacy, guilt, disease, death, nihilism, or deserved punishment.

With mood-incongruent psychotic features: The content of the delusions or hallucinations does not involve typical depressive themes of personal inadequacy, guilt, disease, death, nihilism, or deserved punishment, or the content is a mixture of mood-incongruent and mood-congruent themes.

With catatonia: The catatonia specifier can apply to an episode of depression if catatonic features are present during most of the episode. See criteria for catatonia associated with a mental disorder (for a description of catatonia, see the chapter "Schizophrenia Spectrum and Other Psychotic Disorders").

With peripartum onset: This specifier can be applied to the current or, if full criteria are not currently met for a major depressive episode, most recent episode of major depression if onset of mood symptoms occurs during pregnancy or in the 4 weeks following delivery.

Note: Mood episodes can have their onset either during pregnancy or postpartum. Although the estimates differ according to the period of follow-up after delivery, between 3% and 6% of women will experience the onset of a major depressive episode during pregnancy or in the weeks or months following delivery. Fifty percent of "postpartum" major depressive episodes actually begin prior to delivery. Thus, these episodes are referred to collectively as *peripartum* episodes. Women with peripartum major depressive episodes often have severe anxiety and even panic

attacks. Prospective studies have demonstrated that mood and anxiety symptoms during pregnancy, as well as the "baby blues," increase the risk for a postpartum major depressive episode.

Peripartum-onset mood episodes can present either with or without psychotic features. Infanticide is most often associated with postpartum psychotic episodes that are characterized by command hallucinations to kill the infant or delusions that the infant is possessed, but psychotic symptoms can also occur in severe postpartum mood episodes without such specific delusions or hallucinations.

Postpartum mood (major depressive or manic) episodes with psychotic features appear to occur in from 1 in 500 to 1 in 1,000 deliveries and may be more common in primiparous women. The risk of postpartum episodes with psychotic features is particularly increased for women with prior postpartum mood episodes but is also elevated for those with a prior history of a depressive or bipolar disorder (especially bipolar I disorder) and those with a family history of bipolar disorders.

Once a woman has had a postpartum episode with psychotic features, the risk of recurrence with each subsequent delivery is between 30% and 50%. Postpartum episodes must be differentiated from delirium occurring in the postpartum period, which is distinguished by a fluctuating level of awareness or attention. The postpartum period is unique with respect to the degree of neuroendocrine alterations and psychosocial adjustments, the potential impact of breast-feeding on treatment planning, and the long-term implications of a history of postpartum mood disorder on subsequent family planning.

With seasonal pattern: This specifier applies to recurrent major depressive disorder.

A. There has been a regular temporal relationship between the onset of major depressive episodes in major depressive disorder and a particular time of the year (e.g., in the fall or winter).

   Note: Do not include cases in which there is an obvious effect of seasonally related psychosocial stressors (e.g., regularly being unemployed every winter).

B. Full remissions (or a change from major depression to mania or hypomania) also occur at a characteristic time of the year (e.g., depression disappears in the spring).

C. In the last 2 years, two major depressive episodes have occurred that demonstrate the temporal seasonal relationships defined above and no nonseasonal major depressive episodes have occurred during that same period.

D. Seasonal major depressive episodes (as described above) substantially outnumber the nonseasonal major depressive episodes that may have occurred over the individual's lifetime.

Note: The specifier "with seasonal pattern" can be applied to the pattern of major depressive episodes in major depressive disorder, recurrent. The essential feature is the onset and remission of major depressive episodes at characteristic times of the year. In most cases, the episodes begin in fall or winter and remit in spring. Less commonly, there may be recurrent summer depressive episodes. This pattern of onset and remission of episodes must have occurred during at least a 2-year period, without any nonseasonal episodes occurring during this period. In addition, the seasonal depressive episodes must substantially outnumber any nonseasonal depressive episodes over the individual's lifetime.

This specifier does not apply to those situations in which the pattern is better explained by seasonally linked psychosocial stressors (e.g., seasonal unemployment or school schedule). Major depressive episodes that occur in a seasonal pattern are often characterized by prominent energy, hypersomnia, overeating, weight gain, and a craving for carbohydrates. It is unclear whether a seasonal pattern is more likely in recurrent major depressive disorder or in bipolar disorders. However, within the bipolar disorders group, a seasonal pattern appears to be more likely in bipolar II disorder than

in bipolar I disorder. In some individuals, the onset of manic or hypomanic episodes may also be linked to a particular season.

The prevalence of winter-type seasonal pattern appears to vary with latitude, age, and sex. Prevalence increases with higher latitudes. Age is also a strong predictor of seasonality, with younger persons at higher risk for winter depressive episodes.

Specify if:

In partial remission: Symptoms of the immediately previous major depressive episode are present, but full criteria are not met, or there is a period lasting less than 2 months without any significant symptoms of a major depressive episode following the end of such an episode.

In full remission: During the past 2 months, no significant signs or symptoms of the disturbance were present.

Specify current severity:

Severity is based on the number of criterion symptoms, the severity of those symptoms, and the degree of functional disability.

Mild: Few, if any, symptoms in excess of those required to make the diagnosis are present, the intensity of the symptoms is distressing but manageable, and the symptoms result in minor impairment in social or occupational functioning.

Moderate: The number of symptoms, intensity of symptoms, and/or functional impairment are between those specified for "mild" and "severe."

Severe: The number of symptoms is substantially in excess of that required to make the diagnosis, the intensity of the symptoms is seriously distressing and unmanageable, and the symptoms markedly interfere with social and occupational functioning.

Anxiety Disorders

Anxiety disorders include disorders that share features of excessive fear and anxiety and related behavioral disturbances. *Fear* is the emotional response to real or perceived imminent threat, whereas *anxiety* is anticipation of future threat. Obviously, these two states overlap, but they also differ, with fear more often associated with surges of autonomic arousal necessary for fight or flight, thoughts of immediate danger, and escape behaviors, and anxiety more often associated with muscle tension and vigilance in preparation for future danger and cautious or avoidant behaviors. Sometimes the level of fear or anxiety is reduced by pervasive avoidance behaviors. *Panic attacks* feature prominently within the anxiety disorders as a particular type of fear response. Panic attacks are not limited to anxiety disorders but rather can be seen in other mental disorders as well.

The anxiety disorders differ from one another in the types of objects or situations that induce fear, anxiety, or avoidance behavior, and the associated cognitive ideation. Thus, while the anxiety disorders tend to be highly comorbid with each other, they can be differentiated by close examination of the types of situations that are feared or avoided and the content of the associated thoughts or beliefs.

Anxiety disorders differ from developmentally normative fear or anxiety by being excessive or persisting beyond developmentally appropriate periods. They differ from transient fear or anxiety, often stress-induced, by being persistent (e.g., typically lasting 6 months or more), although the criterion for duration is intended as a general guide with allowance for some degree of flexibility and is sometimes of shorter duration in children (as in separation anxiety disorder and selective mutism). Since individuals with anxiety disorders typically overestimate the danger in situations they fear or avoid, the primary determination of whether the fear or anxiety is excessive or out of proportion is made by the clinician, taking cultural contextual factors into account. Many of the anxiety disorders develop in childhood and tend to persist if not treated. Most occur more frequently in females than in males (approximately 2:1 ratio). Each anxiety disorder is diagnosed only when the symptoms are not attributable to the physiological effects of a substance/medication or to another medical condition or are not better explained by another mental disorder.

The chapter is arranged developmentally, with disorders sequenced according to the typical age at onset. The individual with separation anxiety disorder is fearful or anxious about separation from attachment figures to a degree that is developmentally inappropriate. There is persistent fear or anxiety about harm coming to attachment figures and events that could lead to loss of or separation from attachment figures and reluctance to go away from attachment figures, as well as nightmares and physical symptoms of distress. Although the symptoms often develop in childhood, they can be expressed throughout adulthood as well.

Selective mutism is characterized by a consistent failure to speak in social situations in which there is an expectation to speak (e.g., school) even though the individual speaks in other situations. The failure to speak has significant consequences on achievement in academic or occupational settings or otherwise interferes with normal social communication.

Individuals with specific phobia are fearful or anxious about or avoidant of circumscribed objects or situations. A specific cognitive ideation is not featured in this disorder, as it is in other anxiety disorders. The fear, anxiety, or avoidance is almost always imme-

diately induced by the phobic situation, to a degree that is persistent and out of proportion to the actual risk posed. There are various types of specific phobias: animal; natural environment; blood-injection-injury; situational; and other situations.

In social anxiety disorder (social phobia), the individual is fearful or anxious about or avoidant of social interactions and situations that involve the possibility of being scrutinized. These include social interactions such as meeting unfamiliar people, situations in which the individual may be observed eating or drinking, and situations in which the individual performs in front of others. The cognitive ideation is of being negatively evaluated by others, by being embarrassed, humiliated, or rejected, or offending others.

In panic disorder, the individual experiences recurrent unexpected panic attacks and is persistently concerned or worried about having more panic attacks or changes his or her behavior in maladaptive ways because of the panic attacks (e.g., avoidance of exercise or of unfamiliar locations). Panic attacks are abrupt surges of intense fear or intense discomfort that reach a peak within minutes, accompanied by physical and/or cognitive symptoms. Limited-symptom panic attacks include fewer than four symptoms. Panic attacks may be *expected*, such as in response to a typically feared object or situation, or *unexpected*, meaning that the panic attack occurs for no apparent reason. Panic attacks function as a marker and prognostic factor for severity of diagnosis, course, and comorbidity across an array of disorders, including, but not limited to, the anxiety disorders (e.g., substance use, depressive and psychotic disorders). Panic attack may therefore be used as a descriptive specifier for any anxiety disorder as well as other mental disorders.

Individuals with agoraphobia are fearful and anxious about two or more of the following situations: using public transportation; being in open spaces; being in enclosed places; standing in line or being in a crowd; or being outside of the home alone in other situations. The individual fears these situations because of thoughts that escape might be difficult or help might not be available in the event of developing panic-like symptoms or other incapacitating or embarrassing symptoms. These situations almost always induce fear or anxiety and are often avoided and require the presence of a companion.

The key features of generalized anxiety disorder are persistent and excessive anxiety and worry about various domains, including work and school performance, that the individual finds difficult to control. In addition, the individual experiences physical symptoms, including restlessness or feeling keyed up or on edge; being easily fatigued; difficulty concentrating or mind going blank; irritability; muscle tension; and sleep disturbance.

Substance/medication-induced anxiety disorder involves anxiety due to substance intoxication or withdrawal or to a medication treatment. In anxiety disorder due to another medical condition, anxiety symptoms are the physiological consequence of another medical condition.

Disorder-specific scales are available to better characterize the severity of each anxiety disorder and to capture change in severity over time. For ease of use, particularly for individuals with more than one anxiety disorder, these scales have been developed to have the same format (but different focus) across the anxiety disorders, with ratings of behavioral symptoms, cognitive ideation symptoms, and physical symptoms relevant to each disorder.

Separation Anxiety Disorder

Diagnostic Criteria **309.21 (F93.0)**

A. Developmentally inappropriate and excessive fear or anxiety concerning separation from those to whom the individual is attached, as evidenced by at least three of the following:

1. Recurrent excessive distress when anticipating or experiencing separation from home or from major attachment figures.

2. Persistent and excessive worry about losing major attachment figures or about possible harm to them, such as illness, injury, disasters, or death.
3. Persistent and excessive worry about experiencing an untoward event (e.g., getting lost, being kidnapped, having an accident, becoming ill) that causes separation from a major attachment figure.
4. Persistent reluctance or refusal to go out, away from home, to school, to work, or elsewhere because of fear of separation.
5. Persistent and excessive fear of or reluctance about being alone or without major attachment figures at home or in other settings.
6. Persistent reluctance or refusal to sleep away from home or to go to sleep without being near a major attachment figure.
7. Repeated nightmares involving the theme of separation.
8. Repeated complaints of physical symptoms (e.g., headaches, stomachaches, nausea, vomiting) when separation from major attachment figures occurs or is anticipated.

B. The fear, anxiety, or avoidance is persistent, lasting at least 4 weeks in children and adolescents and typically 6 months or more in adults.

C. The disturbance causes clinically significant distress or impairment in social, academic, occupational, or other important areas of functioning.

D. The disturbance is not better explained by another mental disorder, such as refusing to leave home because of excessive resistance to change in autism spectrum disorder; delusions or hallucinations concerning separation in psychotic disorders; refusal to go outside without a trusted companion in agoraphobia; worries about ill health or other harm befalling significant others in generalized anxiety disorder; or concerns about having an illness in illness anxiety disorder.

Diagnostic Features

The essential feature of separation anxiety disorder is excessive fear or anxiety concerning separation from home or attachment figures. The anxiety exceeds what may be expected given the person's developmental level (Criterion A). Individuals with separation anxiety disorder have symptoms that meet at least three of the following criteria: They experience recurrent excessive distress when separation from home or major attachment figures is anticipated or occurs (Criterion A1). They worry about the well-being or death of attachment figures, particularly when separated from them, and they need to know the whereabouts of their attachment figures and want to stay in touch with them (Criterion A2). They also worry about untoward events to themselves, such as getting lost, being kidnapped, or having an accident, that would keep them from ever being reunited with their major attachment figure (Criterion A3). Individuals with separation anxiety disorder are reluctant or refuse to go out by themselves because of separation fears (Criterion A4). They have persistent and excessive fear or reluctance about being alone or without major attachment figures at home or in other settings. Children with separation anxiety disorder may be unable to stay or go in a room by themselves and may display "clinging" behavior, staying close to or "shadowing" the parent around the house, or requiring someone to be with them when going to another room in the house (Criterion A5). They have persistent reluctance or refusal to go to sleep without being near a major attachment figure or to sleep away from home (Criterion A6). Children with this disorder often have difficulty at bedtime and may insist that someone stay with them until they fall asleep. During the night, they may make their way to their parents' bed (or that of a significant other, such as a sibling). Children may be reluctant or refuse to attend camp, to sleep at friends' homes, or to go on errands. Adults may be uncomfortable when traveling independently (e.g., sleeping in a hotel room). There may be repeated nightmares in which the content expresses the in-

dividual's separation anxiety (e.g., destruction of the family through fire, murder, or other catastrophe) (Criterion A7). Physical symptoms (e.g., headaches, abdominal complaints, nausea, vomiting) are common in children when separation from major attachment figures occurs or is anticipated (Criterion A8). Cardiovascular symptoms such as palpitations, dizziness, and feeling faint are rare in younger children but may occur in adolescents and adults.

The disturbance must last for a period of at least 4 weeks in children and adolescents younger than 18 years and is typically 6 months or longer in adults (Criterion B). However, the duration criterion for adults should be used as a general guide, with allowance for some degree of flexibility. The disturbance must cause clinically significant distress or impairment in social, academic, occupational, or other important areas of functioning (Criterion C).

Associated Features Supporting Diagnosis

When separated from major attachment figures, children with separation anxiety disorder may exhibit social withdrawal, apathy, sadness, or difficulty concentrating on work or play. Depending on their age, individuals may have fears of animals, monsters, the dark, muggers, burglars, kidnappers, car accidents, plane travel, and other situations that are perceived as presenting danger to the family or themselves. Some individuals become homesick and uncomfortable to the point of misery when away from home. Separation anxiety disorder in children may lead to school refusal, which in turn may lead to academic difficulties and social isolation. When extremely upset at the prospect of separation, children may show anger or occasionally aggression toward someone who is forcing separation. When alone, especially in the evening or the dark, young children may report unusual perceptual experiences (e.g., seeing people peering into their room, frightening creatures reaching for them, feeling eyes staring at them). Children with this disorder may be described as demanding, intrusive, and in need of constant attention, and, as adults, may appear dependent and overprotective. The individual's excessive demands often become a source of frustration for family members, leading to resentment and conflict in the family.

Prevalence

The 12-month prevalence of separation anxiety disorder among adults in the United States is 0.9%–1.9%. In children, 6- to 12-month prevalence is estimated to be approximately 4%. In adolescents in the United States, the 12-month prevalence is 1.6%. Separation anxiety disorder decreases in prevalence from childhood through adolescence and adulthood and is the most prevalent anxiety disorder in children younger than 12 years. In clinical samples of children, the disorder is equally common in males and females. In the community, the disorder is more frequent in females.

Development and Course

Periods of heightened separation anxiety from attachment figures are part of normal early development and may indicate the development of secure attachment relationships (e.g., around 1 year of age, when infants may suffer from stranger anxiety). Onset of separation anxiety disorder may be as early as preschool age and may occur at any time during childhood and more rarely in adolescence. Typically there are periods of exacerbation and remission. In some cases, both the anxiety about possible separation and the avoidance of situations involving separation from the home or nuclear family (e.g., going away to college, moving away from attachment figures) may persist through adulthood. However, the majority of children with separation anxiety disorder are free of impairing anxiety disorders over their lifetimes. Many adults with separation anxiety disorder do not recall a childhood onset of separation anxiety disorder, although they may recall symptoms.

The manifestations of separation anxiety disorder vary with age. Younger children are more reluctant to go to school or may avoid school altogether. Younger children may not express worries or specific fears of definite threats to parents, home, or themselves, and the anxiety is manifested only when separation is experienced. As children age, worries emerge; these are often worries about specific dangers (e.g., accidents, kidnapping, mugging, death) or vague concerns about not being reunited with attachment figures. In adults, separation anxiety disorder may limit their ability to cope with changes in circumstances (e.g., moving, getting married). Adults with the disorder are typically overconcerned about their offspring and spouses and experience marked discomfort when separated from them. They may also experience significant disruption in work or social experiences because of needing to continuously check on the whereabouts of a significant other.

Risk and Prognostic Factors

Environmental. Separation anxiety disorder often develops after life stress, especially a loss (e.g., the death of a relative or pet; an illness of the individual or a relative; a change of schools; parental divorce; a move to a new neighborhood; immigration; a disaster that involved periods of separation from attachment figures). In young adults, other examples of life stress include leaving the parental home, entering into a romantic relationship, and becoming a parent. Parental overprotection and intrusiveness may be associated with separation anxiety disorder.

Genetic and physiological. Separation anxiety disorder in children may be heritable. Heritability was estimated at 73% in a community sample of 6-year-old twins, with higher rates in girls. Children with separation anxiety disorder display particularly enhanced sensitivity to respiratory stimulation using CO_2-enriched air.

Culture-Related Diagnostic Issues

There are cultural variations in the degree to which it is considered desirable to tolerate separation, so that demands and opportunities for separation between parents and children are avoided in some cultures. For example, there is wide variation across countries and cultures with respect to the age at which it is expected that offspring should leave the parental home. It is important to differentiate separation anxiety disorder from the high value some cultures place on strong interdependence among family members.

Gender-Related Diagnostic Issues

Girls manifest greater reluctance to attend or avoidance of school than boys. Indirect expression of fear of separation may be more common in males than in females, for example, by limited independent activity, reluctance to be away from home alone, or distress when spouse or offspring do things independently or when contact with spouse or offspring is not possible.

Suicide Risk

Separation anxiety disorder in children may be associated with an increased risk for suicide. In a community sample, the presence of mood disorders, anxiety disorders, or substance use has been associated with suicidal ideation and attempts. However, this association is not specific to separation anxiety disorder and is found in several anxiety disorders.

Functional Consequences of Separation Anxiety Disorder

Individuals with separation anxiety disorder often limit independent activities away from home or attachment figures (e.g., in children, avoiding school, not going to camp, having

difficulty sleeping alone; in adolescents, not going away to college; in adults, not leaving the parental home, not traveling, not working outside the home).

Differential Diagnosis

Generalized anxiety disorder. Separation anxiety disorder is distinguished from generalized anxiety disorder in that the anxiety predominantly concerns separation from attachment figures, and if other worries occur, they do not predominate the clinical picture.

Panic disorder. Threats of separation may lead to extreme anxiety and even a panic attack. In separation anxiety disorder, in contrast to panic disorder, the anxiety concerns the possibility of being away from attachment figures and worry about untoward events befalling them, rather than being incapacitated by an unexpected panic attack.

Agoraphobia. Unlike individuals with agoraphobia, those with separation anxiety disorder are not anxious about being trapped or incapacitated in situations from which escape is perceived as difficult in the event of panic-like symptoms or other incapacitating symptoms.

Conduct disorder. School avoidance (truancy) is common in conduct disorder, but anxiety about separation is not responsible for school absences, and the child or adolescent usually stays away from, rather than returns to, the home.

Social anxiety disorder. School refusal may be due to social anxiety disorder (social phobia). In such instances, the school avoidance is due to fear of being judged negatively by others rather than to worries about being separated from the attachment figures.

Posttraumatic stress disorder. Fear of separation from loved ones is common after traumatic events such as a disasters, particularly when periods of separation from loved ones were experienced during the traumatic event. In posttraumatic stress disorder (PTSD), the central symptoms concern intrusions about, and avoidance of, memories associated with the traumatic event itself, whereas in separation anxiety disorder, the worries and avoidance concern the well-being of attachment figures and separation from them.

Illness anxiety disorder. Individuals with illness anxiety disorder worry about specific illnesses they may have, but the main concern is about the medical diagnosis itself, not about being separated from attachment figures.

Bereavement. Intense yearning or longing for the deceased, intense sorrow and emotional pain, and preoccupation with the deceased or the circumstances of the death are expected responses occurring in bereavement, whereas fear of separation from other attachment figures is central in separation anxiety disorder.

Depressive and bipolar disorders. These disorders may be associated with reluctance to leave home, but the main concern is not worry or fear of untoward events befalling attachment figures, but rather low motivation for engaging with the outside world. However, individuals with separation anxiety disorder may become depressed while being separated or in anticipation of separation.

Oppositional defiant disorder. Children and adolescents with separation anxiety disorder may be oppositional in the context of being forced to separate from attachment figures. Oppositional defiant disorder should be considered only when there is persistent oppositional behavior unrelated to the anticipation or occurrence of separation from attachment figures.

Psychotic disorders. Unlike the hallucinations in psychotic disorders, the unusual perceptual experiences that may occur in separation anxiety disorder are usually based on a misperception of an actual stimulus, occur only in certain situations (e.g., nighttime), and are reversed by the presence of an attachment figure.

Personality disorders. Dependent personality disorder is characterized by an indiscriminate tendency to rely on others, whereas separation anxiety disorder involves concern about the proximity and safety of main attachment figures. Borderline personality disorder is characterized by fear of abandonment by loved ones, but problems in identity, self-direction, interpersonal functioning, and impulsivity are additionally central to that disorder, whereas they are not central to separation anxiety disorder.

Comorbidity

In children, separation anxiety disorder is highly comorbid with generalized anxiety disorder and specific phobia. In adults, common comorbidities include specific phobia, PTSD, panic disorder, generalized anxiety disorder, social anxiety disorder, agoraphobia, obsessive-compulsive disorder, and personality disorders. Depressive and bipolar disorders are also comorbid with separation anxiety disorder in adults.

Selective Mutism

Diagnostic Criteria **312.23 (F94.0)**

A. Consistent failure to speak in specific social situations in which there is an expectation for speaking (e.g., at school) despite speaking in other situations.
B. The disturbance interferes with educational or occupational achievement or with social communication.
C. The duration of the disturbance is at least 1 month (not limited to the first month of school).
D. The failure to speak is not attributable to a lack of knowledge of, or comfort with, the spoken language required in the social situation.
E. The disturbance is not better explained by a communication disorder (e.g., childhood-onset fluency disorder) and does not occur exclusively during the course of autism spectrum disorder, schizophrenia, or another psychotic disorder.

Diagnostic Features

When encountering other individuals in social interactions, children with selective mutism do not initiate speech or reciprocally respond when spoken to by others. Lack of speech occurs in social interactions with children or adults. Children with selective mutism will speak in their home in the presence of immediate family members but often not even in front of close friends or second-degree relatives, such as grandparents or cousins. The disturbance is often marked by high social anxiety. Children with selective mutism often refuse to speak at school, leading to academic or educational impairment, as teachers often find it difficult to assess skills such as reading. The lack of speech may interfere with social communication, although children with this disorder sometimes use nonspoken or nonverbal means (e.g., grunting, pointing, writing) to communicate and may be willing or eager to perform or engage in social encounters when speech is not required (e.g., nonverbal parts in school plays).

Associated Features Supporting Diagnosis

Associated features of selective mutism may include excessive shyness, fear of social embarrassment, social isolation and withdrawal, clinging, compulsive traits, negativism, temper tantrums, or mild oppositional behavior. Although children with this disorder generally have normal language skills, there may occasionally be an associated commu-

nication disorder, although no particular association with a specific communication disorder has been identified. Even when these disorders are present, anxiety is present as well. In clinical settings, children with selective mutism are almost always given an additional diagnosis of another anxiety disorder—most commonly, social anxiety disorder (social phobia).

Prevalence

Selective mutism is a relatively rare disorder and has not been included as a diagnostic category in epidemiological studies of prevalence of childhood disorders. Point prevalence using various clinic or school samples ranges between 0.03% and 1% depending on the setting (e.g., clinic vs. school vs. general population) and ages of the individuals in the sample. The prevalence of the disorder does not seem to vary by sex or race/ethnicity. The disorder is more likely to manifest in young children than in adolescents and adults.

Development and Course

The onset of selective mutism is usually before age 5 years, but the disturbance may not come to clinical attention until entry into school, where there is an increase in social interaction and performance tasks, such as reading aloud. The persistence of the disorder is variable. Although clinical reports suggest that many individuals "outgrow" selective mutism, the longitudinal course of the disorder is unknown. In some cases, particularly in individuals with social anxiety disorder, selective mutism may disappear, but symptoms of social anxiety disorder remain.

Risk and Prognostic Factors

Temperamental. Temperamental risk factors for selective mutism are not well identified. Negative affectivity (neuroticism) or behavioral inhibition may play a role, as may parental history of shyness, social isolation, and social anxiety. Children with selective mutism may have subtle receptive language difficulties compared with their peers, although receptive language is still within the normal range.

Environmental. Social inhibition on the part of parents may serve as a model for social reticence and selective mutism in children. Furthermore, parents of children with selective mutism have been described as overprotective or more controlling than parents of children with other anxiety disorders or no disorder.

Genetic and physiological factors. Because of the significant overlap between selective mutism and social anxiety disorder, there may be shared genetic factors between these conditions.

Culture-Related Diagnostic Issues

Children in families who have immigrated to a country where a different language is spoken may refuse to speak the new language because of lack of knowledge of the language. If comprehension of the new language is adequate but refusal to speak persists, a diagnosis of selective mutism may be warranted.

Functional Consequences of Selective Mutism

Selective mutism may result in social impairment, as children may be too anxious to engage in reciprocal social interaction with other children. As children with selective mutism mature, they may face increasing social isolation. In school settings, these children may suffer academic impairment, because often they do not communicate with teachers regarding their academic or personal needs (e.g., not understanding a class assignment, not

asking to use the restroom). Severe impairment in school and social functioning, including that resulting from teasing by peers, is common. In certain instances, selective mutism may serve as a compensatory strategy to decrease anxious arousal in social encounters.

Differential Diagnosis

Communication disorders. Selective mutism should be distinguished from speech disturbances that are better explained by a communication disorder, such as language disorder, speech sound disorder (previously phonological disorder), childhood-onset fluency disorder (stuttering), or pragmatic (social) communication disorder. Unlike selective mutism, the speech disturbance in these conditions is not restricted to a specific social situation.

Neurodevelopmental disorders and schizophrenia and other psychotic disorders. Individuals with an autism spectrum disorder, schizophrenia or another psychotic disorder, or severe intellectual disability may have problems in social communication and be unable to speak appropriately in social situations. In contrast, selective mutism should be diagnosed only when a child has an established capacity to speak in some social situations (e.g., typically at home).

Social anxiety disorder (social phobia). The social anxiety and social avoidance in social anxiety disorder may be associated with selective mutism. In such cases, both diagnoses may be given.

Comorbidity

The most common comorbid conditions are other anxiety disorders, most commonly social anxiety disorder, followed by separation anxiety disorder and specific phobia. Oppositional behaviors have been noted to occur in children with selective mutism, although oppositional behavior may be limited to situations requiring speech. Communication delays or disorders also may appear in some children with selective mutism.

Specific Phobia

Diagnostic Criteria

A. Marked fear or anxiety about a specific object or situation (e.g., flying, heights, animals, receiving an injection, seeing blood).

   Note: In children, the fear or anxiety may be expressed by crying, tantrums, freezing, or clinging.

B. The phobic object or situation almost always provokes immediate fear or anxiety.

C. The phobic object or situation is actively avoided or endured with intense fear or anxiety.

D. The fear or anxiety is out of proportion to the actual danger posed by the specific object or situation and to the sociocultural context.

E. The fear, anxiety, or avoidance is persistent, typically lasting for 6 months or more.

F. The fear, anxiety, or avoidance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

G. The disturbance is not better explained by the symptoms of another mental disorder, including fear, anxiety, and avoidance of situations associated with panic-like symptoms or other incapacitating symptoms (as in agoraphobia); objects or situations related to obsessions (as in obsessive-compulsive disorder); reminders of traumatic events (as in posttraumatic stress disorder); separation from home or attachment figures (as in separation anxiety disorder); or social situations (as in social anxiety disorder).

Specify if:

Code based on the phobic stimulus:

300.29 (F40.218) Animal (e.g., spiders, insects, dogs).

300.29 (F40.228) Natural environment (e.g., heights, storms, water).

300.29 (F40.23x) Blood-injection-injury (e.g., needles, invasive medical procedures).

Coding note: Select specific ICD-10-CM code as follows: **F40.230** fear of blood; **F40.231** fear of injections and transfusions; **F40.232** fear of other medical care; or **F40.233** fear of injury.

300.29 (F40.248) Situational (e.g., airplanes, elevators, enclosed places).

300.29 (F40.298) Other (e.g., situations that may lead to choking or vomiting; in children, e.g., loud sounds or costumed characters).

Coding note: When more than one phobic stimulus is present, code all ICD-10-CM codes that apply (e.g., for fear of snakes and flying, F40.218 specific phobia, animal, and F40.248 specific phobia, situational).

Specifiers

It is common for individuals to have multiple specific phobias. The average individual with specific phobia fears three objects or situations, and approximately 75% of individuals with specific phobia fear more than one situation or object. In such cases, multiple specific phobia diagnoses, each with its own diagnostic code reflecting the phobic stimulus, would need to be given. For example, if an individual fears thunderstorms and flying, then two diagnoses would be given: specific phobia, natural environment, and specific phobia, situational.

Diagnostic Features

A key feature of this disorder is that the fear or anxiety is circumscribed to the presence of a particular situation or object (Criterion A), which may be termed the *phobic stimulus*. The categories of feared situations or objects are provided as specifiers. Many individuals fear objects or situations from more than one category, or phobic stimulus. For the diagnosis of specific phobia, the response must differ from normal, transient fears that commonly occur in the population. To meet the criteria for a diagnosis, the fear or anxiety must be intense or severe (i.e., "marked") (Criterion A). The amount of fear experienced may vary with proximity to the feared object or situation and may occur in anticipation of or in the actual presence of the object or situation. Also, the fear or anxiety may take the form of a full or limited symptom panic attack (i.e., expected panic attack). Another characteristic of specific phobias is that fear or anxiety is evoked nearly every time the individual comes into contact with the phobic stimulus (Criterion B). Thus, an individual who becomes anxious only occasionally upon being confronted with the situation or object (e.g., becomes anxious when flying only on one out of every five airplane flights) would not be diagnosed with specific phobia. However, the degree of fear or anxiety expressed may vary (from anticipatory anxiety to a full panic attack) across different occasions of encountering the phobic object or situation because of various contextual factors such as the presence of others, duration of exposure, and other threatening elements such as turbulence on a flight for individuals who fear flying. Fear and anxiety are often expressed differently between children and adults. Also, the fear or anxiety occurs as soon as the phobic object or situation is encountered (i.e., immediately rather than being delayed).

The individual actively avoids the situation, or if he or she either is unable or decides not to avoid it, the situation or object evokes intense fear or anxiety (Criterion C). *Active avoidance* means the individual intentionally behaves in ways that are designed to prevent or minimize contact with phobic objects or situations (e.g., takes tunnels instead of bridges on daily commute to work for fear of heights; avoids entering a dark room for fear of spiders; avoids accepting a job in a locale where a phobic stimulus is more common). Avoid-

ance behaviors are often obvious (e.g., an individual who fears blood refusing to go to the doctor) but are sometimes less obvious (e.g., an individual who fears snakes refusing to look at pictures that resemble the form or shape of snakes). Many individuals with specific phobias have suffered over many years and have changed their living circumstances in ways designed to avoid the phobic object or situation as much as possible (e.g., an individual diagnosed with specific phobia, animal, who moves to reside in an area devoid of the particular feared animal). Therefore, they no longer experience fear or anxiety in their daily life. In such instances, avoidance behaviors or ongoing refusal to engage in activities that would involve exposure to the phobic object or situation (e.g., repeated refusal to accept offers for work-related travel because of fear of flying) may be helpful in confirming the diagnosis in the absence of overt anxiety or panic.

The fear or anxiety is out of proportion to the actual danger that the object or situation poses, or more intense than is deemed necessary (Criterion D). Although individuals with specific phobia often recognize their reactions as disproportionate, they tend to overestimate the danger in their feared situations, and thus the judgment of being out of proportion is made by the clinician. The individual's sociocultural context should also be taken into account. For example, fears of the dark may be reasonable in a context of ongoing violence, and fear of insects may be more disproportionate in settings where insects are consumed in the diet. The fear, anxiety, or avoidance is persistent, typically lasting for 6 months or more (Criterion E), which helps distinguish the disorder from transient fears that are common in the population, particularly among children. However, the duration criterion should be used as a general guide, with allowance for some degree of flexibility. The specific phobia must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning in order for the disorder to be diagnosed (Criterion F).

Associated Features Supporting Diagnosis

Individuals with specific phobia typically experience an increase in physiological arousal in anticipation of or during exposure to a phobic object or situation. However, the physiological response to the feared situation or object varies. Whereas individuals with situational, natural environment, and animal specific phobias are likely to show sympathetic nervous system arousal, individuals with blood-injection-injury specific phobia often demonstrate a vasovagal fainting or near-fainting response that is marked by initial brief acceleration of heart rate and elevation of blood pressure followed by a deceleration of heart rate and a drop in blood pressure. Current neural systems models for specific phobia emphasize the amygdala and related structures, much as in other anxiety disorders.

Prevalence

In the United States, the 12-month community prevalence estimate for specific phobia is approximately 7%–9%. Prevalence rates in European countries are largely similar to those in the United States (e.g., about 6%), but rates are generally lower in Asian, African, and Latin American countries (2%–4%). Prevalence rates are approximately 5% in children and are approximately 16% in 13- to 17-year-olds. Prevalence rates are lower in older individuals (about 3%–5%), possibly reflecting diminishing severity to subclinical levels. Females are more frequently affected than males, at a rate of approximately 2:1, although rates vary across different phobic stimuli. That is, animal, natural environment, and situational specific phobias are predominantly experienced by females, whereas blood-injection-injury phobia is experienced nearly equally by both genders.

Development and Course

Specific phobia sometimes develops following a traumatic event (e.g., being attacked by an animal or stuck in an elevator), observation of others going through a traumatic event (e.g.,

watching someone drown), an unexpected panic attack in the to be feared situation (e.g., an unexpected panic attack while on the subway), or informational transmission (e.g., extensive media coverage of a plane crash). However, many individuals with specific phobia are unable to recall the specific reason for the onset of their phobias. Specific phobia usually develops in early childhood, with the majority of cases developing prior to age 10 years. The median age at onset is between 7 and 11 years, with the mean at about 10 years. Situational specific phobias tend to have a later age at onset than natural environment, animal, or blood-injection-injury specific phobias. Specific phobias that develop in childhood and adolescence are likely to wax and wane during that period. However, phobias that do persist into adulthood are unlikely to remit for the majority of individuals.

When specific phobia is being diagnosed in children, two issues should be considered. First, young children may express their fear and anxiety by crying, tantrums, freezing, or clinging. Second, young children typically are not able to understand the concept of avoidance. Therefore, the clinician should assemble additional information from parents, teachers, or others who know the child well. Excessive fears are quite common in young children but are usually transitory and only mildly impairing and thus considered developmentally appropriate. In such cases a diagnosis of specific phobia would not be made. When the diagnosis of specific phobia is being considered in a child, it is important to assess the degree of impairment and the duration of the fear, anxiety, or avoidance, and whether it is typical for the child's particular developmental stage.

Although the prevalence of specific phobia is lower in older populations, it remains one of the more commonly experienced disorders in late life. Several issues should be considered when diagnosing specific phobia in older populations. First, older individuals may be more likely to endorse natural environment specific phobias, as well as phobias of falling. Second, specific phobia (like all anxiety disorders) tends to co-occur with medical concerns in older individuals, including coronary heart disease and chronic obstructive pulmonary disease. Third, older individuals may be more likely to attribute the symptoms of anxiety to medical conditions. Fourth, older individuals may be more likely to manifest anxiety in an atypical manner (e.g., involving symptoms of both anxiety and depression) and thus be more likely to warrant a diagnosis of unspecified anxiety disorder. Additionally, the presence of specific phobia in older adults is associated with decreased quality of life and may serve as a risk factor for major neurocognitive disorder.

Although most specific phobias develop in childhood and adolescence, it is possible for a specific phobia to develop at any age, often as the result of experiences that are traumatic. For example, phobias of choking almost always follow a near-choking event at any age.

Risk and Prognostic Factors

Temperamental. Temperamental risk factors for specific phobia, such as negative affectivity (neuroticism) or behavioral inhibition, are risk factors for other anxiety disorders as well.

Environmental. Environmental risk factors for specific phobias, such as parental overprotectiveness, parental loss and separation, and physical and sexual abuse, tend to predict other anxiety disorders as well. As noted earlier, negative or traumatic encounters with the feared object or situation sometimes (but not always) precede the development of specific phobia.

Genetic and physiological. There may be a genetic susceptibility to a certain category of specific phobia (e.g., an individual with a first-degree relative with a specific phobia of animals is significantly more likely to have the same specific phobia than any other category of phobia). Individuals with blood-injection-injury phobia show a unique propensity to vasovagal syncope (fainting) in the presence of the phobic stimulus.

Culture-Related Diagnostic Issues

In the United States, Asians and Latinos report significantly lower rates of specific phobia than non-Latino whites, African Americans, and Native Americans. In addition to having lower prevalence rates of specific phobia, some countries outside of the United States, particularly Asian and African countries, show differing phobia content, age at onset, and gender ratios.

Suicide Risk

Individuals with specific phobia are up to 60% more likely to make a suicide attempt than are individuals without the diagnosis. However, it is likely that these elevated rates are primarily due to comorbidity with personality disorders and other anxiety disorders.

Functional Consequences of Specific Phobia

Individuals with specific phobia show similar patterns of impairment in psychosocial functioning and decreased quality of life as individuals with other anxiety disorders and alcohol and substance use disorders, including impairments in occupational and interpersonal functioning. In older adults, impairment may be seen in caregiving duties and volunteer activities. Also, fear of falling in older adults can lead to reduced mobility and reduced physical and social functioning, and may lead to receiving formal or informal home support. The distress and impairment caused by specific phobias tend to increase with the number of feared objects and situations. Thus, an individual who fears four objects or situations is likely to have more impairment in his or her occupational and social roles and a lower quality of life than an individual who fears only one object or situation. Individuals with blood-injection-injury specific phobia are often reluctant to obtain medical care even when a medical concern is present. Additionally, fear of vomiting and choking may substantially reduce dietary intake.

Differential Diagnosis

Agoraphobia. Situational specific phobia may resemble agoraphobia in its clinical presentation, given the overlap in feared situations (e.g., flying, enclosed places, elevators). If an individual fears only one of the agoraphobia situations, then specific phobia, situational, may be diagnosed. If two or more agoraphobic situations are feared, a diagnosis of agoraphobia is likely warranted. For example, an individual who fears airplanes and elevators (which overlap with the "public transportation" agoraphobic situation) but does not fear other agoraphobic situations would be diagnosed with specific phobia, situational, whereas an individual who fears airplanes, elevators, and crowds (which overlap with two agoraphobic situations, "using public transportation" and "standing in line and or being in a crowd") would be diagnosed with agoraphobia. Criterion B of agoraphobia (the situations are feared or avoided "because of thoughts that escape might be difficult or help might not be available in the event of developing panic-like symptoms or other incapacitating or embarrassing symptoms") can also be useful in differentiating agoraphobia from specific phobia. If the situations are feared for other reasons, such as fear of being harmed directly by the object or situations (e.g., fear of the plane crashing, fear of the animal biting), a specific phobia diagnosis may be more appropriate.

Social anxiety disorder. If the situations are feared because of negative evaluation, social anxiety disorder should be diagnosed instead of specific phobia.

Separation anxiety disorder. If the situations are feared because of separation from a primary caregiver or attachment figure, separation anxiety disorder should be diagnosed instead of specific phobia.

Panic disorder. Individuals with specific phobia may experience panic attacks when confronted with their feared situation or object. A diagnosis of specific phobia would be given if the panic attacks only occurred in response to the specific object or situation, whereas a diagnosis of panic disorder would be given if the individual also experienced panic attacks that were unexpected (i.e., not in response to the specific phobia object or situation).

Obsessive-compulsive disorder. If an individual's primary fear or anxiety is of an object or situation as a result of obsessions (e.g., fear of blood due to obsessive thoughts about contamination from blood-borne pathogens [i.e., HIV]; fear of driving due to obsessive images of harming others), and if other diagnostic criteria for obsessive-compulsive disorder are met, then obsessive-compulsive disorder should be diagnosed.

Trauma- and stressor-related disorders. If the phobia develops following a traumatic event, posttraumatic stress disorder (PTSD) should be considered as a diagnosis. However, traumatic events can precede the onset of PTSD and specific phobia. In this case, a diagnosis of specific phobia would be assigned only if all of the criteria for PTSD are not met.

Eating disorders. A diagnosis of specific phobia is not given if the avoidance behavior is exclusively limited to avoidance of food and food-related cues, in which case a diagnosis of anorexia nervosa or bulimia nervosa should be considered.

Schizophrenia spectrum and other psychotic disorders. When the fear and avoidance are due to delusional thinking (as in schizophrenia or other schizophrenia spectrum and other psychotic disorders), a diagnosis of specific phobia is not warranted.

Comorbidity

Specific phobia is rarely seen in medical-clinical settings in the absence of other psychopathology and is more frequently seen in nonmedical mental health settings. Specific phobia is frequently associated with a range of other disorders, especially depression in older adults. Because of early onset, specific phobia is typically the temporally primary disorder. Individuals with specific phobia are at increased risk for the development of other disorders, including other anxiety disorders, depressive and bipolar disorders, substance-related disorders, somatic symptom and related disorders, and personality disorders (particularly dependent personality disorder).

Social Anxiety Disorder (Social Phobia)

Diagnostic Criteria **300.23 (F40.10)**

A. Marked fear or anxiety about one or more social situations in which the individual is exposed to possible scrutiny by others. Examples include social interactions (e.g., having a conversation, meeting unfamiliar people), being observed (e.g., eating or drinking), and performing in front of others (e.g., giving a speech).

   Note: In children, the anxiety must occur in peer settings and not just during interactions with adults.

B. The individual fears that he or she will act in a way or show anxiety symptoms that will be negatively evaluated (i.e., will be humiliating or embarrassing; will lead to rejection or offend others).

C. The social situations almost always provoke fear or anxiety.

   Note: In children, the fear or anxiety may be expressed by crying, tantrums, freezing, clinging, shrinking, or failing to speak in social situations.

D. The social situations are avoided or endured with intense fear or anxiety.

E. The fear or anxiety is out of proportion to the actual threat posed by the social situation and to the sociocultural context.
F. The fear, anxiety, or avoidance is persistent, typically lasting for 6 months or more.
G. The fear, anxiety, or avoidance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
H. The fear, anxiety, or avoidance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.
I. The fear, anxiety, or avoidance is not better explained by the symptoms of another mental disorder, such as panic disorder, body dysmorphic disorder, or autism spectrum disorder.
J. If another medical condition (e.g., Parkinson's disease, obesity, disfigurement from burns or injury) is present, the fear, anxiety, or avoidance is clearly unrelated or is excessive.

Specify if:

Performance only: If the fear is restricted to speaking or performing in public.

Specifiers

Individuals with the performance only type of social anxiety disorder have performance fears that are typically most impairing in their professional lives (e.g., musicians, dancers, performers, athletes) or in roles that require regular public speaking. Performance fears may also manifest in work, school, or academic settings in which regular public presentations are required. Individuals with performance only social anxiety disorder do not fear or avoid nonperformance social situations.

Diagnostic Features

The essential feature of social anxiety disorder is a marked, or intense, fear or anxiety of social situations in which the individual may be scrutinized by others. In children the fear or anxiety must occur in peer settings and not just during interactions with adults (Criterion A). When exposed to such social situations, the individual fears that he or she will be negatively evaluated. The individual is concerned that he or she will be judged as anxious, weak, crazy, stupid, boring, intimidating, dirty, or unlikable. The individual fears that he or she will act or appear in a certain way or show anxiety symptoms, such as blushing, trembling, sweating, stumbling over one's words, or staring, that will be negatively evaluated by others (Criterion B). Some individuals fear offending others or being rejected as a result. Fear of offending others—for example, by a gaze or by showing anxiety symptoms—may be the predominant fear in individuals from cultures with strong collectivistic orientations. An individual with fear of trembling of the hands may avoid drinking, eating, writing, or pointing in public; an individual with fear of sweating may avoid shaking hands or eating spicy foods; and an individual with fear of blushing may avoid public performance, bright lights, or discussion about intimate topics. Some individuals fear and avoid urinating in public restrooms when other individuals are present (i.e., paruresis, or "shy bladder syndrome").

The social situations almost always provoke fear or anxiety (Criterion C). Thus, an individual who becomes anxious only occasionally in the social situation(s) would not be diagnosed with social anxiety disorder. However, the degree and type of fear and anxiety may vary (e.g., anticipatory anxiety, a panic attack) across different occasions. The anticipatory anxiety may occur sometimes far in advance of upcoming situations (e.g., worrying every day for weeks before attending a social event, repeating a speech for days in advance). In children, the fear or anxiety may be expressed by crying, tantrums, freezing, clinging, or shrinking in social situations. The individual will often avoid the feared social situations. Alternatively, the situations are endured with intense fear or anxiety (Criterion D). Avoid-

ance can be extensive (e.g., not going to parties, refusing school) or subtle (e.g., overpreparing the text of a speech, diverting attention to others, limiting eye contact).

The fear or anxiety is judged to be out of proportion to the actual risk of being negatively evaluated or to the consequences of such negative evaluation (Criterion E). Sometimes, the anxiety may not be judged to be excessive, because it is related to an actual danger (e.g., being bullied or tormented by others). However, individuals with social anxiety disorder often overestimate the negative consequences of social situations, and thus the judgment of being out of proportion is made by the clinician. The individual's sociocultural context needs to be taken into account when this judgment is being made. For example, in certain cultures, behavior that might otherwise appear socially anxious may be considered appropriate in social situations (e.g., might be seen as a sign of respect).

The duration of the disturbance is typically at least 6 months (Criterion F). This duration threshold helps distinguish the disorder from transient social fears that are common, particularly among children and in the community. However, the duration criterion should be used as a general guide, with allowance for some degree of flexibility. The fear, anxiety, and avoidance must interfere significantly with the individual's normal routine, occupational or academic functioning, or social activities or relationships, or must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion G). For example, an individual who is afraid to speak in public would not receive a diagnosis of social anxiety disorder if this activity is not routinely encountered on the job or in classroom work, and if the individual is not significantly distressed about it. However, if the individual avoids, or is passed over for, the job or education he or she really wants because of social anxiety symptoms, Criterion G is met.

Associated Features Supporting Diagnosis

Individuals with social anxiety disorder may be inadequately assertive or excessively submissive or, less commonly, highly controlling of the conversation. They may show overly rigid body posture or inadequate eye contact, or speak with an overly soft voice. These individuals may be shy or withdrawn, and they may be less open in conversations and disclose little about themselves. They may seek employment in jobs that do not require social contact, although this is not the case for individuals with social anxiety disorder, performance only. They may live at home longer. Men may be delayed in marrying and having a family, whereas women who would want to work outside the home may live a life as homemaker and mother. Self-medication with substances is common (e.g., drinking before going to a party). Social anxiety among older adults may also include exacerbation of symptoms of medical illnesses, such as increased tremor or tachycardia. Blushing is a hallmark physical response of social anxiety disorder.

Prevalence

The 12-month prevalence estimate of social anxiety disorder for the United States is approximately 7%. Lower 12-month prevalence estimates are seen in much of the world using the same diagnostic instrument, clustering around 0.5%–2.0%; median prevalence in Europe is 2.3%. The 12-month prevalence rates in children and adolescents are comparable to those in adults. Prevalence rates decrease with age. The 12-month prevalence for older adults ranges from 2% to 5%. In general, higher rates of social anxiety disorder are found in females than in males in the general population (with odds ratios ranging from 1.5 to 2.2), and the gender difference in prevalence is more pronounced in adolescents and young adults. Gender rates are equivalent or slightly higher for males in clinical samples, and it is assumed that gender roles and social expectations play a significant role in explaining the heightened help-seeking behavior in male patients. Prevalence in the United States is higher in American Indians and lower in persons of Asian, Latino, African American, and Afro-Caribbean descent compared with non-Hispanic whites.

Development and Course

Median age at onset of social anxiety disorder in the United States is 13 years, and 75% of individuals have an age at onset between 8 and 15 years. The disorder sometimes emerges out of a childhood history of social inhibition or shyness in U.S. and European studies. Onset can also occur in early childhood. Onset of social anxiety disorder may follow a stressful or humiliating experience (e.g., being bullied, vomiting during a public speech), or it may be insidious, developing slowly. First onset in adulthood is relatively rare and is more likely to occur after a stressful or humiliating event or after life changes that require new social roles (e.g., marrying someone from a different social class, receiving a job promotion). Social anxiety disorder may diminish after an individual with fear of dating marries and may reemerge after divorce. Among individuals presenting to clinical care, the disorder tends to be particularly persistent.

Adolescents endorse a broader pattern of fear and avoidance, including of dating, compared with younger children. Older adults express social anxiety at lower levels but across a broader range of situations, whereas younger adults express higher levels of social anxiety for specific situations. In older adults, social anxiety may concern disability due to declining sensory functioning (hearing, vision) or embarrassment about one's appearance (e.g., tremor as a symptom of Parkinson's disease) or functioning due to medical conditions, incontinence, or cognitive impairment (e.g., forgetting people's names). In the community approximately 30% of individuals with social anxiety disorder experience remission of symptoms within 1 year, and about 50% experience remission within a few years. For approximately 60% of individuals without a specific treatment for social anxiety disorder, the course takes several years or longer.

Detection of social anxiety disorder in older adults may be challenging because of several factors, including a focus on somatic symptoms, comorbid medical illness, limited insight, changes to social environment or roles that may obscure impairment in social functioning, or reticence about describing psychological distress.

Risk and Prognostic Factors

Temperamental. Underlying traits that predispose individuals to social anxiety disorder include behavioral inhibition and fear of negative evaluation.

Environmental. There is no causative role of increased rates of childhood maltreatment or other early-onset psychosocial adversity in the development of social anxiety disorder. However, childhood maltreatment and adversity are risk factors for social anxiety disorder.

Genetic and physiological. Traits predisposing individuals to social anxiety disorder, such as behavioral inhibition, are strongly genetically influenced. The genetic influence is subject to gene-environment interaction; that is, children with high behavioral inhibition are more susceptible to environmental influences, such as socially anxious modeling by parents. Also, social anxiety disorder is heritable (but performance-only anxiety less so). First-degree relatives have a two to six times greater chance of having social anxiety disorder, and liability to the disorder involves the interplay of disorder-specific (e.g., fear of negative evaluation) and nonspecific (e.g., neuroticism) genetic factors.

Culture-Related Diagnostic Issues

The syndrome of *taijin kyofusho* (e.g., in Japan and Korea) is often characterized by social-evaluative concerns, fulfilling criteria for social anxiety disorder, that are associated with the fear that the individual makes *other* people uncomfortable (e.g., "My gaze upsets people so they look away and avoid me"), a fear that is at times experienced with delusional intensity. This symptom may also be found in non-Asian settings. Other presentations of *taijin kyofusho* may fulfill criteria for body dysmorphic disorder or delusional disorder.

Immigrant status is associated with significantly lower rates of social anxiety disorder in both Latino and non-Latino white groups. Prevalence rates of social anxiety disorder may not be in line with self-reported social anxiety levels in the same culture—that is, societies with strong collectivistic orientations may report high levels of social anxiety but low prevalence of social anxiety disorder.

Gender-Related Diagnostic Issues

Females with social anxiety disorder report a greater number of social fears and comorbid depressive, bipolar, and anxiety disorders, whereas males are more likely to fear dating, have oppositional defiant disorder or conduct disorder, and use alcohol and illicit drugs to relieve symptoms of the disorder. Paruresis is more common in males.

Functional Consequences of Social Anxiety Disorder

Social anxiety disorder is associated with elevated rates of school dropout and with decreased well-being, employment, workplace productivity, socioeconomic status, and quality of life. Social anxiety disorder is also associated with being single, unmarried, or divorced and with not having children, particularly among men. In older adults, there may be impairment in caregiving duties and volunteer activities. Social anxiety disorder also impedes leisure activities. Despite the extent of distress and social impairment associated with social anxiety disorder, only about half of individuals with the disorder in Western societies ever seek treatment, and they tend to do so only after 15–20 years of experiencing symptoms. Not being employed is a strong predictor for the persistence of social anxiety disorder.

Differential Diagnosis

Normative shyness. Shyness (i.e., social reticence) is a common personality trait and is not by itself pathological. In some societies, shyness is even evaluated positively. However, when there is a significant adverse impact on social, occupational, and other important areas of functioning, a diagnosis of social anxiety disorder should be considered, and when full diagnostic criteria for social anxiety disorder are met, the disorder should be diagnosed. Only a minority (12%) of self-identified shy individuals in the United States have symptoms that meet diagnostic criteria for social anxiety disorder.

Agoraphobia. Individuals with agoraphobia may fear and avoid social situations (e.g., going to a movie) because escape might be difficult or help might not be available in the event of incapacitation or panic-like symptoms, whereas individuals with social anxiety disorder are most fearful of scrutiny by others. Moreover, individuals with social anxiety disorder are likely to be calm when left entirely alone, which is often not the case in agoraphobia.

Panic disorder. Individuals with social anxiety disorder may have panic attacks, but the concern is about fear of negative evaluation, whereas in panic disorder the concern is about the panic attacks themselves.

Generalized anxiety disorder. Social worries are common in generalized anxiety disorder, but the focus is more on the nature of ongoing relationships rather than on fear of negative evaluation. Individuals with generalized anxiety disorder, particularly children, may have excessive worries about the quality of their social performance, but these worries also pertain to nonsocial performance and when the individual is not being evaluated by others. In social anxiety disorder, the worries focus on social performance and others' evaluation.

Separation anxiety disorder. Individuals with separation anxiety disorder may avoid social settings (including school refusal) because of concerns about being separated from attachment figures or, in children, about requiring the presence of a parent when it is not developmentally appropriate. Individuals with separation anxiety disorder are usually comfortable in social settings when their attachment figure is present or when they are at

home, whereas those with social anxiety disorder may be uncomfortable when social situations occur at home or in the presence of attachment figures.

Specific phobias. Individuals with specific phobias may fear embarrassment or humiliation (e.g., embarrassment about fainting when they have their blood drawn), but they do not generally fear negative evaluation in other social situations.

Selective mutism. Individuals with selective mutism may fail to speak because of fear of negative evaluation, but they do not fear negative evaluation in social situations where no speaking is required (e.g., nonverbal play).

Major depressive disorder. Individuals with major depressive disorder may be concerned about being negatively evaluated by others because they feel they are bad or not worthy of being liked. In contrast, individuals with social anxiety disorder are worried about being negatively evaluated because of certain social behaviors or physical symptoms.

Body dysmorphic disorder. Individuals with body dysmorphic disorder are preoccupied with one or more perceived defects or flaws in their physical appearance that are not observable or appear slight to others; this preoccupation often causes social anxiety and avoidance. If their social fears and avoidance are caused only by their beliefs about their appearance, a separate diagnosis of social anxiety disorder is not warranted.

Delusional disorder. Individuals with delusional disorder may have nonbizarre delusions and/or hallucinations related to the delusional theme that focus on being rejected by or offending others. Although extent of insight into beliefs about social situations may vary, many individuals with social anxiety disorder have good insight that their beliefs are out of proportion to the actual threat posed by the social situation.

Autism spectrum disorder. Social anxiety and social communication deficits are hallmarks of autism spectrum disorder. Individuals with social anxiety disorder typically have adequate age-appropriate social relationships and social communication capacity, although they may appear to have impairment in these areas when first interacting with unfamiliar peers or adults.

Personality disorders. Given its frequent onset in childhood and its persistence into and through adulthood, social anxiety disorder may resemble a personality disorder. The most apparent overlap is with avoidant personality disorder. Individuals with avoidant personality disorder have a broader avoidance pattern than those with social anxiety disorder. Nonetheless, social anxiety disorder is typically more comorbid with avoidant personality disorder than with other personality disorders, and avoidant personality disorder is more comorbid with social anxiety disorder than with other anxiety disorders.

Other mental disorders. Social fears and discomfort can occur as part of schizophrenia, but other evidence for psychotic symptoms is usually present. In individuals with an eating disorder, it is important to determine that fear of negative evaluation about eating disorder symptoms or behaviors (e.g., purging and vomiting) is not the sole source of social anxiety before applying a diagnosis of social anxiety disorder. Similarly, obsessive-compulsive disorder may be associated with social anxiety, but the additional diagnosis of social anxiety disorder is used only when social fears and avoidance are independent of the foci of the obsessions and compulsions.

Other medical conditions. Medical conditions may produce symptoms that may be embarrassing (e.g. trembling in Parkinson's disease). When the fear of negative evaluation due to other medical conditions is excessive, a diagnosis of social anxiety disorder should be considered.

Oppositional defiant disorder. Refusal to speak due to opposition to authority figures should be differentiated from failure to speak due to fear of negative evaluation.

Comorbidity

Social anxiety disorder is often comorbid with other anxiety disorders, major depressive disorder, and substance use disorders, and the onset of social anxiety disorder generally precedes that of the other disorders, except for specific phobia and separation anxiety disorder. Chronic social isolation in the course of a social anxiety disorder may result in major depressive disorder. Comorbidity with depression is high also in older adults. Substances may be used as self-medication for social fears, but the symptoms of substance intoxication or withdrawal, such as trembling, may also be a source of (further) social fear. Social anxiety disorder is frequently comorbid with bipolar disorder or body dysmorphic disorder; for example, an individual has body dysmorphic disorder concerning a preoccupation with a slight irregularity of her nose, as well as social anxiety disorder because of a severe fear of sounding unintelligent. The more generalized form of social anxiety disorder, but not social anxiety disorder, performance only, is often comorbid with avoidant personality disorder. In children, comorbidities with high-functioning autism and selective mutism are common.

Panic Disorder

Diagnostic Criteria **300.01 (F41.0)**

A. Recurrent unexpected panic attacks. A panic attack is an abrupt surge of intense fear or intense discomfort that reaches a peak within minutes, and during which time four (or more) of the following symptoms occur:

Note: The abrupt surge can occur from a calm state or an anxious state.

1. Palpitations, pounding heart, or accelerated heart rate.
2. Sweating.
3. Trembling or shaking.
4. Sensations of shortness of breath or smothering.
5. Feelings of choking.
6. Chest pain or discomfort.
7. Nausea or abdominal distress.
8. Feeling dizzy, unsteady, light-headed, or faint.
9. Chills or heat sensations.
10. Paresthesias (numbness or tingling sensations).
11. Derealization (feelings of unreality) or depersonalization (being detached from oneself).
12. Fear of losing control or "going crazy."
13. Fear of dying.

Note: Culture-specific symptoms (e.g., tinnitus, neck soreness, headache, uncontrollable screaming or crying) may be seen. Such symptoms should not count as one of the four required symptoms.

B. At least one of the attacks has been followed by 1 month (or more) of one or both of the following:

1. Persistent concern or worry about additional panic attacks or their consequences (e.g., losing control, having a heart attack, "going crazy").
2. A significant maladaptive change in behavior related to the attacks (e.g., behaviors designed to avoid having panic attacks, such as avoidance of exercise or unfamiliar situations).

C. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g., hyperthyroidism, cardiopulmonary disorders).
D. The disturbance is not better explained by another mental disorder (e.g., the panic attacks do not occur only in response to feared social situations, as in social anxiety disorder; in response to circumscribed phobic objects or situations, as in specific phobia; in response to obsessions, as in obsessive-compulsive disorder; in response to reminders of traumatic events, as in posttraumatic stress disorder; or in response to separation from attachment figures, as in separation anxiety disorder).

Diagnostic Features

Panic disorder refers to recurrent unexpected panic attacks (Criterion A). A panic attack is an abrupt surge of intense fear or intense discomfort that reaches a peak within minutes, and during which time four or more of a list of 13 physical and cognitive symptoms occur. The term *recurrent* literally means more than one unexpected panic attack. The term *unexpected* refers to a panic attack for which there is no obvious cue or trigger at the time of occurrence—that is, the attack appears to occur from out of the blue, such as when the individual is relaxing or emerging from sleep (nocturnal panic attack). In contrast, *expected* panic attacks are attacks for which there is an obvious cue or trigger, such as a situation in which panic attacks typically occur. The determination of whether panic attacks are expected or unexpected is made by the clinician, who makes this judgment based on a combination of careful questioning as to the sequence of events preceding or leading up to the attack and the individual's own judgment of whether or not the attack seemed to occur for no apparent reason. Cultural interpretations may influence the assignment of panic attacks as expected or unexpected (see section "Culture-Related Diagnostic Issues" for this disorder). In the United States and Europe, approximately one-half of individuals with panic disorder have expected panic attacks as well as unexpected panic attacks. Thus, the presence of expected panic attacks does not rule out the diagnosis of panic disorder. For more details regarding expected versus unexpected panic attacks, see the text accompanying panic attacks (pp. 214–217).

The frequency and severity of panic attacks vary widely. In terms of frequency, there may be moderately frequent attacks (e.g., one per week) for months at a time, or short bursts of more frequent attacks (e.g., daily) separated by weeks or months without any attacks or with less frequent attacks (e.g., two per month) over many years. Persons who have infrequent panic attacks resemble persons with more frequent panic attacks in terms of panic attack symptoms, demographic characteristics, comorbidity with other disorders, family history, and biological data. In terms of severity, individuals with panic disorder may have both full-symptom (four or more symptoms) and limited-symptom (fewer than four symptoms) attacks, and the number and type of panic attack symptoms frequently differ from one panic attack to the next. However, more than one unexpected full-symptom panic attack is required for the diagnosis of panic disorder.

The worries about panic attacks or their consequences usually pertain to physical concerns, such as worry that panic attacks reflect the presence of life-threatening illnesses (e.g., cardiac disease, seizure disorder); social concerns, such as embarrassment or fear of being judged negatively by others because of visible panic symptoms; and concerns about mental functioning, such as "going crazy" or losing control (Criterion B). The maladaptive changes in behavior represent attempts to minimize or avoid panic attacks or their consequences. Examples include avoiding physical exertion, reorganizing daily life to ensure that help is available in the event of a panic attack, restricting usual daily activities, and avoiding agoraphobia-type situations, such as leaving home, using public transportation, or shopping. If agoraphobia is present, a separate diagnosis of agoraphobia is given.

Associated Features Supporting Diagnosis

One type of unexpected panic attack is a *nocturnal* panic attack (i.e., waking from sleep in a state of panic, which differs from panicking after fully waking from sleep). In the United States, this type of panic attack has been estimated to occur at least one time in roughly one-quarter to one-third of individuals with panic disorder, of whom the majority also have daytime panic attacks. In addition to worry about panic attacks and their consequences, many individuals with panic disorder report constant or intermittent feelings of anxiety that are more broadly related to health and mental health concerns. For example, individuals with panic disorder often anticipate a catastrophic outcome from a mild physical symptom or medication side effect (e.g., thinking that they may have heart disease or that a headache means presence of a brain tumor). Such individuals often are relatively intolerant of medication side effects. In addition, there may be pervasive concerns about abilities to complete daily tasks or withstand daily stressors, excessive use of drugs (e.g., alcohol, prescribed medications or illicit drugs) to control panic attacks, or extreme behaviors aimed at controlling panic attacks (e.g., severe restrictions on food intake or avoidance of specific foods or medications because of concerns about physical symptoms that provoke panic attacks).

Prevalence

In the general population, the 12-month prevalence estimate for panic disorder across the United States and several European countries is about 2%–3% in adults and adolescents. In the United States, significantly lower rates of panic disorder are reported among Latinos, African Americans, Caribbean blacks, and Asian Americans, compared with non-Latino whites; American Indians, by contrast, have significantly higher rates. Lower estimates have been reported for Asian, African, and Latin American countries, ranging from 0.1% to 0.8%. Females are more frequently affected than males, at a rate of approximately 2:1. The gender differentiation occurs in adolescence and is already observable before age 14 years. Although panic attacks occur in children, the overall prevalence of panic disorder is low before age 14 years (<0.4%). The rates of panic disorder show a gradual increase during adolescence, particularly in females, and possibly following the onset of puberty, and peak during adulthood. The prevalence rates decline in older individuals (i.e., 0.7% in adults over the age of 64), possibly reflecting diminishing severity to subclinical levels.

Development and Course

The median age at onset for panic disorder in the United States is 20–24 years. A small number of cases begin in childhood, and onset after age 45 years is unusual but can occur. The usual course, if the disorder is untreated, is chronic but waxing and waning. Some individuals may have episodic outbreaks with years of remission in between, and others may have continuous severe symptomatology. Only a minority of individuals have full remission without subsequent relapse within a few years. The course of panic disorder typically is complicated by a range of other disorders, in particular other anxiety disorders, depressive disorders, and substance use disorders (see section "Comorbidity" for this disorder).

Although panic disorder is very rare in childhood, first occurrence of "fearful spells" is often dated retrospectively back to childhood. As in adults, panic disorder in adolescents tends to have a chronic course and is frequently comorbid with other anxiety, depressive, and bipolar disorders. To date, no differences in the clinical presentation between adolescents and adults have been found. However, adolescents may be less worried about additional panic attacks than are young adults. Lower prevalence of panic disorder in older adults appears to be attributable to age-related "dampening" of the autonomic nervous system response. Many older individuals with "panicky feelings" are observed to have a "hybrid" of limited-symptom panic attacks and generalized anxiety. Also, older adults

tend to attribute their panic attacks to certain stressful situations, such as a medical procedure or social setting. Older individuals may retrospectively endorse explanations for the panic attack (which would preclude the diagnosis of panic disorder), even if an attack might actually have been unexpected in the moment (and thus qualify as the basis for a panic disorder diagnosis). This may result in under-endorsement of unexpected panic attacks in older individuals. Thus, careful questioning of older adults is required to assess whether panic attacks were expected before entering the situation, so that unexpected panic attacks and the diagnosis of panic disorder are not overlooked.

While the low rate of panic disorder in children could relate to difficulties in symptom reporting, this seems unlikely given that children are capable of reporting intense fear or panic in relation to separation and to phobic objects or phobic situations. Adolescents might be less willing than adults to openly discuss panic attacks. Therefore, clinicians should be aware that unexpected panic attacks do occur in adolescents, much as they do in adults, and be attuned to this possibility when encountering adolescents presenting with episodes of intense fear or distress.

Risk and Prognostic Factors

Temperamental. Negative affectivity (neuroticism) (i.e., proneness to experiencing negative emotions) and anxiety sensitivity (i.e., the disposition to believe that symptoms of anxiety are harmful) are risk factors for the onset of panic attacks and, separately, for worry about panic, although their risk status for the diagnosis of panic disorder is unknown. History of "fearful spells" (i.e., limited-symptom attacks that do not meet full criteria for a panic attack) may be a risk factor for later panic attacks and panic disorder. Although separation anxiety in childhood, especially when severe, may precede the later development of panic disorder, it is not a consistent risk factor.

Environmental. Reports of childhood experiences of sexual and physical abuse are more common in panic disorder than in certain other anxiety disorders. Smoking is a risk factor for panic attacks and panic disorder. Most individuals report identifiable stressors in the months before their first panic attack (e.g., interpersonal stressors and stressors related to physical well-being, such as negative experiences with illicit or prescription drugs, disease, or death in the family).

Genetic and physiological. It is believed that multiple genes confer vulnerability to panic disorder. However, the exact genes, gene products, or functions related to the genetic regions implicated remain unknown. Current neural systems models for panic disorder emphasize the amygdala and related structures, much as in other anxiety disorders. There is an increased risk for panic disorder among offspring of parents with anxiety, depressive, and bipolar disorders. Respiratory disturbance, such as asthma, is associated with panic disorder, in terms of past history, comorbidity, and family history.

Culture-Related Diagnostic Issues

The rate of fears about mental and somatic symptoms of anxiety appears to vary across cultures and may influence the rate of panic attacks and panic disorder. Also, cultural expectations may influence the classification of panic attacks as expected or unexpected. For example, a Vietnamese individual who has a panic attack after walking out into a windy environment (*trúng gió;* "hit by the wind") may attribute the panic attack to exposure to wind as a result of the cultural syndrome that links these two experiences, resulting in classification of the panic attack as expected. Various other cultural syndromes are associated with panic disorder, including *ataque de nervios* ("attack of nerves") among Latin Americans and *khyâl* attacks and "soul loss" among Cambodians. *Ataque de nervios* may involve trembling, uncontrollable screaming or crying, aggressive or suicidal behavior, and depersonalization or derealization, which may be experienced longer than the few minutes typical

of panic attacks. Some clinical presentations of *ataque de nervios* fulfill criteria for conditions other than panic attack (e.g., other specified dissociative disorder). These syndromes impact the symptoms and frequency of panic disorder, including the individual's attribution of unexpectedness, as cultural syndromes may create fear of certain situations, ranging from interpersonal arguments (associated with *ataque de nervios*), to types of exertion (associated with *khyâl* attacks), to atmospheric wind (associated with *trúng gió* attacks). Clarification of the details of cultural attributions may aid in distinguishing expected and unexpected panic attacks. For more information regarding cultural syndromes, refer to the "Glossary of Cultural Concepts of Distress" in the Appendix.

The specific worries about panic attacks or their consequences are likely to vary from one culture to another (and across different age groups and gender). For panic disorder, U.S. community samples of non-Latino whites have significantly less functional impairment than African Americans. There are also higher rates of objectively defined severity in non-Latino Caribbean blacks with panic disorder, and lower rates of panic disorder overall in both African American and Afro-Caribbean groups, suggesting that among individuals of African descent, the criteria for panic disorder may be met only when there is substantial severity and impairment.

Gender-Related Diagnostic Issues

The clinical features of panic disorder do not appear to differ between males and females. There is some evidence for sexual dimorphism, with an association between panic disorder and the catechol-O-methyltransferase (COMT) gene in females only.

Diagnostic Markers

Agents with disparate mechanisms of action, such as sodium lactate, caffeine, isoproterenol, yohimbine, carbon dioxide, and cholecystokinin, provoke panic attacks in individuals with panic disorder to a much greater extent than in healthy control subjects (and in some cases, than in individuals with other anxiety, depressive, or bipolar disorders without panic attacks). Also, for a proportion of individuals with panic disorder, panic attacks are related to hypersensitive medullary carbon dioxide detectors, resulting in hypocapnia and other respiratory irregularities. However, none of these laboratory findings are considered diagnostic of panic disorder.

Suicide Risk

Panic attacks and a diagnosis of panic disorder in the past 12 months are related to a higher rate of suicide attempts and suicidal ideation in the past 12 months even when comorbidity and a history of childhood abuse and other suicide risk factors are taken into account.

Functional Consequences of Panic Disorder

Panic disorder is associated with high levels of social, occupational, and physical disability; considerable economic costs; and the highest number of medical visits among the anxiety disorders, although the effects are strongest with the presence of agoraphobia. Individuals with panic disorder may be frequently absent from work or school for doctor and emergency room visits, which can lead to unemployment or dropping out of school. In older adults, impairment may be seen in caregiving duties or volunteer activities. Full-symptom panic attacks typically are associated with greater morbidity (e.g., greater health care utilization, more disability, poorer quality of life) than limited-symptom attacks.

Differential Diagnosis

Other specified anxiety disorder or unspecified anxiety disorder. Panic disorder should not be diagnosed if full-symptom (unexpected) panic attacks have never been experienced. In

the case of only limited-symptom unexpected panic attacks, an other specified anxiety disorder or unspecified anxiety disorder diagnosis should be considered.

Anxiety disorder due to another medical condition. Panic disorder is not diagnosed if the panic attacks are judged to be a direct physiological consequence of another medical condition. Examples of medical conditions that can cause panic attacks include hyperthyroidism, hyperparathyroidism, pheochromocytoma, vestibular dysfunctions, seizure disorders, and cardiopulmonary conditions (e.g., arrhythmias, supraventricular tachycardia, asthma, chronic obstructive pulmonary disease [COPD]). Appropriate laboratory tests (e.g., serum calcium levels for hyperparathyroidism; Holter monitor for arrhythmias) or physical examinations (e.g., for cardiac conditions) may be helpful in determining the etiological role of another medical condition.

Substance/medication-induced anxiety disorder. Panic disorder is not diagnosed if the panic attacks are judged to be a direct physiological consequence of a substance. Intoxication with central nervous system stimulants (e.g., cocaine, amphetamines, caffeine) or cannabis and withdrawal from central nervous system depressants (e.g., alcohol, barbiturates) can precipitate a panic attack. However, if panic attacks continue to occur outside of the context of substance use (e.g., long after the effects of intoxication or withdrawal have ended), a diagnosis of panic disorder should be considered. In addition, because panic disorder may precede substance use in some individuals and may be associated with increased substance use, especially for purposes of self-medication, a detailed history should be taken to determine if the individual had panic attacks prior to excessive substance use. If this is the case, a diagnosis of panic disorder should be considered in addition to a diagnosis of substance use disorder. Features such as onset after age 45 years or the presence of atypical symptoms during a panic attack (e.g., vertigo, loss of consciousness, loss of bladder or bowel control, slurred speech, amnesia) suggest the possibility that another medical condition or a substance may be causing the panic attack symptoms.

Other mental disorders with panic attacks as an associated feature (e.g., other anxiety disorders and psychotic disorders). Panic attacks that occur as a symptom of other anxiety disorders are expected (e.g., triggered by social situations in social anxiety disorder, by phobic objects or situations in specific phobia or agoraphobia, by worry in generalized anxiety disorder, by separation from home or attachment figures in separation anxiety disorder) and thus would not meet criteria for panic disorder. (**Note:** Sometimes an unexpected panic attack is associated with the onset of another anxiety disorder, but then the attacks become expected, whereas panic disorder is characterized by recurrent unexpected panic attacks.) If the panic attacks occur only in response to specific triggers, then only the relevant anxiety disorder is assigned. However, if the individual experiences unexpected panic attacks as well and shows persistent concern and worry or behavioral change because of the attacks, then an additional diagnosis of panic disorder should be considered.

Comorbidity

Panic disorder infrequently occurs in clinical settings in the absence of other psychopathology. The prevalence of panic disorder is elevated in individuals with other disorders, particularly other anxiety disorders (and especially agoraphobia), major depression, bipolar disorder, and possibly mild alcohol use disorder. While panic disorder often has an earlier age at onset than the comorbid disorder(s), onset sometimes occurs after the comorbid disorder and may be seen as a severity marker of the comorbid illness.

Reported lifetime rates of comorbidity between major depressive disorder and panic disorder vary widely, ranging from 10% to 65% in individuals with panic disorder. In approximately one-third of individuals with both disorders, the depression precedes the onset of panic disorder. In the remaining two-thirds, depression occurs coincident with or following the onset of panic disorder. A subset of individuals with panic disorder develop a substance-related disorder, which for some represents an attempt to treat their anxiety

with alcohol or medications. Comorbidity with other anxiety disorders and illness anxiety disorder is also common.

Panic disorder is significantly comorbid with numerous general medical symptoms and conditions, including, but not limited to, dizziness, cardiac arrhythmias, hyperthyroidism, asthma, COPD, and irritable bowel syndrome. However, the nature of the association (e.g., cause and effect) between panic disorder and these conditions remains unclear. Although mitral valve prolapse and thyroid disease are more common among individuals with panic disorder than in the general population, the differences in prevalence are not consistent.

Panic Attack Specifier

Note: Symptoms are presented for the purpose of identifying a panic attack; however, panic attack is not a mental disorder and cannot be coded. Panic attacks can occur in the context of any anxiety disorder as well as other mental disorders (e.g., depressive disorders, posttraumatic stress disorder, substance use disorders) and some medical conditions (e.g., cardiac, respiratory, vestibular, gastrointestinal). When the presence of a panic attack is identified, it should be noted as a specifier (e.g., "posttraumatic stress disorder with panic attacks"). For panic disorder, the presence of panic attack is contained within the criteria for the disorder and panic attack is not used as a specifier.

An abrupt surge of intense fear or intense discomfort that reaches a peak within minutes, and during which time four (or more) of the following symptoms occur:

Note: The abrupt surge can occur from a calm state or an anxious state.

1. Palpitations, pounding heart, or accelerated heart rate.
2. Sweating.
3. Trembling or shaking.
4. Sensations of shortness of breath or smothering.
5. Feelings of choking.
6. Chest pain or discomfort.
7. Nausea or abdominal distress.
8. Feeling dizzy, unsteady, light-headed, or faint.
9. Chills or heat sensations.
10. Paresthesias (numbness or tingling sensations).
11. Derealization (feelings of unreality) or depersonalization (being detached from oneself).
12. Fear of losing control or "going crazy."
13. Fear of dying.

Note: Culture-specific symptoms (e.g., tinnitus, neck soreness, headache, uncontrollable screaming or crying) may be seen. Such symptoms should not count as one of the four required symptoms.

Features

The essential feature of a panic attack is an abrupt surge of intense fear or intense discomfort that reaches a peak within minutes and during which time four or more of 13 physical and cognitive symptoms occur. Eleven of these 13 symptoms are physical (e.g., palpitations, sweating), while two are cognitive (i.e., fear of losing control or going crazy, fear of dying). "Fear of going crazy" is a colloquialism often used by individuals with panic attacks and is not intended as a pejorative or diagnostic term. The term *within minutes* means that the time to peak

intensity is literally only a few minutes. A panic attack can arise from either a calm state or an anxious state, and time to peak intensity should be assessed independently of any preceding anxiety. That is, the start of the panic attack is the point at which there is an abrupt increase in discomfort rather than the point at which anxiety first developed. Likewise, a panic attack can return to either an anxious state or a calm state and possibly peak again. A panic attack is distinguished from ongoing anxiety by its time to peak intensity, which occurs within minutes; its discrete nature; and its typically greater severity. Attacks that meet all other criteria but have fewer than four physical and/or cognitive symptoms are referred to as *limited-symptom attacks.*

There are two characteristic types of panic attacks: expected and unexpected. *Expected panic attacks* are attacks for which there is an obvious cue or trigger, such as situations in which panic attacks have typically occurred. *Unexpected panic attacks* are those for which there is no obvious cue or trigger at the time of occurrence (e.g., when relaxing or out of sleep [nocturnal panic attack]). The determination of whether panic attacks are expected or unexpected is made by the clinician, who makes this judgment based on a combination of careful questioning as to the sequence of events preceding or leading up to the attack and the individual's own judgment of whether or not the attack seemed to occur for no apparent reason. Cultural interpretations may influence their determination as expected or unexpected. Culture-specific symptoms (e.g., tinnitus, neck soreness, headache, uncontrollable screaming or crying) may be seen; however, such symptoms should not count as one of the four required symptoms. Panic attacks can occur in the context of any mental disorder (e.g., anxiety disorders, depressive disorders, bipolar disorders, eating disorders, obsessive-compulsive and related disorders, personality disorders, psychotic disorders, substance use disorders) and some medical conditions (e.g., cardiac, respiratory, vestibular, gastrointestinal), with the majority never meeting criteria for panic disorder. Recurrent unexpected panic attacks are required for a diagnosis of panic disorder.

Associated Features

One type of unexpected panic attack is a *nocturnal panic attack* (i.e., waking from sleep in a state of panic), which differs from panicking after fully waking from sleep. Panic attacks are related to a higher rate of suicide attempts and suicidal ideation even when comorbidity and other suicide risk factors are taken into account.

Prevalence

In the general population, 12-month prevalence estimates for panic attacks in the United States is 11.2% in adults. Twelve-month prevalence estimates do not appear to differ significantly among African Americans, Asian Americans, and Latinos. Lower 12-month prevalence estimates for European countries appear to range from 2.7% to 3.3%. Females are more frequently affected than males, although this gender difference is more pronounced for panic disorder. Panic attacks can occur in children but are relatively rare until the age of puberty, when the prevalence rates increase. The prevalence rates decline in older individuals, possibly reflecting diminishing severity to subclinical levels.

Development and Course

The mean age at onset for panic attacks in the United States is approximately 22–23 years among adults. However, the course of panic attacks is likely influenced by the course of any co-occurring mental disorder(s) and stressful life events. Panic attacks are uncommon, and unexpected panic attacks are rare, in preadolescent children. Adolescents might be less willing than adults to openly discuss panic attacks, even though they present with episodes of intense fear or discomfort. Lower prevalence of panic attacks in older individuals may be related to a weaker autonomic response to emotional states relative to younger individuals. Older individuals may be less inclined to use the word "fear" and more inclined

to use the word "discomfort" to describe panic attacks. Older individuals with "panicky feelings" may have a hybrid of limited-symptom attacks and generalized anxiety. In addition, older individuals tend to attribute panic attacks to certain situations that are stressful (e.g., medical procedures, social settings) and may retrospectively endorse explanations for the panic attack even if it was unexpected in the moment. This may result in under-endorsement of unexpected panic attacks in older individuals.

Risk and Prognostic Factors

Temperamental. Negative affectivity (neuroticism) (i.e., proneness to experiencing negative emotions) and anxiety sensitivity (i.e., the disposition to believe that symptoms of anxiety are harmful) are risk factors for the onset of panic attacks. History of "fearful spells" (i.e., limited-symptom attacks that do not meet full criteria for a panic attack) may be a risk factor for later panic attacks.

Environmental. Smoking is a risk factor for panic attacks. Most individuals report identifiable stressors in the months before their first panic attack (e.g., interpersonal stressors and stressors related to physical well-being, such as negative experiences with illicit or prescription drugs, disease, or death in the family).

Culture-Related Diagnostic issues

Cultural interpretations may influence the determination of panic attacks as expected or unexpected. Culture-specific symptoms (e.g., tinnitus, neck soreness, headache, and uncontrollable screaming or crying) may be seen; however, such symptoms should not count as one of the four required symptoms. Frequency of each of the 13 symptoms varies cross-culturally (e.g., higher rates of paresthesias in African Americans and of dizziness in several Asian groups). Cultural syndromes also influence the cross-cultural presentation of panic attacks, resulting in different symptom profiles across different cultural groups. Examples include *khyâl* (wind) attacks, a Cambodian cultural syndrome involving dizziness, tinnitus, and neck soreness; and *trûng gió* (wind-related) attacks, a Vietnamese cultural syndrome associated with headaches. *Ataque de nervios* (attack of nerves) is a cultural syndrome among Latin Americans that may involve trembling, uncontrollable screaming or crying, aggressive or suicidal behavior, and depersonalization or derealization, and which may be experienced for longer than only a few minutes. Some clinical presentations of *ataque de nervios* fulfill criteria for conditions other than panic attack (e.g., other specified dissociative disorder). Also, cultural expectations may influence the classification of panic attacks as expected or unexpected, as cultural syndromes may create fear of certain situations, ranging from interpersonal arguments (associated with *ataque de nervios*), to types of exertion (associated with *khyâl* attacks), to atmospheric wind (associated with *trûng gió* attacks). Clarification of the details of cultural attributions may aid in distinguishing expected and unexpected panic attacks. For more information about cultural syndromes, see "Glossary of Cultural Concepts of Distress" in the Appendix to this manual.

Gender-Related Diagnostic Issues

Panic attacks are more common in females than in males, but clinical features or symptoms of panic attacks do not differ between males and females.

Diagnostic Markers

Physiological recordings of naturally occurring panic attacks in individuals with panic disorder indicate abrupt surges of arousal, usually of heart rate, that reach a peak within minutes and subside within minutes, and for a proportion of these individuals the panic attack may be preceded by cardiorespiratory instabilities.

Functional Consequences of Panic Attacks

In the context of co-occurring mental disorders, including anxiety disorders, depressive disorders, bipolar disorder, substance use disorders, psychotic disorders, and personality disorders, panic attacks are associated with increased symptom severity, higher rates of comorbidity and suicidality, and poorer treatment response. Also, full-symptom panic attacks typically are associated with greater morbidity (e.g., greater health care utilization, more disability, poorer quality of life) than limited-symptom attacks.

Differential Diagnosis

Other paroxysmal episodes (e.g., "anger attacks"). Panic attacks should not be diagnosed if the episodes do not involve the essential feature of an abrupt surge of intense fear or intense discomfort, but rather other emotional states (e.g., anger, grief).

Anxiety disorder due to another medical condition. Medical conditions that can cause or be misdiagnosed as panic attacks include hyperthyroidism, hyperparathyroidism, pheochromocytoma, vestibular dysfunctions, seizure disorders, and cardiopulmonary conditions (e.g., arrhythmias, supraventricular tachycardia, asthma, chronic obstructive pulmonary disease). Appropriate laboratory tests (e.g., serum calcium levels for hyperparathyroidism; Holter monitor for arrhythmias) or physical examinations (e.g., for cardiac conditions) may be helpful in determining the etiological role of another medical condition.

Substance/medication-induced anxiety disorder. Intoxication with central nervous system stimulants (e.g., cocaine, amphetamines, caffeine) or cannabis and withdrawal from central nervous system depressants (e.g., alcohol, barbiturates) can precipitate a panic attack. A detailed history should be taken to determine if the individual had panic attacks prior to excessive substance use. Features such as onset after age 45 years or the presence of atypical symptoms during a panic attack (e.g., vertigo, loss of consciousness, loss of bladder or bowel control, slurred speech, or amnesia) suggest the possibility that a medical condition or a substance may be causing the panic attack symptoms.

Panic disorder. Repeated unexpected panic attacks are required but are not sufficient for the diagnosis of panic disorder (i.e., full diagnostic criteria for panic disorder must be met).

Comorbidity

Panic attacks are associated with increased likelihood of various comorbid mental disorders, including anxiety disorders, depressive disorders, bipolar disorders, impulse-control disorders, and substance use disorders. Panic attacks are associated with increased likelihood of later developing anxiety disorders, depressive disorders, bipolar disorders, and possibly other disorders.

Agoraphobia

Diagnostic Criteria **300.22 (F40.00)**

A. Marked fear or anxiety about two (or more) of the following five situations:

1. Using public transportation (e.g., automobiles, buses, trains, ships, planes).
2. Being in open spaces (e.g., parking lots, marketplaces, bridges).
3. Being in enclosed places (e.g., shops, theaters, cinemas).
4. Standing in line or being in a crowd.
5. Being outside of the home alone.

B. The individual fears or avoids these situations because of thoughts that escape might be difficult or help might not be available in the event of developing panic-like symp-

toms or other incapacitating or embarrassing symptoms (e.g., fear of falling in the elderly; fear of incontinence).

C. The agoraphobic situations almost always provoke fear or anxiety.

D. The agoraphobic situations are actively avoided, require the presence of a companion, or are endured with intense fear or anxiety.

E. The fear or anxiety is out of proportion to the actual danger posed by the agoraphobic situations and to the sociocultural context.

F. The fear, anxiety, or avoidance is persistent, typically lasting for 6 months or more.

G. The fear, anxiety, or avoidance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

H. If another medical condition (e.g., inflammatory bowel disease, Parkinson's disease) is present, the fear, anxiety, or avoidance is clearly excessive.

I. The fear, anxiety, or avoidance is not better explained by the symptoms of another mental disorder—for example, the symptoms are not confined to specific phobia, situational type; do not involve only social situations (as in social anxiety disorder); and are not related exclusively to obsessions (as in obsessive-compulsive disorder), perceived defects or flaws in physical appearance (as in body dysmorphic disorder), reminders of traumatic events (as in posttraumatic stress disorder), or fear of separation (as in separation anxiety disorder).

Note: Agoraphobia is diagnosed irrespective of the presence of panic disorder. If an individual's presentation meets criteria for panic disorder and agoraphobia, both diagnoses should be assigned.

Diagnostic Features

The essential feature of agoraphobia is marked, or intense, fear or anxiety triggered by the real or anticipated exposure to a wide range of situations (Criterion A). The diagnosis requires endorsement of symptoms occurring in at least two of the following five situations: 1) using public transporation, such as automobiles, buses, trains, ships, or planes; 2) being in open spaces, such as parking lots, marketplaces, or bridges; 3) being in enclosed spaces, such as shops, theaters, or cinemas; 4) standing in line or being in a crowd; or 5) being outside of the home alone. The examples for each situation are not exhaustive; other situations may be feared. When experiencing fear and anxiety cued by such situations, individuals typically experience thoughts that something terrible might happen (Criterion B). Individuals frequently believe that escape from such situations might be difficult (e.g., "can't get out of here") or that help might be unavailable (e.g., "there is nobody to help me") when panic-like symptoms or other incapacitating or embarrassing symptoms occur. "Panic-like symptoms" refer to any of the 13 symptoms included in the criteria for panic attack, such as dizziness, faintness, and fear of dying. "Other incapacitating or embarrassing symptoms" include symptoms such as vomiting and inflammatory bowel symptoms, as well as, in older adults, a fear of falling or, in children, a sense of disorientation and getting lost.

The amount of fear experienced may vary with proximity to the feared situation and may occur in anticipation of or in the actual presence of the agoraphobic situation. Also, the fear or anxiety may take the form of a full- or limited-symptom panic attack (i.e., an expected panic attack). Fear or anxiety is evoked nearly every time the individual comes into contact with the feared situation (Criterion C). Thus, an individual who becomes anxious only occasionally in an agoraphobic situation (e.g., becomes anxious when standing in line on only one out of every five occasions) would not be diagnosed with agoraphobia. The individual actively avoids the situation or, if he or she either is unable or decides not to avoid it, the situation evokes intense fear or anxiety (Criterion D). *Active avoidance* means the individual is currently behaving in ways that are intentionally designed to prevent or minimize contact with agoraphobic situations. Avoidance can be behavioral (e.g., changing

daily routines, choosing a job nearby to avoid using public transportation, arranging for food delivery to avoid entering shops and supermarkets) as well as cognitive (e.g., using distraction to get through agoraphobic situations) in nature. The avoidance can become so severe that the person is completely homebound. Often, an individual is better able to confront a feared situation when accompanied by a companion, such as a partner, friend, or health professional.

The fear, anxiety, or avoidance must be out of proportion to the actual danger posed by the agoraphobic situations and to the sociocultural context (Criterion E). Differentiating clinically significant agoraphobic fears from reasonable fears (e.g., leaving the house during a bad storm) or from situations that are deemed dangerous (e.g., walking in a parking lot or using public transportation in a high-crime area) is important for a number of reasons. First, what constitutes avoidance may be difficult to judge across cultures and sociocultural contexts (e.g., it is socioculturally appropriate for orthodox Muslim women in certain parts of the world to avoid leaving the house alone, and thus such avoidance would not be considered indicative of agoraphobia). Second, older adults are likely to overattribute their fears to age-related constraints and are less likely to judge their fears as being out of proportion to the actual risk. Third, individuals with agoraphobia are likely to overestimate danger in relation to panic-like or other bodily symptoms. Agoraphobia should be diagnosed only if the fear, anxiety, or avoidance persists (Criterion F) and if it causes clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion G). The duration of "typically lasting for 6 months or more" is meant to exclude individuals with short-lived, transient problems. However, the duration criterion should be used as a general guide, with allowance for some degree of flexibility.

Associated Features Supporting Diagnosis

In its most severe forms, agoraphobia can cause individuals to become completely homebound, unable to leave their home and dependent on others for services or assistance to provide even for basic needs. Demoralization and depressive symptoms, as well as abuse of alcohol and sedative medication as inappropriate self-medication strategies, are common.

Prevalence

Every year approximately 1.7% of adolescents and adults have a diagnosis of agoraphobia. Females are twice as likely as males to experience agoraphobia. Agoraphobia may occur in childhood, but incidence peaks in late adolescence and early adulthood. Twelve-month prevalence in individuals older than 65 years is 0.4%. Prevalence rates do not appear to vary systematically across cultural/racial groups.

Development and Course

The percentage of individuals with agoraphobia reporting panic attacks or panic disorder preceding the onset of agoraphobia ranges from 30% in community samples to more than 50% in clinic samples. The majority of individuals with panic disorder show signs of anxiety and agoraphobia before the onset of panic disorder.

In two-thirds of all cases of agoraphobia, initial onset is before age 35 years. There is a substantial incidence risk in late adolescence and early adulthood, with indications for a second high incidence risk phase after age 40 years. First onset in childhood is rare. The overall mean age at onset for agoraphobia is 17 years, although the age at onset without preceding panic attacks or panic disorder is 25–29 years.

The course of agoraphobia is typically persistent and chronic. Complete remission is rare (10%), unless the agoraphobia is treated. With more severe agoraphobia, rates of full remission decrease, whereas rates of relapse and chronicity increase. A range of other disorders, in particular other anxiety disorders, depressive disorders, substance use disorders, and personality disorders, may complicate the course of agoraphobia. The long-term

course and outcome of agoraphobia are associated with substantially elevated risk of secondary major depressive disorder, persistent depressive disorder (dysthymia), and substance use disorders.

The clinical features of agoraphobia are relatively consistent across the lifespan, although the type of agoraphobic situations triggering fear, anxiety, or avoidance, as well as the type of cognitions, may vary. For example, in children, being outside of the home alone is the most frequent situation feared, whereas in older adults, being in shops, standing in line, and being in open spaces are most often feared. Also, cognitions often pertain to becoming lost (in children), to experiencing panic-like symptoms (in adults), to falling (in older adults).

The low prevalence of agoraphobia in children could reflect difficulties in symptom reporting, and thus assessments in young children may require solicitation of information from multiple sources, including parents or teachers. Adolescents, particularly males, may be less willing than adults to openly discuss agoraphobic fears and avoidance; however, agoraphobia can occur prior to adulthood and should be assessed in children and adolescents. In older adults, comorbid somatic symptom disorders, as well as motor disturbances (e.g., sense of falling or having medical complications), are frequently mentioned by individuals as the reason for their fear and avoidance. In these instances, care is to be taken in evaluating whether the fear and avoidance are out of proportion to the real danger involved.

Risk and Prognostic Factors

Temperamental. Behavioral inhibition and neurotic disposition (i.e., negative affectivity [neuroticism] and anxiety sensitivity) are closely associated with agoraphobia but are relevant to most anxiety disorders (phobic disorders, panic disorder, generalized anxiety disorder). Anxiety sensitivity (the disposition to believe that symptoms of anxiety are harmful) is also characteristic of individuals with agoraphobia.

Environmental. Negative events in childhood (e.g., separation, death of parent) and other stressful events, such as being attacked or mugged, are associated with the onset of agoraphobia. Furthermore, individuals with agoraphobia describe the family climate and child-rearing behavior as being characterized by reduced warmth and increased overprotection.

Genetic and physiological. Heritability for agoraphobia is 61%. Of the various phobias, agoraphobia has the strongest and most specific association with the genetic factor that represents proneness to phobias.

Gender-Reiated Diagnostic Issues

Females have different patterns of comorbid disorders than males. Consistent with gender differences in the prevalence of mental disorders, males have higher rates of comorbid substance use disorders.

Functional Consequences of Agoraphobia

Agoraphobia is associated with considerable impairment and disability in terms of role functioning, work productivity, and disability days. Agoraphobia severity is a strong determinant of the degree of disability, irrespective of the presence of comorbid panic disorder, panic attacks, and other comorbid conditions. More than one-third of individuals with agoraphobia are completely homebound and unable to work.

Differential Diagnosis

When diagnostic criteria for agoraphobia and another disorder are fully met, both diagnoses should be assigned, unless the fear, anxiety, or avoidance of agoraphobia is attributable to the other disorder. Weighting of criteria and clinical judgment may be helpful in some cases.

Specific phobia, situational type. Differentiating agoraphobia from situational specific phobia can be challenging in some cases, because these conditions share several symptom characteristics and criteria. Specific phobia, situational type, should be diagnosed versus agoraphobia if the fear, anxiety, or avoidance is limited to one of the agoraphobic situations. Requiring fears from two or more of the agoraphobic situations is a robust means for differentiating agoraphobia from specific phobias, particularly the situational subtype. Additional differentiating features include the cognitive ideation. Thus, if the situation is feared for reasons other than panic-like symptoms or other incapacitating or embarrassing symptoms (e.g., fears of being directly harmed by the situation itself, such as fear of the plane crashing for individuals who fear flying), then a diagnosis of specific phobia may be more appropriate.

Separation anxiety disorder. Separation anxiety disorder can be best differentiated from agoraphobia by examining cognitive ideation. In separation anxiety disorder, the thoughts are about detachment from significant others and the home environment (i.e., parents or other attachment figures), whereas in agoraphobia the focus is on panic-like symptoms or other incapacitating or embarrassing symptoms in the feared situations.

Social anxiety disorder (social phobia). Agoraphobia should be differentiated from social anxiety disorder based primarily on the situational clusters that trigger fear, anxiety, or avoidance and the cognitive ideation. In social anxiety disorder, the focus is on fear of being negatively evaluated.

Panic disorder. When criteria for panic disorder are met, agoraphobia should not be diagnosed if the avoidance behaviors associated with the panic attacks do not extend to avoidance of two or more agoraphobic situations.

Acute stress disorder and posttraumatic stress disorder. Acute stress disorder and posttraumatic stress disorder (PTSD) can be differentiated from agoraphobia by examining whether the fear, anxiety, or avoidance is related only to situations that remind the individual of a traumatic event. If the fear, anxiety, or avoidance is restricted to trauma reminders, and if the avoidance behavior does not extend to two or more agoraphobic situations, then a diagnosis of agoraphobia is not warranted.

Major depressive disorder. In major depressive disorder, the individual may avoid leaving home because of apathy, loss of energy, low self-esteem, and anhedonia. If the avoidance is unrelated to fears of panic-like or other incapacitating or embarrassing symptoms, then agoraphobia should not be diagnosed.

Other medical conditions. Agoraphobia is not diagnosed if the avoidance of situations is judged to be a physiological consequence of a medical condition. This determination is based on history, laboratory findings, and a physical examination. Other relevant medical conditions may include neurodegenerative disorders with associated motor disturbances (e.g., Parkinson's disease, multiple sclerosis), as well as cardiovascular disorders. Individuals with certain medical conditions may avoid situations because of realistic concerns about being incapacitated (e.g., fainting in an individual with transient ischemic attacks) or being embarrassed (e.g., diarrhea in an individual with Crohn's disease). The diagnosis of agoraphobia should be given only when the fear or avoidance is clearly in excess of that usually associated with these medical conditions.

Comorbidity

The majority of individuals with agoraphobia also have other mental disorders. The most frequent additional diagnoses are other anxiety disorders (e.g., specific phobias, panic disorder, social anxiety disorder), depressive disorders (major depressive disorder), PTSD, and alcohol use disorder. Whereas other anxiety disorders (e.g., separation anxiety disorder, specific phobias, panic disorder) frequently precede onset of agoraphobia, depressive disorders and substance use disorders typically occur secondary to agoraphobia.

Generalized Anxiety Disorder

Diagnostic Criteria 300.02 (F41.1)

A. Excessive anxiety and worry (apprehensive expectation), occurring more days than not for at least 6 months, about a number of events or activities (such as work or school performance).

B. The individual finds it difficult to control the worry.

C. The anxiety and worry are associated with three (or more) of the following six symptoms (with at least some symptoms having been present for more days than not for the past 6 months):

Note: Only one item is required in children.

1. Restlessness or feeling keyed up or on edge.
2. Being easily fatigued.
3. Difficulty concentrating or mind going blank.
4. Irritability.
5. Muscle tension.
6. Sleep disturbance (difficulty falling or staying asleep, or restless, unsatisfying sleep).

D. The anxiety, worry, or physical symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

E. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g., hyperthyroidism).

F. The disturbance is not better explained by another mental disorder (e.g., anxiety or worry about having panic attacks in panic disorder, negative evaluation in social anxiety disorder [social phobia], contamination or other obsessions in obsessive-compulsive disorder, separation from attachment figures in separation anxiety disorder, reminders of traumatic events in posttraumatic stress disorder, gaining weight in anorexia nervosa, physical complaints in somatic symptom disorder, perceived appearance flaws in body dysmorphic disorder, having a serious illness in illness anxiety disorder, or the content of delusional beliefs in schizophrenia or delusional disorder).

Diagnostic Features

The essential feature of generalized anxiety disorder is excessive anxiety and worry (apprehensive expectation) about a number of events or activities. The intensity, duration, or frequency of the anxiety and worry is out of proportion to the actual likelihood or impact of the anticipated event. The individual finds it difficult to control the worry and to keep worrisome thoughts from interfering with attention to tasks at hand. Adults with generalized anxiety disorder often worry about everyday, routine life circumstances, such as possible job responsibilities, health and finances, the health of family members, misfortune to their children, or minor matters (e.g., doing household chores or being late for appointments). Children with generalized anxiety disorder tend to worry excessively about their competence or the quality of their performance. During the course of the disorder, the focus of worry may shift from one concern to another.

Several features distinguish generalized anxiety disorder from nonpathological anxiety. First, the worries associated with generalized anxiety disorder are excessive and typically interfere significantly with psychosocial functioning, whereas the worries of everyday life are not excessive and are perceived as more manageable and may be put off when more pressing matters arise. Second, the worries associated with generalized anxiety disorder are

more pervasive, pronounced, and distressing; have longer duration; and frequently occur without precipitants. The greater the range of life circumstances about which a person worries (e.g., finances, children's safety, job performance), the more likely his or her symptoms are to meet criteria for generalized anxiety disorder. Third, everyday worries are much less likely to be accompanied by physical symptoms (e.g., restlessness or feeling keyed up or on edge). Individuals with generalized anxiety disorder report subjective distress due to constant worry and related impairment in social, occupational, or other important areas of functioning.

The anxiety and worry are accompanied by at least three of the following additional symptoms: restlessness or feeling keyed up or on edge, being easily fatigued, difficulty concentrating or mind going blank, irritability, muscle tension, and disturbed sleep, although only one additional symptom is required in children.

Associated Features Supporting Diagnosis

Associated with muscle tension, there may be trembling, twitching, feeling shaky, and muscle aches or soreness. Many individuals with generalized anxiety disorder also experience somatic symptoms (e.g., sweating, nausea, diarrhea) and an exaggerated startle response. Symptoms of autonomic hyperarousal (e.g., accelerated heart rate, shortness of breath, dizziness) are less prominent in generalized anxiety disorder than in other anxiety disorders, such as panic disorder. Other conditions that may be associated with stress (e.g., irritable bowel syndrome, headaches) frequently accompany generalized anxiety disorder.

Prevalence

The 12-month prevalence of generalized anxiety disorder is 0.9% among adolescents and 2.9% among adults in the general community of the United States. The 12-month prevalence for the disorder in other countries ranges from 0.4% to 3.6%. The lifetime morbid risk is 9.0%. Females are twice as likely as males to experience generalized anxiety disorder. The prevalence of the diagnosis peaks in middle age and declines across the later years of life.

Individuals of European descent tend to experience generalized anxiety disorder more frequently than do individuals of non-European descent (i.e., Asian, African, Native American and Pacific Islander). Furthermore, individuals from developed countries are more likely than individuals from nondeveloped countries to report that they have experienced symptoms that meet criteria for generalized anxiety disorder in their lifetime.

Development and Course

Many individuals with generalized anxiety disorder report that they have felt anxious and nervous all of their lives. The median age at onset for generalized anxiety disorder is 30 years; however, age at onset is spread over a very broad range. The median age at onset is later than that for the other anxiety disorders. The symptoms of excessive worry and anxiety may occur early in life but are then manifested as an anxious temperament. Onset of the disorder rarely occurs prior to adolescence. The symptoms of generalized anxiety disorder tend to be chronic and wax and wane across the lifespan, fluctuating between syndromal and subsyndromal forms of the disorder. Rates of full remission are very low.

The clinical expression of generalized anxiety disorder is relatively consistent across the lifespan. The primary difference across age groups is in the content of the individual's worry. Children and adolescents tend to worry more about school and sporting performance, whereas older adults report greater concern about the well-being of family or their own physical heath. Thus, the content of an individual's worry tends to be age appropriate. Younger adults experience greater severity of symptoms than do older adults.

The earlier in life individuals have symptoms that meet criteria for generalized anxiety disorder, the more comorbidity they tend to have and the more impaired they are likely to

be. The advent of chronic physical disease can be a potent issue for excessive worry in the elderly. In the frail elderly, worries about safety—and especially about falling—may limit activities. In those with early cognitive impairment, what appears to be excessive worry about, for example, the whereabouts of things is probably better regarded as realistic given the cognitive impairment.

In children and adolescents with generalized anxiety disorder, the anxieties and worries often concern the quality of their performance or competence at school or in sporting events, even when their performance is not being evaluated by others. There may be excessive concerns about punctuality. They may also worry about catastrophic events, such as earthquakes or nuclear war. Children with the disorder may be overly conforming, perfectionist, and unsure of themselves and tend to redo tasks because of excessive dissatisfaction with less-than-perfect performance. They are typically overzealous in seeking reassurance and approval and require excessive reassurance about their performance and other things they are worried about.

Generalized anxiety disorder may be overdiagnosed in children. When this diagnosis is being considered in children, a thorough evaluation for the presence of other childhood anxiety disorders and other mental disorders should be done to determine whether the worries may be better explained by one of these disorders. Separation anxiety disorder, social anxiety disorder (social phobia), and obsessive-compulsive disorder are often accompanied by worries that may mimic those described in generalized anxiety disorder. For example, a child with social anxiety disorder may be concerned about school performance because of fear of humiliation. Worries about illness may also be better explained by separation anxiety disorder or obsessive-compulsive disorder.

Risk and Prognostic Factors

Temperamental. Behavioral inhibition, negative affectivity (neuroticism), and harm avoidance have been associated with generalized anxiety disorder.

Environmental. Although childhood adversities and parental overprotection have been associated with generalized anxiety disorder, no environmental factors have been identified as specific to generalized anxiety disorder or necessary or sufficient for making the diagnosis.

Genetic and physiological. One-third of the risk of experiencing generalized anxiety disorder is genetic, and these genetic factors overlap with the risk of neuroticism and are shared with other anxiety and mood disorders, particularly major depressive disorder.

Culture-Related Diagnostic Issues

There is considerable cultural variation in the expression of generalized anxiety disorder. For example, in some cultures, somatic symptoms predominate in the expression of the disorder, whereas in other cultures cognitive symptoms tend to predominate. This difference may be more evident on initial presentation than subsequently, as more symptoms are reported over time. There is no information as to whether the propensity for excessive worrying is related to culture, although the topic being worried about can be culture specific. It is important to consider the social and cultural context when evaluating whether worries about certain situations are excessive.

Gender-Related Diagnostic Issues

In clinical settings, generalized anxiety disorder is diagnosed somewhat more frequently in females than in males (about 55%–60% of those presenting with the disorder are female). In epidemiological studies, approximately two-thirds are female. Females and males who experience generalized anxiety disorder appear to have similar symptoms but

demonstrate different patterns of comorbidity consistent with gender differences in the prevalence of disorders. In females, comorbidity is largely confined to the anxiety disorders and unipolar depression, whereas in males, comorbidity is more likely to extend to the substance use disorders as well.

Functional Consequences of Generalized Anxiety Disorder

Excessive worrying impairs the individual's capacity to do things quickly and efficiently, whether at home or at work. The worrying takes time and energy; the associated symptoms of muscle tension and feeling keyed up or on edge, tiredness, difficulty concentrating, and disturbed sleep contribute to the impairment. Importantly the excessive worrying may impair the ability of individuals with generalized anxiety disorder to encourage confidence in their children.

Generalized anxiety disorder is associated with significant disability and distress that is independent of comorbid disorders, and most non-institutionalized adults with the disorder are moderately to seriously disabled. Generalized anxiety disorder accounts for 110 million disability days per annum in the U.S. population.

Differential Diagnosis

Anxiety disorder due to another medical condition. The diagnosis of anxiety disorder associated with another medical condition should be assigned if the individual's anxiety and worry are judged, based on history, laboratory findings, or physical examination, to be a physiological effect of another specific medical condition (e.g., pheochromocytoma, hyperthyroidism).

Substance/medication-induced anxiety disorder. A substance/medication-induced anxiety disorder is distinguished from generalized anxiety disorder by the fact that a substance or medication (e.g., a drug of abuse, exposure to a toxin) is judged to be etiologically related to the anxiety. For example, severe anxiety that occurs only in the context of heavy coffee consumption would be diagnosed as caffeine-induced anxiety disorder.

Social anxiety disorder. Individuals with social anxiety disorder often have anticipatory anxiety that is focused on upcoming social situations in which they must perform or be evaluated by others, whereas individuals with generalized anxiety disorder worry, whether or not they are being evaluated.

Obsessive-compulsive disorder. Several features distinguish the excessive worry of generalized anxiety disorder from the obsessional thoughts of obsessive-compulsive disorder. In generalized anxiety disorder the focus of the worry is about forthcoming problems, and it is the excessiveness of the worry about future events that is abnormal. In obsessive-compulsive disorder, the obsessions are inappropriate ideas that take the form of intrusive and unwanted thoughts, urges, or images.

Posttraumatic stress disorder and adjustment disorders. Anxiety is invariably present in posttraumatic stress disorder. Generalized anxiety disorder is not diagnosed if the anxiety and worry are better explained by symptoms of posttraumatic stress disorder. Anxiety may also be present in adjustment disorder, but this residual category should be used only when the criteria are not met for any other disorder (including generalized anxiety disorder). Moreover, in adjustment disorders, the anxiety occurs in response to an identifiable stressor within 3 months of the onset of the stressor and does not persist for more than 6 months after the termination of the stressor or its consequences.

Depressive, bipolar, and psychotic disorders. Generalized anxiety/worry is a common associated feature of depressive, bipolar, and psychotic disorders and should not be di-

agnosed separately if the excessive worry has occurred only during the course of these conditions.

Comorbidity

Individuals whose presentation meets criteria for generalized anxiety disorder are likely to have met, or currently meet, criteria for other anxiety and unipolar depressive disorders. The neuroticism or emotional liability that underpins this pattern of comorbidity is associated with temperamental antecedents and genetic and environmental risk factors shared between these disorders, although independent pathways are also possible. Comorbidity with substance use, conduct, psychotic, neurodevelopmental, and neurocognitive disorders is less common.

Substance/Medication-Induced Anxiety Disorder

Diagnostic Criteria

A. Panic attacks or anxiety is predominant in the clinical picture.

B. There is evidence from the history, physical examination, or laboratory findings of both (1) and (2):

   1. The symptoms in Criterion A developed during or soon after substance intoxication or withdrawal or after exposure to a medication.
   2. The involved substance/medication is capable of producing the symptoms in Criterion A.

C. The disturbance is not better explained by an anxiety disorder that is not substance/medication-induced. Such evidence of an independent anxiety disorder could include the following:

   The symptoms precede the onset of the substance/medication use; the symptoms persist for a substantial period of time (e.g., about 1 month) after the cessation of acute withdrawal or severe intoxication; or there is other evidence suggesting the existence of an independent non-substance/medication-induced anxiety disorder (e.g., a history of recurrent non-substance/medication-related episodes).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Note: This diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and they are sufficiently severe to warrant clinical attention.

Coding note: The ICD-9-CM and ICD-10-CM codes for the [specific substance/medication]-induced anxiety disorders are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. If a mild substance use disorder is comorbid with the substance-induced anxiety disorder, the 4th position character is "1," and the clinician should record "mild [substance] use disorder" before the substance-induced anxiety disorder (e.g., "mild cocaine use disorder with cocaine-induced anxiety disorder"). If a moderate or severe substance use disorder is comorbid with the substance-induced anxiety disorder, the 4th position character is "2," and the clinician should record "moderate [substance] use disorder" or "severe [substance] use disorder," depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder (e.g., after a one-

time heavy use of the substance), then the 4th position character is "9," and the clinician should record only the substance-induced anxiety disorder.

		ICD-10-CM		
	ICD-9-CM	With use disorder, mild	With use disorder, moderate or severe	Without use disorder
Alcohol	291.89	F10.180	F10.280	F10.980
Caffeine	292.89	F15.180	F15.280	F15.980
Cannabis	292.89	F12.180	F12.280	F12.980
Phencyclidine	292.89	F16.180	F16.280	F16.980
Other hallucinogen	292.89	F16.180	F16.280	F16.980
Inhalant	292.89	F18.180	F18.280	F18.980
Opioid	292.89	F11.188	F11.288	F11.988
Sedative, hypnotic, or anxiolytic	292.89	F13.180	F13.280	F13.980
Amphetamine (or other stimulant)	292.89	F15.180	F15.280	F15.980
Cocaine	292.89	F14.180	F14.280	F14.980
Other (or unknown) substance	292.89	F19.180	F19.280	F19.980

Specify if (see Table 1 in the chapter "Substance-Related and Addictive Disorders" for diagnoses associated with substance class):

With onset during intoxication: This specifier applies if criteria are met for intoxication with the substance and the symptoms develop during intoxication.

With onset during withdrawal: This specifier applies if criteria are met for withdrawal from the substance and the symptoms develop during, or shortly after, withdrawal.

With onset after medication use: Symptoms may appear either at initiation of medication or after a modification or change in use.

Recording Procedures

ICD-9-CM. The name of the substance/medication-induced anxiety disorder begins with the specific substance (e.g., cocaine, salbutamol) that is presumed to be causing the anxiety symptoms. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class. For substances that do not fit into any of the classes (e.g., salbutamol), the code for "other substance" should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category "unknown substance" should be used.

The name of the disorder is followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal, with onset during medication use). Unlike the recording procedures for ICD-10-CM, which combine the substance-induced disorder and substance use disorder into a single code, for ICD-9-CM a separate diagnostic code is given for the substance use disorder. For example, in the case of anxiety symptoms occurring during withdrawal in a man with a severe lorazepam use disorder, the diagnosis is 292.89 lorazepam-induced anxiety disorder, with onset during withdrawal. An additional diagnosis of 304.10 severe lorazepam use disorder is also given. When more than one substance is judged to play a significant role in the development of anxiety symptoms, each should be listed sep-

arately (e.g., 292.89 methylphenidate-induced anxiety disorder, with onset during intoxication; 292.89 salbutamol-induced anxiety disorder, with onset after medication use).

ICD-10-CM. The name of the substance/medication-induced anxiety disorder begins with the specific substance (e.g., cocaine, salbutamol) that is presumed to be causing the anxiety symptoms. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class and presence or absence of a comorbid substance use disorder. For substances that do not fit into any of the classes (e.g., salbutamol), the code for "other substance" should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category "unknown substance" should be used.

When recording the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by the word "with," followed by the name of the substance-induced anxiety disorder, followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal, with onset during medication use). For example, in the case of anxiety symptoms occurring during withdrawal in a man with a severe lorazepam use disorder, the diagnosis is F13.280 severe lorazepam use disorder with lorazepam-induced anxiety disorder, with onset during withdrawal. A separate diagnosis of the comorbid severe lorazepam use disorder is not given. If the substance-induced anxiety disorder occurs without a comorbid substance use disorder (e.g., after a one-time heavy use of the substance), no accompanying substance use disorder is noted (e.g., F16.980 psilocybin-induced anxiety disorder, with onset during intoxication). When more than one substance is judged to play a significant role in the development of anxiety symptoms, each should be listed separately (e.g., F15.280 severe methylphenidate use disorder with methylphenidate-induced anxiety disorder, with onset during intoxication; F19.980 salbutamol-induced anxiety disorder, with onset after medication use).

Diagnostic Features

The essential features of substance/medication-induced anxiety disorder are prominent symptoms of panic or anxiety (Criterion A) that are judged to be due to the effects of a substance (e.g., a drug of abuse, a medication, or a toxin exposure). The panic or anxiety symptoms must have developed during or soon after substance intoxication or withdrawal or after exposure to a medication, and the substances or medications must be capable of producing the symptoms (Criterion B2). Substance/medication-induced anxiety disorder due to a prescribed treatment for a mental disorder or another medical condition must have its onset while the individual is receiving the medication (or during withdrawal, if a withdrawal is associated with the medication). Once the treatment is discontinued, the panic or anxiety symptoms will usually improve or remit within days to several weeks to a month (depending on the half-life of the substance/medication and the presence of withdrawal). The diagnosis of substance/medication-induced anxiety disorder should not be given if the onset of the panic or anxiety symptoms precedes the substance/medication intoxication or withdrawal, or if the symptoms persist for a substantial period of time (i.e., usually longer than 1 month) from the time of severe intoxication or withdrawal. If the panic or anxiety symptoms persist for substantial periods of time, other causes for the symptoms should be considered.

The substance/medication-induced anxiety disorder diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A are predominant in the clinical picture and are sufficiently severe to warrant independent clinical attention.

Associated Features Supporting Diagnosis

Panic or anxiety can occur in association with intoxication with the following classes of substances: alcohol, caffeine, cannabis, phencyclidine, other hallucinogens, inhalants, stimu-

lants (including cocaine), and other (or unknown) substances. Panic or anxiety can occur in association with withdrawal from the following classes of substances: alcohol; opioids; sedatives, hypnotics, and anxiolytics; stimulants (including cocaine); and other (or unknown) substances. Some medications that evoke anxiety symptoms include anesthetics and analgesics, sympathomimetics or other bronchodilators, anticholinergics, insulin, thyroid preparations, oral contraceptives, antihistamines, antiparkinsonian medications, corticosteroids, antihypertensive and cardiovascular medications, anticonvulsants, lithium carbonate, antipsychotic medications, and antidepressant medications. Heavy metals and toxins (e.g., organophosphate insecticide, nerve gases, carbon monoxide, carbon dioxide, volatile substances such as gasoline and paint) may also cause panic or anxiety symptoms.

Prevalence

The prevalence of substance/medication-induced anxiety disorder is not clear. General population data suggest that it may be rare, with a 12-month prevalence of approximately 0.002%. However, in clinical populations, the prevalence is likely to be higher.

Diagnostic Markers

Laboratory assessments (e.g., urine toxicology) may be useful to measure substance intoxication as part of an assessment for substance/medication-induced anxiety disorder.

Differential Diagnosis

Substance intoxication and substance withdrawal. Anxiety symptoms commonly occur in substance intoxication and substance withdrawal. The diagnosis of the substance-specific intoxication or substance-specific withdrawal will usually suffice to categorize the symptom presentation. A diagnosis of substance/medication-induced anxiety disorder should be made in addition to substance intoxication or substance withdrawal when the panic or anxiety symptoms are predominant in the clinical picture and are sufficiently severe to warrant independent clinical attention. For example, panic or anxiety symptoms are characteristic of alcohol withdrawal.

Anxiety disorder (i.e., not induced by a substance/medication). Substance/medication-induced anxiety disorder is judged to be etiologically related to the substance/medication. Substance/medication-induced anxiety disorder is distinguished from a primary anxiety disorder based on the onset, course, and other factors with respect to substances/medications. For drugs of abuse, there must be evidence from the history, physical examination, or laboratory findings for use, intoxication, or withdrawal. Substance/medication-induced anxiety disorders arise only in association with intoxication or withdrawal states, whereas primary anxiety disorders may precede the onset of substance/medication use. The presence of features that are atypical of a primary anxiety disorder, such as atypical age at onset (e.g., onset of panic disorder after age 45 years) or symptoms (e.g., atypical panic attack symptoms such as true vertigo, loss of balance, loss of consciousness, loss of bladder control, headaches, slurred speech) may suggest a substance/medication-induced etiology. A primary anxiety disorder diagnosis is warranted if the panic or anxiety symptoms persist for a substantial period of time (about 1 month or longer) after the end of the substance intoxication or acute withdrawal or there is a history of an anxiety disorder.

Delirium. If panic or anxiety symptoms occur exclusively during the course of delirium, they are considered to be an associated feature of the delirium and are not diagnosed separately.

Anxiety disorder due to another medical condition. If the panic or anxiety symptoms are attributed to the physiological consequences of another medical condition (i.e., rather than to the medication taken for the medical condition), anxiety disorder due to another

medical condition should be diagnosed. The history often provides the basis for such a judgment. At times, a change in the treatment for the other medical condition (e.g., medication substitution or discontinuation) may be needed to determine whether the medication is the causative agent (in which case the symptoms may be better explained by substance/medication-induced anxiety disorder). If the disturbance is attributable to both another medical condition and substance use, both diagnoses (i.e., anxiety disorder due to another medical condition and substance/medication-induced anxiety disorder) may be given. When there is insufficient evidence to determine whether the panic or anxiety symptoms are attributable to a substance/medication or to another medical condition or are primary (i.e., not attributable to either a substance or another medical condition), a diagnosis of other specified or unspecified anxiety disorder would be indicated.

Anxiety Disorder Due to Another Medical Condition

Diagnostic Criteria **293.84 (F06.4)**

A. Panic attacks or anxiety is predominant in the clinical picture.
B. There is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct pathophysiological consequence of another medical condition.
C. The disturbance is not better explained by another mental disorder.
D. The disturbance does not occur exclusively during the course of a delirium.
E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Coding note: Include the name of the other medical condition within the name of the mental disorder (e.g., 293.84 [F06.4] anxiety disorder due to pheochromocytoma). The other medical condition should be coded and listed separately immediately before the anxiety disorder due to the medical condition (e.g., 227.0 [D35.00] pheochromocytoma; 293.84 [F06.4] anxiety disorder due to pheochromocytoma.

Diagnostic Features

The essential feature of anxiety disorder due to another medical condition is clinically significant anxiety that is judged to be best explained as a physiological effect of another medical condition. Symptoms can include prominent anxiety symptoms or panic attacks (Criterion A). The judgment that the symptoms are best explained by the associated physical condition must be based on evidence from the history, physical examination, or laboratory findings (Criterion B). Additionally, it must be judged that the symptoms are not better accounted for by another mental disorder, in particular, adjustment disorder, with anxiety, in which the stressor is the medical condition (Criterion C). In this case, an individual with adjustment disorder is especially distressed about the meaning or the consequences of the associated medical condition. By contrast, there is often a prominent physical component to the anxiety (e.g., shortness of breath) when the anxiety is due to another medical condition. The diagnosis is not made if the anxiety symptoms occur only during the course of a delirium (Criterion D). The anxiety symptoms must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion E).

In determining whether the anxiety symptoms are attributable to another medical condition, the clinician must first establish the presence of the medical condition. Furthermore, it must be established that anxiety symptoms can be etiologically related to the medical condition through a physiological mechanism before making a judgment that this is the best explanation for the symptoms in a specific individual. A careful and compre-

hensive assessment of multiple factors is necessary to make this judgment. Several aspects of the clinical presentation should be considered: 1) the presence of a clear temporal association between the onset, exacerbation, or remission of the medical condition and the anxiety symptoms; 2) the presence of features that are atypical of a primary anxiety disorder (e.g., atypical age at onset or course); and 3) evidence in the literature that a known physiological mechanism (e.g., hyperthyroidism) causes anxiety. In addition, the disturbance must not be better explained by a primary anxiety disorder, a substance/medication-induced anxiety disorder, or another primary mental disorder (e.g., adjustment disorder).

Associated Features Supporting Diagnosis

A number of medical conditions are known to include anxiety as a symptomatic manifestation. Examples include endocrine disease (e.g., hyperthyroidism, pheochromocytoma, hypoglycemia, hyperadrenocortisolism), cardiovascular disorders (e.g., congestive heart failure, pulmonary embolism, arrhythmia such as atrial fibrillation), respiratory illness (e.g., chronic obstructive pulmonary disease, asthma, pneumonia), metabolic disturbances (e.g., vitamin B_{12} deficiency, porphyria), and neurological illness (e.g., neoplasms, vestibular dysfunction, encephalitis, seizure disorders). Anxiety due to another medical condition is diagnosed when the medical condition is known to induce anxiety and when the medical condition preceded the onset of the anxiety.

Prevalence

The prevalence of anxiety disorder due to another medical condition is unclear. There appears to be an elevated prevalence of anxiety disorders among individuals with a variety of medical conditions, including asthma, hypertension, ulcers, and arthritis. However, this increased prevalence may be due to reasons other than the anxiety disorder directly causing the medical condition.

Development and Course

The development and course of anxiety disorder due to another medical condition generally follows the course of the underlying illness. This diagnosis is not meant to include primary anxiety disorders that arise in the context of chronic medical illness. This is important to consider with older adults, who may experience chronic medical illness and then develop independent anxiety disorders secondary to the chronic medical illness.

Diagnostic Markers

Laboratory assessments and/or medical examinations are necessary to confirm the diagnosis of the associated medical condition.

Differential Diagnosis

Delirium. A separate diagnosis of anxiety disorder due to another medical condition is not given if the anxiety disturbance occurs exclusively during the course of a delirium. However, a diagnosis of anxiety disorder due to another medical condition may be given in addition to a diagnosis of major neurocognitive disorder (dementia) if the etiology of anxiety is judged to be a physiological consequence of the pathological process causing the neurocognitive disorder and if anxiety is a prominent part of the clinical presentation.

Mixed presentation of symptoms (e.g., mood and anxiety). If the presentation includes a mix of different types of symptoms, the specific mental disorder due to another medical condition depends on which symptoms predominate in the clinical picture.

Substance/medication-induced anxiety disorder. If there is evidence of recent or prolonged substance use (including medications with psychoactive effects), withdrawal from

a substance, or exposure to a toxin, a substance/medication-induced anxiety disorder should be considered. Certain medications are known to increase anxiety (e.g., corticosteroids, estrogens, metoclopramide), and when this is the case, the medication may be the most likely etiology, although it may be difficult to distinguish whether the anxiety is attributable to the medications or to the medical illness itself. When a diagnosis of substance-induced anxiety is being made in relation to recreational or nonprescribed drugs, it may be useful to obtain a urine or blood drug screen or other appropriate laboratory evaluation. Symptoms that occur during or shortly after (i.e., within 4 weeks of) substance intoxication or withdrawal or after medication use may be especially indicative of a substance/medication-induced anxiety disorder, depending on the type, duration, or amount of the substance used. If the disturbance is associated with both another medical condition and substance use, both diagnoses (i.e., anxiety disorder due to another medical condition and substance/medication-induced anxiety disorder) can be given. Features such as onset after age 45 years or the presence of atypical symptoms during a panic attack (e.g., vertigo, loss of consciousness, loss of bladder or bowel control, slurred speech, amnesia) suggest the possibility that another medical condition or a substance may be causing the panic attack symptoms.

Anxiety disorder (not due to a known medical condition). Anxiety disorder due to another medical condition should be distinguished from other anxiety disorders (especially panic disorder and generalized anxiety disorder). In other anxiety disorders, no specific and direct causative physiological mechanisms associated with another medical condition can be demonstrated. Late age at onset, atypical symptoms, and the absence of a personal or family history of anxiety disorders suggest the need for a thorough assessment to rule out the diagnosis of anxiety disorder due to another medical condition. Anxiety disorders can exacerbate or pose increased risk for medical conditions such as cardiovascular events and myocardial infarction and should not be diagnosed as anxiety disorder due to another medical condition in these cases.

Illness anxiety disorder. Anxiety disorder due to another medical condition should be distinguished from illness anxiety disorder. Illness anxiety disorder is characterized by worry about illness, concern about pain, and bodily preoccupations. In the case of illness anxiety disorder, individuals may or may not have diagnosed medical conditions. Although an individual with illness anxiety disorder and a diagnosed medical condition is likely to experience anxiety about the medical condition, the medical condition is not physiologically related to the anxiety symptoms.

Adjustment disorders. Anxiety disorder due to another medical condition should be distinguished from adjustment disorders, with anxiety, or with anxiety and depressed mood. Adjustment disorder is warranted when individuals experience a maladaptive response to the stress of having another medical condition. The reaction to stress usually concerns the meaning or consequences of the stress, as compared with the experience of anxiety or mood symptoms that occur as a physiological consequence of the other medical condition. In adjustment disorder, the anxiety symptoms are typically related to coping with the stress of having a general medical condition, whereas in anxiety disorder due to another medical condition, individuals are more likely to have prominent physical symptoms and to be focused on issues other than the stress of the illness itself.

Associated feature of another mental disorder. Anxiety symptoms may be an associated feature of another mental disorder (e.g., schizophrenia, anorexia nervosa).

Other specified or unspecified anxiety disorder. This diagnosis is given if it cannot be determined whether the anxiety symptoms are primary, substance-induced, or associated with another medical condition.

Other Specified Anxiety Disorder

300.09 (F41.8)

This category applies to presentations in which symptoms characteristic of an anxiety disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the anxiety disorders diagnostic class. The other specified anxiety disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific anxiety disorder. This is done by recording "other specified anxiety disorder" followed by the specific reason (e.g., "generalized anxiety not occurring more days than not").

Examples of presentations that can be specified using the "other specified" designation include the following:

1. **Limited-symptom attacks.**
2. **Generalized anxiety not occurring more days than not.**
3. ***Khyâl cap* (wind attacks):** See "Glossary of Cultural Concepts of Distress" in the Appendix.
4. ***Ataque de nervios* (attack of nerves):** See "Glossary of Cultural Concepts of Distress" in the Appendix.

Unspecified Anxiety Disorder

300.00 (F41.9)

This category applies to presentations in which symptoms characteristic of an anxiety disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the anxiety disorders diagnostic class. The unspecified anxiety disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific anxiety disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Obsessive-Compulsive and Related Disorders

Obsessive-compulsive and related disorders include obsessive-compulsive disorder (OCD), body dysmorphic disorder, hoarding disorder, trichotillomania (hair-pulling disorder), excoriation (skin-picking) disorder, substance/medication-induced obsessive-compulsive and related disorder, obsessive-compulsive and related disorder due to another medical condition, and other specified obsessive-compulsive and related disorder and unspecified obsessive-compulsive and related disorder (e.g., body-focused repetitive behavior disorder, obsessional jealousy).

OCD is characterized by the presence of obsessions and/or compulsions. *Obsessions* are recurrent and persistent thoughts, urges, or images that are experienced as intrusive and unwanted, whereas *compulsions* are repetitive behaviors or mental acts that an individual feels driven to perform in response to an obsession or according to rules that must be applied rigidly. Some other obsessive-compulsive and related disorders are also characterized by preoccupations and by repetitive behaviors or mental acts in response to the preoccupations. Other obsessive-compulsive and related disorders are characterized primarily by recurrent body-focused repetitive behaviors (e.g., hair pulling, skin picking) and repeated attempts to decrease or stop the behaviors.

The inclusion of a chapter on obsessive-compulsive and related disorders in DSM-5 reflects the increasing evidence of these disorders' relatedness to one another in terms of a range of diagnostic validators as well as the clinical utility of grouping these disorders in the same chapter. Clinicians are encouraged to screen for these conditions in individuals who present with one of them and be aware of overlaps between these conditions. At the same time, there are important differences in diagnostic validators and treatment approaches across these disorders. Moreover, there are close relationships between the anxiety disorders and some of the obsessive-compulsive and related disorders (e.g., OCD), which is reflected in the sequence of DSM-5 chapters, with obsessive-compulsive and related disorders following anxiety disorders.

The obsessive-compulsive and related disorders differ from developmentally normative preoccupations and rituals by being excessive or persisting beyond developmentally appropriate periods. The distinction between the presence of subclinical symptoms and a clinical disorder requires assessment of a number of factors, including the individual's level of distress and impairment in functioning.

The chapter begins with OCD. It then covers body dysmorphic disorder and hoarding disorder, which are characterized by cognitive symptoms such as perceived defects or flaws in physical appearance or the perceived need to save possessions, respectively. The chapter then covers trichotillomania (hair-pulling disorder) and excoriation (skin-picking) disorder, which are characterized by recurrent body-focused repetitive behaviors. Finally, it covers substance/medication-induced obsessive-compulsive and related disorder, obsessive-compulsive and related disorder due to another medical condition, and other specified obsessive-compulsive and related disorder and unspecified obsessive-compulsive and related disorder.

While the specific content of obsessions and compulsions varies among individuals, certain symptom dimensions are common in OCD, including those of cleaning (contamination obsessions and cleaning compulsions); symmetry (symmetry obsessions and repeat-

ing, ordering, and counting compulsions); forbidden or taboo thoughts (e.g., aggressive, sexual, and religious obsessions and related compulsions); and harm (e.g., fears of harm to oneself or others and related checking compulsions). The tic-related specifier of OCD is used when an individual has a current or past history of a tic disorder.

Body dysmorphic disorder is characterized by preoccupation with one or more perceived defects or flaws in physical appearance that are not observable or appear only slight to others, and by repetitive behaviors (e.g., mirror checking, excessive grooming, skin picking, or reassurance seeking) or mental acts (e.g., comparing one's appearance with that of other people) in response to the appearance concerns. The appearance preoccupations are not better explained by concerns with body fat or weight in an individual with an eating disorder. Muscle dysmorphia is a form of body dysmorphic disorder that is characterized by the belief that one's body build is too small or is insufficiently muscular.

Hoarding disorder is characterized by persistent difficulty discarding or parting with possessions, regardless of their actual value, as a result of a strong perceived need to save the items and to distress associated with discarding them. Hoarding disorder differs from normal collecting. For example, symptoms of hoarding disorder result in the accumulation of a large number of possessions that congest and clutter active living areas to the extent that their intended use is substantially compromised. The excessive acquisition form of hoarding disorder, which characterizes most but not all individuals with hoarding disorder, consists of excessive collecting, buying, or stealing of items that are not needed or for which there is no available space.

Trichotillomania (hair-pulling disorder) is characterized by recurrent pulling out of one's hair resulting in hair loss, and repeated attempts to decrease or stop hair pulling. Excoriation (skin-picking) disorder is characterized by recurrent picking of one's skin resulting in skin lesions and repeated attempts to decrease or stop skin picking. The body-focused repetitive behaviors that characterize these two disorders are not triggered by obsessions or preoccupations; however, they may be preceded or accompanied by various emotional states, such as feelings of anxiety or boredom. They may also be preceded by an increasing sense of tension or may lead to gratification, pleasure, or a sense of relief when the hair is pulled out or the skin is picked. Individuals with these disorders may have varying degrees of conscious awareness of the behavior while engaging in it, with some individuals displaying more focused attention on the behavior (with preceding tension and subsequent relief) and other individuals displaying more automatic behavior (with the behaviors seeming to occur without full awareness).

Substance/medication-induced obsessive-compulsive and related disorder consists of symptoms that are due to substance intoxication or withdrawal or to a medication. Obsessive-compulsive and related disorder due to another medical condition involves symptoms characteristic of obsessive-compulsive and related disorders that are the direct pathophysiological consequence of a medical disorder. Other specified obsessive-compulsive and related disorder and unspecified obsessive-compulsive and related disorder consist of symptoms that do not meet criteria for a specific obsessive-compulsive and related disorder because of atypical presentation or uncertain etiology; these categories are also used for other specific syndromes that are not listed in Section II and when insufficient information is available to diagnose the presentation as another obsessive-compulsive and related disorder. Examples of specific syndromes not listed in Section II, and therefore diagnosed as other specified obsessive-compulsive and related disorder or as unspecified obsessive-compulsive and related disorder include body-focused repetitive behavior disorder and obsessional jealousy.

Obsessive-compulsive and related disorders that have a cognitive component have insight as the basis for specifiers; in each of these disorders, insight ranges from "good or fair insight" to "poor insight" to "absent insight/delusional beliefs" with respect to disorder-related beliefs. For individuals whose obsessive-compulsive and related disorder symptoms warrant the "with absent insight/delusional beliefs" specifier, these symptoms should not be diagnosed as a psychotic disorder.

Obsessive-Compulsive Disorder

Diagnostic Criteria 300.3 (F42)

A. Presence of obsessions, compulsions, or both:

Obsessions are defined by (1) and (2):

1. Recurrent and persistent thoughts, urges, or images that are experienced, at some time during the disturbance, as intrusive and unwanted, and that in most individuals cause marked anxiety or distress.
2. The individual attempts to ignore or suppress such thoughts, urges, or images, or to neutralize them with some other thought or action (i.e., by performing a compulsion).

Compulsions are defined by (1) and (2):

1. Repetitive behaviors (e.g., hand washing, ordering, checking) or mental acts (e.g., praying, counting, repeating words silently) that the individual feels driven to perform in response to an obsession or according to rules that must be applied rigidly.
2. The behaviors or mental acts are aimed at preventing or reducing anxiety or distress, or preventing some dreaded event or situation; however, these behaviors or mental acts are not connected in a realistic way with what they are designed to neutralize or prevent, or are clearly excessive.

   Note: Young children may not be able to articulate the aims of these behaviors or mental acts.

B. The obsessions or compulsions are time-consuming (e.g., take more than 1 hour per day) or cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The obsessive-compulsive symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.

D. The disturbance is not better explained by the symptoms of another mental disorder (e.g., excessive worries, as in generalized anxiety disorder; preoccupation with appearance, as in body dysmorphic disorder; difficulty discarding or parting with possessions, as in hoarding disorder; hair pulling, as in trichotillomania [hair-pulling disorder]; skin picking, as in excoriation [skin-picking] disorder; stereotypies, as in stereotypic movement disorder; ritualized eating behavior, as in eating disorders; preoccupation with substances or gambling, as in substance-related and addictive disorders; preoccupation with having an illness, as in illness anxiety disorder; sexual urges or fantasies, as in paraphilic disorders; impulses, as in disruptive, impulse-control, and conduct disorders; guilty ruminations, as in major depressive disorder; thought insertion or delusional preoccupations, as in schizophrenia spectrum and other psychotic disorders; or repetitive patterns of behavior, as in autism spectrum disorder).

Specify if:

With good or fair insight: The individual recognizes that obsessive-compulsive disorder beliefs are definitely or probably not true or that they may or may not be true.

With poor insight: The individual thinks obsessive-compulsive disorder beliefs are probably true.

With absent insight/delusional beliefs: The individual is completely convinced that obsessive-compulsive disorder beliefs are true.

Specify if:

Tic-related: The individual has a current or past history of a tic disorder.

Specifiers

Many individuals with obsessive-compulsive disorder (OCD) have dysfunctional beliefs. These beliefs can include an inflated sense of responsibility and the tendency to overestimate threat; perfectionism and intolerance of uncertainty; and over-importance of thoughts (e.g., believing that having a forbidden thought is as bad as acting on it) and the need to control thoughts.

Individuals with OCD vary in the degree of insight they have about the accuracy of the beliefs that underlie their obsessive-compulsive symptoms. Many individuals have *good or fair insight* (e.g., the individual believes that the house definitely will not, probably will not, or may or may not burn down if the stove is not checked 30 times). Some have *poor insight* (e.g., the individual believes that the house will probably burn down if the stove is not checked 30 times), and a few (4% or less) have *absent insight/delusional beliefs* (e.g., the individual is convinced that the house will burn down if the stove is not checked 30 times). Insight can vary within an individual over the course of the illness. Poorer insight has been linked to worse long-term outcome.

Up to 30% of individuals with OCD have a lifetime tic disorder. This is most common in males with onset of OCD in childhood. These individuals tend to differ from those without a history of tic disorders in the themes of their OCD symptoms, comorbidity, course, and pattern of familial transmission.

Diagnostic Features

The characteristic symptoms of OCD are the presence of obsessions and compulsions (Criterion A). *Obsessions* are repetitive and persistent thoughts (e.g., of contamination), images (e.g., of violent or horrific scenes), or urges (e.g., to stab someone). Importantly, obsessions are not pleasurable or experienced as voluntary: they are intrusive and unwanted and cause marked distress or anxiety in most individuals. The individual attempts to ignore or suppress these obsessions (e.g., avoiding triggers or using thought suppression) or to neutralize them with another thought or action (e.g., performing a compulsion). *Compulsions* (or rituals) are repetitive behaviors (e.g., washing, checking) or mental acts (e.g., counting, repeating words silently) that the individual feels driven to perform in response to an obsession or according to rules that must be applied rigidly. Most individuals with OCD have both obsessions and compulsions. Compulsions are typically performed in response to an obsession (e.g., thoughts of contamination leading to washing rituals or that something is incorrect leading to repeating rituals until it feels "just right"). The aim is to reduce the distress triggered by obsessions or to prevent a feared event (e.g., becoming ill). However, these compulsions either are not connected in a realistic way to the feared event (e.g., arranging items symmetrically to prevent harm to a loved one) or are clearly excessive (e.g., showering for hours each day). Compulsions are not done for pleasure, although some individuals experience relief from anxiety or distress.

Criterion B emphasizes that obsessions and compulsions must be time-consuming (e.g., more than 1 hour per day) or cause clinically significant distress or impairment to warrant a diagnosis of OCD. This criterion helps to distinguish the disorder from the occasional intrusive thoughts or repetitive behaviors that are common in the general population (e.g., double-checking that a door is locked). The frequency and severity of obsessions and compulsions vary across individuals with OCD (e.g., some have mild to moderate symptoms, spending 1–3 hours per day obsessing or doing compulsions, whereas others have nearly constant intrusive thoughts or compulsions that can be incapacitating).

Associated Features Supporting Diagnosis

The specific content of obsessions and compulsions varies between individuals. However, certain themes, or dimensions, are common, including those of cleaning (contamination obsessions and cleaning compulsions); symmetry (symmetry obsessions and repeating,

ordering, and counting compulsions); forbidden or taboo thoughts (e.g., aggressive, sexual, or religious obsessions and related compulsions); and harm (e.g., fears of harm to oneself or others and checking compulsions). Some individuals also have difficulties discarding and accumulate (hoard) objects as a consequence of typical obsessions and compulsions, such as fears of harming others. These themes occur across different cultures, are relatively consistent over time in adults with the disorder, and may be associated with different neural substrates. Importantly, individuals often have symptoms in more than one dimension.

Individuals with OCD experience a range of affective responses when confronted with situations that trigger obsessions and compulsions. For example, many individuals experience marked anxiety that can include recurrent panic attacks. Others report strong feelings of disgust. While performing compulsions, some individuals report a distressing sense of "incompleteness" or uneasiness until things look, feel, or sound "just right."

It is common for individuals with the disorder to avoid people, places, and things that trigger obsessions and compulsions. For example, individuals with contamination concerns might avoid public situations (e.g., restaurants, public restrooms) to reduce exposure to feared contaminants; individuals with intrusive thoughts about causing harm might avoid social interactions.

Prevalence

The 12-month prevalence of OCD in the United States is 1.2%, with a similar prevalence internationally (1.1%–1.8%). Females are affected at a slightly higher rate than males in adulthood, although males are more commonly affected in childhood.

Development and Course

In the United States, the mean age at onset of OCD is 19.5 years, and 25% of cases start by age 14 years. Onset after age 35 years is unusual but does occur. Males have an earlier age at onset than females: nearly 25% of males have onset before age 10 years. The onset of symptoms is typically gradual; however, acute onset has also been reported.

If OCD is untreated, the course is usually chronic, often with waxing and waning symptoms. Some individuals have an episodic course, and a minority have a deteriorating course. Without treatment, remission rates in adults are low (e.g., 20% for those reevaluated 40 years later). Onset in childhood or adolescence can lead to a lifetime of OCD. However, 40% of individuals with onset of OCD in childhood or adolescence may experience remission by early adulthood. The course of OCD is often complicated by the co-occurrence of other disorders (see section "Comorbidity" for this disorder).

Compulsions are more easily diagnosed in children than obsessions are because compulsions are observable. However, most children have both obsessions and compulsions (as do most adults). The pattern of symptoms in adults can be stable over time, but it is more variable in children. Some differences in the content of obsessions and compulsions have been reported when children and adolescent samples have been compared with adult samples. These differences likely reflect content appropriate to different developmental stages (e.g., higher rates of sexual and religious obsessions in adolescents than in children; higher rates of harm obsessions [e.g., fears of catastrophic events, such as death or illness to self or loved ones] in children and adolescents than in adults).

Risk and Prognostic Factors

Temperamental. Greater internalizing symptoms, higher negative emotionality, and behavioral inhibition in childhood are possible temperamental risk factors.

Environmental. Physical and sexual abuse in childhood and other stressful or traumatic events have been associated with an increased risk for developing OCD. Some children

may develop the sudden onset of obsessive-compulsive symptoms, which has been associated with different environmental factors, including various infectious agents and a post-infectious autoimmune syndrome.

Genetic and physiological. The rate of OCD among first-degree relatives of adults with OCD is approximately two times that among first-degree relatives of those without the disorder; however, among first-degree relatives of individuals with onset of OCD in childhood or adolescence, the rate is increased 10-fold. Familial transmission is due in part to genetic factors (e.g., a concordance rate of 0.57 for monozygotic vs. 0.22 for dizygotic twins). Dysfunction in the orbitofrontal cortex, anterior cingulate cortex, and striatum have been most strongly implicated.

Culture-Related Diagnostic Issues

OCD occurs across the world. There is substantial similarity across cultures in the gender distribution, age at onset, and comorbidity of OCD. Moreover, around the globe, there is a similar symptom structure involving cleaning, symmetry, hoarding, taboo thoughts, or fear of harm. However, regional variation in symptom expression exists, and cultural factors may shape the content of obsessions and compulsions.

Gender-Related Diagnostic Issues

Males have an earlier age at onset of OCD than females and are more likely to have comorbid tic disorders. Gender differences in the pattern of symptom dimensions have been reported, with, for example, females more likely to have symptoms in the cleaning dimension and males more likely to have symptoms in the forbidden thoughts and symmetry dimensions. Onset or exacerbation of OCD, as well as symptoms that can interfere with the mother-infant relationship (e.g., aggressive obsessions leading to avoidance of the infant), have been reported in the peripartum period.

Suicide Risk

Suicidal thoughts occur at some point in as many as about half of individuals with OCD. Suicide attempts are also reported in up to one-quarter of individuals with OCD; the presence of comorbid major depressive disorder increases the risk.

Functional Consequences of Obsessive-Compulsive Disorder

OCD is associated with reduced quality of life as well as high levels of social and occupational impairment. Impairment occurs across many different domains of life and is associated with symptom severity. Impairment can be caused by the time spent obsessing and doing compulsions. Avoidance of situations that can trigger obsessions or compulsions can also severely restrict functioning. In addition, specific symptoms can create specific obstacles. For example, obsessions about harm can make relationships with family and friends feel hazardous; the result can be avoidance of these relationships. Obsessions about symmetry can derail the timely completion of school or work projects because the project never feels "just right," potentially resulting in school failure or job loss. Health consequences can also occur. For example, individuals with contamination concerns may avoid doctors' offices and hospitals (e.g., because of fears of exposure to germs) or develop dermatological problems (e.g., skin lesions due to excessive washing). Sometimes the symptoms of the disorder interfere with its own treatment (e.g., when medications are considered contaminated). When the disorder starts in childhood or adolescence, individuals may experience developmental difficulties. For example, adolescents may avoid socializing with peers; young adults may struggle when they leave home to live independently.

The result can be few significant relationships outside the family and a lack of autonomy and financial independence from their family of origin. In addition, some individuals with OCD try to impose rules and prohibitions on family members because of their disorder (e.g., no one in the family can have visitors to the house for fear of contamination), and this can lead to family dysfunction.

Differential Diagnosis

Anxiety disorders. Recurrent thoughts, avoidant behaviors, and repetitive requests for reassurance can also occur in anxiety disorders. However, the recurrent thoughts that are present in generalized anxiety disorder (i.e., worries) are usually about real-life concerns, whereas the obsessions of OCD usually do not involve real-life concerns and can include content that is odd, irrational, or of a seemingly magical nature; moreover, compulsions are often present and usually linked to the obsessions. Like individuals with OCD, individuals with specific phobia can have a fear reaction to specific objects or situations; however, in specific phobia the feared object is usually much more circumscribed, and rituals are not present. In social anxiety disorder (social phobia), the feared objects or situations are limited to social interactions, and avoidance or reassurance seeking is focused on reducing this social fear.

Major depressive disorder. OCD can be distinguished from the rumination of major depressive disorder, in which thoughts are usually mood-congruent and not necessarily experienced as intrusive or distressing; moreover, ruminations are not linked to compulsions, as is typical in OCD.

Other obsessive-compulsive and related disorders. In body dysmorphic disorder, the obsessions and compulsions are limited to concerns about physical appearance; and in trichotillomania (hair-pulling disorder), the compulsive behavior is limited to hair pulling in the absence of obsessions. Hoarding disorder symptoms focus exclusively on the persistent difficulty discarding or parting with possessions, marked distress associated with discarding items, and excessive accumulation of objects. However, if an individual has obsessions that are typical of OCD (e.g., concerns about incompleteness or harm), and these obsessions lead to compulsive hoarding behaviors (e.g., acquiring all objects in a set to attain a sense of completeness or not discarding old newspapers because they may contain information that could prevent harm), a diagnosis of OCD should be given instead.

Eating disorders. OCD can be distinguished from anorexia nervosa in that in OCD the obsessions and compulsions are not limited to concerns about weight and food.

Tics (in tic disorder) and stereotyped movements. A *tic* is a sudden, rapid, recurrent, nonrhythmic motor movement or vocalization (e.g., eye blinking, throat clearing). A *stereotyped movement* is a repetitive, seemingly driven, nonfunctional motor behavior (e.g., head banging, body rocking, self-biting). Tics and stereotyped movements are typically less complex than compulsions and are not aimed at neutralizing obsessions. However, distinguishing between complex tics and compulsions can be difficult. Whereas compulsions are usually preceded by obsessions, tics are often preceded by premonitory sensory urges. Some individuals have symptoms of both OCD and a tic disorder, in which case both diagnoses may be warranted.

Psychotic disorders. Some individuals with OCD have poor insight or even delusional OCD beliefs. However, they have obsessions and compulsions (distinguishing their condition from delusional disorder) and do not have other features of schizophrenia or schizoaffective disorder (e.g., hallucinations or formal thought disorder).

Other compulsive-like behaviors. Certain behaviors are sometimes described as "compulsive," including sexual behavior (in the case of paraphilias), gambling (i.e., gambling

disorder), and substance use (e.g., alcohol use disorder). However, these behaviors differ from the compulsions of OCD in that the person usually derives pleasure from the activity and may wish to resist it only because of its deleterious consequences.

Obsessive-compulsive personality disorder. Although obsessive-compulsive personality disorder and OCD have similar names, the clinical manifestations of these disorders are quite different. Obsessive-compulsive personality disorder is not characterized by intrusive thoughts, images, or urges or by repetitive behaviors that are performed in response to these intrusions; instead, it involves an enduring and pervasive maladaptive pattern of excessive perfectionism and rigid control. If an individual manifests symptoms of both OCD and obsessive-compulsive personality disorder, both diagnoses can be given.

Comorbidity

Individuals with OCD often have other psychopathology. Many adults with the disorder have a lifetime diagnosis of an anxiety disorder (76%; e.g., panic disorder, social anxiety disorder, generalized anxiety disorder, specific phobia) or a depressive or bipolar disorder (63% for any depressive or bipolar disorder, with the most common being major depressive disorder [41%]). Onset of OCD is usually later than for most comorbid anxiety disorders (with the exception of separation anxiety disorder) and PTSD but often precedes that of depressive disorders. Comorbid obsessive-compulsive personality disorder is also common in individuals with OCD (e.g., ranging from 23% to 32%).

Up to 30% of individuals with OCD also have a lifetime tic disorder. A comorbid tic disorder is most common in males with onset of OCD in childhood. These individuals tend to differ from those without a history of tic disorders in the themes of their OCD symptoms, comorbidity, course, and pattern of familial transmission. A triad of OCD, tic disorder, and attention-deficit/hyperactivity disorder can also be seen in children.

Disorders that occur more frequently in individuals with OCD than in those without the disorder include several obsessive-compulsive and related disorders such as body dysmorphic disorder, trichotillomania (hair-pulling disorder), and excoriation (skin-picking) disorder. Finally, an association between OCD and some disorders characterized by impulsivity, such as oppositional defiant disorder, has been reported.

OCD is also much more common in individuals with certain other disorders than would be expected based on its prevalence in the general population; when one of those other disorders is diagnosed, the individual should be assessed for OCD as well. For example, in individuals with schizophrenia or schizoaffective disorder, the prevalence of OCD is approximately 12%. Rates of OCD are also elevated in bipolar disorder; eating disorders, such as anorexia nervosa and bulimia nervosa; and Tourette's disorder.

Body Dysmorphic Disorder

Diagnostic Criteria **300.7 (F45.22)**

A. Preoccupation with one or more perceived defects or flaws in physical appearance that are not observable or appear slight to others.

B. At some point during the course of the disorder, the individual has performed repetitive behaviors (e.g., mirror checking, excessive grooming, skin picking, reassurance seeking) or mental acts (e.g., comparing his or her appearance with that of others) in response to the appearance concerns.

C. The preoccupation causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The appearance preoccupation is not better explained by concerns with body fat or weight in an individual whose symptoms meet diagnostic criteria for an eating disorder.

Specify if:

With muscle dysmorphia: The individual is preoccupied with the idea that his or her body build is too small or insufficiently muscular. This specifier is used even if the individual is preoccupied with other body areas, which is often the case.

Specify if:

Indicate degree of insight regarding body dysmorphic disorder beliefs (e.g., "I look ugly" or "I look deformed").

With good or fair insight: The individual recognizes that the body dysmorphic disorder beliefs are definitely or probably not true or that they may or may not be true.

With poor insight: The individual thinks that the body dysmorphic disorder beliefs are probably true.

With absent insight/delusional beliefs: The individual is completely convinced that the body dysmorphic disorder beliefs are true.

Diagnostic Features

Individuals with body dysmorphic disorder (formerly known as *dysmorphophobia*) are preoccupied with one or more perceived defects or flaws in their physical appearance, which they believe look ugly, unattractive, abnormal, or deformed (Criterion A). The perceived flaws are not observable or appear only slight to other individuals. Concerns range from looking "unattractive" or "not right" to looking "hideous" or "like a monster." Preoccupations can focus on one or many body areas, most commonly the skin (e.g., perceived acne, scars, lines, wrinkles, paleness), hair (e.g., "thinning" hair or "excessive" body or facial hair), or nose (e.g., size or shape). However, any body area can be the focus of concern (e.g., eyes, teeth, weight, stomach, breasts, legs, face size or shape, lips, chin, eyebrows, genitals). Some individuals are concerned about perceived asymmetry of body areas. The preoccupations are intrusive, unwanted, time-consuming (occurring, on average, 3–8 hours per day), and usually difficult to resist or control.

Excessive repetitive behaviors or mental acts (e.g., comparing) are performed in response to the preoccupation (Criterion B). The individual feels driven to perform these behaviors, which are not pleasurable and may increase anxiety and dysphoria. They are typically time-consuming and difficult to resist or control. Common behaviors are comparing one's appearance with that of other individuals; repeatedly checking perceived defects in mirrors or other reflecting surfaces or examining them directly; excessively grooming (e.g., combing, styling, shaving, plucking, or pulling hair); camouflaging (e.g., repeatedly applying makeup or covering disliked areas with such things as a hat, clothing, makeup, or hair); seeking reassurance about how the perceived flaws look; touching disliked areas to check them; excessively exercising or weight lifting; and seeking cosmetic procedures. Some individuals excessively tan (e.g., to darken "pale" skin or diminish perceived acne), repeatedly change their clothes (e.g., to camouflage perceived defects), or compulsively shop (e.g., for beauty products). Compulsive skin picking intended to improve perceived skin defects is common and can cause skin damage, infections, or ruptured blood vessels. The preoccupation must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion C); usually both are present. Body dysmorphic disorder must be differentiated from an eating disorder.

Muscle dysmorphia, a form of body dysmorphic disorder occurring almost exclusively in males, consists of preoccupation with the idea that one's body is too small or insufficiently lean or muscular. Individuals with this form of the disorder actually have a normal-looking body or are even very muscular. They may also be preoccupied with other body areas, such as skin or hair. A majority (but not all) diet, exercise, and/or lift weights excessively, sometimes causing bodily damage. Some use potentially dangerous anabolic-

androgenic steroids and other substances to try to make their body bigger and more muscular. Body dysmorphic disorder by proxy is a form of body dysmorphic disorder in which individuals are preoccupied with defects they perceive in another person's appearance.

Insight regarding body dysmorphic disorder beliefs can range from good to absent/delusional (i.e., delusional beliefs consisting of complete conviction that the individual's view of their appearance is accurate and undistorted). On average, insight is poor; one-third or more of individuals currently have delusional body dysmorphic disorder beliefs. Individuals with delusional body dysmorphic disorder tend to have greater morbidity in some areas (e.g., suicidality), but this appears accounted for by their tendency to have more severe body dysmorphic disorder symptoms.

Associated Features Supporting Diagnosis

Many individuals with body dysmorphic disorder have ideas or delusions of reference, believing that other people take special notice of them or mock them because of how they look. Body dysmorphic disorder is associated with high levels of anxiety, social anxiety, social avoidance, depressed mood, neuroticism, and perfectionism as well as low extroversion and low self-esteem. Many individuals are ashamed of their appearance and their excessive focus on how they look, and are reluctant to reveal their concerns to others. A majority of individuals receive cosmetic treatment to try to improve their perceived defects. Dermatological treatment and surgery are most common, but any type (e.g., dental, electrolysis) may be received. Occasionally, individuals may perform surgery on themselves. Body dysmorphic disorder appears to respond poorly to such treatments and sometimes becomes worse. Some individuals take legal action or are violent toward the clinician because they are dissatisfied with the cosmetic outcome.

Body dysmorphic disorder has been associated with executive dysfunction and visual processing abnormalities, with a bias for analyzing and encoding details rather than holistic or configural aspects of visual stimuli. Individuals with this disorder tend to have a bias for negative and threatening interpretations of facial expressions and ambiguous scenarios.

Prevalence

The point prevalence among U.S. adults is 2.4% (2.5% in females and 2.2% in males). Outside the United States (i.e., Germany), current prevalence is approximately 1.7%–1.8%, with a gender distribution similar to that in the United States. The current prevalence is 9%–15% among dermatology patients, 7%–8% among U.S. cosmetic surgery patients, 3%–16% among international cosmetic surgery patients (most studies), 8% among adult orthodontia patients, and 10% among patients presenting for oral or maxillofacial surgery.

Development and Course

The mean age at disorder onset is 16–17 years, the median age at onset is 15 years, and the most common age at onset is 12–13 years. Two-thirds of individuals have disorder onset before age 18. Subclinical body dysmorphic disorder symptoms begin, on average, at age 12 or 13 years. Subclinical concerns usually evolve gradually to the full disorder, although some individuals experience abrupt onset of body dysmorphic disorder. The disorder appears to usually be chronic, although improvement is likely when evidence-based treatment is received. The disorder's clinical features appear largely similar in children/adolescents and adults. Body dysmorphic disorder occurs in the elderly, but little is known about the disorder in this age group. Individuals with disorder onset before age 18 years are more likely to attempt suicide, have more comorbidity, and have gradual (rather than acute) disorder onset than those with adult-onset body dysmorphic disorder.

Risk and Prognostic Factors

Environmental. Body dysmorphic disorder has been associated with high rates of childhood neglect and abuse.

Genetic and physiological. The prevalence of body dysmorphic disorder is elevated in first-degree relatives of individuals with obsessive-compulsive disorder (OCD).

Culture-Related Diagnostic Issues

Body dysmorphic disorder has been reported internationally. It appears that the disorder may have more similarities than differences across races and cultures but that cultural values and preferences may influence symptom content to some degree. *Taijin kyofusho*, included in the traditional Japanese diagnostic system, has a subtype similar to body dysmorphic disorder: *shubo-kyofu* ("the phobia of a deformed body").

Gender-Related Diagnostic Issues

Females and males appear to have more similarities than differences in terms of most clinical features— for example, disliked body areas, types of repetitive behaviors, symptom severity, suicidality, comorbidity, illness course, and receipt of cosmetic procedures for body dysmorphic disorder. However, males are more likely to have genital preoccupations, and females are more likely to have a comorbid eating disorder. Muscle dysmorphia occurs almost exclusively in males.

Suicide Risk

Rates of suicidal ideation and suicide attempts are high in both adults and children/adolescents with body dysmorphic disorder. Furthermore, risk for suicide appears high in adolescents. A substantial proportion of individuals attribute suicidal ideation or suicide attempts primarily to their appearance concerns. Individuals with body dysmorphic disorder have many risk factors for completed suicide, such as high rates of suicidal ideation and suicide attempts, demographic characteristics associated with suicide, and high rates of comorbid major depressive disorder.

Functional Consequences of Body Dysmorphic Disorder

Nearly all individuals with body dysmorphic disorder experience impaired psychosocial functioning because of their appearance concerns. Impairment can range from moderate (e.g., avoidance of some social situations) to extreme and incapacitating (e.g., being completely housebound). On average, psychosocial functioning and quality of life are markedly poor. More severe body dysmorphic disorder symptoms are associated with poorer functioning and quality of life. Most individuals experience impairment in their job, academic, or role functioning (e.g., as a parent or caregiver), which is often severe (e.g., performing poorly, missing school or work, not working). About 20% of youths with body dysmorphic disorder report dropping out of school primarily because of their body dysmorphic disorder symptoms. Impairment in social functioning (e.g., social activities, relationships, intimacy), including avoidance, is common. Individuals may be housebound because of their body dysmorphic disorder symptoms, sometimes for years. A high proportion of adults and adolescents have been psychiatrically hospitalized.

Differential Diagnosis

Normal appearance concerns and clearly noticeable physical defects. Body dysmorphic disorder differs from normal appearance concerns in being characterized by exces-

sive appearance-related preoccupations and repetitive behaviors that are time-consuming, are usually difficult to resist or control, and cause clinically significant distress or impairment in functioning. Physical defects that are clearly noticeable (i.e., not slight) are not diagnosed as body dysmorphic disorder. However, skin picking as a symptom of body dysmorphic disorder can cause noticeable skin lesions and scarring; in such cases, body dysmorphic disorder should be diagnosed.

Eating disorders. In an individual with an eating disorder, concerns about being fat are considered a symptom of the eating disorder rather than body dysmorphic disorder. However, weight concerns may occur in body dysmorphic disorder. Eating disorders and body dysmorphic disorder can be comorbid, in which case both should be diagnosed.

Other obsessive-compulsive and related disorders. The preoccupations and repetitive behaviors of body dysmorphic disorder differ from obsessions and compulsions in OCD in that the former focus only on appearance. These disorders have other differences, such as poorer insight in body dysmorphic disorder. When skin picking is intended to improve the appearance of perceived skin defects, body dysmorphic disorder, rather than excoriation (skin-picking) disorder, is diagnosed. When hair removal (plucking, pulling, or other types of removal) is intended to improve perceived defects in the appearance of facial or body hair, body dysmorphic disorder is diagnosed rather than trichotillomania (hair-pulling disorder).

Illness anxiety disorder. Individuals with body dysmorphic disorder are not preoccupied with having or acquiring a serious illness and do not have particularly elevated levels of somatization.

Major depressive disorder. The prominent preoccupation with appearance and excessive repetitive behaviors in body dysmorphic disorder differentiate it from major depressive disorder. However, major depressive disorder and depressive symptoms are common in individuals with body dysmorphic disorder, often appearing to be secondary to the distress and impairment that body dysmorphic disorder causes. Body dysmorphic disorder should be diagnosed in depressed individuals if diagnostic criteria for body dysmorphic disorder are met.

Anxiety disorders. Social anxiety and avoidance are common in body dysmorphic disorder. However, unlike social anxiety disorder (social phobia), agoraphobia, and avoidant personality disorder, body dysmorphic disorder includes prominent appearance-related preoccupation, which may be delusional, and repetitive behaviors, and the social anxiety and avoidance are due to concerns about perceived appearance defects and the belief or fear that other people will consider these individuals ugly, ridicule them, or reject them because of their physical features. Unlike generalized anxiety disorder, anxiety and worry in body dysmorphic disorder focus on perceived appearance flaws.

Psychotic disorders. Many individuals with body dysmorphic disorder have delusional appearance beliefs (i.e., complete conviction that their view of their perceived defects is accurate), which is diagnosed as body dysmorphic disorder, with absent insight/delusional beliefs, not as delusional disorder. Appearance-related ideas or delusions of reference are common in body dysmorphic disorder; however, unlike schizophrenia or schizoaffective disorder, body dysmorphic disorder involves prominent appearance preoccupations and related repetitive behaviors, and disorganized behavior and other psychotic symptoms are absent (except for appearance beliefs, which may be delusional).

Other disorders and symptoms. Body dysmorphic disorder should not be diagnosed if the preoccupation is limited to discomfort with or a desire to be rid of one's primary and/or secondary sex characteristics in an individual with gender dysphoria or if the preoccupation focuses on the belief that one emits a foul or offensive body odor as in olfactory reference syndrome (which is not a DSM-5 disorder). Body identity integrity disorder

(apotemnophilia) (which is not a DSM-5 disorder) involves a desire to have a limb amputated to correct an experience of mismatch between a person's sense of body identity and his or her actual anatomy. However, the concern does not focus on the limb's appearance, as it would in body dysmorphic disorder. *Koro*, a culturally related disorder that usually occurs in epidemics in Southeastern Asia, consists of a fear that the penis (labia, nipples, or breasts in females) is shrinking or retracting and will disappear into the abdomen, often accompanied by a belief that death will result. Koro differs from body dysmorphic disorder in several ways, including a focus on death rather than preoccupation with perceived ugliness. *Dysmorphic concern* (which is not a DSM-5 disorder) is a much broader construct than, and is not equivalent to, body dysmorphic disorder. It involves symptoms reflecting an overconcern with slight or imagined flaws in appearance.

Comorbidity

Major depressive disorder is the most common comorbid disorder, with onset usually after that of body dysmorphic disorder. Comorbid social anxiety disorder (social phobia), OCD, and substance-related disorders are also common.

Hoarding Disorder

Diagnostic Criteria **300.3 (F42)**

A. Persistent difficulty discarding or parting with possessions, regardless of their actual value.
B. This difficulty is due to a perceived need to save the items and to distress associated with discarding them.
C. The difficulty discarding possessions results in the accumulation of possessions that congest and clutter active living areas and substantially compromises their intended use. If living areas are uncluttered, it is only because of the interventions of third parties (e.g., family members, cleaners, authorities).
D. The hoarding causes clinically significant distress or impairment in social, occupational, or other important areas of functioning (including maintaining a safe environment for self and others).
E. The hoarding is not attributable to another medical condition (e.g., brain injury, cerebrovascular disease, Prader-Willi syndrome).
F. The hoarding is not better explained by the symptoms of another mental disorder (e.g., obsessions in obsessive-compulsive disorder, decreased energy in major depressive disorder, delusions in schizophrenia or another psychotic disorder, cognitive deficits in major neurocognitive disorder, restricted interests in autism spectrum disorder).

Specify if:

With excessive acquisition: If difficulty discarding possessions is accompanied by excessive acquisition of items that are not needed or for which there is no available space.

Specify if:

With good or fair insight: The individual recognizes that hoarding-related beliefs and behaviors (pertaining to difficulty discarding items, clutter, or excessive acquisition) are problematic.

With poor insight: The individual is mostly convinced that hoarding-related beliefs and behaviors (pertaining to difficulty discarding items, clutter, or excessive acquisition) are not problematic despite evidence to the contrary.

With absent insight/delusional beliefs: The individual is completely convinced that hoarding-related beliefs and behaviors (pertaining to difficulty discarding items, clutter, or excessive acquisition) are not problematic despite evidence to the contrary.

Specifiers

With excessive acquisition. Approximately 80%–90% of individuals with hoarding disorder display excessive acquisition. The most frequent form of acquisition is excessive buying, followed by acquisition of free items (e.g., leaflets, items discarded by others). Stealing is less common. Some individuals may deny excessive acquisition when first assessed, yet it may appear later during the course of treatment. Individuals with hoarding disorder typically experience distress if they are unable to or are prevented from acquiring items.

Diagnostic Features

The essential feature of hoarding disorder is persistent difficulties discarding or parting with possessions, regardless of their actual value (Criterion A). The term *persistent* indicates a long-standing difficulty rather than more transient life circumstances that may lead to excessive clutter, such as inheriting property. The difficulty in discarding possessions noted in Criterion A refers to any form of discarding, including throwing away, selling, giving away, or recycling. The main reasons given for these difficulties are the perceived utility or aesthetic value of the items or strong sentimental attachment to the possessions. Some individuals feel responsible for the fate of their possessions and often go to great lengths to avoid being wasteful. Fears of losing important information are also common. The most commonly saved items are newspapers, magazines, old clothing, bags, books, mail, and paperwork, but virtually any item can be saved. The nature of items is not limited to possessions that most other people would define as useless or of limited value. Many individuals collect and save large numbers of valuable things as well, which are often found in piles mixed with other less valuable items.

Individuals with hoarding disorder purposefully save possessions and experience distress when facing the prospect of discarding them (Criterion B). This criterion emphasizes that the saving of possessions is intentional, which discriminates hoarding disorder from other forms of psychopathology that are characterized by the passive accumulation of items or the absence of distress when possessions are removed.

Individuals accumulate large numbers of items that fill up and clutter active living areas to the extent that their intended use is no longer possible (Criterion C). For example, the individual may not be able to cook in the kitchen, sleep in his or her bed, or sit in a chair. If the space can be used, it is only with great difficulty. *Clutter* is defined as a large group of usually unrelated or marginally related objects piled together in a disorganized fashion in spaces designed for other purposes (e.g., tabletops, floor, hallway). Criterion C emphasizes the "active" living areas of the home, rather than more peripheral areas, such as garages, attics, or basements, that are sometimes cluttered in homes of individuals without hoarding disorder. However, individuals with hoarding disorder often have possessions that spill beyond the active living areas and can occupy and impair the use of other spaces, such as vehicles, yards, the workplace, and friends' and relatives' houses. In some cases, living areas may be uncluttered because of the intervention of third parties (e.g., family members, cleaners, local authorities). Individuals who have been forced to clear their homes still have a symptom picture that meets criteria for hoarding disorder because the lack of clutter is due to a third-party intervention. Hoarding disorder contrasts with normative collecting behavior, which is organized and systematic, even if in some cases the actual amount of possessions may be similar to the amount accumulated by an individual with hoarding disorder. Normative collecting does not produce the clutter, distress, or impairment typical of hoarding disorder.

Symptoms (i.e., difficulties discarding and/or clutter) must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning, including maintaining a safe environment for self and others (Criterion D). In some cases,

particularly when there is poor insight, the individual may not report distress, and the impairment may be apparent only to those around the individual. However, any attempts to discard or clear the possessions by third parties result in high levels of distress.

Associated Features Supporting Diagnosis

Other common features of hoarding disorder include indecisiveness, perfectionism, avoidance, procrastination, difficulty planning and organizing tasks, and distractibility. Some individuals with hoarding disorder live in unsanitary conditions that may be a logical consequence of severely cluttered spaces and/or that are related to planning and organizing difficulties. *Animal hoarding* can be defined as the accumulation of a large number of animals and a failure to provide minimal standards of nutrition, sanitation, and veterinary care and to act on the deteriorating condition of the animals (including disease, starvation, or death) and the environment (e.g., severe overcrowding, extremely unsanitary conditions). Animal hoarding may be a special manifestation of hoarding disorder. Most individuals who hoard animals also hoard inanimate objects. The most prominent differences between animal and object hoarding are the extent of unsanitary conditions and the poorer insight in animal hoarding.

Prevalence

Nationally representative prevalence studies of hoarding disorder are not available. Community surveys estimate the point prevalence of clinically significant hoarding in the United States and Europe to be approximately 2%–6%. Hoarding disorder affects both males and females, but some epidemiological studies have reported a significantly greater prevalence among males. This contrasts with clinical samples, which are predominantly female. Hoarding symptoms appear to be almost three times more prevalent in older adults (ages 55–94 years) compared with younger adults (ages 34–44 years).

Development and Course

Hoarding appears to begin early in life and spans well into the late stages. Hoarding symptoms may first emerge around ages 11–15 years, start interfering with the individual's everyday functioning by the mid-20s, and cause clinically significant impairment by the mid-30s. Participants in clinical research studies are usually in their 50s. Thus, the severity of hoarding increases with each decade of life. Once symptoms begin, the course of hoarding is often chronic, with few individuals reporting a waxing and waning course.

Pathological hoarding in children appears to be easily distinguished from developmentally adaptive saving and collecting behaviors. Because children and adolescents typically do not control their living environment and discarding behaviors, the possible intervention of third parties (e.g., parents keeping the spaces usable and thus reducing interference) should be considered when making the diagnosis.

Risk and Prognostic Factors

Temperamental. Indecisiveness is a prominent feature of individuals with hoarding disorder and their first-degree relatives.

Environmental. Individuals with hoarding disorder often retrospectively report stressful and traumatic life events preceding the onset of the disorder or causing an exacerbation.

Genetic and physiological. Hoarding behavior is familial, with about 50% of individuals who hoard reporting having a relative who also hoards. Twin studies indicate that approximately 50% of the variability in hoarding behavior is attributable to additive genetic factors.

Culture-Related Diagnostic Issues

While most of the research has been done in Western, industrialized countries and urban communities, the available data from non-Western and developing countries suggest that hoarding is a universal phenomenon with consistent clinical features.

Gender-Related Diagnostic Issues

The key features of hoarding disorder (i.e., difficulties discarding, excessive amount of clutter) are generally comparable in males and females, but females tend to display more excessive acquisition, particularly excessive buying, than do males.

Functional Consequences of Hoarding Disorder

Clutter impairs basic activities, such as moving through the house, cooking, cleaning, personal hygiene, and even sleeping. Appliances may be broken, and utilities such as water and electricity may be disconnected, as access for repair work may be difficult. Quality of life is often considerably impaired. In severe cases, hoarding can put individuals at risk for fire, falling (especially elderly individuals), poor sanitation, and other health risks. Hoarding disorder is associated with occupational impairment, poor physical health, and high social service utilization. Family relationships are frequently under great strain. Conflict with neighbors and local authorities is common, and a substantial proportion of individuals with severe hoarding disorder have been involved in legal eviction proceedings, and some have a history of eviction.

Differential Diagnosis

Other medical conditions. Hoarding disorder is not diagnosed if the symptoms are judged to be a direct consequence of another medical condition (Criterion E), such as traumatic brain injury, surgical resection for treatment of a tumor or seizure control, cerebrovascular disease, infections of the central nervous system (e.g., herpes simplex encephalitis), or neurogenetic conditions such as Prader-Willi syndrome. Damage to the anterior ventromedial prefrontal and cingulate cortices has been particularly associated with the excessive accumulation of objects. In these individuals, the hoarding behavior is not present prior to the onset of the brain damage and appears shortly after the brain damage occurs. Some of these individuals appear to have little interest in the accumulated items and are able to discard them easily or do not care if others discard them, whereas others appear to be very reluctant to discard anything.

Neurodevelopmental disorders. Hoarding disorder is not diagnosed if the accumulation of objects is judged to be a direct consequence of a neurodevelopmental disorder, such as autism spectrum disorder or intellectual disability (intellectual developmental disorder).

Schizophrenia spectrum and other psychotic disorders. Hoarding disorder is not diagnosed if the accumulation of objects is judged to be a direct consequence of delusions or negative symptoms in schizophrenia spectrum and other psychotic disorders.

Major depressive episode. Hoarding disorder is not diagnosed if the accumulation of objects is judged to be a direct consequence of psychomotor retardation, fatigue, or loss of energy during a major depressive episode.

Obsessive-compulsive disorder. Hoarding disorder is not diagnosed if the symptoms are judged to be a direct consequence of typical obsessions or compulsions, such as fears of contamination, harm, or feelings of incompleteness in obsessive-compulsive disorder (OCD). Feelings of incompleteness (e.g., losing one's identity, or having to document and preserve all life experiences) are the most frequent OCD symptoms associated with this form of hoarding. The accumulation of objects can also be the result of persistently avoid-

ing onerous rituals (e.g., not discarding objects in order to avoid endless washing or checking rituals).

In OCD, the behavior is generally unwanted and highly distressing, and the individual experiences no pleasure or reward from it. Excessive acquisition is usually not present; if excessive acquisition is present, items are acquired because of a specific obsession (e.g., the need to buy items that have been accidentally touched in order to avoid contaminating other people), not because of a genuine desire to possess the items. Individuals who hoard in the context of OCD are also more likely to accumulate bizarre items, such as trash, feces, urine, nails, hair, used diapers, or rotten food. Accumulation of such items is very unusual in hoarding disorder.

When severe hoarding appears concurrently with other typical symptoms of OCD but is judged to be independent from these symptoms, both hoarding disorder and OCD may be diagnosed.

Neurocognitive disorders. Hoarding disorder is not diagnosed if the accumulation of objects is judged to be a direct consequence of a degenerative disorder, such as neurocognitive disorder associated with frontotemporal lobar degeneration or Alzheimer's disease. Typically, onset of the accumulating behavior is gradual and follows onset of the neurocognitive disorder. The accumulating behavior may be accompanied by self-neglect and severe domestic squalor, alongside other neuropsychiatric symptoms, such as disinhibition, gambling, rituals/stereotypies, tics, and self-injurious behaviors.

Comorbidity

Approximately 75% of individuals with hoarding disorder have a comorbid mood or anxiety disorder. The most common comorbid conditions are major depressive disorder (up to 50% of cases), social anxiety disorder (social phobia), and generalized anxiety disorder. Approximately 20% of individuals with hoarding disorder also have symptoms that meet diagnostic criteria for OCD. These comorbidities may often be the main reason for consultation, because individuals are unlikely to spontaneously report hoarding symptoms, and these symptoms are often not asked about in routine clinical interviews.

Trichotillomania (Hair-Pulling Disorder)

Diagnostic Criteria **312.39 (F63.2)**

A. Recurrent pulling out of one's hair, resulting in hair loss.
B. Repeated attempts to decrease or stop hair pulling.
C. The hair pulling causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
D. The hair pulling or hair loss is not attributable to another medical condition (e.g., a dermatological condition).
E. The hair pulling is not better explained by the symptoms of another mental disorder (e.g., attempts to improve a perceived defect or flaw in appearance in body dysmorphic disorder).

Diagnostic Features

The essential feature of trichotillomania (hair-pulling disorder) is the recurrent pulling out of one's own hair (Criterion A). Hair pulling may occur from any region of the body in which hair grows; the most common sites are the scalp, eyebrows, and eyelids, while less common sites are axillary, facial, pubic, and peri-rectal regions. Hair-pulling sites may vary over time. Hair pulling may occur in brief episodes scattered throughout the day or during less frequent but more sustained periods that can continue for hours, and such hair

pulling may endure for months or years. Criterion A requires that hair pulling lead to hair loss, although individuals with this disorder may pull hair in a widely distributed pattern (i.e., pulling single hairs from all over a site) such that hair loss may not be clearly visible. Alternatively, individuals may attempt to conceal or camouflage hair loss (e.g., by using makeup, scarves, or wigs). Individuals with trichotillomania have made repeated attempts to decrease or stop hair pulling (Criterion B). Criterion C indicates that hair pulling causes clinically significant distress or impairment in social, occupational, or other important areas of functioning. The term *distress* includes negative affects that may be experienced by individuals with hair pulling, such as feeling a loss of control, embarrassment, and shame. Significant impairment may occur in several different areas of functioning (e.g., social, occupational, academic, and leisure), in part because of avoidance of work, school, or other public situations.

Associated Features Supporting Diagnosis

Hair pulling may be accompanied by a range of behaviors or rituals involving hair. Thus, individuals may search for a particular kind of hair to pull (e.g., hairs with a specific texture or color), may try to pull out hair in a specific way (e.g., so that the root comes out intact), or may visually examine or tactilely or orally manipulate the hair after it has been pulled (e.g., rolling the hair between the fingers, pulling the strand between the teeth, biting the hair into pieces, or swallowing the hair).

Hair pulling may also be preceded or accompanied by various emotional states; it may be triggered by feelings of anxiety or boredom, may be preceded by an increasing sense of tension (either immediately before pulling out the hair or when attempting to resist the urge to pull), or may lead to gratification, pleasure, or a sense of relief when the hair is pulled out. Hair-pulling behavior may involve varying degrees of conscious awareness, with some individuals displaying more focused attention on the hair pulling (with preceding tension and subsequent relief), and other individuals displaying more automatic behavior (in which the hair pulling seems to occur without full awareness). Many individuals report a mix of both behavioral styles. Some individuals experience an "itch-like" or tingling sensation in the scalp that is alleviated by the act of pulling hair. Pain does not usually accompany hair pulling.

Patterns of hair loss are highly variable. Areas of complete alopecia, as well as areas of thinned hair density, are common. When the scalp is involved, there may be a predilection for pulling out hair in the crown or parietal regions. There may be a pattern of nearly complete baldness except for a narrow perimeter around the outer margins of the scalp, particularly at the nape of the neck ("tonsure trichotillomania"). Eyebrows and eyelashes may be completely absent.

Hair pulling does not usually occur in the presence of other individuals, except immediate family members. Some individuals have urges to pull hair from other individuals and may sometimes try to find opportunities to do so surreptitiously. Some individuals may pull hairs from pets, dolls, and other fibrous materials (e.g., sweaters or carpets). Some individuals may deny their hair pulling to others. The majority of individuals with trichotillomania also have one or more other body-focused repetitive behaviors, including skin picking, nail biting, and lip chewing.

Prevalence

In the general population, the 12-month prevalence estimate for trichotillomania in adults and adolescents is 1%–2%. Females are more frequently affected than males, at a ratio of approximately 10:1. This estimate likely reflects the true gender ratio of the condition, although it may also reflect differential treatment seeking based on gender or cultural attitudes regarding appearance (e.g., acceptance of normative hair loss among males). Among children with trichotillomania, males and females are more equally represented.

Development and Course

Hair pulling may be seen in infants, and this behavior typically resolves during early development. Onset of hair pulling in trichotillomania most commonly coincides with, or follows the onset of, puberty. Sites of hair pulling may vary over time. The usual course of trichotillomania is chronic, with some waxing and waning if the disorder is untreated. Symptoms may possibly worsen in females accompanying hormonal changes (e.g., menstruation, perimenopause). For some individuals, the disorder may come and go for weeks, months, or years at a time. A minority of individuals remit without subsequent relapse within a few years of onset.

Risk and Prognostic Factors

Genetic and physiological. There is evidence for a genetic vulnerability to trichotillomania. The disorder is more common in individuals with obsessive-compulsive disorder (OCD) and their first-degree relatives than in the general population.

Culture-Related Diagnostic Issues

Trichotillomania appears to manifest similarly across cultures, although there is a paucity of data from non-Western regions.

Diagnostic Markers

Most individuals with trichotillomania admit to hair pulling; thus, dermatopathological diagnosis is rarely required. Skin biopsy and dermoscopy (or trichoscopy) of trichotillomania are able to differentiate the disorder from other causes of alopecia. In trichotillomania, dermoscopy shows a range of characteristic features, including decreased hair density, short vellus hair, and broken hairs with different shaft lengths.

Functional Consequences of Trichotiiiomania (Hair-Puliing Disorder)

Trichotillomania is associated with distress as well as with social and occupational impairment. There may be irreversible damage to hair growth and hair quality. Infrequent medical consequences of trichotillomania include digit purpura, musculoskeletal injury (e.g., carpal tunnel syndrome; back, shoulder and neck pain), blepharitis, and dental damage (e.g., worn or broken teeth due to hair biting). Swallowing of hair (trichophagia) may lead to trichobezoars, with subsequent anemia, abdominal pain, hematemesis, nausea and vomiting, bowel obstruction, and even perforation.

Differential Diagnosis

Normative hair removal/manipulation. Trichotillomania should not be diagnosed when hair removal is performed solely for cosmetic reasons (i.e., to improve one's physical appearance). Many individuals twist and play with their hair, but this behavior does not usually qualify for a diagnosis of trichotillomania. Some individuals may bite rather than pull hair; again, this does not qualify for a diagnosis of trichotillomania.

Other obsessive-compulsive and related disorders. Individuals with OCD and symmetry concerns may pull out hairs as part of their symmetry rituals, and individuals with body dysmorphic disorder may remove body hair that they perceive as ugly, asymmetrical, or abnormal; in such cases a diagnosis of trichotillomania is not given. The description of body-focused repetitive behavior disorder in other specified obsessive-compulsive and related disorder excludes individuals who meet diagnostic criteria for trichotillomania.

Neurodevelopmental disorders. In neurodevelopmental disorders, hair pulling may meet the definition of stereotypies (e.g., in stereotypic movement disorder). Tics (in tic disorders) rarely lead to hair pulling.

Psychotic disorder. Individuals with a psychotic disorder may remove hair in response to a delusion or hallucination. Trichotillomania is not diagnosed in such cases.

Another medical condition. Trichotillomania is not diagnosed if the hair pulling or hair loss is attributable to another medical condition (e.g., inflammation of the skin or other dermatological conditions). Other causes of scarring alopecia (e.g., alopecia areata, androgenic alopecia, telogen effluvium) or nonscarring alopecia (e.g., chronic discoid lupus erythematosus, lichen planopilaris, central centrifugal cicatricial alopecia, pseudopelade, folliculitis decalvans, dissecting folliculitis, acne keloidalis nuchae) should be considered in individuals with hair loss who deny hair pulling. Skin biopsy or dermoscopy can be used to differentiate individuals with trichotillomania from those with dermatological disorders.

Substance-related disorders. Hair-pulling symptoms may be exacerbated by certain substances—for example, stimulants—but it is less likely that substances are the primary cause of persistent hair pulling.

Comorbidity

Trichotillomania is often accompanied by other mental disorders, most commonly major depressive disorder and excoriation (skin-picking) disorder. Repetitive body-focused symptoms other than hair pulling or skin picking (e.g. nail biting) occur in the majority of individuals with trichotillomania and may deserve an additional diagnosis of other specified obsessive-compulsive and related disorder (i.e., body-focused repetitive behavior disorder).

Excoriation (Skin-Picking) Disorder

Diagnostic Criteria **698.4 (L98.1)**

A. Recurrent skin picking resulting in skin lesions.
B. Repeated attempts to decrease or stop skin picking.
C. The skin picking causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
D. The skin picking is not attributable to the physiological effects of a substance (e.g., cocaine) or another medical condition (e.g., scabies).
E. The skin picking is not better explained by symptoms of another mental disorder (e.g., delusions or tactile hallucinations in a psychotic disorder, attempts to improve a perceived defect or flaw in appearance in body dysmorphic disorder, stereotypies in stereotypic movement disorder, or intention to harm oneself in nonsuicidal self-injury).

Diagnostic Features

The essential feature of excoriation (skin-picking) disorder is recurrent picking at one's own skin (Criterion A). The most commonly picked sites are the face, arms, and hands, but many individuals pick from multiple body sites. Individuals may pick at healthy skin, at minor skin irregularities, at lesions such as pimples or calluses, or at scabs from previous picking. Most individuals pick with their fingernails, although many use tweezers, pins, or other objects. In addition to skin picking, there may be skin rubbing, squeezing, lancing, and biting. Individuals with excoriation disorder often spend significant amounts of time on their picking behavior, sometimes several hours per day, and such skin picking may

endure for months or years. Criterion A requires that skin picking lead to skin lesions, although individuals with this disorder often attempt to conceal or camouflage such lesions (e.g., with makeup or clothing). Individuals with excoriation disorder have made repeated attempts to decrease or stop skin picking (Criterion B).

Criterion C indicates that skin picking causes clinically significant distress or impairment in social, occupational, or other important areas of functioning. The term *distress* includes negative affects that may be experienced by individuals with skin picking, such as feeling a loss of control, embarrassment, and shame. Significant impairment may occur in several different areas of functioning (e.g., social, occupational, academic, and leisure), in part because of avoidance of social situations.

Associated Features Supporting Diagnosis

Skin picking may be accompanied by a range of behaviors or rituals involving skin or scabs. Thus, individuals may search for a particular kind of scab to pull, and they may examine, play with, or mouth or swallow the skin after it has been pulled. Skin picking may also be preceded or accompanied by various emotional states. Skin picking may be triggered by feelings of anxiety or boredom, may be preceded by an increasing sense of tension (either immediately before picking the skin or when attempting to resist the urge to pick), and may lead to gratification, pleasure, or a sense of relief when the skin or scab has been picked. Some individuals report picking in response to a minor skin irregularity or to relieve an uncomfortable bodily sensation. Pain is not routinely reported to accompany skin picking. Some individuals engage in skin picking that is more focused (i.e., with preceding tension and subsequent relief), whereas others engage in more automatic picking (i.e., when skin picking occurs without preceding tension and without full awareness), and many have a mix of both behavioral styles. Skin picking does not usually occur in the presence of other individuals, except immediate family members. Some individuals report picking the skin of others.

Prevaience

In the general population, the lifetime prevalence for excoriation disorder in adults is 1.4% or somewhat higher. Three-quarters or more of individuals with the disorder are female. This likely reflects the true gender ratio of the condition, although it may also reflect differential treatment seeking based on gender or cultural attitudes regarding appearance.

Development and Course

Although individuals with excoriation disorder may present at various ages, the skin picking most often has onset during adolescence, commonly coinciding with or following the onset of puberty. The disorder frequently begins with a dermatological condition, such as acne. Sites of skin picking may vary over time. The usual course is chronic, with some waxing and waning if untreated. For some individuals, the disorder may come and go for weeks, months, or years at a time.

Risk and Prognostic Factors

Genetic and physiological. Excoriation disorder is more common in individuals with obsessive-compulsive disorder (OCD) and their first-degree family members than in the general population.

Diagnostic Markers

Most individuals with excoriation disorder admit to skin picking; therefore, dermatopathological diagnosis is rarely required. However, the disorder may have characteristic features on histopathology.

Functional Consequences of Excoriation (Skin-Picking) Disorder

Excoriation disorder is associated with distress as well as with social and occupational impairment. The majority of individuals with this condition spend at least 1 hour per day picking, thinking about picking, and resisting urges to pick. Many individuals report avoiding social or entertainment events as well as going out in public. A majority of individuals with the disorder also report experiencing work interference from skin picking on at least a daily or weekly basis. A significant proportion of students with excoriation disorder report having missed school, having experienced difficulties managing responsibilities at school, or having had difficulties studying because of skin picking. Medical complications of skin picking include tissue damage, scarring, and infection and can be life-threatening. Rarely, synovitis of the wrists due to chronic picking has been reported. Skin picking often results in significant tissue damage and scarring. It frequently requires antibiotic treatment for infection, and on occasion it may require surgery.

Differential Diagnosis

Psychotic disorder. Skin picking may occur in response to a delusion (i.e., parasitosis) or tactile hallucination (i.e., formication) in a psychotic disorder. In such cases, excoriation disorder should not be diagnosed.

Other obsessive-compulsive and related disorders. Excessive washing compulsions in response to contamination obsessions in individuals with OCD may lead to skin lesions, and skin picking may occur in individuals with body dysmorphic disorder who pick their skin solely because of appearance concerns; in such cases, excoriation disorder should not be diagnosed. The description of body-focused repetitive behavior disorder in other specified obsessive-compulsive and related disorder excludes individuals whose symptoms meet diagnostic criteria for excoriation disorder.

Neurodevelopmental disorders. While stereotypic movement disorder may be characterized by repetitive self-injurious behavior, onset is in the early developmental period. For example, individuals with the neurogenetic condition Prader-Willi syndrome may have early onset of skin picking, and their symptoms may meet criteria for stereotypic movement disorder. While tics in individuals with Tourette's disorder may lead to self-injury, the behavior is not tic-like in excoriation disorder.

Somatic symptom and related disorders. Excoriation disorder is not diagnosed if the skin lesion is primarily attributable to deceptive behaviors in factitious disorder.

Other disorders. Excoriation disorder is not diagnosed if the skin picking is primarily attributable to the intention to harm oneself that is characteristic of nonsuicidal self-injury.

Other medical conditions. Excoriation disorder is not diagnosed if the skin picking is primarily attributable to another medical condition. For example, scabies is a dermatological condition invariably associated with severe itching and scratching. However, excoriation disorder may be precipitated or exacerbated by an underlying dermatological condition. For example, acne may lead to some scratching and picking, which may also be associated with comorbid excoriation disorder. The differentiation between these two clinical situations (acne with some scratching and picking vs. acne with comorbid excoriation disorder) requires an assessment of the extent to which the individual's skin picking has become independent of the underlying dermatological condition.

Substance/medication-induced disorders. Skin-picking symptoms may also be induced by certain substances (e.g., cocaine), in which case excoriation disorder should not be diagnosed. If such skin picking is clinically significant, then a diagnosis of substance/medication-induced obsessive-compulsive and related disorder should be considered.

Comorbidity

Excoriation disorder is often accompanied by other mental disorders. Such disorders include OCD and trichotillomania (hair-pulling disorder), as well as major depressive disorder. Repetitive body-focused symptoms other than skin picking and hair pulling (e.g., nail biting) occur in many individuals with excoriation disorder and may deserve an additional diagnosis of other specified obsessive-compulsive and related disorder (i.e., body-focused repetitive behavior disorder).

Substance/Medication-Induced Obsessive-Compulsive and Related Disorder

Diagnostic Criteria

A. Obsessions, compulsions, skin picking, hair pulling, other body-focused repetitive behaviors, or other symptoms characteristic of the obsessive-compulsive and related disorders predominate in the clinical picture.

B. There is evidence from the history, physical examination, or laboratory findings of both (1) and (2):

   1. The symptoms in Criterion A developed during or soon after substance intoxication or withdrawal or after exposure to a medication.
   2. The involved substance/medication is capable of producing the symptoms in Criterion A.

C. The disturbance is not better explained by an obsessive-compulsive and related disorder that is not substance/medication-induced. Such evidence of an independent obsessive-compulsive and related disorder could include the following:

   The symptoms precede the onset of the substance/medication use; the symptoms persist for a substantial period of time (e.g., about 1 month) after the cessation of acute withdrawal or severe intoxication; or there is other evidence suggesting the existence of an independent non-substance/medication-induced obsessive-compulsive and related disorder (e.g., a history of recurrent non-substance/medication-related episodes).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Note: This diagnosis should be made in addition to a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and are sufficiently severe to warrant clinical attention.

Coding note: The ICD-9-CM and ICD-10-CM codes for the [specific substance/medication]-induced obsessive-compulsive and related disorders are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. If a mild substance use disorder is comorbid with the substance-induced obsessive-compulsive and related disorder, the 4th position character is "1," and the clinician should record "mild [substance] use disorder" before the substance-induced obsessive-compulsive and related disorder (e.g., "mild cocaine use disorder with cocaine-induced obsessive-compulsive and related disorder"). If a moderate or severe substance use disorder is comorbid with the substance-induced obsessive-compulsive and related disorder, the 4th position character is "2," and the clinician should record "moderate [substance] use disorder" or "severe [substance] use disorder," depending on the severity of the comorbid substance use disorder. If there is no comorbid

substance use disorder (e.g., after a one-time heavy use of the substance), then the 4th position character is "9," and the clinician should record only the substance-induced obsessive-compulsive and related disorder.

		ICD-10-CM		
	ICD-9-CM	With use disorder, mild	With use disorder, moderate or severe	Without use disorder
Amphetamine (or other stimulant)	292.89	F15.188	F15.288	F15.988
Cocaine	292.89	F14.188	F14.288	F14.988
Other (or unknown) substance	292.89	F19.188	F19.288	F19.988

Specify if (see Table 1 in the chapter "Substance-Related and Addictive Disorders" for diagnoses associated with substance class):

With onset during intoxication: If the criteria are met for intoxication with the substance and the symptoms develop during intoxication.

With onset during withdrawal: If criteria are met for withdrawal from the substance and the symptoms develop during, or shortly after, withdrawal.

With onset after medication use: Symptoms may appear either at initiation of medication or after a modification or change in use.

Recording Procedures

ICD-9-CM. The name of the substance/medication-induced obsessive-compulsive and related disorder begins with the specific substance (e.g., cocaine) that is presumed to be causing the obsessive-compulsive and related symptoms. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class. For substances that do not fit into any of the classes, the code for "other substance" should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category "unknown substance" should be used.

The name of the disorder is followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal, with onset after medication use). Unlike the recording procedures for ICD-10-CM, which combine the substance-induced disorder and substance use disorder into a single code, for ICD-9-CM a separate diagnostic code is given for the substance use disorder. For example, in the case of repetitive behaviors occurring during intoxication in a man with a severe cocaine use disorder, the diagnosis is 292.89 cocaine-induced obsessive-compulsive and related disorder, with onset during intoxication. An additional diagnosis of 304.20 severe cocaine use disorder is also given. When more than one substance is judged to play a significant role in the development of the obsessive-compulsive and related disorder, each should be listed separately.

ICD-10-CM. The name of the substance/medication-induced obsessive-compulsive and related disorder begins with the specific substance (e.g., cocaine) that is presumed to be causing the obsessive-compulsive and related symptoms. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class and presence or absence of a comorbid substance use disorder. For substances that do not fit into any of the classes, the code for "other substance" with no comorbid substance use should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category "unknown substance" with no comorbid substance use should be used.

When recording the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by the word "with," followed by the name of the substance-induced obsessive-compulsive and related disorder, followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal, with onset after medication use). For example, in the case of repetitive behaviors occurring during intoxication in a man with a severe cocaine use disorder, the diagnosis is F14.288 severe cocaine use disorder with cocaine-induced obsessive-compulsive and related disorder, with onset during intoxication. A separate diagnosis of the comorbid severe cocaine use disorder is not given. If the substance-induced obsessive-compulsive and related disorder occurs without a comorbid substance use disorder (e.g., after a one-time heavy use of the substance), no accompanying substance use disorder is noted (e.g., F15.988 amphetamine-induced obsessive-compulsive and related disorder, with onset during intoxication). When more than one substance is judged to play a significant role in the development of the obsessive-compulsive and related disorder, each should be listed separately.

Diagnostic Features

The essential features of substance/medication-induced obsessive-compulsive and related disorder are prominent symptoms of an obsessive-compulsive and related disorder (Criterion A) that are judged to be attributable to the effects of a substance (e.g., drug of abuse, medication). The obsessive-compulsive and related disorder symptoms must have developed during or soon after substance intoxication or withdrawal or after exposure to a medication or toxin, and the substance/medication must be capable of producing the symptoms (Criterion B). Substance/medication-induced obsessive-compulsive and related disorder due to a prescribed treatment for a mental disorder or general medical condition must have its onset while the individual is receiving the medication. Once the treatment is discontinued, the obsessive-compulsive and related disorder symptoms will usually improve or remit within days to several weeks to 1 month (depending on the half-life of the substance/medication). The diagnosis of substance/medication-induced obsessive-compulsive and related disorder should not be given if onset of the obsessive-compulsive and related disorder symptoms precedes the substance intoxication or medication use, or if the symptoms persist for a substantial period of time, usually longer than 1 month, from the time of severe intoxication or withdrawal. If the obsessive-compulsive and related disorder symptoms persist for a substantial period of time, other causes for the symptoms should be considered. The substance/medication-induced obsessive-compulsive and related disorder diagnosis should be made in addition to a diagnosis of substance intoxication only when the symptoms in Criterion A predominate in the clinical picture and are sufficiently severe to warrant independent clinical attention

Associated Features Supporting Diagnosis

Obsessions, compulsions, hair pulling, skin picking, or other body-focused repetitive behaviors can occur in association with intoxication with the following classes of substances: stimulants (including cocaine) and other (or unknown) substances. Heavy metals and toxins may also cause obsessive-compulsive and related disorder symptoms. Laboratory assessments (e.g., urine toxicology) may be useful to measure substance intoxication as part of an assessment for obsessive-compulsive and related disorders.

Prevalence

In the general population, the very limited data that are available indicate that substance-induced obsessive-compulsive and related disorder is very rare.

Differential Diagnosis

Substance intoxication. Obsessive-compulsive and related disorder symptoms may occur in substance intoxication. The diagnosis of the substance-specific intoxication will usu-

ally suffice to categorize the symptom presentation. A diagnosis of an obsessive-compulsive and related disorder should be made in addition to substance intoxication when the symptoms are judged to be in excess of those usually associated with intoxication and are sufficiently severe to warrant independent clinical attention.

Obsessive-compulsive and related disorder (i.e., not induced by a substance). Substance/medication-induced obsessive-compulsive and related disorder is judged to be etiologically related to the substance/medication. Substance/medication-induced obsessive-compulsive and related disorder is distinguished from a primary obsessive-compulsive and related disorder by considering the onset, course, and other factors with respect to substances/medications. For drugs of abuse, there must be evidence from the history, physical examination, or laboratory findings for use or intoxication. Substance/medication-induced obsessive-compulsive and related disorder arises only in association with intoxication, whereas a primary obsessive-compulsive and related disorder may precede the onset of substance/medication use. The presence of features that are atypical of a primary obsessive-compulsive and related disorder, such as atypical age at onset of symptoms, may suggest a substance-induced etiology. A primary obsessive-compulsive and related disorder diagnosis is warranted if the symptoms persist for a substantial period of time (about 1 month or longer) after the end of the substance intoxication or the individual has a history of an obsessive-compulsive and related disorder.

Obsessive-compulsive and related disorder due to another medical condition. If the obsessive-compulsive and related disorder symptoms are attributable to another medical condition (i.e., rather than to the medication taken for the other medical condition), obsessive-compulsive and related disorder due to another medical condition should be diagnosed. The history often provides the basis for judgment. At times, a change in the treatment for the other medical condition (e.g., medication substitution or discontinuation) may be needed to determine whether or not the medication is the causative agent (in which case the symptoms may be better explained by substance/medication-induced obsessive-compulsive and related disorder). If the disturbance is attributable to both another medical condition and substance use, both diagnoses (i.e., obsessive-compulsive and related disorder due to another medical condition and substance/medication-induced obsessive-compulsive and related disorder) may be given. When there is insufficient evidence to determine whether the symptoms are attributable to either a substance/medication or another medical condition or are primary (i.e., attributable to neither a substance/medication nor another medical condition), a diagnosis of other specified or unspecified obsessive-compulsive and related disorder would be indicated.

Delirium. If obsessive-compulsive and related disorder symptoms occur exclusively during the course of delirium, they are considered to be an associated feature of the delirium and are not diagnosed separately.

Obsessive-Compulsive and Related Disorder Due to Another Medical Condition

Diagnostic Criteria **294.8 (F06.8)**

A. Obsessions, compulsions, preoccupations with appearance, hoarding, skin picking, hair pulling, other body-focused repetitive behaviors, or other symptoms characteristic of obsessive-compulsive and related disorder predominate in the clinical picture.

B. There is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct pathophysiological consequence of another medical condition.

C. The disturbance is not better explained by another mental disorder.

D. The disturbance does not occur exclusively during the course of a delirium.
E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if:

With obsessive-compulsive disorder–like symptoms: If obsessive-compulsive disorder–like symptoms predominate in the clinical presentation.

With appearance preoccupations: If preoccupation with perceived appearance defects or flaws predominates in the clinical presentation.

With hoarding symptoms: If hoarding predominates in the clinical presentation.

With hair-pulling symptoms: If hair pulling predominates in the clinical presentation.

With skin-picking symptoms: If skin picking predominates in the clinical presentation.

Coding note: Include the name of the other medical condition in the name of the mental disorder (e.g., 294.8 [F06.8] obsessive-compulsive and related disorder due to cerebral infarction). The other medical condition should be coded and listed separately immediately before the obsessive-compulsive and related disorder due to the medical condition (e.g., 438.89 [I69.398] cerebral infarction; 294.8 [F06.8] obsessive-compulsive and related disorder due to cerebral infarction).

Diagnostic Features

The essential feature of obsessive-compulsive and related disorder due to another medical condition is clinically significant obsessive-compulsive and related symptoms that are judged to be best explained as the direct pathophysiological consequence of another medical condition. Symptoms can include prominent obsessions, compulsions, preoccupations with appearance, hoarding, hair pulling, skin picking, or other body-focused repetitive behaviors (Criterion A). The judgment that the symptoms are best explained by the associated medical condition must be based on evidence from the history, physical examination, or laboratory findings (Criterion B). Additionally, it must be judged that the symptoms are not better explained by another mental disorder (Criterion C). The diagnosis is not made if the obsessive-compulsive and related symptoms occur only during the course of a delirium (Criterion D). The obsessive-compulsive and related symptoms must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion E).

In determining whether the obsessive-compulsive and related symptoms are attributable to another medical condition, a relevant medical condition must be present. Furthermore, it must be established that obsessive-compulsive and related symptoms can be etiologically related to the medical condition through a pathophysiological mechanism and that this best explains the symptoms in the individual. Although there are no infallible guidelines for determining whether the relationship between the obsessive-compulsive and related symptoms and the medical condition is etiological, considerations that may provide some guidance in making this diagnosis include the presence of a clear temporal association between the onset, exacerbation, or remission of the medical condition and the obsessive-compulsive and related symptoms; the presence of features that are atypical of a primary obsessive-compulsive and related disorder (e.g., atypical age at onset or course); and evidence in the literature that a known physiological mechanism (e.g., striatal damage) causes obsessive-compulsive and related symptoms. In addition, the disturbance cannot be better explained by a primary obsessive-compulsive and related disorder, a substance/medication-induced obsessive-compulsive and related disorder, or another mental disorder.

There is some controversy about whether obsessive-compulsive and related disorders can be attributed to Group A streptococcal infection. Sydenham's chorea is the neurolog-

ical manifestation of rheumatic fever, which is in turn due to Group A streptococcal infection. Sydenham's chorea is characterized by a combination of motor and nonmotor features. Nonmotor features include obsessions, compulsions, attention deficit, and emotional lability. Although individuals with Sydenham's chorea may present with nonneuropsychiatric features of acute rheumatic fever, such as carditis and arthritis, they may present with obsessive-compulsive disorder–like symptoms; such individuals should be diagnosed with obsessive-compulsive and related disorder due to another medical condition.

Pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (PANDAS) has been identified as another post-infectious autoimmune disorder characterized by the sudden onset of obsessions, compulsions, and/or tics accompanied by a variety of acute neuropsychiatric symptoms in the absence of chorea, carditis, or arthritis, after Group A streptococcal infection. Although there is a body of evidence that supports the existence of PANDAS, it remains a controversial diagnosis. Given this ongoing controversy, the description of PANDAS has been modified to eliminate etiological factors and to designate an expanded clinical entity: pediatric acute-onset neuropsychiatric syndrome (PANS) or idiopathic childhood acute neuropsychiatric symptoms (CANS), which deserves further study.

Associated Features Supporting Diagnosis

A number of other medical disorders are known to include obsessive-compulsive and related symptoms as a manifestation. Examples include disorders leading to striatal damage, such as cerebral infarction.

Development and Course

The development and course of obsessive-compulsive and related disorder due to another medical condition generally follows the course of the underlying illness.

Diagnostic Markers

Laboratory assessments and/or medical examinations are necessary to confirm the diagnosis of another medical condition.

Differential Diagnosis

Delirium. A separate diagnosis of obsessive-compulsive and related disorder due to another medical condition is not given if the disturbance occurs exclusively during the course of a delirium. However, a diagnosis of obsessive-compulsive and related disorder due to another medical condition may be given in addition to a diagnosis of major neurocognitive disorder (dementia) if the etiology of the obsessive-compulsive symptoms is judged to be a physiological consequence of the pathological process causing the dementia and if obsessive-compulsive symptoms are a prominent part of the clinical presentation.

Mixed presentation of symptoms (e.g., mood and obsessive-compulsive and related disorder symptoms). If the presentation includes a mix of different types of symptoms, the specific mental disorder due to another medical condition depends on which symptoms predominate in the clinical picture.

Substance/medication-induced obsessive-compulsive and related disorders. If there is evidence of recent or prolonged substance use (including medications with psychoactive effects), withdrawal from a substance, or exposure to a toxin, a substance/medication-induced obsessive-compulsive and related disorder should be considered. When a substance/medication-induced obsessive-compulsive and related disorder is being diagnosed in relation to drugs of abuse, it may be useful to obtain a urine or blood drug screen

or other appropriate laboratory evaluation. Symptoms that occur during or shortly after (i.e., within 4 weeks of) substance intoxication or withdrawal or after medication use may be especially indicative of a substance/medication-induced obsessive-compulsive and related disorder, depending on the type, duration, or amount of the substance used.

Obsessive-compulsive and related disorders (primary). Obsessive-compulsive and related disorder due to another medical condition should be distinguished from a primary obsessive-compulsive and related disorder. In primary mental disorders, no specific and direct causative physiological mechanisms associated with a medical condition can be demonstrated. Late age at onset or atypical symptoms suggest the need for a thorough assessment to rule out the diagnosis of obsessive-compulsive and related disorder due to another medical condition.

Illness anxiety disorder. Illness anxiety disorder is characterized by a preoccupation with having or acquiring a serious illness. In the case of illness anxiety disorder, individuals may or may not have diagnosed medical conditions.

Associated feature of another mental disorder. Obsessive-compulsive and related symptoms may be an associated feature of another mental disorder (e.g., schizophrenia, anorexia nervosa).

Other specified obsessive-compulsive and related disorder or unspecified obsessive-compulsive and related disorder. These diagnoses are given if it is unclear whether the obsessive-compulsive and related symptoms are primary, substance-induced, or due to another medical condition.

Other Specified Obsessive-Compulsive and Related Disorder

300.3 (F42)

This category applies to presentations in which symptoms characteristic of an obsessive-compulsive and related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the obsessive-compulsive and related disorders diagnostic class. The other specified obsessive-compulsive and related disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific obsessive-compulsive and related disorder. This is done by recording "other specified obsessive-compulsive and related disorder" followed by the specific reason (e.g., "body-focused repetitive behavior disorder").

Examples of presentations that can be specified using the "other specified" designation include the following:

1. **Body dysmorphic–like disorder with actual flaws:** This is similar to body dysmorphic disorder except that the defects or flaws in physical appearance are clearly observable by others (i.e., they are more noticeable than "slight"). In such cases, the preoccupation with these flaws is clearly excessive and causes significant impairment or distress.
2. **Body dysmorphic–like disorder without repetitive behaviors:** Presentations that meet body dysmorphic disorder except that the individual has not performed repetitive behaviors or mental acts in response to the appearance concerns.
3. **Body-focused repetitive behavior disorder:** This is characterized by recurrent body-focused repetitive behaviors (e.g., nail biting, lip biting, cheek chewing) and repeated attempts to decrease or stop the behaviors. These symptoms cause clinically significant

distress or impairment in social, occupational, or other important areas of functioning and are not better explained by trichotillomania (hair-pulling disorder), excoriation (skin-picking) disorder, stereotypic movement disorder, or nonsuicidal self-injury.

4. **Obsessional jealousy:** This is characterized by nondelusional preoccupation with a partner's perceived infidelity. The preoccupations may lead to repetitive behaviors or mental acts in response to the infidelity concerns; they cause clinically significant distress or impairment in social, occupational, or other important areas of functioning; and they are not better explained by another mental disorder such as delusional disorder, jealous type, or paranoid personality disorder.
5. ***Shubo-kyofu:*** A variant of *taijin kyofusho* (see "Glossary of Cultural Concepts of Distress" in the Appendix) that is similar to body dysmorphic disorder and is characterized by excessive fear of having a bodily deformity.
6. ***Koro:*** Related to *dhat syndrome* (see "Glossary of Cultural Concepts of Distress" in the Appendix), an episode of sudden and intense anxiety that the penis (or the vulva and nipples in females) will recede into the body, possibly leading to death.
7. ***Jikoshu-kyofu:*** A variant of *taijin kyofusho* (see "Glossary of Cultural Concepts of Distress" in the Appendix) characterized by fear of having an offensive body odor (also termed *olfactory reference syndrome*).

Unspecified Obsessive-Compulsive and Related Disorder

300.3 (F42)

This category applies to presentations in which symptoms characteristic of an obsessive-compulsive and related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the obsessive-compulsive and related disorders diagnostic class. The unspecified obsessive-compulsive and related disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific obsessive-compulsive and related disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Trauma- and Stressor-Related Disorders

Trauma- and stressor-related disorders include disorders in which exposure to a traumatic or stressful event is listed explicitly as a diagnostic criterion. These include reactive attachment disorder, disinhibited social engagement disorder, posttraumatic stress disorder (PTSD), acute stress disorder, and adjustment disorders. Placement of this chapter reflects the close relationship between these diagnoses and disorders in the surrounding chapters on anxiety disorders, obsessive-compulsive and related disorders, and dissociative disorders.

Psychological distress following exposure to a traumatic or stressful event is quite variable. In some cases, symptoms can be well understood within an anxiety- or fear-based context. It is clear, however, that many individuals who have been exposed to a traumatic or stressful event exhibit a phenotype in which, rather than anxiety- or fear-based symptoms, the most prominent clinical characteristics are anhedonic and dysphoric symptoms, externalizing angry and aggressive symptoms, or dissociative symptoms. Because of these variable expressions of clinical distress following exposure to catastrophic or aversive events, the aforementioned disorders have been grouped under a separate category: *trauma- and stressor-related disorders*. Furthermore, it is not uncommon for the clinical picture to include some combination of the above symptoms (with or without anxiety- or fear-based symptoms). Such a heterogeneous picture has long been recognized in adjustment disorders, as well. Social neglect—that is, the absence of adequate caregiving during childhood—is a diagnostic requirement of both reactive attachment disorder and disinhibited social engagement disorder. Although the two disorders share a common etiology, the former is expressed as an internalizing disorder with depressive symptoms and withdrawn behavior, while the latter is marked by disinhibition and externalizing behavior.

Reactive Attachment Disorder

Diagnostic Criteria — 313.89 (F94.1)

A. A consistent pattern of inhibited, emotionally withdrawn behavior toward adult caregivers, manifested by both of the following:
   1. The child rarely or minimally seeks comfort when distressed.
   2. The child rarely or minimally responds to comfort when distressed.

B. A persistent social and emotional disturbance characterized by at least two of the following:
   1. Minimal social and emotional responsiveness to others.
   2. Limited positive affect.
   3. Episodes of unexplained irritability, sadness, or fearfulness that are evident even during nonthreatening interactions with adult caregivers.

C. The child has experienced a pattern of extremes of insufficient care as evidenced by at least one of the following:
   1. Social neglect or deprivation in the form of persistent lack of having basic emotional needs for comfort, stimulation, and affection met by caregiving adults.

2. Repeated changes of primary caregivers that limit opportunities to form stable attachments (e.g., frequent changes in foster care).
3. Rearing in unusual settings that severely limit opportunities to form selective attachments (e.g., institutions with high child-to-caregiver ratios).

D. The care in Criterion C is presumed to be responsible for the disturbed behavior in Criterion A (e.g., the disturbances in Criterion A began following the lack of adequate care in Criterion C).

E. The criteria are not met for autism spectrum disorder.

F. The disturbance is evident before age 5 years.

G. The child has a developmental age of at least 9 months.

Specify if:

Persistent: The disorder has been present for more than 12 months.

Specify current severity:

Reactive attachment disorder is specified as **severe** when a child exhibits all symptoms of the disorder, with each symptom manifesting at relatively high levels.

Diagnostic Features

Reactive attachment disorder of infancy or early childhood is characterized by a pattern of markedly disturbed and developmentally inappropriate attachment behaviors, in which a child rarely or minimally turns preferentially to an attachment figure for comfort, support, protection, and nurturance. The essential feature is absent or grossly underdeveloped attachment between the child and putative caregiving adults. Children with reactive attachment disorder are believed to have the capacity to form selective attachments. However, because of limited opportunities during early development, they fail to show the behavioral manifestations of selective attachments. That is, when distressed, they show no consistent effort to obtain comfort, support, nurturance, or protection from caregivers. Furthermore, when distressed, children with this disorder do not respond more than minimally to comforting efforts of caregivers. Thus, the disorder is associated with the absence of expected comfort seeking and response to comforting behaviors. As such, children with reactive attachment disorder show diminished or absent expression of positive emotions during routine interactions with caregivers. In addition, their emotion regulation capacity is compromised, and they display episodes of negative emotions of fear, sadness, or irritability that are not readily explained. A diagnosis of reactive attachment disorder should not be made in children who are developmentally unable to form selective attachments. For this reason, the child must have a developmental age of at least 9 months.

Associated Features Supporting Diagnosis

Because of the shared etiological association with social neglect, reactive attachment disorder often co-occurs with developmental delays, especially in delays in cognition and language. Other associated features include stereotypies and other signs of severe neglect (e.g., malnutrition or signs of poor care).

Prevaience

The prevalence of reactive attachment disorder is unknown, but the disorder is seen relatively rarely in clinical settings. The disorder has been found in young children exposed to severe neglect before being placed in foster care or raised in institutions. However, even in populations of severely neglected children, the disorder is uncommon, occurring in less than 10% of such children.

Development and Course

Conditions of social neglect are often present in the first months of life in children diagnosed with reactive attachment disorder, even before the disorder is diagnosed. The clinical features of the disorder manifest in a similar fashion between the ages of 9 months and 5 years. That is, signs of absent-to-minimal attachment behaviors and associated emotionally aberrant behaviors are evident in children throughout this age range, although differing cognitive and motor abilities may affect how these behaviors are expressed. Without remediation and recovery through normative caregiving environments, it appears that signs of the disorder may persist, at least for several years.

It is unclear whether reactive attachment disorder occurs in older children and, if so, how it differs from its presentation in young children. Because of this, the diagnosis should be made with caution in children older than 5 years.

Risk and Prognostic Factors

Environmental. Serious social neglect is a diagnostic requirement for reactive attachment disorder and is also the only known risk factor for the disorder. However, the majority of severely neglected children do not develop the disorder. Prognosis appears to depend on the quality of the caregiving environment following serious neglect.

Cuiture-Related Diagnostic Issues

Similar attachment behaviors have been described in young children in many different cultures around the world. However, caution should be exercised in making the diagnosis of reactive attachment disorder in cultures in which attachment has not been studied.

Functional Consequences of Reactive Attachment Disorder

Reactive attachment disorder significantly impairs young children's abilities to relate interpersonally to adults or peers and is associated with functional impairment across many domains of early childhood.

Differential Diagnosis

Autism spectrum disorder. Aberrant social behaviors manifest in young children with reactive attachment disorder, but they also are key features of autism spectrum disorder. Specifically, young children with either condition can manifest dampened expression of positive emotions, cognitive and language delays, and impairments in social reciprocity. As a result, reactive attachment disorder must be differentiated from autism spectrum disorder. These two disorders can be distinguished based on differential histories of neglect and on the presence of restricted interests or ritualized behaviors, specific deficit in social communication, and selective attachment behaviors. Children with reactive attachment disorder have experienced a history of severe social neglect, although it is not always possible to obtain detailed histories about the precise nature of their experiences, especially in initial evaluations. Children with autistic spectrum disorder will only rarely have a history of social neglect. The restricted interests and repetitive behaviors characteristic of autism spectrum disorder are not a feature of reactive attachment disorder. These clinical features manifest as excessive adherence to rituals and routines; restricted, fixated interests; and unusual sensory reactions. However, it is important to note that children with either condition can exhibit stereotypic behaviors such as rocking or flapping. Children with either disorder also may exhibit a range of intellectual functioning, but only children with autis-

tic spectrum disorder exhibit selective impairments in social communicative behaviors, such as intentional communication (i.e., impairment in communication that is deliberate, goal-directed, and aimed at influencing the behavior of the recipient). Children with reactive attachment disorder show social communicative functioning comparable to their overall level of intellectual functioning. Finally, children with autistic spectrum disorder regularly show attachment behavior typical for their developmental level. In contrast, children with reactive attachment disorder do so only rarely or inconsistently, if at all.

Intellectual disability (intellectual developmental disorder). Developmental delays often accompany reactive attachment disorder, but they should not be confused with the disorder. Children with intellectual disability should exhibit social and emotional skills comparable to their cognitive skills and do not demonstrate the profound reduction in positive affect and emotion regulation difficulties evident in children with reactive attachment disorder. In addition, developmentally delayed children who have reached a cognitive age of 7–9 months should demonstrate selective attachments regardless of their chronological age. In contrast, children with reactive attachment disorder show lack of preferred attachment despite having attained a developmental age of at least 9 months.

Depressive disorders. Depression in young children is also associated with reductions in positive affect. There is limited evidence, however, to suggest that children with depressive disorders have impairments in attachment. That is, young children who have been diagnosed with depressive disorders still should seek and respond to comforting efforts by caregivers.

Comorbidity

Conditions associated with neglect, including cognitive delays, language delays, and stereotypies, often co-occur with reactive attachment disorder. Medical conditions, such as severe malnutrition, may accompany signs of the disorder. Depressive symptoms also may co-occur with reactive attachment disorder.

Disinhibited Social Engagement Disorder

Diagnostic Criteria **313.89 (F94.2)**

A. A pattern of behavior in which a child actively approaches and interacts with unfamiliar adults and exhibits at least two of the following:
   1. Reduced or absent reticence in approaching and interacting with unfamiliar adults.
   2. Overly familiar verbal or physical behavior (that is not consistent with culturally sanctioned and with age-appropriate social boundaries).
   3. Diminished or absent checking back with adult caregiver after venturing away, even in unfamiliar settings.
   4. Willingness to go off with an unfamiliar adult with minimal or no hesitation.

B. The behaviors in Criterion A are not limited to impulsivity (as in attention-deficit/hyperactivity disorder) but include socially disinhibited behavior.

C. The child has experienced a pattern of extremes of insufficient care as evidenced by at least one of the following:
   1. Social neglect or deprivation in the form of persistent lack of having basic emotional needs for comfort, stimulation, and affection met by caregiving adults.
   2. Repeated changes of primary caregivers that limit opportunities to form stable attachments (e.g., frequent changes in foster care).
   3. Rearing in unusual settings that severely limit opportunities to form selective attachments (e.g., institutions with high child-to-caregiver ratios).

D. The care in Criterion C is presumed to be responsible for the disturbed behavior in Criterion A (e.g., the disturbances in Criterion A began following the pathogenic care in Criterion C).
E. The child has a developmental age of at least 9 months.

Specify if:

Persistent: The disorder has been present for more than 12 months.

Specify current severity:

Disinhibited social engagement disorder is specified as **severe** when the child exhibits all symptoms of the disorder, with each symptom manifesting at relatively high levels.

Diagnostic Features

The essential feature of disinhibited social engagement disorder is a pattern of behavior that involves culturally inappropriate, overly familiar behavior with relative strangers (Criterion A). This overly familiar behavior violates the social boundaries of the culture. A diagnosis of disinhibited social engagement disorder should not be made before children are developmentally able to form selective attachments. For this reason, the child must have a developmental age of at least 9 months.

Associated Features Supporting Diagnosis

Because of the shared etiological association with social neglect, disinhibited social engagement disorder may co-occur with developmental delays, especially cognitive and language delays, stereotypies, and other signs of severe neglect, such as malnutrition or poor care. However, signs of the disorder often persist even after these other signs of neglect are no longer present. Therefore, it is not uncommon for children with the disorder to present with no current signs of neglect. Moreover, the condition can present in children who show no signs of disordered attachment. Thus, disinhibited social engagement disorder may be seen in children with a history of neglect who lack attachments or whose attachments to their caregivers range from disturbed to secure.

Prevalence

The prevalence of disinhibited social attachment disorder is unknown. Nevertheless, the disorder appears to be rare, occurring in a minority of children, even those who have been severely neglected and subsequently placed in foster care or raised in institutions. In such high-risk populations, the condition occurs in only about 20% of children. The condition is seen rarely in other clinical settings.

Development and Course

Conditions of social neglect are often present in the first months of life in children diagnosed with disinhibited social engagement disorder, even before the disorder is diagnosed. However, there is no evidence that neglect beginning after age 2 years is associated with manifestations of the disorder. If neglect occurs early and signs of the disorder appear, clinical features of the disorder are moderately stable over time, particularly if conditions of neglect persist. Indiscriminate social behavior and lack of reticence with unfamiliar adults in toddlerhood are accompanied by attention-seeking behaviors in preschoolers. When the disorder persists into middle childhood, clinical features manifest as verbal and physical overfamiliarity as well as inauthentic expression of emotions. These signs appear particularly apparent when the child interacts with adults. Peer relationships are most affected in adolescence, with both indiscriminate behavior and conflicts apparent. The disorder has not been described in adults.

Disinhibited social engagement disorder has been described from the second year of life through adolescence. There are some differences in manifestations of the disorder from early childhood through adolescence. At the youngest ages, across many cultures, children show reticence when interacting with strangers. Young children with the disorder fail to show reticence to approach, engage with, and even accompany adults. In preschool children, verbal and social intrusiveness appear most prominent, often accompanied by attention-seeking behavior. Verbal and physical overfamiliarity continue through middle childhood, accompanied by inauthentic expressions of emotion. In adolescence, indiscriminate behavior extends to peers. Relative to healthy adolescents, adolescents with the disorder have more "superficial" peer relationships and more peer conflicts. Adult manifestations of the disorder are unknown.

Risk and Prognostic Factors

Environmental. Serious social neglect is a diagnostic requirement for disinhibited social engagement disorder and is also the only known risk factor for the disorder. However, the majority of severely neglected children do not develop the disorder. Neurobiological vulnerability may differentiate neglected children who do and do not develop the disorder. However, no clear link with any specific neurobiological factors has been established. The disorder has not been identified in children who experience social neglect only after age 2 years. Prognosis is only modestly associated with quality of the caregiving environment following serious neglect. In many cases, the disorder persists, even in children whose caregiving environment becomes markedly improved.

Course modifiers. Caregiving quality seems to moderate the course of disinhibited social engagement disorder. Nevertheless, even after placement in normative caregiving environments, some children show persistent signs of the disorder, at least through adolescence.

Functional Consequences of Disinhibited Social Engagement Disorder

Disinhibited social engagement disorder significantly impairs young children's abilities to relate interpersonally to adults and peers.

Differential Diagnosis

Attention-deficit/hyperactivity disorder. Because of social impulsivity that sometimes accompanies attention-deficit/hyperactivity disorder (ADHD), it is necessary to differentiate the two disorders. Children with disinhibited social engagement disorder may be distinguished from those with ADHD because the former do not show difficulties with attention or hyperactivity.

Comorbidity

Limited research has examined the issue of disorders comorbid with disinhibited social engagement disorder. Conditions associated with neglect, including cognitive delays, language delays, and stereotypies, may co-occur with disinhibited social engagement disorder. In addition, children may be diagnosed with ADHD and disinhibited social engagement disorder concurrently.

Posttraumatic Stress Disorder

Diagnostic Criteria 309.81 (F43.10)

Posttraumatic Stress Disorder

Note: The following criteria apply to adults, adolescents, and children older than 6 years. For children 6 years and younger, see corresponding criteria below.

A. Exposure to actual or threatened death, serious injury, or sexual violence in one (or more) of the following ways:

1. Directly experiencing the traumatic event(s).
2. Witnessing, in person, the event(s) as it occurred to others.
3. Learning that the traumatic event(s) occurred to a close family member or close friend. In cases of actual or threatened death of a family member or friend, the event(s) must have been violent or accidental.
4. Experiencing repeated or extreme exposure to aversive details of the traumatic event(s) (e.g., first responders collecting human remains; police officers repeatedly exposed to details of child abuse).

   Note: Criterion A4 does not apply to exposure through electronic media, television, movies, or pictures, unless this exposure is work related.

B. Presence of one (or more) of the following intrusion symptoms associated with the traumatic event(s), beginning after the traumatic event(s) occurred:

1. Recurrent, involuntary, and intrusive distressing memories of the traumatic event(s).

   Note: In children older than 6 years, repetitive play may occur in which themes or aspects of the traumatic event(s) are expressed.

2. Recurrent distressing dreams in which the content and/or affect of the dream are related to the traumatic event(s).

   Note: In children, there may be frightening dreams without recognizable content.

3. Dissociative reactions (e.g., flashbacks) in which the individual feels or acts as if the traumatic event(s) were recurring. (Such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings.)

   Note: In children, trauma-specific reenactment may occur in play.

4. Intense or prolonged psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).
5. Marked physiological reactions to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).

C. Persistent avoidance of stimuli associated with the traumatic event(s), beginning after the traumatic event(s) occurred, as evidenced by one or both of the following:

1. Avoidance of or efforts to avoid distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s).
2. Avoidance of or efforts to avoid external reminders (people, places, conversations, activities, objects, situations) that arouse distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s).

D. Negative alterations in cognitions and mood associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:

1. Inability to remember an important aspect of the traumatic event(s) (typically due to dissociative amnesia and not to other factors such as head injury, alcohol, or drugs).

2. Persistent and exaggerated negative beliefs or expectations about oneself, others, or the world (e.g., "I am bad," "No one can be trusted," "The world is completely dangerous," "My whole nervous system is permanently ruined").
3. Persistent, distorted cognitions about the cause or consequences of the traumatic event(s) that lead the individual to blame himself/herself or others.
4. Persistent negative emotional state (e.g., fear, horror, anger, guilt, or shame).
5. Markedly diminished interest or participation in significant activities.
6. Feelings of detachment or estrangement from others.
7. Persistent inability to experience positive emotions (e.g., inability to experience happiness, satisfaction, or loving feelings).

E. Marked alterations in arousal and reactivity associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:

1. Irritable behavior and angry outbursts (with little or no provocation) typically expressed as verbal or physical aggression toward people or objects.
2. Reckless or self-destructive behavior.
3. Hypervigilance.
4. Exaggerated startle response.
5. Problems with concentration.
6. Sleep disturbance (e.g., difficulty falling or staying asleep or restless sleep).

F. Duration of the disturbance (Criteria B, C, D, and E) is more than 1 month.

G. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

H. The disturbance is not attributable to the physiological effects of a substance (e.g., medication, alcohol) or another medical condition.

Specify whether:

With dissociative symptoms: The individual's symptoms meet the criteria for posttraumatic stress disorder, and in addition, in response to the stressor, the individual experiences persistent or recurrent symptoms of either of the following:

1. **Depersonalization:** Persistent or recurrent experiences of feeling detached from, and as if one were an outside observer of, one's mental processes or body (e.g., feeling as though one were in a dream; feeling a sense of unreality of self or body or of time moving slowly).
2. **Derealization:** Persistent or recurrent experiences of unreality of surroundings (e.g., the world around the individual is experienced as unreal, dreamlike, distant, or distorted).

Note: To use this subtype, the dissociative symptoms must not be attributable to the physiological effects of a substance (e.g., blackouts, behavior during alcohol intoxication) or another medical condition (e.g., complex partial seizures).

Specify if:

With delayed expression: If the full diagnostic criteria are not met until at least 6 months after the event (although the onset and expression of some symptoms may be immediate).

Posttraumatic Stress Disorder for Children 6 Years and Younger

A. In children 6 years and younger, exposure to actual or threatened death, serious injury, or sexual violence in one (or more) of the following ways:

1. Directly experiencing the traumatic event(s).
2. Witnessing, in person, the event(s) as it occurred to others, especially primary caregivers.

Note: Witnessing does not include events that are witnessed only in electronic media, television, movies, or pictures.

3. Learning that the traumatic event(s) occurred to a parent or caregiving figure.

B. Presence of one (or more) of the following intrusion symptoms associated with the traumatic event(s), beginning after the traumatic event(s) occurred:

1. Recurrent, involuntary, and intrusive distressing memories of the traumatic event(s).

   Note: Spontaneous and intrusive memories may not necessarily appear distressing and may be expressed as play reenactment.

2. Recurrent distressing dreams in which the content and/or affect of the dream are related to the traumatic event(s).

   Note: It may not be possible to ascertain that the frightening content is related to the traumatic event.

3. Dissociative reactions (e.g., flashbacks) in which the child feels or acts as if the traumatic event(s) were recurring. (Such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings.) Such trauma-specific reenactment may occur in play.
4. Intense or prolonged psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).
5. Marked physiological reactions to reminders of the traumatic event(s).

C. One (or more) of the following symptoms, representing either persistent avoidance of stimuli associated with the traumatic event(s) or negative alterations in cognitions and mood associated with the traumatic event(s), must be present, beginning after the event(s) or worsening after the event(s):

Persistent Avoidance of Stimuli

1. Avoidance of or efforts to avoid activities, places, or physical reminders that arouse recollections of the traumatic event(s).
2. Avoidance of or efforts to avoid people, conversations, or interpersonal situations that arouse recollections of the traumatic event(s).

Negative Alterations in Cognitions

3. Substantially increased frequency of negative emotional states (e.g., fear, guilt, sadness, shame, confusion).
4. Markedly diminished interest or participation in significant activities, including constriction of play.
5. Socially withdrawn behavior.
6. Persistent reduction in expression of positive emotions.

D. Alterations in arousal and reactivity associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:

1. Irritable behavior and angry outbursts (with little or no provocation) typically expressed as verbal or physical aggression toward people or objects (including extreme temper tantrums).
2. Hypervigilance.
3. Exaggerated startle response.
4. Problems with concentration.
5. Sleep disturbance (e.g., difficulty falling or staying asleep or restless sleep).

E. The duration of the disturbance is more than 1 month.

F. The disturbance causes clinically significant distress or impairment in relationships with parents, siblings, peers, or other caregivers or with school behavior.
G. The disturbance is not attributable to the physiological effects of a substance (e.g., medication or alcohol) or another medical condition.

Specify whether:

With dissociative symptoms: The individual's symptoms meet the criteria for posttraumatic stress disorder, and the individual experiences persistent or recurrent symptoms of either of the following:

1. **Depersonalization:** Persistent or recurrent experiences of feeling detached from, and as if one were an outside observer of, one's mental processes or body (e.g., feeling as though one were in a dream; feeling a sense of unreality of self or body or of time moving slowly).
2. **Derealization:** Persistent or recurrent experiences of unreality of surroundings (e.g., the world around the individual is experienced as unreal, dreamlike, distant, or distorted).

Note: To use this subtype, the dissociative symptoms must not be attributable to the physiological effects of a substance (e.g., blackouts) or another medical condition (e.g., complex partial seizures).

Specify if:

With delayed expression: If the full diagnostic criteria are not met until at least 6 months after the event (although the onset and expression of some symptoms may be immediate).

Diagnostic Features

The essential feature of posttraumatic stress disorder (PTSD) is the development of characteristic symptoms following exposure to one or more traumatic events. Emotional reactions to the traumatic event (e.g., fear, helplessness, horror) are no longer a part of Criterion A. The clinical presentation of PTSD varies. In some individuals, fear-based reexperiencing, emotional, and behavioral symptoms may predominate. In others, anhedonic or dysphoric mood states and negative cognitions may be most distressing. In some other individuals, arousal and reactive-externalizing symptoms are prominent, while in others, dissociative symptoms predominate. Finally, some individuals exhibit combinations of these symptom patterns.

The directly experienced traumatic events in Criterion A include, but are not limited to, exposure to war as a combatant or civilian, threatened or actual physical assault (e.g., physical attack, robbery, mugging, childhood physical abuse), threatened or actual sexual violence (e.g., forced sexual penetration, alcohol/drug-facilitated sexual penetration, abusive sexual contact, noncontact sexual abuse, sexual trafficking), being kidnapped, being taken hostage, terrorist attack, torture, incarceration as a prisoner of war, natural or human-made disasters, and severe motor vehicle accidents. For children, sexually violent events may include developmentally inappropriate sexual experiences without physical violence or injury. A life-threatening illness or debilitating medical condition is not necessarily considered a traumatic event. Medical incidents that qualify as traumatic events involve sudden, catastrophic events (e.g., waking during surgery, anaphylactic shock). Witnessed events include, but are not limited to, observing threatened or serious injury, unnatural death, physical or sexual abuse of another person due to violent assault, domestic violence, accident, war or disaster, or a medical catastrophe in one's child (e.g., a life-threatening hemorrhage). Indirect exposure through learning about an event is limited to experiences affecting close relatives or friends and experiences that are violent or accidental (e.g., death due to natural causes does not qualify). Such events include violent per-

sonal assault, suicide, serious accident, and serious injury. The disorder may be especially severe or long-lasting when the stressor is interpersonal and intentional (e.g., torture, sexual violence).

The traumatic event can be reexperienced in various ways. Commonly, the individual has recurrent, involuntary, and intrusive recollections of the event (Criterion B1). Intrusive recollections in PTSD are distinguished from depressive rumination in that they apply only to involuntary and intrusive distressing memories. The emphasis is on recurrent memories of the event that usually include sensory, emotional, or physiological behavioral components. A common reexperiencing symptom is distressing dreams that replay the event itself or that are representative or thematically related to the major threats involved in the traumatic event (Criterion B2). The individual may experience dissociative states that last from a few seconds to several hours or even days, during which components of the event are relived and the individual behaves as if the event were occurring at that moment (Criterion B3). Such events occur on a continuum from brief visual or other sensory intrusions about part of the traumatic event without loss of reality orientation, to complete loss of awareness of present surroundings. These episodes, often referred to as "flashbacks," are typically brief but can be associated with prolonged distress and heightened arousal. For young children, reenactment of events related to trauma may appear in play or in dissociative states. Intense psychological distress (Criterion B4) or physiological reactivity (Criterion B5) often occurs when the individual is exposed to triggering events that resemble or symbolize an aspect of the traumatic event (e.g., windy days after a hurricane; seeing someone who resembles one's perpetrator). The triggering cue could be a physical sensation (e.g., dizziness for survivors of head trauma; rapid heartbeat for a previously traumatized child), particularly for individuals with highly somatic presentations.

Stimuli associated with the trauma are persistently (e.g., always or almost always) avoided. The individual commonly makes deliberate efforts to avoid thoughts, memories, feelings, or talking about the traumatic event (e.g., utilizing distraction techniques to avoid internal reminders) (Criterion C1) and to avoid activities, objects, situations, or people who arouse recollections of it (Criterion C2).

Negative alterations in cognitions or mood associated with the event begin or worsen after exposure to the event. These negative alterations can take various forms, including an inability to remember an important aspect of the traumatic event; such amnesia is typically due to dissociative amnesia and is not due to head injury, alcohol, or drugs (Criterion D1). Another form is persistent (i.e., always or almost always) and exaggerated negative expectations regarding important aspects of life applied to oneself, others, or the future (e.g., "I have always had bad judgment"; "People in authority can't be trusted") that may manifest as a negative change in perceived identity since the trauma (e.g., "I can't trust anyone ever again"; Criterion D2). Individuals with PTSD may have persistent erroneous cognitions about the causes of the traumatic event that lead them to blame themselves or others (e.g., "It's all my fault that my uncle abused me") (Criterion D3). A persistent negative mood state (e.g., fear, horror, anger, guilt, shame) either began or worsened after exposure to the event (Criterion D4). The individual may experience markedly diminished interest or participation in previously enjoyed activities (Criterion D5), feeling detached or estranged from other people (Criterion D6), or a persistent inability to feel positive emotions (especially happiness, joy, satisfaction, or emotions associated with intimacy, tenderness, and sexuality) (Criterion D7).

Individuals with PTSD may be quick tempered and may even engage in aggressive verbal and/or physical behavior with little or no provocation (e.g., yelling at people, getting into fights, destroying objects) (Criterion E1). They may also engage in reckless or self-destructive behavior such as dangerous driving, excessive alcohol or drug use, or self-injurious or suicidal behavior (Criterion E2). PTSD is often characterized by a heightened sensitivity to potential threats, including those that are related to the traumatic experience (e.g., following a motor vehicle accident, being especially sensitive to the threat potentially

caused by cars or trucks) and those not related to the traumatic event (e.g., being fearful of suffering a heart attack) (Criterion E3). Individuals with PTSD may be very reactive to unexpected stimuli, displaying a heightened startle response, or jumpiness, to loud noises or unexpected movements (e.g., jumping markedly in response to a telephone ringing) (Criterion E4). Concentration difficulties, including difficulty remembering daily events (e.g., forgetting one's telephone number) or attending to focused tasks (e.g., following a conversation for a sustained period of time), are commonly reported (Criterion E5). Problems with sleep onset and maintenance are common and may be associated with nightmares and safety concerns or with generalized elevated arousal that interferes with adequate sleep (Criterion E6). Some individuals also experience persistent dissociative symptoms of detachment from their bodies (depersonalization) or the world around them (derealization); this is reflected in the "with dissociative symptoms" specifier.

Associated Features Supporting Diagnosis

Developmental regression, such as loss of language in young children, may occur. Auditory pseudo-hallucinations, such as having the sensory experience of hearing one's thoughts spoken in one or more different voices, as well as paranoid ideation, can be present. Following prolonged, repeated, and severe traumatic events (e.g., childhood abuse, torture), the individual may additionally experience difficulties in regulating emotions or maintaining stable interpersonal relationships, or dissociative symptoms. When the traumatic event produces violent death, symptoms of both problematic bereavement and PTSD may be present.

Prevalence

In the United States, projected lifetime risk for PTSD using DSM-IV criteria at age 75 years is 8.7%. Twelve-month prevalence among U.S. adults is about 3.5%. Lower estimates are seen in Europe and most Asian, African, and Latin American countries, clustering around 0.5%–1.0%. Although different groups have different levels of exposure to traumatic events, the conditional probability of developing PTSD following a similar level of exposure may also vary across cultural groups. Rates of PTSD are higher among veterans and others whose vocation increases the risk of traumatic exposure (e.g., police, firefighters, emergency medical personnel). Highest rates (ranging from one-third to more than one-half of those exposed) are found among survivors of rape, military combat and captivity, and ethnically or politically motivated internment and genocide. The prevalence of PTSD may vary across development; children and adolescents, including preschool children, generally have displayed lower prevalence following exposure to serious traumatic events; however, this may be because previous criteria were insufficiently developmentally informed. The prevalence of full-threshold PTSD also appears to be lower among older adults compared with the general population; there is evidence that subthreshold presentations are more common than full PTSD in later life and that these symptoms are associated with substantial clinical impairment. Compared with U.S. non-Latino whites, higher rates of PTSD have been reported among U.S. Latinos, African Americans, and American Indians, and lower rates have been reported among Asian Americans, after adjustment for traumatic exposure and demographic variables.

Development and Course

PTSD can occur at any age, beginning after the first year of life. Symptoms usually begin within the first 3 months after the trauma, although there may be a delay of months, or even years, before criteria for the diagnosis are met. There is abundant evidence for what DSM-IV called "delayed onset" but is now called "delayed expression," with the recognition that some symptoms typically appear immediately and that the delay is in meeting full criteria.

Frequently, an individual's reaction to a trauma initially meets criteria for acute stress disorder in the immediate aftermath of the trauma. The symptoms of PTSD and the relative predominance of different symptoms may vary over time. Duration of the symptoms also varies, with complete recovery within 3 months occurring in approximately one-half of adults, while some individuals remain symptomatic for longer than 12 months and sometimes for more than 50 years. Symptom recurrence and intensification may occur in response to reminders of the original trauma, ongoing life stressors, or newly experienced traumatic events. For older individuals, declining health, worsening cognitive functioning, and social isolation may exacerbate PTSD symptoms.

The clinical expression of reexperiencing can vary across development. Young children may report new onset of frightening dreams without content specific to the traumatic event. Before age 6 years (see criteria for preschool subtype), young children are more likely to express reexperiencing symptoms through play that refers directly or symbolically to the trauma. They may not manifest fearful reactions at the time of the exposure or during reexperiencing. Parents may report a wide range of emotional or behavioral changes in young children. Children may focus on imagined interventions in their play or storytelling. In addition to avoidance, children may become preoccupied with reminders. Because of young children's limitations in expressing thoughts or labeling emotions, negative alterations in mood or cognition tend to involve primarily mood changes. Children may experience co-occurring traumas (e.g., physical abuse, witnessing domestic violence) and in chronic circumstances may not be able to identify onset of symptomatology. Avoidant behavior may be associated with restricted play or exploratory behavior in young children; reduced participation in new activities in school-age children; or reluctance to pursue developmental opportunities in adolescents (e.g., dating, driving). Older children and adolescents may judge themselves as cowardly. Adolescents may harbor beliefs of being changed in ways that make them socially undesirable and estrange them from peers (e.g., "Now I'll never fit in") and lose aspirations for the future. Irritable or aggressive behavior in children and adolescents can interfere with peer relationships and school behavior. Reckless behavior may lead to accidental injury to self or others, thrill-seeking, or high-risk behaviors. Individuals who continue to experience PTSD into older adulthood may express fewer symptoms of hyperarousal, avoidance, and negative cognitions and mood compared with younger adults with PTSD, although adults exposed to traumatic events during later life may display more avoidance, hyperarousal, sleep problems, and crying spells than do younger adults exposed to the same traumatic events. In older individuals, the disorder is associated with negative health perceptions, primary care utilization, and suicidal ideation.

Risk and Prognostic Factors

Risk (and protective) factors are generally divided into pretraumatic, peritraumatic, and posttraumatic factors.

Pretraumatic factors

Temperamental. These include childhood emotional problems by age 6 years (e.g., prior traumatic exposure, externalizing or anxiety problems) and prior mental disorders (e.g., panic disorder, depressive disorder, PTSD, or obsessive-compulsive disorder [OCD]).

Environmental. These include lower socioeconomic status; lower education; exposure to prior trauma (especially during childhood); childhood adversity (e.g., economic deprivation, family dysfunction, parental separation or death); cultural characteristics (e.g., fatalistic or self-blaming coping strategies); lower intelligence; minority racial/ethnic status; and a family psychiatric history. Social support prior to event exposure is protective.

Genetic and physiological. These include female gender and younger age at the time of trauma exposure (for adults). Certain genotypes may either be protective or increase risk of PTSD after exposure to traumatic events.

Peritraumatic factors

Environmental. These include severity (dose) of the trauma (the greater the magnitude of trauma, the greater the likelihood of PTSD), perceived life threat, personal injury, interpersonal violence (particularly trauma perpetrated by a caregiver or involving a witnessed threat to a caregiver in children), and, for military personnel, being a perpetrator, witnessing atrocities, or killing the enemy. Finally, dissociation that occurs during the trauma and persists afterward is a risk factor.

Posttraumatic factors

Temperamental. These include negative appraisals, inappropriate coping strategies, and development of acute stress disorder.

Environmental. These include subsequent exposure to repeated upsetting reminders, subsequent adverse life events, and financial or other trauma-related losses. Social support (including family stability, for children) is a protective factor that moderates outcome after trauma.

Culture-Related Diagnostic Issues

The risk of onset and severity of PTSD may differ across cultural groups as a result of variation in the type of traumatic exposure (e.g., genocide), the impact on disorder severity of the meaning attributed to the traumatic event (e.g., inability to perform funerary rites after a mass killing), the ongoing sociocultural context (e.g., residing among unpunished perpetrators in postconflict settings), and other cultural factors (e.g., acculturative stress in immigrants). The relative risk for PTSD of particular exposures (e.g., religious persecution) may vary across cultural groups. The clinical expression of the symptoms or symptom clusters of PTSD may vary culturally, particularly with respect to avoidance and numbing symptoms, distressing dreams, and somatic symptoms (e.g., dizziness, shortness of breath, heat sensations).

Cultural syndromes and idioms of distress influence the expression of PTSD and the range of comorbid disorders in different cultures by providing behavioral and cognitive templates that link traumatic exposures to specific symptoms. For example, panic attack symptoms may be salient in PTSD among Cambodians and Latin Americans because of the association of traumatic exposure with panic-like *khyâl* attacks and *ataque de nervios*. Comprehensive evaluation of local expressions of PTSD should include assessment of cultural concepts of distress (see the chapter "Cultural Formulation" in Section III).

Gender-Related Diagnostic Issues

PTSD is more prevalent among females than among males across the lifespan. Females in the general population experience PTSD for a longer duration than do males. At least some of the increased risk for PTSD in females appears to be attributable to a greater likelihood of exposure to traumatic events, such as rape, and other forms of interpersonal violence. Within populations exposed specifically to such stressors, gender differences in risk for PTSD are attenuated or nonsignificant.

Suicide Risk

Traumatic events such as childhood abuse increase a person's suicide risk. PTSD is associated with suicidal ideation and suicide attempts, and presence of the disorder may indicate which individuals with ideation eventually make a suicide plan or actually attempt suicide.

Functional Consequences of Posttraumatic Stress Disorder

PTSD is associated with high levels of social, occupational, and physical disability, as well as considerable economic costs and high levels of medical utilization. Impaired function-

ing is exhibited across social, interpersonal, developmental, educational, physical health, and occupational domains. In community and veteran samples, PTSD is associated with poor social and family relationships, absenteeism from work, lower income, and lower educational and occupational success.

Differential Diagnosis

Adjustment disorders. In adjustment disorders, the stressor can be of any severity or type rather than that required by PTSD Criterion A. The diagnosis of an adjustment disorder is used when the response to a stressor that meets PTSD Criterion A does not meet all other PTSD criteria (or criteria for another mental disorder). An adjustment disorder is also diagnosed when the symptom pattern of PTSD occurs in response to a stressor that does not meet PTSD Criterion A (e.g., spouse leaving, being fired).

Other posttraumatic disorders and conditions. Not all psychopathology that occurs in individuals exposed to an extreme stressor should necessarily be attributed to PTSD. The diagnosis requires that trauma exposure precede the onset or exacerbation of pertinent symptoms. Moreover, if the symptom response pattern to the extreme stressor meets criteria for another mental disorder, these diagnoses should be given instead of, or in addition to, PTSD. Other diagnoses and conditions are excluded if they are better explained by PTSD (e.g., symptoms of panic disorder that occur only after exposure to traumatic reminders). If severe, symptom response patterns to the extreme stressor may warrant a separate diagnosis (e.g., dissociative amnesia).

Acute stress disorder. Acute stress disorder is distinguished from PTSD because the symptom pattern in acute stress disorder is restricted to a duration of 3 days to 1 month following exposure to the traumatic event.

Anxiety disorders and obsessive-compulsive disorder. In OCD, there are recurrent intrusive thoughts, but these meet the definition of an obsession. In addition, the intrusive thoughts are not related to an experienced traumatic event, compulsions are usually present, and other symptoms of PTSD or acute stress disorder are typically absent. Neither the arousal and dissociative symptoms of panic disorder nor the avoidance, irritability, and anxiety of generalized anxiety disorder are associated with a specific traumatic event. The symptoms of separation anxiety disorder are clearly related to separation from home or family, rather than to a traumatic event.

Major depressive disorder. Major depression may or may not be preceded by a traumatic event and should be diagnosed if other PTSD symptoms are absent. Specifically, major depressive disorder does not include any PTSD Criterion B or C symptoms. Nor does it include a number of symptoms from PTSD Criterion D or E.

Personality disorders. Interpersonal difficulties that had their onset, or were greatly exacerbated, after exposure to a traumatic event may be an indication of PTSD, rather than a personality disorder, in which such difficulties would be expected independently of any traumatic exposure.

Dissociative disorders. Dissociative amnesia, dissociative identity disorder, and depersonalization-derealization disorder may or may not be preceded by exposure to a traumatic event or may or may not have co-occurring PTSD symptoms. When full PTSD criteria are also met, however, the PTSD "with dissociative symptoms" subtype should be considered.

Conversion disorder (functional neurological symptom disorder). New onset of somatic symptoms within the context of posttraumatic distress might be an indication of PTSD rather than conversion disorder (functional neurological symptom disorder).

Psychotic disorders. Flashbacks in PTSD must be distinguished from illusions, hallucinations, and other perceptual disturbances that may occur in schizophrenia, brief psychotic disorder, and other psychotic disorders; depressive and bipolar disorders with

psychotic features; delirium; substance/medication-induced disorders; and psychotic disorders due to another medical condition.

Traumatic brain injury. When a brain injury occurs in the context of a traumatic event (e.g., traumatic accident, bomb blast, acceleration/deceleration trauma), symptoms of PTSD may appear. An event causing head trauma may also constitute a psychological traumatic event, and tramautic brain injury (TBI)–related neurocognitive symptoms are not mutually exclusive and may occur concurrently. Symptoms previously termed *postconcussive* (e.g., headaches, dizziness, sensitivity to light or sound, irritability, concentration deficits) can occur in brain-injured and non-brain-injured populations, including individuals with PTSD. Because symptoms of PTSD and TBI-related neurocognitive symptoms can overlap, a differential diagnosis between PTSD and neurocognitive disorder symptoms attributable to TBI may be possible based on the presence of symptoms that are distinctive to each presentation. Whereas reexperiencing and avoidance are characteristic of PTSD and not the effects of TBI, persistent disorientation and confusion are more specific to TBI (neurocognitive effects) than to PTSD.

Comorbidity

Individuals with PTSD are 80% more likely than those without PTSD to have symptoms that meet diagnostic criteria for at least one other mental disorder (e.g., depressive, bipolar, anxiety, or substance use disorders). Comorbid substance use disorder and conduct disorder are more common among males than among females. Among U.S. military personnel and combat veterans who have been deployed to recent wars in Afghanistan and Iraq, co-occurrence of PTSD and mild TBI is 48%. Although most young children with PTSD also have at least one other diagnosis, the patterns of comorbidity are different than in adults, with oppositional defiant disorder and separation anxiety disorder predominating. Finally, there is considerable comorbidity between PTSD and major neurocognitive disorder and some overlapping symptoms between these disorders.

Acute Stress Disorder

Diagnostic Criteria **308.3 (F43.0)**

A. Exposure to actual or threatened death, serious injury, or sexual violation in one (or more) of the following ways:

1. Directly experiencing the traumatic event(s).
2. Witnessing, in person, the event(s) as it occurred to others.
3. Learning that the event(s) occurred to a close family member or close friend. **Note:** In cases of actual or threatened death of a family member or friend, the event(s) must have been violent or accidental.
4. Experiencing repeated or extreme exposure to aversive details of the traumatic event(s) (e.g., first responders collecting human remains, police officers repeatedly exposed to details of child abuse).

   Note: This does not apply to exposure through electronic media, television, movies, or pictures, unless this exposure is work related.

B. Presence of nine (or more) of the following symptoms from any of the five categories of intrusion, negative mood, dissociation, avoidance, and arousal, beginning or worsening after the traumatic event(s) occurred:

Intrusion Symptoms

1. Recurrent, involuntary, and intrusive distressing memories of the traumatic event(s). **Note:** In children, repetitive play may occur in which themes or aspects of the traumatic event(s) are expressed.

2. Recurrent distressing dreams in which the content and/or affect of the dream are related to the event(s). **Note:** In children, there may be frightening dreams without recognizable content.
3. Dissociative reactions (e.g., flashbacks) in which the individual feels or acts as if the traumatic event(s) were recurring. (Such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings.) **Note:** In children, trauma-specific reenactment may occur in play.
4. Intense or prolonged psychological distress or marked physiological reactions in response to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).

Negative Mood

5. Persistent inability to experience positive emotions (e.g., inability to experience happiness, satisfaction, or loving feelings).

Dissociative Symptoms

6. An altered sense of the reality of one's surroundings or oneself (e.g., seeing oneself from another's perspective, being in a daze, time slowing).
7. Inability to remember an important aspect of the traumatic event(s) (typically due to dissociative amnesia and not to other factors such as head injury, alcohol, or drugs).

Avoidance Symptoms

8. Efforts to avoid distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s).
9. Efforts to avoid external reminders (people, places, conversations, activities, objects, situations) that arouse distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s).

Arousal Symptoms

10. Sleep disturbance (e.g., difficulty falling or staying asleep, restless sleep).
11. Irritable behavior and angry outbursts (with little or no provocation), typically expressed as verbal or physical aggression toward people or objects.
12. Hypervigilance.
13. Problems with concentration.
14. Exaggerated startle response.

C. Duration of the disturbance (symptoms in Criterion B) is 3 days to 1 month after trauma exposure.

Note: Symptoms typically begin immediately after the trauma, but persistence for at least 3 days and up to a month is needed to meet disorder criteria.

D. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

E. The disturbance is not attributable to the physiological effects of a substance (e.g., medication or alcohol) or another medical condition (e.g., mild traumatic brain injury) and is not better explained by brief psychotic disorder.

Diagnostic Features

The essential feature of acute stress disorder is the development of characteristic symptoms lasting from 3 days to 1 month following exposure to one or more traumatic events. Traumatic events that are experienced directly include, but are not limited to, exposure to war as a combatant or civilian, threatened or actual violent personal assault (e.g., sexual

violence, physical attack, active combat, mugging, childhood physical and/or sexual violence, being kidnapped, being taken hostage, terrorist attack, torture), natural or human-made disasters (e.g., earthquake, hurricane, airplane crash), and severe accident (e.g., severe motor vehicle, industrial accident). For children, sexually traumatic events may include inappropriate sexual experiences without violence or injury. A life-threatening illness or debilitating medical condition is not necessarily considered a traumatic event. Medical incidents that qualify as traumatic events involve sudden, catastrophic events (e.g., waking during surgery, anaphylactic shock). Stressful events that do not possess the severe and traumatic components of events encompassed by Criterion A may lead to an adjustment disorder but not to acute stress disorder.

The clinical presentation of acute stress disorder may vary by individual but typically involves an anxiety response that includes some form of reexperiencing of or reactivity to the traumatic event. In some individuals, a dissociative or detached presentation can predominate, although these individuals typically will also display strong emotional or physiological reactivity in response to trauma reminders. In other individuals, there can be a strong anger response in which reactivity is characterized by irritable or possibly aggressive responses. The full symptom picture must be present for at least 3 days after the traumatic event and can be diagnosed only up to 1 month after the event. Symptoms that occur immediately after the event but resolve in less than 3 days would not meet criteria for acute stress disorder.

Witnessed events include, but are not limited to, observing threatened or serious injury, unnatural death, physical or sexual violence inflicted on another individual as a result of violent assault, severe domestic violence, severe accident, war, and disaster; it may also include witnessing a medical catastrophe (e.g., a life-threatening hemorrhage) involving one's child. Events experienced indirectly through learning about the event are limited to close relatives or close friends. Such events must have been violent or accidental—death due to natural causes does not qualify—and include violent personal assault, suicide, serious accident, or serious injury. The disorder may be especially severe when the stressor is interpersonal and intentional (e.g., torture, rape). The likelihood of developing this disorder may increase as the intensity of and physical proximity to the stressor increase.

The traumatic event can be reexperienced in various ways. Commonly, the individual has recurrent and intrusive recollections of the event (Criterion B1). The recollections are spontaneous or triggered recurrent memories of the event that usually occur in response to a stimulus that is reminiscent of the traumatic experience (e.g., the sound of a backfiring car triggering memories of gunshots). These intrusive memories often include sensory (e.g., sensing the intense heat that was perceived in a house fire), emotional (e.g., experiencing the fear of believing that one was about to be stabbed), or physiological (e.g., experiencing the shortness of breath that one suffered during a near-drowning) components.

Distressing dreams may contain themes that are representative of or thematically related to the major threats involved in the traumatic event. (For example, in the case of a motor vehicle accident survivor, the distressing dreams may involve crashing cars generally; in the case of a combat soldier, the distressing dreams may involve being harmed in ways other than combat.)

Dissociative states may last from a few seconds to several hours, or even days, during which components of the event are relived and the individual behaves as though experiencing the event at that moment. While dissociative responses are common during a traumatic event, only dissociative responses that persist beyond 3 days after trauma exposure are considered for the diagnosis of acute stress disorder. For young children, reenactment of events related to trauma may appear in play and may include dissociative moments (e.g., a child who survives a motor vehicle accident may repeatedly crash toy cars during play in a focused and distressing manner). These episodes, often referred to as *flashbacks*, are typically brief but involve a sense that the traumatic event is occurring in the present rather than being remembered in the past and are associated with significant distress.

Some individuals with the disorder do not have intrusive memories of the event itself, but instead experience intense psychological distress or physiological reactivity when they are exposed to triggering events that resemble or symbolize an aspect of the traumatic event (e.g., windy days for children after a hurricane, entering an elevator for a male or female who was raped in an elevator, seeing someone who resembles one's perpetrator). The triggering cue could be a physical sensation (e.g., a sense of heat for a burn victim, dizziness for survivors of head trauma), particularly for individuals with highly somatic presentations. The individual may have a persistent inability to feel positive emotions (e.g., happiness, joy, satisfaction, or emotions associated with intimacy, tenderness, or sexuality) but can experience negative emotions such as fear, sadness, anger, guilt, or shame.

Alterations in awareness can include *depersonalization*, a detached sense of oneself (e.g., seeing oneself from the other side of the room), or *derealization*, having a distorted view of one's surroundings (e.g., perceiving that things are moving in slow motion, seeing things in a daze, not being aware of events that one would normally encode). Some individuals also report an inability to remember an important aspect of the traumatic event that was presumably encoded. This symptom is attributable to dissociative amnesia and is not attributable to head injury, alcohol, or drugs.

Stimuli associated with the trauma are persistently avoided. The individual may refuse to discuss the traumatic experience or may engage in avoidance strategies to minimize awareness of emotional reactions (e.g., excessive alcohol use when reminded of the experience). This behavioral avoidance may include avoiding watching news coverage of the traumatic experience, refusing to return to a workplace where the trauma occurred, or avoiding interacting with others who shared the same traumatic experience.

It is very common for individuals with acute stress disorder to experience problems with sleep onset and maintenance, which may be associated with nightmares or with generalized elevated arousal that prevents adequate sleep. Individuals with acute stress disorder may be quick tempered and may even engage in aggressive verbal and/or physical behavior with little provocation. Acute stress disorder is often characterized by a heightened sensitivity to potential threats, including those that are related to the traumatic experience (e.g., a motor vehicle accident victim may be especially sensitive to the threat potentially caused by any cars or trucks) or those not related to the traumatic event (e.g., fear of having a heart attack). Concentration difficulties, including difficulty remembering daily events (e.g., forgetting one's telephone number) or attending to focused tasks (e.g., following a conversation for a sustained period of time), are commonly reported. Individuals with acute stress disorder may be very reactive to unexpected stimuli, displaying a heightened startle response or jumpiness to loud noises or unexpected movements (e.g., the individual may jump markedly in the response to a telephone ringing).

Associated Features Supporting Diagnosis

Individuals with acute stress disorder commonly engage in catastrophic or extremely negative thoughts about their role in the traumatic event, their response to the traumatic experience, or the likelihood of future harm. For example, an individual with acute stress disorder may feel excessively guilty about not having prevented the traumatic event or about not adapting to the experience more successfully. Individuals with acute stress disorder may also interpret their symptoms in a catastrophic manner, such that flashback memories or emotional numbing may be interpreted as a sign of diminished mental capacity. It is common for individuals with acute stress disorder to experience panic attacks in the initial month after trauma exposure that may be triggered by trauma reminders or may apparently occur spontaneously. Additionally, individuals with acute stress disorder may display chaotic or impulsive behavior. For example, individuals may drive recklessly, make irrational decisions, or gamble excessively. In children, there may be significant separation anxiety, possibly manifested by excessive needs for attention from

caregivers. In the case of bereavement following a death that occurred in traumatic circumstances, the symptoms of acute stress disorder can involve acute grief reactions. In such cases, reexperiencing, dissociative, and arousal symptoms may involve reactions to the loss, such as intrusive memories of the circumstances of the individual's death, disbelief that the individual has died, and anger about the death. Postconcussive symptoms (e.g., headaches, dizziness, sensitivity to light or sound, irritability, concentration deficits), which occur frequently following mild traumatic brain injury, are also frequently seen in individuals with acute stress disorder. Postconcussive symptoms are equally common in brain-injured and non–brain-injured populations, and the frequent occurrence of postconcussive symptoms could be attributable to acute stress disorder symptoms.

Prevalence

The prevalence of acute stress disorder in recently trauma-exposed populations (i.e., within 1 month of trauma exposure) varies according to the nature of the event and the context in which it is assessed. In both U.S. and non-U.S. populations, acute stress disorder tends to be identified in less than 20% of cases following traumatic events that do not involve interpersonal assault; 13%–21% of motor vehicle accidents, 14% of mild traumatic brain injury, 19% of assault, 10% of severe burns, and 6%–12% of industrial accidents. Higher rates (i.e., 20%–50%) are reported following interpersonal traumatic events, including assault, rape, and witnessing a mass shooting.

Development and Course

Acute stress disorder cannot be diagnosed until 3 days after a traumatic event. Although acute stress disorder may progress to posttraumatic stress disorder (PTSD) after 1 month, it may also be a transient stress response that remits within 1 month of trauma exposure and does not result in PTSD. Approximately half of individuals who eventually develop PTSD initially present with acute stress disorder. Symptom worsening during the initial month can occur, often as a result of ongoing life stressors or further traumatic events.

The forms of reexperiencing can vary across development. Unlike adults or adolescents, young children may report frightening dreams without content that clearly reflects aspects of the trauma (e.g., waking in fright in the aftermath of the trauma but being unable to relate the content of the dream to the traumatic event). Children age 6 years and younger are more likely than older children to express reexperiencing symptoms through play that refers directly or symbolically to the trauma. For example, a very young child who survived a fire may draw pictures of flames. Young children also do not necessarily manifest fearful reactions at the time of the exposure or even during reexperiencing. Parents typically report a range of emotional expressions, such as anger, shame, or withdrawal, and even excessively bright positive affect, in young children who are traumatized. Although children may avoid reminders of the trauma, they sometimes become preoccupied with reminders (e.g., a young child bitten by a dog may talk about dogs constantly yet avoid going outside because of fear of coming into contact with a dog).

Risk and Prognostic Factors

Temperamental. Risk factors include prior mental disorder, high levels of negative affectivity (neuroticism), greater perceived severity of the traumatic event, and an avoidant coping style. Catastrophic appraisals of the traumatic experience, often characterized by exaggerated appraisals of future harm, guilt, or hopelessness, are strongly predictive of acute stress disorder.

Environmental. First and foremost, an individual must be exposed to a traumatic event to be at risk for acute stress disorder. Risk factors for the disorder include a history of prior trauma.

Genetic and physiological. Females are at greater risk for developing acute stress disorder.

Elevated reactivity, as reflected by acoustic startle response, prior to trauma exposure increases the risk for developing acute stress disorder.

Culture-Related Diagnostic Issues

The profile of symptoms of acute stress disorder may vary cross-culturally, particularly with respect to dissociative symptoms, nightmares, avoidance, and somatic symptoms (e.g., dizziness, shortness of breath, heat sensations). Cultural syndromes and idioms of distress shape the local symptom profiles of acute stress disorder. Some cultural groups may display variants of dissociative responses, such as possession or trancelike behaviors in the initial month after trauma exposure. Panic symptoms may be salient in acute stress disorder among Cambodians because of the association of traumatic exposure with panic-like *khyâl* attacks, and *ataque de nervios* among Latin Americans may also follow a traumatic exposure.

Gender-Related Diagnostic Issues

Acute stress disorder is more prevalent among females than among males. Sex-linked neurobiological differences in stress response may contribute to females' increased risk for acute stress disorder. The increased risk for the disorder in females may be attributable in part to a greater likelihood of exposure to the types of traumatic events with a high conditional risk for acute stress disorder, such as rape and other interpersonal violence.

Functional Consequences of Acute Stress Disorder

Impaired functioning in social, interpersonal, or occupational domains has been shown across survivors of accidents, assault, and rape who develop acute stress disorder. The extreme levels of anxiety that may be associated with acute stress disorder may interfere with sleep, energy levels, and capacity to attend to tasks. Avoidance in acute stress disorder can result in generalized withdrawal from many situations that are perceived as potentially threatening, which can lead to nonattendance of medical appointments, avoidance of driving to important appointments, and absenteeism from work.

Differential Diagnosis

Adjustment disorders. In acute stress disorder, the stressor can be of any severity rather than of the severity and type required by Criterion A of acute stress disorder. The diagnosis of an adjustment disorder is used when the response to a Criterion A event does not meet the criteria for acute stress disorder (or another specific mental disorder) and when the symptom pattern of acute stress disorder occurs in response to a stressor that does not meet Criterion A for exposure to actual or threatened death, serious injury, or sexual violence (e.g., spouse leaving, being fired). For example, severe stress reactions to life-threatening illnesses that may include some acute stress disorder symptoms may be more appropriately described as an adjustment disorder. Some forms of acute stress response do not include acute stress disorder symptoms and may be characterized by anger, depression, or guilt. These responses are more appropriately described as primarily an adjustment disorder. Depressive or anger responses in an adjustment disorder may involve rumination about the traumatic event, as opposed to involuntary and intrusive distressing memories in acute stress disorder.

Panic disorder. Spontaneous panic attacks are very common in acute stress disorder. However, panic disorder is diagnosed only if panic attacks are unexpected and there is anxiety about future attacks or maladaptive changes in behavior associated with fear of dire consequences of the attacks.

Dissociative disorders. Severe dissociative responses (in the absence of characteristic acute stress disorder symptoms) may be diagnosed as derealization/depersonalization disorder. If severe amnesia of the trauma persists in the absence of characteristic acute stress disorder symptoms, the diagnosis of dissociative amnesia may be indicated.

Posttraumatic stress disorder. Acute stress disorder is distinguished from PTSD because the symptom pattern in acute stress disorder must occur within 1 month of the traumatic event and resolve within that 1-month period. If the symptoms persist for more than 1 month and meet criteria for PTSD, the diagnosis is changed from acute stress disorder to PTSD.

Obsessive-compulsive disorder. In obsessive-compulsive disorder, there are recurrent intrusive thoughts, but these meet the definition of an obsession. In addition, the intrusive thoughts are not related to an experienced traumatic event, compulsions are usually present, and other symptoms of acute stress disorder are typically absent.

Psychotic disorders. Flashbacks in acute stress disorder must be distinguished from illusions, hallucinations, and other perceptual disturbances that may occur in schizophrenia, other psychotic disorders, depressive or bipolar disorder with psychotic features, a delirium, substance/medication-induced disorders, and psychotic disorders due to another medical condition. Acute stress disorder flashbacks are distinguished from these other perceptual disturbances by being directly related to the traumatic experience and by occurring in the absence of other psychotic or substance-induced features.

Traumatic brain injury. When a brain injury occurs in the context of a traumatic event (e.g., traumatic accident, bomb blast, acceleration/deceleration trauma), symptoms of acute stress disorder may appear. An event causing head trauma may also constitute a psychological traumatic event, and tramautic brain injury (TBI)–related neurocognitive symptoms are not mutually exclusive and may occur concurrently. Symptoms previously termed *postconcussive* (e.g., headaches, dizziness, sensitivity to light or sound, irritability, concentration deficits) can occur in brain-injured and non–brain injured populations, including individuals with acute stress disorder. Because symptoms of acute stress disorder and TBI-related neurocognitive symptoms can overlap, a differential diagnosis between acute stress disorder and neurocognitive disorder symptoms attributable to TBI may be possible based on the presence of symptoms that are distinctive to each presentation. Whereas reexperiencing and avoidance are characteristic of acute stress disorder and not the effects of TBI, persistent disorientation and confusion are more specific to TBI (neurocognitive effects) than to acute stress disorder. Furthermore, differential is aided by the fact that symptoms of acute stress disorder persist for up to only 1 month following trauma exposure.

Adjustment Disorders

Diagnostic Criteria

A. The development of emotional or behavioral symptoms in response to an identifiable stressor(s) occurring within 3 months of the onset of the stressor(s).

B. These symptoms or behaviors are clinically significant, as evidenced by one or both of the following:

   1. Marked distress that is out of proportion to the severity or intensity of the stressor, taking into account the external context and the cultural factors that might influence symptom severity and presentation.
   2. Significant impairment in social, occupational, or other important areas of functioning.

C. The stress-related disturbance does not meet the criteria for another mental disorder and is not merely an exacerbation of a preexisting mental disorder.

D. The symptoms do not represent normal bereavement.
E. Once the stressor or its consequences have terminated, the symptoms do not persist for more than an additional 6 months.

Specify whether:

309.0 (F43.21) With depressed mood: Low mood, tearfulness, or feelings of hopelessness are predominant.

309.24 (F43.22) With anxiety: Nervousness, worry, jitteriness, or separation anxiety is predominant.

309.28 (F43.23) With mixed anxiety and depressed mood: A combination of depression and anxiety is predominant.

309.3 (F43.24) With disturbance of conduct: Disturbance of conduct is predominant.

309.4 (F43.25) With mixed disturbance of emotions and conduct: Both emotional symptoms (e.g., depression, anxiety) and a disturbance of conduct are predominant.

309.9 (F43.20) Unspecified: For maladaptive reactions that are not classifiable as one of the specific subtypes of adjustment disorder.

Diagnostic Features

The presence of emotional or behavioral symptoms in response to an identifiable stressor is the essential feature of adjustment disorders (Criterion A). The stressor may be a single event (e.g., a termination of a romantic relationship), or there may be multiple stressors (e.g., marked business difficulties and marital problems). Stressors may be recurrent (e.g., associated with seasonal business crises, unfulfilling sexual relationships) or continuous (e.g., a persistent painful illness with increasing disability, living in a crime-ridden neighborhood). Stressors may affect a single individual, an entire family, or a larger group or community (e.g., a natural disaster). Some stressors may accompany specific developmental events (e.g., going to school, leaving a parental home, reentering a parental home, getting married, becoming a parent, failing to attain occupational goals, retirement).

Adjustment disorders may be diagnosed following the death of a loved one when the intensity, quality, or persistence of grief reactions exceeds what normally might be expected, when cultural, religious, or age-appropriate norms are taken into account. A more specific set of bereavement-related symptoms has been designated *persistent complex bereavement disorder*.

Adjustment disorders are associated with an increased risk of suicide attempts and completed suicide.

Prevalence

Adjustment disorders are common, although prevalence may vary widely as a function of the population studied and the assessment methods used. The percentage of individuals in outpatient mental health treatment with a principal diagnosis of an adjustment disorder ranges from approximately 5% to 20%. In a hospital psychiatric consultation setting, it is often the most common diagnosis, frequently reaching 50%.

Development and Course

By definition, the disturbance in adjustment disorders begins within 3 months of onset of a stressor and lasts no longer than 6 months after the stressor or its consequences have ceased. If the stressor is an acute event (e.g., being fired from a job), the onset of the disturbance is usually immediate (i.e., within a few days) and the duration is relatively brief (i.e., no more than a few months). If the stressor or its consequences persist, the adjustment disorder may also continue to be present and become the persistent form.

Risk and Prognostic Factors

Environmental. Individuals from disadvantaged life circumstances experience a high rate of stressors and may be at increased risk for adjustment disorders.

Culture-Reiated Diagnostic Issues

The context of the individual's cultural setting should be taken into account in making the clinical judgment of whether the individual's response to the stressor is maladaptive or whether the associated distress is in excess of what would be expected. The nature, meaning, and experience of the stressors and the evaluation of the response to the stressors may vary across cultures.

Functional Consequences of Adjustment Disorders

The subjective distress or impairment in functioning associated with adjustment disorders is frequently manifested as decreased performance at work or school and temporary changes in social relationships. An adjustment disorder may complicate the course of illness in individuals who have a general medical condition (e.g., decreased compliance with the recommended medical regimen; increased length of hospital stay).

Differential Diagnosis

Major depressive disorder. If an individual has symptoms that meet criteria for a major depressive disorder in response to a stressor, the diagnosis of an adjustment disorder is not applicable. The symptom profile of major depressive disorder differentiates it from adjustment disorders.

Posttraumatic stress disorder and acute stress disorder. In adjustment disorders, the stressor can be of any severity rather than of the severity and type required by Criterion A of acute stress disorder and posttraumatic stress disorder (PTSD). In distinguishing adjustment disorders from these two posttraumatic diagnoses, there are both timing and symptom profile considerations. Adjustment disorders can be diagnosed immediately and persist up to 6 months after exposure to the traumatic event, whereas acute stress disorder can only occur between 3 days and 1 month of exposure to the stressor, and PTSD cannot be diagnosed until at least 1 month has passed since the occurrence of the traumatic stressor. The required symptom profile for PTSD and acute stress disorder differentiates them from the adjustment disorders. With regard to symptom profiles, an adjustment disorder may be diagnosed following a traumatic event when an individual exhibits symptoms of either acute stress disorder or PTSD that do not meet or exceed the diagnostic threshold for either disorder. An adjustment disorder should also be diagnosed for individuals who have not been exposed to a traumatic event but who otherwise exhibit the full symptom profile of either acute stress disorder or PTSD.

Personality disorders. With regard to personality disorders, some personality features may be associated with a vulnerability to situational distress that may resemble an adjustment disorder. The lifetime history of personality functioning will help inform the interpretation of distressed behaviors to aid in distinguishing a long-standing personality disorder from an adjustment disorder. In addition to some personality disorders incurring vulnerability to distress, stressors may also exacerbate personality disorder symptoms. In the presence of a personality disorder, if the symptom criteria for an adjustment disorder are met, and the stress-related disturbance exceeds what may be attributable to maladaptive personality disorder symptoms (i.e., Criterion C is met), then the diagnosis of an adjustment disorder should be made.

Psychological factors affecting other medical conditions. In psychological factors affecting other medical conditions, specific psychological entities (e.g., psychological symptoms, behaviors, other factors) exacerbate a medical condition. These psychological factors can precipitate, exacerbate, or put an individual at risk for medical illness, or they can worsen an existing condition. In contrast, an adjustment disorder is a reaction to the stressor (e.g., having a medical illness).

Normative stress reactions. When bad things happen, most people get upset. This is not an adjustment disorder. The diagnosis should only be made when the magnitude of the distress (e.g., alterations in mood, anxiety, or conduct) exceeds what would normally be expected (which may vary in different cultures) or when the adverse event precipitates functional impairment.

Comorbidity

Adjustment disorders can accompany most mental disorders and any medical disorder. Adjustment disorders can be diagnosed in addition to another mental disorder only if the latter does not explain the particular symptoms that occur in reaction to the stressor. For example, an individual may develop an adjustment disorder, with depressed mood, after losing a job and at the same time have a diagnosis of obsessive-compulsive disorder. Or, an individual may have a depressive or bipolar disorder and an adjustment disorder as long as the criteria for both are met. Adjustment disorders are common accompaniments of medical illness and may be the major psychological response to a medical disorder.

Other Specified Trauma- and Stressor-Related Disorder

309.89 (F43.8)

This category applies to presentations in which symptoms characteristic of a trauma- and stressor-related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the trauma- and stressor-related disorders diagnostic class. The other specified trauma- and stressor-related disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific trauma- and stressor-related disorder. This is done by recording "other specified trauma- and stressor-related disorder" followed by the specific reason (e.g., "persistent complex bereavement disorder").

Examples of presentations that can be specified using the "other specified" designation include the following:

1. **Adjustment-like disorders with delayed onset of symptoms that occur more than 3 months after the stressor.**
2. **Adjustment-like disorders with prolonged duration of more than 6 months without prolonged duration of stressor.**
3. ***Ataque de nervios:*** See "Glossary of Cultural Concepts of Distress" in the Appendix.
4. **Other cultural syndromes:** See "Glossary of Cultural Concepts of Distress" in the Appendix.
5. **Persistent complex bereavement disorder:** This disorder is characterized by severe and persistent grief and mourning reactions (see the chapter "Conditions for Further Study").

Unspecified Trauma- and Stressor-Related Disorder

309.9 (F43.9)

This category applies to presentations in which symptoms characteristic of a trauma- and stressor-related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the trauma- and stressor-related disorders diagnostic class. The unspecified trauma- or stressor-related disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific trauma- and stressor-related disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Dissociative Disorders

Dissociative disorders are characterized by a disruption of and/or discontinuity in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control, and behavior. Dissociative symptoms can potentially disrupt every area of psychological functioning. This chapter includes dissociative identity disorder, dissociative amnesia, depersonalization/derealization disorder, other specified dissociative disorder, and unspecified dissociative disorder.

Dissociative symptoms are experienced as a) unbidden intrusions into awareness and behavior, with accompanying losses of continuity in subjective experience (i.e., "positive" dissociative symptoms such as fragmentation of identity, depersonalization, and derealization) and/or b) inability to access information or to control mental functions that normally are readily amenable to access or control (i.e., "negative" dissociative symptoms such as amnesia).

The dissociative disorders are frequently found in the aftermath of trauma, and many of the symptoms, including embarrassment and confusion about the symptoms or a desire to hide them, are influenced by the proximity to trauma. In DSM-5, the dissociative disorders are placed next to, but are not part of, the trauma- and stressor-related disorders, reflecting the close relationship between these diagnostic classes. Both acute stress disorder and posttraumatic stress disorder contain dissociative symptoms, such as amnesia, flashbacks, numbing, and depersonalization/derealization.

Depersonalization/derealization disorder is characterized by clinically significant persistent or recurrent depersonalization (i.e., experiences of unreality or detachment from one's mind, self, or body) and/or derealization (i.e., experiences of unreality or detachment from one's surroundings). These alterations of experience are accompanied by intact reality testing. There is no evidence of any distinction between individuals with predominantly depersonalization versus derealization symptoms. Therefore, individuals with this disorder can have depersonalization, derealization, or both.

Dissociative amnesia is characterized by an inability to recall autobiographical information. This amnesia may be localized (i.e., an event or period of time), selective (i.e., a specific aspect of an event), or generalized (i.e., identity and life history). Dissociative amnesia is fundamentally an inability to recall autobiographical information that is inconsistent with normal forgetting. It may or may not involve purposeful travel or bewildered wandering (i.e., fugue). Although some individuals with amnesia promptly notice that they have "lost time" or that they have a gap in their memory, most individuals with dissociative disorders are initially unaware of their amnesias. For them, awareness of amnesia occurs only when personal identity is lost or when circumstances make these individuals aware that autobiographical information is missing (e.g., when they discover evidence of events they cannot recall or when others tell them or ask them about events they cannot recall). Until and unless this happens, these individuals have "amnesia for their amnesia." Amnesia is experienced as an essential feature of dissociative amnesia; individuals may experience localized or selective amnesia most commonly, or generalized amnesia rarely. Dissociative fugue is rare in persons with dissociative amnesia but common in dissociative identity disorder.

Dissociative identity disorder is characterized by a) the presence of two or more distinct personality states or an experience of possession and b) recurrent episodes of amnesia. The

fragmentation of identity may vary with culture (e.g., possession-form presentations) and circumstance. Thus, individuals may experience discontinuities in identity and memory that may not be immediately evident to others or are obscured by attempts to hide dysfunction. Individuals with dissociative identity disorder experience a) recurrent, inexplicable intrusions into their conscious functioning and sense of self (e.g., voices; dissociated actions and speech; intrusive thoughts, emotions, and impulses), b) alterations of sense of self (e.g., attitudes, preferences, and feeling like one's body or actions are not one's own), c) odd changes of perception (e.g., depersonalization or derealization, such as feeling detached from one's body while cutting), and d) intermittent functional neurological symptoms. Stress often produces transient exacerbation of dissociative symptoms that makes them more evident.

The residual category of other specified dissociative disorder has seven examples: chronic or recurrent mixed dissociative symptoms that approach, but fall short of, the diagnostic criteria for dissociative identity disorder; dissociative states secondary to brainwashing or thought reform; two acute presentations, of less than 1 month's duration, of mixed dissociative symptoms, one of which is also marked by the presence of psychotic symptoms; and three single-symptom dissociative presentations—dissociative trance, dissociative stupor or coma, and Ganser's syndrome (the giving of approximate and vague answers).

Dissociative Identity Disorder

Diagnostic Criteria **300.14 (F44.81)**

A. Disruption of identity characterized by two or more distinct personality states, which may be described in some cultures as an experience of possession. The disruption in identity involves marked discontinuity in sense of self and sense of agency, accompanied by related alterations in affect, behavior, consciousness, memory, perception, cognition, and/or sensory-motor functioning. These signs and symptoms may be observed by others or reported by the individual.

B. Recurrent gaps in the recall of everyday events, important personal information, and/or traumatic events that are inconsistent with ordinary forgetting.

C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The disturbance is not a normal part of a broadly accepted cultural or religious practice. **Note:** In children, the symptoms are not better explained by imaginary playmates or other fantasy play.

E. The symptoms are not attributable to the physiological effects of a substance (e.g., blackouts or chaotic behavior during alcohol intoxication) or another medical condition (e.g., complex partial seizures).

Diagnostic Features

The defining feature of dissociative identity disorder is the presence of two or more distinct personality states or an experience of possession (Criterion A). The overtness or covertness of these personality states, however, varies as a function of psychological motivation, current level of stress, culture, internal conflicts and dynamics, and emotional resilience. Sustained periods of identity disruption may occur when psychosocial pressures are severe and/or prolonged. In many possession-form cases of dissociative identity disorder, and in a small proportion of non-possession-form cases, manifestations of alternate identities are highly overt. Most individuals with non-possession-form dissociative identity disorder do not overtly display their discontinuity of identity for long periods of time; only a small minority present to clinical attention with observable alternation of

identities. When alternate personality states are not directly observed, the disorder can be identified by two clusters of symptoms: 1) sudden alterations or discontinuities in sense of self and sense of agency (Criterion A), and 2) recurrent dissociative amnesias (Criterion B).

Criterion A symptoms are related to discontinuities of experience that can affect any aspect of an individual's functioning. Individuals with dissociative identity disorder may report the feeling that they have suddenly become depersonalized observers of their "own" speech and actions, which they may feel powerless to stop (sense of self). Such individuals may also report perceptions of voices (e.g., a child's voice; crying; the voice of a spiritual being). In some cases, voices are experienced as multiple, perplexing, independent thought streams over which the individual experiences no control. Strong emotions, impulses, and even speech or other actions may suddenly emerge, without a sense of personal ownership or control (sense of agency). These emotions and impulses are frequently reported as ego-dystonic and puzzling. Attitudes, outlooks, and personal preferences (e.g., about food, activities, dress) may suddenly shift and then shift back. Individuals may report that their bodies feel different (e.g., like a small child, like the opposite gender, huge and muscular). Alterations in sense of self and loss of personal agency may be accompanied by a feeling that these attitudes, emotions, and behaviors—even one's body—are "not mine" and/or are "not under my control." Although most Criterion A symptoms are subjective, many of these sudden discontinuities in speech, affect, and behavior can be witnessed by family, friends, or the clinician. Non-epileptic seizures and other conversion symptoms are prominent in some presentations of dissociative identity disorder, especially in some non-Western settings.

The dissociative amnesia of individuals with dissociative identity disorder manifests in three primary ways: as 1) gaps in remote memory of personal life events (e.g., periods of childhood or adolescence; some important life events, such as the death of a grandparent, getting married, giving birth); 2) lapses in dependable memory (e.g., of what happened today, of well-learned skills such as how to do their job, use a computer, read, drive); and 3) discovery of evidence of their everyday actions and tasks that they do not recollect doing (e.g., finding unexplained objects in their shopping bags or among their possessions; finding perplexing writings or drawings that they must have created; discovering injuries; "coming to" in the midst of doing something). Dissociative fugues, wherein the person discovers dissociated travel, are common. Thus, individuals with dissociative identity disorder may report that they have suddenly found themselves at the beach, at work, in a nightclub, or somewhere at home (e.g., in the closet, on a bed or sofa, in the corner) with no memory of how they came to be there. Amnesia in individuals with dissociative identity disorder is not limited to stressful or traumatic events; these individuals often cannot recall everyday events as well.

Individuals with dissociative identity disorder vary in their awareness and attitude toward their amnesias. It is common for these individuals to minimize their amnestic symptoms. Some of their amnestic behaviors may be apparent to others—as when these persons do not recall something they were witnessed to have done or said, when they cannot remember their own name, or when they do not recognize their spouse, children, or close friends.

Possession-form identities in dissociative identity disorder typically manifest as behaviors that appear as if a "spirit," supernatural being, or outside person has taken control, such that the individual begins speaking or acting in a distinctly different manner. For example, an individual's behavior may give the appearance that her identity has been replaced by the "ghost" of a girl who committed suicide in the same community years before, speaking and acting as though she were still alive. Or an individual may be "taken over" by a demon or deity, resulting in profound impairment, and demanding that the individual or a relative be punished for a past act, followed by more subtle periods of identity alteration. However, the majority of possession states around the world are normal, usually part of spiritual practice, and do not meet criteria for dissociative identity disor-

der. The identities that arise during possession-form dissociative identity disorder present recurrently, are unwanted and involuntary, cause clinically significant distress or impairment (Criterion C), and are not a normal part of a broadly accepted cultural or religious practice (Criterion D).

Associated Features Supporting Diagnosis

Individuals with dissociative identity disorder typically present with comorbid depression, anxiety, substance abuse, self-injury, non-epileptic seizures, or another common symptom. They often conceal, or are not fully aware of, disruptions in consciousness, amnesia, or other dissociative symptoms. Many individuals with dissociative identity disorder report dissociative flashbacks during which they undergo a sensory reliving of a previous event as though it were occurring in the present, often with a change of identity, a partial or complete loss of contact with or disorientation to current reality during the flashback, and a subsequent amnesia for the content of the flashback. Individuals with the disorder typically report multiple types of interpersonal maltreatment during childhood and adulthood. Nonmaltreatment forms of overwhelming early life events, such as multiple long, painful, early-life medical procedures, also may be reported. Self-mutilation and suicidal behavior are frequent. On standardized measures, these individuals report higher levels of hypnotizability and dissociativity compared with other clinical groups and healthy control subjects. Some individuals experience transient psychotic phenomena or episodes. Several brain regions have been implicated in the pathophysiology of dissociative identity disorder, including the orbitofrontal cortex, hippocampus, parahippocampal gyrus, and amygdala.

Prevalence

The 12-month prevalence of dissociative identity disorder among adults in a small U.S. community study was 1.5%. The prevalence across genders in that study was 1.6% for males and 1.4% for females.

Development and Course

Dissociative identity disorder is associated with overwhelming experiences, traumatic events, and/or abuse occurring in childhood. The full disorder may first manifest at almost any age (from earliest childhood to late life). Dissociation in children may generate problems with memory, concentration, attachment, and traumatic play. Nevertheless, children usually do not present with identity changes; instead they present primarily with overlap and interference among mental states (Criterion A phenomena), with symptoms related to discontinuities of experience. Sudden changes in identity during adolescence may appear to be just adolescent turmoil or the early stages of another mental disorder. Older individuals may present to treatment with what appear to be late-life mood disorders, obsessive-compulsive disorder, paranoia, psychotic mood disorders, or even cognitive disorders due to dissociative amnesia. In some cases, disruptive affects and memories may increasingly intrude into awareness with advancing age.

Psychological decompensation and overt changes in identity may be triggered by 1) removal from the traumatizing situation (e.g., through leaving home); 2) the individual's children reaching the same age at which the individual was originally abused or traumatized; 3) later traumatic experiences, even seemingly inconsequential ones, like a minor motor vehicle accident; or 4) the death of, or the onset of a fatal illness in, their abuser(s).

Risk and Prognostic Factors

Environmental. Interpersonal physical and sexual abuse is associated with an increased risk of dissociative identity disorder. Prevalence of childhood abuse and neglect in the

United States, Canada, and Europe among those with the disorder is about 90%. Other forms of traumatizing experiences, including childhood medical and surgical procedures, war, childhood prostitution, and terrorism, have been reported.

Course modifiers. Ongoing abuse, later-life retraumatization, comorbidity with mental disorders, severe medical illness, and delay in appropriate treatment are associated with poorer prognosis.

Culture-Related Diagnostic Issues

Many features of dissociative identity disorder can be influenced by the individual's cultural background. Individuals with this disorder may present with prominent medically unexplained neurological symptoms, such as non-epileptic seizures, paralyses, or sensory loss, in cultural settings where such symptoms are common. Similarly, in settings where normative possession is common (e.g., rural areas in the developing world, among certain religious groups in the United States and Europe), the fragmented identities may take the form of possessing spirits, deities, demons, animals, or mythical figures. Acculturation or prolonged intercultural contact may shape the characteristics of the other identities (e.g., identities in India may speak English exclusively and wear Western clothes). Possession-form dissociative identity disorder can be distinguished from culturally accepted possession states in that the former is involuntary, distressing, uncontrollable, and often recurrent or persistent; involves conflict between the individual and his or her surrounding family, social, or work milieu; and is manifested at times and in places that violate the norms of the culture or religion.

Gender-Related Diagnostic Issues

Females with dissociative identity disorder predominate in adult clinical settings but not in child clinical settings. Adult males with dissociative identity disorder may deny their symptoms and trauma histories, and this can lead to elevated rates of false negative diagnosis. Females with dissociative identity disorder present more frequently with acute dissociative states (e.g., flashbacks, amnesia, fugue, functional neurological [conversion] symptoms, hallucinations, self-mutilation). Males commonly exhibit more criminal or violent behavior than females; among males, common triggers of acute dissociative states include combat, prison conditions, and physical or sexual assaults.

Suicide Risk

Over 70% of outpatients with dissociative identity disorder have attempted suicide; multiple attempts are common, and other self-injurious behavior is frequent. Assessment of suicide risk may be complicated when there is amnesia for past suicidal behavior or when the presenting identity does not feel suicidal and is unaware that other dissociated identities do.

Functional Consequences of Dissociative Identity Disorder

Impairment varies widely, from apparently minimal (e.g., in high-functioning professionals) to profound. Regardless of level of disability, individuals with dissociative identity disorder commonly minimize the impact of their dissociative and posttraumatic symptoms. The symptoms of higher-functioning individuals may impair their relational, marital, family, and parenting functions more than their occupational and professional life (although the latter also may be affected). With appropriate treatment, many impaired individuals show marked improvement in occupational and personal functioning. However, some remain highly impaired in most activities of living. These individuals may only respond to treatment very slowly, with gradual reduction in or improved tolerance of

their dissociative and posttraumatic symptoms. Long-term supportive treatment may slowly increase these individuals' ability to manage their symptoms and decrease use of more restrictive levels of care.

Differential Diagnosis

Other specified dissociative disorder. The core of dissociative identity disorder is the division of identity, with recurrent disruption of conscious functioning and sense of self. This central feature is shared with one form of other specified dissociative disorder, which may be distinguished from dissociative identity disorder by the presence of chronic or recurrent mixed dissociative symptoms that do not meet Criterion A for dissociative identity disorder or are not accompanied by recurrent amnesia.

Major depressive disorder. Individuals with dissociative identity disorder are often depressed, and their symptoms may appear to meet the criteria for a major depressive episode. Rigorous assessment indicates that this depression in some cases does not meet full criteria for major depressive disorder. Other specified depressive disorder in individuals with dissociative identity disorder often has an important feature: the depressed mood and cognitions *fluctuate* because they are experienced in some identity states but not others.

Bipolar disorders. Individuals with dissociative identity disorder are often misdiagnosed with a bipolar disorder, most often bipolar II disorder. The relatively rapid shifts in mood in individuals with this disorder—typically within minutes or hours, in contrast to the slower mood changes typically seen in individuals with bipolar disorders—are due to the rapid, subjective shifts in mood commonly reported across dissociative states, sometimes accompanied by fluctuation in levels of activation. Furthermore, in dissociative identity disorder, elevated or depressed mood may be displayed in conjunction with overt identities, so one or the other mood may predominate for a relatively long period of time (often for days) or may shift within minutes.

Posttraumatic stress disorder. Some traumatized individuals have both posttraumatic stress disorder (PTSD) and dissociative identity disorder. Accordingly, it is crucial to distinguish between individuals with PTSD only and individuals who have both PTSD and dissociative identity disorder. This differential diagnosis requires that the clinician establish the presence or absence of dissociative symptoms that are not characteristic of acute stress disorder or PTSD. Some individuals with PTSD manifest dissociative symptoms that also occur in dissociative identity disorder: 1) amnesia for some aspects of trauma, 2) dissociative flashbacks (i.e., reliving of the trauma, with reduced awareness of one's current orientation), and 3) symptoms of intrusion and avoidance, negative alterations in cognition and mood, and hyperarousal that are focused around the traumatic event. On the other hand, individuals with dissociative identity disorder manifest dissociative symptoms that are not a manifestation of PTSD: 1) amnesias for many everyday (i.e., nontraumatic) events, 2) dissociative flashbacks that may be followed by amnesia for the content of the flashback, 3) disruptive intrusions (unrelated to traumatic material) by dissociated identity states into the individual's sense of self and agency, and 4) infrequent, full-blown changes among different identity states.

Psychotic disorders. Dissociative identity disorder may be confused with schizophrenia or other psychotic disorders. The personified, internally communicative inner voices of dissociative identity disorder, especially of a child (e.g., "I hear a little girl crying in a closet and an angry man yelling at her"), may be mistaken for psychotic hallucinations. Dissociative experiences of identity fragmentation or possession, and of perceived loss of control over thoughts, feelings, impulses, and acts, may be confused with signs of formal thought disorder, such as thought insertion or withdrawal. Individuals with dissociative identity disorder may also report visual, tactile, olfactory, gustatory, and somatic hallucinations, which are usually related to posttraumatic and dissociative factors, such as partial

flashbacks. Individuals with dissociative identity disorder experience these symptoms as caused by alternate identities, do not have delusional explanations for the phenomena, and often describe the symptoms in a personified way (e.g., "I feel like someone else wants to cry with my eyes"). Persecutory and derogatory internal voices in dissociative identity disorder associated with depressive symptoms may be misdiagnosed as major depression with psychotic features. Chaotic identity change and acute intrusions that disrupt thought processes may be distinguished from brief psychotic disorder by the predominance of dissociative symptoms and amnesia for the episode, and diagnostic evaluation after cessation of the crisis can help confirm the diagnosis.

Substance/medication-induced disorders. Symptoms associated with the physiological effects of a substance can be distinguished from dissociative identity disorder if the substance in question is judged to be etiologically related to the disturbance.

Personality disorders. Individuals with dissociative identity disorder often present identities that appear to encapsulate a variety of severe personality disorder features, suggesting a differential diagnosis of personality disorder, especially of the borderline type. Importantly, however, the individual's longitudinal variability in personality style (due to inconsistency among identities) differs from the pervasive and persistent dysfunction in affect management and interpersonal relationships typical of those with personality disorders.

Conversion disorder (functional neurological symptom disorder). This disorder may be distinguished from dissociative identity disorder by the absence of an identity disruption characterized by two or more distinct personality states or an experience of possession. Dissociative amnesia in conversion disorder is more limited and circumscribed (e.g., amnesia for a non-epileptic seizure).

Seizure disorders. Individuals with dissociative identity disorder may present with seizurelike symptoms and behaviors that resemble complex partial seizures with temporal lobe foci. These include déjà vu, jamais vu, depersonalization, derealization, out-of-body experiences, amnesia, disruptions of consciousness, hallucinations, and other intrusion phenomena of sensation, affect, and thought. Normal electroencephalographic findings, including telemetry, differentiate non-epileptic seizures from the seizurelike symptoms of dissociative identity disorder. Also, individuals with dissociative identity disorder obtain very high dissociation scores, whereas individuals with complex partial seizures do not.

Factitious disorder and malingering. Individuals who feign dissociative identity disorder do not report the subtle symptoms of intrusion characteristic of the disorder; instead they tend to overreport well-publicized symptoms of the disorder, such as dissociative amnesia, while underreporting less-publicized comorbid symptoms, such as depression. Individuals who feign dissociative identity disorder tend to be relatively undisturbed by or may even seem to enjoy "having" the disorder. In contrast, individuals with genuine dissociative identity disorder tend to be ashamed of and overwhelmed by their symptoms and to underreport their symptoms or deny their condition. Sequential observation, corroborating history, and intensive psychometric and psychological assessment may be helpful in assessment.

Individuals who malinger dissociative identity disorder usually create limited, stereotyped alternate identities, with feigned amnesia, related to the events for which gain is sought. For example, they may present an "all-good" identity and an "all-bad" identity in hopes of gaining exculpation for a crime.

Comorbidity

Many individuals with dissociative identity disorder present with a comorbid disorder. If not assessed and treated specifically for the dissociative disorder, these individuals often receive prolonged treatment for the comorbid diagnosis only, with limited overall treatment response and resultant demoralization, and disability.

Individuals with dissociative identity disorder usually exhibit a large number of comorbid disorders. In particular, most develop PTSD. Other disorders that are highly comorbid with dissociative identity disorder include depressive disorders, trauma- and stressor-related disorders, personality disorders (especially avoidant and borderline personality disorders), conversion disorder (functional neurological symptom disorder), somatic symptom disorder, eating disorders, substance-related disorders, obsessive-compulsive disorder, and sleep disorders. Dissociative alterations in identity, memory, and consciousness may affect the symptom presentation of comorbid disorders.

Dissociative Amnesia

Diagnostic Criteria **300.12 (F44.0)**

A. An inability to recall important autobiographical information, usually of a traumatic or stressful nature, that is inconsistent with ordinary forgetting.
   Note: Dissociative amnesia most often consists of localized or selective amnesia for a specific event or events; or generalized amnesia for identity and life history.
B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
C. The disturbance is not attributable to the physiological effects of a substance (e.g., alcohol or other drug of abuse, a medication) or a neurological or other medical condition (e.g., partial complex seizures, transient global amnesia, sequelae of a closed head injury/traumatic brain injury, other neurological condition).
D. The disturbance is not better explained by dissociative identity disorder, posttraumatic stress disorder, acute stress disorder, somatic symptom disorder, or major or mild neurocognitive disorder.

Coding note: The code for dissociative amnesia without dissociative fugue is **300.12 (F44.0)**. The code for dissociative amnesia with dissociative fugue is **300.13 (F44.1)**.

Specify if:

300.13 (F44.1) With dissociative fugue: Apparently purposeful travel or bewildered wandering that is associated with amnesia for identity or for other important autobiographical information.

Diagnostic Features

The defining characteristic of dissociative amnesia is an inability to recall important autobiographical information that 1) should be successfully stored in memory and 2) ordinarily would be readily remembered (Criterion A). Dissociative amnesia differs from the permanent amnesias due to neurobiological damage or toxicity that prevent memory storage or retrieval in that it is always potentially reversible because the memory has been successfully stored.

Localized amnesia, a failure to recall events during a circumscribed period of time, is the most common form of dissociative amnesia. Localized amnesia may be broader than amnesia for a single traumatic event (e.g., months or years associated with child abuse or intense combat). In *selective amnesia,* the individual can recall some, but not all, of the events during a circumscribed period of time. Thus, the individual may remember part of a traumatic event but not other parts. Some individuals report both localized and selective amnesias.

Generalized amnesia, a complete loss of memory for one's life history, is rare. Individuals with generalized amnesia may forget personal identity. Some lose previous knowledge about the world (i.e., semantic knowledge) and can no longer access well-learned skills

(i.e., procedural knowledge). Generalized amnesia has an acute onset; the perplexity, disorientation, and purposeless wandering of individuals with generalized amnesia usually bring them to the attention of the police or psychiatric emergency services. Generalized amnesia may be more common among combat veterans, sexual assault victims, and individuals experiencing extreme emotional stress or conflict.

Individuals with dissociative amnesia are frequently unaware (or only partially aware) of their memory problems. Many, especially those with localized amnesia, minimize the importance of their memory loss and may become uncomfortable when prompted to address it. In *systematized amnesia*, the individual loses memory for a specific category of information (e.g., all memories relating to one's family, a particular person, or childhood sexual abuse). In *continuous amnesia*, an individual forgets each new event as it occurs.

Associated Features Supporting Diagnosis

Many individuals with dissociative amnesia are chronically impaired in their ability to form and sustain satisfactory relationships. Histories of trauma, child abuse, and victimization are common. Some individuals with dissociative amnesia report dissociative flashbacks (i.e., behavioral reexperiencing of traumatic events). Many have a history of self-mutilation, suicide attempts, and other high-risk behaviors. Depressive and functional neurological symptoms are common, as are depersonalization, auto-hypnotic symptoms, and high hypnotizability. Sexual dysfunctions are common. Mild traumatic brain injury may precede dissociative amnesia.

Prevalence

The 12-month prevalence for dissociative amnesia among adults in a small U.S. community study was 1.8% (1.0% for males; 2.6% for females).

Development and Course

Onset of generalized amnesia is usually sudden. Less is known about the onset of localized and selective amnesias because these amnesias are seldom evident, even to the individual. Although overwhelming or intolerable events typically precede localized amnesia, its onset may be delayed for hours, days, or longer.

Individuals may report multiple episodes of dissociative amnesia. A single episode may predispose to future episodes. In between episodes of amnesia, the individual may or may not appear to be acutely symptomatic. The duration of the forgotten events can range from minutes to decades. Some episodes of dissociative amnesia resolve rapidly (e.g., when the person is removed from combat or some other stressful situation), whereas other episodes persist for long periods of time. Some individuals may gradually recall the dissociated memories years later. Dissociative capacities may decline with age, but not always. As the amnesia remits, there may be considerable distress, suicidal behavior, and symptoms of posttraumatic stress disorder (PTSD).

Dissociative amnesia has been observed in young children, adolescents, and adults. Children may be the most difficult to evaluate because they often have difficulty understanding questions about amnesia, and interviewers may find it difficult to formulate child-friendly questions about memory and amnesia. Observations of apparent dissociative amnesia are often difficult to differentiate from inattention, absorption, anxiety, oppositional behavior, and learning disorders. Reports from several different sources (e.g., teacher, therapist, case worker) may be needed to diagnose amnesia in children.

Risk and Prognostic Factors

Environmental. Single or repeated traumatic experiences (e.g., war, childhood maltreatment, natural disaster, internment in concentration camps, genocide) are common ante-

cedents. Dissociative amnesia is more likely to occur with 1) a greater number of adverse childhood experiences, particularly physical and/or sexual abuse, 2) interpersonal violence; and 3) increased severity, frequency, and violence of the trauma.

Genetic and physiological. There are no genetic studies of dissociative amnesia. Studies of dissociation report significant genetic and environmental factors in both clinical and nonclinical samples.

Course modifiers. Removal from the traumatic circumstances underlying the dissociative amnesia (e.g., combat) may bring about a rapid return of memory. The memory loss of individuals with dissociative fugue may be particularly refractory. Onset of PTSD symptoms may decrease localized, selective, or systematized amnesia. The returning memory, however, may be experienced as flashbacks that alternate with amnesia for the content of the flashbacks.

Culture-Related Diagnostic Issues

In Asia, the Middle East, and Latin America, non-epileptic seizures and other functional neurological symptoms may accompany dissociative amnesia. In cultures with highly restrictive social traditions, the precipitants of dissociative amnesia often do not involve frank trauma. Instead, the amnesia is preceded by severe psychological stresses or conflicts (e.g., marital conflict, other family disturbances, attachment problems, conflicts due to restriction or oppression).

Suicide Risk

Suicidal and other self-destructive behaviors are common in individuals with dissociative amnesia. Suicidal behavior may be a particular risk when the amnesia remits suddenly and overwhelms the individual with intolerable memories.

Functional Consequences of Dissociative Amnesia

The impairment of individuals with localized, selective, or systematized dissociative amnesia ranges from limited to severe. Individuals with chronic generalized dissociative amnesia usually have impairment in all aspects of functioning. Even when these individuals "re-learn" aspects of their life history, autobiographical memory remains very impaired. Most become vocationally and interpersonally disabled.

Differential Diagnosis

Dissociative identity disorder. Individuals with dissociative amnesia may report depersonalization and auto-hypnotic symptoms. Individuals with dissociative identity disorder report pervasive discontinuities in sense of self and agency, accompanied by many other dissociative symptoms. The amnesias of individuals with localized, selective, and/or systematized dissociative amnesias are relatively stable. Amnesias in dissociative identity disorder include amnesia for everyday events, finding of unexplained possessions, sudden fluctuations in skills and knowledge, major gaps in recall of life history, and brief amnesic gaps in interpersonal interactions.

Posttraumatic stress disorder. Some individuals with PTSD cannot recall part or all of a specific traumatic event (e.g., a rape victim with depersonalization and/or derealization symptoms who cannot recall most events for the entire day of the rape). When that amnesia extends beyond the immediate time of the trauma, a comorbid diagnosis of dissociative amnesia is warranted.

Neurocognitive disorders. In neurocognitive disorders, memory loss for personal information is usually embedded in cognitive, linguistic, affective, attentional, and behavioral

disturbances. In dissociative amnesia, memory deficits are primarily for autobiographical information; intellectual and cognitive abilities are preserved.

Substance-related disorders. In the context of repeated intoxication with alcohol or other substances/medications, there may be episodes of "black outs" or periods for which the individual has no memory. To aid in distinguishing these episodes from dissociative amnesia, a longitudinal history noting that the amnestic episodes occur only in the context of intoxication and do not occur in other situations would help identify the source as substance-induced; however the distinction may be difficult when the individual with dissociative amnesia may also misuse alcohol or other substances in the context of stressful situations that may also exacerbate dissociative symptoms. Some individuals with comorbid dissociative amnesia and substance use disorders will attribute their memory problems solely to the substance use. Prolonged use of alcohol or other substances may result in a substance-induced neurocognitive disorder that may be associated with impaired cognitive function, but in this context the protracted history of substance use and the persistent deficits associated with the neurocognitive disorder would serve to distinguish it from dissociative amnesia, where there is typically no evidence of persistent impairment in intellectual functioning.

Posttraumatic amnesia due to brain injury. Amnesia may occur in the context of a traumatic brain injury (TBI) when there has been an impact to the head or other mechanisms of rapid movement or displacement of the brain within the skull TBI. Other characteristics of TBI include loss of consciousness, disorientation and confusion, or, in more severe cases, neurological signs (e.g., abnormalities on neuroimaging, a new onset of seizures or a marked worsening of a preexisting seizure disorder, visual field cuts, anosmia). A neurocognitive disorder attributable to TBI must present either immediately after brain injury occurs or immediately after the individual recovers consciousness after the injury, and persist past the acute post-injury period. The cognitive presentation of a neurocognitive disorder following TBI is variable and includes difficulties in the domains of complex attention, executive function, learning and memory as well as slowed speed of information processing and disturbances in social cognition. These additional features help distinguish it from dissociative amnesia.

Seizure disorders. Individuals with seizure disorders may exhibit complex behavior during seizures or post-ictally with subsequent amnesia. Some individuals with a seizure disorder engage in nonpurposive wandering that is limited to the period of seizure activity. Conversely, behavior during a dissociative fugue is usually purposeful, complex, and goal-directed and may last for days, weeks, or longer. Occasionally, individuals with a seizure disorder will report that earlier autobiographical memories have been "wiped out" as the seizure disorder progresses. Such memory loss is not associated with traumatic circumstances and appears to occur randomly. Serial electroencephalograms usually show abnormalities. Telemetric electroencephalographic monitoring usually shows an association between the episodes of amnesia and seizure activity. Dissociative and epileptic amnesias may coexist.

Catatonic stupor. Mutism in catatonic stupor may suggest dissociative amnesia, but failure of recall is absent. Other catatonic symptoms (e.g., rigidity, posturing, negativism) are usually present.

Factitious disorder and malingering. There is no test, battery of tests, or set of procedures that invariably distinguishes dissociative amnesia from feigned amnesia. Individuals with factitious disorder or malingering have been noted to continue their deception even during hypnotic or barbiturate-facilitated interviews. Feigned amnesia is more common in individuals with 1) acute, florid dissociative amnesia; 2) financial, sexual, or legal problems; or 3) a wish to escape stressful circumstances. True amnesia can be associated with those same circumstances. Many individuals who malinger confess spontaneously or when confronted.

Normal and age-related changes in memory. Memory decrements in major and mild neurocognitive disorders differ from those of dissociative amnesia, which are usually associated with stressful events and are more specific, extensive, and/or complex.

Comorbidity

As dissociative amnesia begins to remit, a wide variety of affective phenomena may surface: dysphoria, grief, rage, shame, guilt, psychological conflict and turmoil, and suicidal and homicidal ideation, impulses, and acts. These individuals may have symptoms that then meet diagnostic criteria for persistent depressive disorder (dysthymia); major depressive disorder; other specified or unspecified depressive disorder; adjustment disorder, with depressed mood; or adjustment disorder, with mixed disturbance of emotions and conduct. Many individuals with dissociative amnesia develop PTSD at some point during their life, especially when the traumatic antecedents of their amnesia are brought into conscious awareness.

Many individuals with dissociative amnesia have symptoms that meet diagnostic criteria for a comorbid somatic symptom or related disorder (and vice versa), including somatic symptom disorder and conversion disorder (functional neurological symptom disorder). Many individuals with dissociative amnesia have symptoms that meet diagnostic criteria for a personality disorder, especially dependent, avoidant, and borderline.

Depersonalization/Derealization Disorder

Diagnostic Criteria **300.6 (F48.1)**

A. The presence of persistent or recurrent experiences of depersonalization, derealization, or both:
   1. **Depersonalization:** Experiences of unreality, detachment, or being an outside observer with respect to one's thoughts, feelings, sensations, body, or actions (e.g., perceptual alterations, distorted sense of time, unreal or absent self, emotional and/or physical numbing).
   2. **Derealization:** Experiences of unreality or detachment with respect to surroundings (e.g., individuals or objects are experienced as unreal, dreamlike, foggy, lifeless, or visually distorted).

B. During the depersonalization or derealization experiences, reality testing remains intact.

C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, medication) or another medical condition (e.g., seizures).

E. The disturbance is not better explained by another mental disorder, such as schizophrenia, panic disorder, major depressive disorder, acute stress disorder, posttraumatic stress disorder, or another dissociative disorder.

Diagnostic Features

The essential features of depersonalization/derealization disorder are persistent or recurrent episodes of depersonalization, derealization, or both. Episodes of depersonalization are characterized by a feeling of unreality or detachment from, or unfamiliarity with, one's whole self or from aspects of the self (Criterion A1). The individual may feel detached from his or her entire being (e.g., "I am no one," "I have no self"). He or she may also feel subjectively detached from aspects of the self, including feelings (e.g., hypoemotionality:

"I know I have feelings but I don't feel them"), thoughts (e.g., "My thoughts don't feel like my own," "head filled with cotton"), whole body or body parts, or sensations (e.g., touch, proprioception, hunger, thirst, libido). There may also be a diminished sense of agency (e.g., feeling robotic, like an automaton; lacking control of one's speech or movements). The depersonalization experience can sometimes be one of a split self, with one part observing and one participating, known as an "out-of-body experience" in its most extreme form. The unitary symptom of "depersonalization" consists of several symptom factors: anomalous body experiences (i.e., unreality of the self and perceptual alterations); emotional or physical numbing; and temporal distortions with anomalous subjective recall.

Episodes of derealization are characterized by a feeling of unreality or detachment from, or unfamiliarity with, the world, be it individuals, inanimate objects, or all surroundings (Criterion A2). The individual may feel as if he or she were in a fog, dream, or bubble, or as if there were a veil or a glass wall between the individual and world around. Surroundings may be experienced as artificial, colorless, or lifeless. Derealization is commonly accompanied by subjective visual distortions, such as blurriness, heightened acuity, widened or narrowed visual field, two-dimensionality or flatness, exaggerated three-dimensionality, or altered distance or size of objects (i.e., macropsia or micropsia). Auditory distortions can also occur, whereby voices or sounds are muted or heightened. In addition, Criterion C requires the presence of clinically significant distress or impairment in social, occupational, or other important areas of functioning, and Criteria D and E describe exclusionary diagnoses.

Associated Features Supporting Diagnosis

Individuals with depersonalization/derealization disorder may have difficulty describing their symptoms and may think they are "crazy" or "going crazy". Another common experience is the fear of irreversible brain damage. A commonly associated symptom is a subjectively altered sense of time (i.e., too fast or too slow), as well as a subjective difficulty in vividly recalling past memories and owning them as personal and emotional. Vague somatic symptoms, such as head fullness, tingling, or lightheadedness, are not uncommon. Individuals may suffer extreme rumination or obsessional preoccupation (e.g., constantly obsessing about whether they really exist, or checking their perceptions to determine whether they appear real). Varying degrees of anxiety and depression are also common associated features. Individuals with the disorder have been found to have physiological hyporeactivity to emotional stimuli. Neural substrates of interest include the hypothalamic-pituitary-adrenocortical axis, inferior parietal lobule, and prefrontal cortical-limbic circuits.

Prevalence

Transient depersonalization/derealization symptoms lasting hours to days are common in the general population. The 12-month prevalence of depersonalization/derealization disorder is thought to be markedly less than for transient symptoms, although precise estimates for the disorder are unavailable. In general, approximately one-half of all adults have experienced at least one lifetime episode of depersonalization/derealization. However, symptomatology that meets full criteria for depersonalization/derealization disorder is markedly less common than transient symptoms. Lifetime prevalence in U.S. and non-U.S. countries is approximately 2% (range of 0.8% to 2.8%). The gender ratio for the disorder is 1:1.

Development and Course

The mean age at onset of depersonalization/derealization disorder is 16 years, although the disorder can start in early or middle childhood; a minority cannot recall ever not having had

the symptoms. Less than 20% of individuals experience onset after age 20 years and only 5% after age 25 years. Onset in the fourth decade of life or later is highly unusual. Onset can range from extremely sudden to gradual. Duration of depersonalization/derealization disorder episodes can vary greatly, from brief (hours or days) to prolonged (weeks, months, or years). Given the rarity of disorder onset after age 40 years, in such cases the individual should be examined more closely for underlying medical conditions (e.g., brain lesions, seizure disorders, sleep apnea). The course of the disorder is often persistent. About one-third of cases involve discrete episodes; another third, continuous symptoms from the start; and still another third, an initially episodic course that eventually becomes continuous.

While in some individuals the intensity of symptoms can wax and wane considerably, others report an unwavering level of intensity that in extreme cases can be constantly present for years or decades. Internal and external factors that affect symptom intensity vary between individuals, yet some typical patterns are reported. Exacerbations can be triggered by stress, worsening mood or anxiety symptoms, novel or overstimulating settings, and physical factors such as lighting or lack of sleep.

Risk and Prognostic Factors

Temperamental. Individuals with depersonalization/derealization disorder are characterized by harm-avoidant temperament, immature defenses, and both disconnection and overconnection schemata. Immature defenses such as idealization/devaluation, projection and acting out result in denial of reality and poor adaptation. *Cognitive disconnection schemata* reflect defectiveness and emotional inhibition and subsume themes of abuse, neglect, and deprivation. *Overconnection schemata* involve impaired autonomy with themes of dependency, vulnerability, and incompetence.

Environmental. There is a clear association between the disorder and childhood interpersonal traumas in a substantial portion of individuals, although this association is not as prevalent or as extreme in the nature of the traumas as in other dissociative disorders, such as dissociative identity disorder. In particular, emotional abuse and emotional neglect have been most strongly and consistently associated with the disorder. Other stressors can include physical abuse; witnessing domestic violence; growing up with a seriously impaired, mentally ill parent; or unexpected death or suicide of a family member or close friend. Sexual abuse is a much less common antecedent but can be encountered. The most common proximal precipitants of the disorder are severe stress (interpersonal, financial, occupational), depression, anxiety (particularly panic attacks), and illicit drug use. Symptoms may be specifically induced by substances such as tetrahydrocannabinol, hallucinogens, ketamine, MDMA (3,4-methylenedioxymethamphetamine; "ecstasy") and salvia. Marijuana use may precipitate new-onset panic attacks and depersonalization/derealization symptoms simultaneously.

Culture-Related Diagnostic Issues

Volitionally induced experiences of depersonalization/derealization can be a part of meditative practices that are prevalent in many religions and cultures and should not be diagnosed as a disorder. However, there are individuals who initially induce these states intentionally but over time lose control over them and may develop a fear and aversion for related practices.

Functional Consequences of Depersonalization/Derealization Disorder

Symptoms of depersonalization/derealization disorder are highly distressing and are associated with major morbidity. The affectively flattened and robotic demeanor that these

individuals often demonstrate may appear incongruent with the extreme emotional pain reported by those with the disorder. Impairment is often experienced in both interpersonal and occupational spheres, largely due to the hypoemotionality with others, subjective difficulty in focusing and retaining information, and a general sense of disconnectedness from life.

Differential Diagnosis

Illness anxiety disorder. Although individuals with depersonalization/derealization disorder can present with vague somatic complaints as well as fears of permanent brain damage, the diagnosis of depersonalization/derealization disorder is characterized by the presence of a constellation of typical depersonalization/derealization symptoms and the absence of other manifestations of illness anxiety disorder.

Major depressive disorder. Feelings of numbness, deadness, apathy, and being in a dream are not uncommon in major depressive episodes. However, in depersonalization/derealization disorder, such symptoms are associated with further symptoms of the disorder. If the depersonalization/derealization clearly precedes the onset of a major depressive episode or clearly continues after its resolution, the diagnosis of depersonalization/derealization disorder applies.

Obsessive-compulsive disorder. Some individuals with depersonalization/derealization disorder can become obsessively preoccupied with their subjective experience or develop rituals checking on the status of their symptoms. However, other symptoms of obsessive-compulsive disorder unrelated to depersonalization/derealization are not present.

Other dissociative disorders. In order to diagnose depersonalization/derealization disorder, the symptoms should not occur in the context of another dissociative disorder, such as dissociative identity disorder. Differentiation from dissociative amnesia and conversion disorder (functional neurological symptom disorder) is simpler, as the symptoms of these disorders do not overlap with those of depersonalization/derealization disorder.

Anxiety disorders. Depersonalization/derealization is one of the symptoms of panic attacks, increasingly common as panic attack severity increases. Therefore, depersonalization/derealization disorder should not be diagnosed when the symptoms occur only during panic attacks that are part of panic disorder, social anxiety disorder, or specific phobia. In addition, it is not uncommon for depersonalization/derealization symptoms to first begin in the context of new-onset panic attacks or as panic disorder progresses and worsens. In such presentations, the diagnosis of depersonalization/derealization disorder can be made if 1) the depersonalization/derealization component of the presentation is very prominent from the start, clearly exceeding in duration and intensity the occurrence of actual panic attacks; or 2) the depersonalization/derealization continues after panic disorder has remitted or has been successfully treated.

Psychotic disorders. The presence of intact reality testing specifically regarding the depersonalization/derealization symptoms is essential to differentiating depersonalization/derealization disorder from psychotic disorders. Rarely, positive-symptom schizophrenia can pose a diagnostic challenge when nihilistic delusions are present. For example, an individual may complain that he or she is dead or the world is not real; this could be either a subjective experience that the individual knows is not true or a delusional conviction.

Substance/medication-induced disorders. Depersonalization/derealization associated with the physiological effects of substances during acute intoxication or withdrawal is not diagnosed as depersonalization/derealization disorder. The most common precipitating substances are the illicit drugs marijuana, hallucinogens, ketamine, ecstasy, and salvia. In

about 15% of all cases of depersonalization/derealization disorder, the symptoms are precipitated by ingestion of such substances. If the symptoms persist for some time in the absence of any further substance or medication use, the diagnosis of depersonalization/derealization disorder applies. This diagnosis is usually easy to establish since the vast majority of individuals with this presentation become highly phobic and aversive to the triggering substance and do not use it again.

Mental disorders due to another medical condition. Features such as onset after age 40 years or the presence of atypical symptoms and course in any individual suggest the possibility of an underlying medical condition. In such cases, it is essential to conduct a thorough medical and neurological evaluation, which may include standard laboratory studies, viral titers, an electroencephalogram, vestibular testing, visual testing, sleep studies, and/or brain imaging. When the suspicion of an underlying seizure disorder proves difficult to confirm, an ambulatory electroencephalogram may be indicated; although temporal lobe epilepsy is most commonly implicated, parietal and frontal lobe epilepsy may also be associated.

Comorbidity

In a convenience sample of adults recruited for a number of depersonalization research studies, lifetime comorbidities were high for unipolar depressive disorder and for any anxiety disorder, with a significant proportion of the sample having both disorders. Comorbidity with posttraumatic stress disorder was low. The three most commonly co-occurring personality disorders were avoidant, borderline, and obsessive-compulsive.

Other Specified Dissociative Disorder

300.15 (F44.89)

This category applies to presentations in which symptoms characteristic of a dissociative disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the dissociative disorders diagnostic class. The other specified dissociative disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific dissociative disorder. This is done by recording "other specified dissociative disorder" followed by the specific reason (e.g., "dissociative trance").

Examples of presentations that can be specified using the "other specified" designation include the following:

1. **Chronic and recurrent syndromes of mixed dissociative symptoms:** This category includes identity disturbance associated with less-than-marked discontinuities in sense of self and agency, or alterations of identity or episodes of possession in an individual who reports no dissociative amnesia.
2. **Identity disturbance due to prolonged and intense coercive persuasion:** Individuals who have been subjected to intense coercive persuasion (e.g., brainwashing, thought reform, indoctrination while captive, torture, long-term political imprisonment, recruitment by sects/cults or by terror organizations) may present with prolonged changes in, or conscious questioning of, their identity.
3. **Acute dissociative reactions to stressful events:** This category is for acute, transient conditions that typically last less than 1 month, and sometimes only a few hours or days. These conditions are characterized by constriction of consciousness; depersonalization; derealization; perceptual disturbances (e.g., time slowing, macropsia);

micro-amnesias; transient stupor; and/or alterations in sensory-motor functioning (e.g., analgesia, paralysis).

4. **Dissociative trance:** This condition is characterized by an acute narrowing or complete loss of awareness of immediate surroundings that manifests as profound unresponsiveness or insensitivity to environmental stimuli. The unresponsiveness may be accompanied by minor stereotyped behaviors (e.g., finger movements) of which the individual is unaware and/or that he or she cannot control, as well as transient paralysis or loss of consciousness. The dissociative trance is not a normal part of a broadly accepted collective cultural or religious practice.

Unspecified Dissociative Disorder

300.15 (F44.9)

This category applies to presentations in which symptoms characteristic of a dissociative disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the dissociative disorders diagnostic class. The unspecified dissociative disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific dissociative disorder, and includes presentations for which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Somatic Symptom and Related Disorders

Somatic symptom disorder and other disorders with prominent somatic symptoms constitute a new category in DSM-5 called *somatic symptom and related disorders.* This chapter includes the diagnoses of somatic symptom disorder, illness anxiety disorder, conversion disorder (functional neurological symptom disorder), psychological factors affecting other medical conditions, factitious disorder, other specified somatic symptom and related disorder, and unspecified somatic symptom and related disorder. All of the disorders in this chapter share a common feature: the prominence of somatic symptoms associated with significant distress and impairment. Individuals with disorders with prominent somatic symptoms are commonly encountered in primary care and other medical settings but are less commonly encountered in psychiatric and other mental health settings. These reconceptualized diagnoses, based on a reorganization of DSM-IV somatoform disorder diagnoses, are more useful for primary care and other medical (nonpsychiatric) clinicians.

The major diagnosis in this diagnostic class, somatic symptom disorder, emphasizes diagnosis made on the basis of positive symptoms and signs (distressing somatic symptoms plus abnormal thoughts, feelings, and behaviors in response to these symptoms) rather than the absence of a medical explanation for somatic symptoms. A distinctive characteristic of many individuals with somatic symptom disorder is not the somatic symptoms per se, but instead the way they present and interpret them. Incorporating affective, cognitive, and behavioral components into the criteria for somatic symptom disorder provides a more comprehensive and accurate reflection of the true clinical picture than can be achieved by assessing the somatic complaints alone.

The principles behind the changes in the somatic symptom and related diagnoses from DSM-IV are crucial in understanding the DSM-5 diagnoses. The DSM-IV term *somatoform disorders* was confusing and is replaced by *somatic symptom and related disorders.* In DSM-IV there was a great deal of overlap across the somatoform disorders and a lack of clarity about the boundaries of diagnoses. Although individuals with these disorders primarily present in medical rather than mental health settings, nonpsychiatric physicians found the DSM-IV somatoform diagnoses difficult to understand and use. The current DSM-5 classification recognizes this overlap by reducing the total number of disorders as well as their subcategories.

The previous criteria overemphasized the centrality of medically unexplained symptoms. Such symptoms are present to various degrees, particularly in conversion disorder, but somatic symptom disorders can also accompany diagnosed medical disorders. The reliability of determining that a somatic symptom is medically unexplained is limited, and grounding a diagnosis on the absence of an explanation is problematic and reinforces mind-body dualism. It is not appropriate to give an individual a mental disorder diagnosis solely because a medical cause cannot be demonstrated. Furthermore, the presence of a medical diagnosis does not exclude the possibility of a comorbid mental disorder, including a somatic symptom and related disorder. Perhaps because of the predominant focus on lack of medical explanation, individuals regarded these diagnoses as pejorative and demeaning, implying that their physical symptoms were not "real." The new classification defines the major diagnosis, somatic symptom disorder, on the basis of positive symptoms (distressing somatic symptoms plus abnormal thoughts, feelings, and behaviors in response

to these symptoms). However, medically unexplained symptoms remain a key feature in conversion disorder and pseudocyesis (other specified somatic symptom and related disorder) because it is possible to demonstrate definitively in such disorders that the symptoms are not consistent with medical pathophysiology.

It is important to note that some other mental disorders may initially manifest with primarily somatic symptoms (e.g., major depressive disorder, panic disorder). Such diagnoses may account for the somatic symptoms, or they may occur alongside one of the somatic symptom and related disorders in this chapter. There is also considerable medical comorbidity among somatizing individuals. Although somatic symptoms are frequently associated with psychological distress and psychopathology, some somatic symptom and related disorders can arise spontaneously, and their causes can remain obscure. Anxiety disorders and depressive disorders may accompany somatic symptom and related disorders. The somatic component adds severity and complexity to depressive and anxiety disorders and results in higher severity, functional impairment, and even refractoriness to traditional treatments. In rare instances, the degree of preoccupation may be so severe as to warrant consideration of a delusional disorder diagnosis.

A number of factors may contribute to somatic symptom and related disorders. These include genetic and biological vulnerability (e.g., increased sensitivity to pain), early traumatic experiences (e.g., violence, abuse, deprivation), and learning (e.g., attention obtained from illness, lack of reinforcement of nonsomatic expressions of distress), as well as cultural/social norms that devalue and stigmatize psychological suffering as compared with physical suffering. Differences in medical care across cultures affect the presentation, recognition, and management of these somatic presentations. Variations in symptom presentation are likely the result of the interaction of multiple factors within cultural contexts that affect how individuals identify and classify bodily sensations, perceive illness, and seek medical attention for them. Thus, somatic presentations can be viewed as expressions of personal suffering inserted in a cultural and social context.

All of these disorders are characterized by the prominent focus on somatic concerns and their initial presentation mainly in medical rather than mental health care settings. Somatic symptom disorder offers a more clinically useful method of characterizing individuals who may have been considered in the past for a diagnosis of somatization disorder. Furthermore, approximately 75% of individuals previously diagnosed with hypochondriasis are subsumed under the diagnosis of somatic symptom disorder. However, about 25% of individuals with hypochondriasis have high health anxiety in the absence of somatic symptoms, and many such individuals' symptoms would not qualify for an anxiety disorder diagnosis. The DSM-5 diagnosis of illness anxiety disorder is for this latter group of individuals. Illness anxiety disorder can be considered either in this diagnostic section or as an anxiety disorder. Because of the strong focus on somatic concerns, and because illness anxiety disorder is most often encountered in medical settings, for utility it is listed with the somatic symptom and related disorders. In conversion disorder, the essential feature is neurological symptoms that are found, after appropriate neurological assessment, to be incompatible with neurological pathophysiology. Psychological factors affecting other medical conditions is also included in this chapter. Its essential feature is the presence of one or more clinically significant psychological or behavioral factors that adversely affect a medical condition by increasing the risk for suffering, death, or disability. Like the other somatic symptom and related disorders, factitious disorder embodies persistent problems related to illness perception and identity. In the great majority of reported cases of factitious disorder, both imposed on self and imposed on another, individuals present with somatic symptoms and medical disease conviction. Consequently, DSM-5 factitious disorder is included among the somatic symptom and related disorders. Other specified somatic symptom and related disorder and unspecified somatic symptom and related disorder include conditions for which some, but not all, of the criteria for somatic symptom disorder or illness anxiety disorder are met, as well as pseudocyesis.

Somatic Symptom Disorder

Diagnostic Criteria **300.82 (F45.1)**

A. One or more somatic symptoms that are distressing or result in significant disruption of daily life.

B. Excessive thoughts, feelings, or behaviors related to the somatic symptoms or associated health concerns as manifested by at least one of the following:

1. Disproportionate and persistent thoughts about the seriousness of one's symptoms.
2. Persistently high level of anxiety about health or symptoms.
3. Excessive time and energy devoted to these symptoms or health concerns.

C. Although any one somatic symptom may not be continuously present, the state of being symptomatic is persistent (typically more than 6 months).

Specify if:

With predominant pain (previously pain disorder): This specifier is for individuals whose somatic symptoms predominantly involve pain.

Specify if:

Persistent: A persistent course is characterized by severe symptoms, marked impairment, and long duration (more than 6 months).

Specify current severity:

Mild: Only one of the symptoms specified in Criterion B is fulfilled.

Moderate: Two or more of the symptoms specified in Criterion B are fulfilled.

Severe: Two or more of the symptoms specified in Criterion B are fulfilled, plus there are multiple somatic complaints (or one very severe somatic symptom).

Diagnostic Features

Individuals with somatic symptom disorder typically have multiple, current, somatic symptoms that are distressing or result in significant disruption of daily life (Criterion A), although sometimes only one severe symptom, most commonly pain, is present. Symptoms may be specific (e.g., localized pain) or relatively nonspecific (e.g., fatigue). The symptoms sometimes represent normal bodily sensations or discomfort that does not generally signify serious disease. Somatic symptoms without an evident medical explanation are not sufficient to make this diagnosis. The individual's suffering is authentic, whether or not it is medically explained.

The symptoms may or may not be associated with another medical condition. The diagnoses of somatic symptom disorder and a concurrent medical illness are not mutually exclusive, and these frequently occur together. For example, an individual may become seriously disabled by symptoms of somatic symptom disorder after an uncomplicated myocardial infarction even if the myocardial infarction itself did not result in any disability. If another medical condition or high risk for developing one is present (e.g., strong family history), the thoughts, feelings, and behaviors associated with this condition are excessive (Criterion B).

Individuals with somatic symptom disorder tend to have very high levels of worry about illness (Criterion B). They appraise their bodily symptoms as unduly threatening, harmful, or troublesome and often think the worst about their health. Even when there is evidence to the contrary, some patients still fear the medical seriousness of their symptoms. In severe somatic symptom disorder, health concerns may assume a central role in the individual's life, becoming a feature of his or her identity and dominating interpersonal relationships.

Individuals typically experience distress that is principally focused on somatic symptoms and their significance. When asked directly about their distress, some individuals describe it in relation to other aspects of their lives, while others deny any source of distress other than the somatic symptoms. Health-related quality of life is often impaired, both physically and mentally. In severe somatic symptom disorder, the impairment is marked, and when persistent, the disorder can lead to invalidism.

There is often a high level of medical care utilization, which rarely alleviates the individual's concerns. Consequently, the patient may seek care from multiple doctors for the same symptoms. These individuals often seem unresponsive to medical interventions, and new interventions may only exacerbate the presenting symptoms. Some individuals with the disorder seem unusually sensitive to medication side effects. Some feel that their medical assessment and treatment have been inadequate.

Associated Features Supporting Diagnosis

Cognitive features include attention focused on somatic symptoms, attribution of normal bodily sensations to physical illness (possibly with catastrophic interpretations), worry about illness, and fear that any physical activity may damage the body. The relevant associated behavioral features may include repeated bodily checking for abnormalities, repeated seeking of medical help and reassurance, and avoidance of physical activity. These behavioral features are most pronounced in severe, persistent somatic symptom disorder. These features are usually associated with frequent requests for medical help for different somatic symptoms. This may lead to medical consultations in which individuals are so focused on their concerns about somatic symptom(s) that they cannot be redirected to other matters. Any reassurance by the doctor that the symptoms are not indicative of serious physical illness tends to be short-lived and/or is experienced by the individuals as the doctor not taking their symptoms with due seriousness. As the focus on somatic symptoms is a primary feature of the disorder, individuals with somatic symptom disorder typically present to general medical health services rather than mental health services. The suggestion of referral to a mental health specialist may be met with surprise or even frank refusal by individuals with somatic symptom disorder.

Since somatic symptom disorder is associated with depressive disorders, there is an increased suicide risk. It is not known whether somatic symptom disorder is associated with suicide risk independent of its association with depressive disorders.

Prevalence

The prevalence of somatic symptom disorder is not known. However, the prevalence of somatic symptom disorder is expected to be higher than that of the more restrictive DSM-IV somatization disorder (<1%) but lower than that of undifferentiated somatoform disorder (approximately 19%). The prevalence of somatic symptom disorder in the general adult population may be around 5%–7%. Females tend to report more somatic symptoms than do males, and the prevalence of somatic symptom disorder is consequently likely to be higher in females.

Development and Course

In older individuals, somatic symptoms and concurrent medical illnesses are common, and a focus on Criterion B is crucial for making the diagnosis. Somatic symptom disorder may be underdiagnosed in older adults either because certain somatic symptoms (e.g., pain, fatigue) are considered part of normal aging or because illness worry is considered "understandable" in older adults who have more general medical illnesses and medications than do younger people. Concurrent depressive disorder is common in older people who present with numerous somatic symptoms.

In children, the most common symptoms are recurrent abdominal pain, headache, fatigue, and nausea. A single prominent symptom is more common in children than in adults. While young children may have somatic complaints, they rarely worry about "illness" per se prior to adolescence. The parents' response to the symptom is important, as this may determine the level of associated distress. It is the parent who may determine the interpretation of symptoms and the associated time off school and medical help seeking.

Risk and Prognostic Factors

Temperamental. The personality trait of negative affectivity (neuroticism) has been identified as an independent correlate/risk factor of a high number of somatic symptoms. Comorbid anxiety or depression is common and may exacerbate symptoms and impairment.

Environmental. Somatic symptom disorder is more frequent in individuals with few years of education and low socioeconomic status, and in those who have recently experienced stressful life events.

Course modifiers. Persistent somatic symptoms are associated with demographic features (female sex, older age, fewer years of education, lower socioeconomic status, unemployment), a reported history of sexual abuse or other childhood adversity, concurrent chronic physical illness or psychiatric disorder (depression, anxiety, persistent depressive disorder [dysthymia], panic), social stress, and reinforcing social factors such as illness benefits. Cognitive factors that affect clinical course include sensitization to pain, heightened attention to bodily sensations, and attribution of bodily symptoms to a possible medical illness rather than recognizing them as a normal phenomenon or psychological stress.

Culture-Related Diagnostic Issues

Somatic symptoms are prominent in various "culture-bound syndromes." High numbers of somatic symptoms are found in population-based and primary care studies around the world, with a similar pattern of the most commonly reported somatic symptoms, impairment, and treatment seeking. The relationship between number of somatic symptoms and illness worry is similar in different cultures, and marked illness worry is associated with impairment and greater treatment seeking across cultures. The relationship between numerous somatic symptoms and depression appears to be very similar around the world and between different cultures within one country.

Despite these similarities, there are differences in somatic symptoms among cultures and ethnic groups. The description of somatic symptoms varies with linguistic and other local cultural factors. These somatic presentations have been described as "idioms of distress" because somatic symptoms may have special meanings and shape patient-clinician interactions in the particular cultural contexts. "Burnout," the sensation of heaviness or the complaints of "gas"; too much heat in the body; or burning in the head are examples of symptoms that are common in some cultures or ethnic groups but rare in others. Explanatory models also vary, and somatic symptoms may be attributed variously to particular family, work, or environmental stresses; general medical illness; the suppression of feelings of anger and resentment; or certain culture-specific phenomena, such as semen loss. There may also be differences in medical treatment seeking among cultural groups, in addition to differences due to variable access to medical care services. Seeking treatment for multiple somatic symptoms in general medical clinics is a worldwide phenomenon and occurs at similar rates among ethnic groups in the same country.

Functional Consequences of Somatic Symptom Disorder

The disorder is associated with marked impairment of health status. Many individuals with severe somatic symptom disorder are likely to have impaired health status scores more than 2 standard deviations below population norms.

Differential Diagnosis

If the somatic symptoms are consistent with another mental disorder (e.g., panic disorder), and the diagnostic criteria for that disorder are fulfilled, then that mental disorder should be considered as an alternative or additional diagnosis. A separate diagnosis of somatic symptom disorder is not made if the somatic symptoms and related thoughts, feelings, or behaviors occur only during major depressive episodes. If, as commonly occurs, the criteria for both somatic symptom disorder and another mental disorder diagnosis are fulfilled, then both should be coded, as both may require treatment.

Other medical conditions. The presence of somatic symptoms of unclear etiology is not in itself sufficient to make the diagnosis of somatic symptom disorder. The symptoms of many individuals with disorders like irritable bowel syndrome or fibromyalgia would not satisfy the criterion necessary to diagnose somatic symptom disorder (Criterion B). Conversely, the presence of somatic symptoms of an established medical disorder (e.g., diabetes or heart disease) does not exclude the diagnosis of somatic symptom disorder if the criteria are otherwise met.

Panic disorder. In panic disorder, somatic symptoms and anxiety about health tend to occur in acute episodes, whereas in somatic symptom disorder, anxiety and somatic symptoms are more persistent.

Generalized anxiety disorder. Individuals with generalized anxiety disorder worry about multiple events, situations, or activities, only one of which may involve their health. The main focus is not usually somatic symptoms or fear of illness as it is in somatic symptom disorder.

Depressive disorders. Depressive disorders are commonly accompanied by somatic symptoms. However, depressive disorders are differentiated from somatic symptom disorder by the core depressive symptoms of low (dysphoric) mood and anhedonia.

Illness anxiety disorder. If the individual has extensive worries about health but no or minimal somatic symptoms, it may be more appropriate to consider illness anxiety disorder.

Conversion disorder (functional neurological symptom disorder). In conversion disorder, the presenting symptom is loss of function (e.g., of a limb), whereas in somatic symptom disorder, the focus is on the distress that particular symptoms cause. The features listed under Criterion B of somatic symptom disorder may be helpful in differentiating the two disorders.

Delusional disorder. In somatic symptom disorder, the individual's beliefs that somatic symptoms might reflect serious underlying physical illness are not held with delusional intensity. Nonetheless, the individual's beliefs concerning the somatic symptoms can be firmly held. In contrast, in delusional disorder, somatic subtype, the somatic symptom beliefs and behavior are stronger than those found in somatic symptom disorder.

Body dysmorphic disorder. In body dysmorphic disorder, the individual is excessively concerned about, and preoccupied by, a perceived defect in his or her physical features. In contrast, in somatic symptom disorder, the concern about somatic symptoms reflects fear of underlying illness, not of a defect in appearance.

Obsessive-compulsive disorder. In somatic symptom disorder, the recurrent ideas about somatic symptoms or illness are less intrusive, and individuals with this disorder do not exhibit the associated repetitive behaviors aimed at reducing anxiety that occur in obsessive-compulsive disorder.

Comorbidity

Somatic symptom disorder is associated with high rates of comorbidity with medical disorders as well as anxiety and depressive disorders. When a concurrent medical illness is

present, the degree of impairment is more marked than would be expected from the physical illness alone. When an individual's symptoms meet diagnostic criteria for somatic symptom disorder, the disorder should be diagnosed; however, in view of the frequent comorbidity, especially with anxiety and depressive disorders, evidence for these concurrent diagnoses should be sought.

Illness Anxiety Disorder

Diagnostic Criteria 300.7 (F45.21)

A. Preoccupation with having or acquiring a serious illness.

B. Somatic symptoms are not present or, if present, are only mild in intensity. If another medical condition is present or there is a high risk for developing a medical condition (e.g., strong family history is present), the preoccupation is clearly excessive or disproportionate.

C. There is a high level of anxiety about health, and the individual is easily alarmed about personal health status.

D. The individual performs excessive health-related behaviors (e.g., repeatedly checks his or her body for signs of illness) or exhibits maladaptive avoidance (e.g., avoids doctor appointments and hospitals).

E. Illness preoccupation has been present for at least 6 months, but the specific illness that is feared may change over that period of time.

F. The illness-related preoccupation is not better explained by another mental disorder, such as somatic symptom disorder, panic disorder, generalized anxiety disorder, body dysmorphic disorder, obsessive-compulsive disorder, or delusional disorder, somatic type.

Specify whether:

Care-seeking type: Medical care, including physician visits or undergoing tests and procedures, is frequently used.

Care-avoidant type: Medical care is rarely used.

Diagnostic Features

Most individuals with hypochondriasis are now classified as having somatic symptom disorder; however, in a minority of cases, the diagnosis of illness anxiety disorder applies instead. Illness anxiety disorder entails a preoccupation with having or acquiring a serious, undiagnosed medical illness (Criterion A). Somatic symptoms are not present or, if present, are only mild in intensity (Criterion B). A thorough evaluation fails to identify a serious medical condition that accounts for the individual's concerns. While the concern may be derived from a nonpathological physical sign or sensation, the individual's distress emanates not primarily from the physical complaint itself but rather from his or her anxiety about the meaning, significance, or cause of the complaint (i.e., the suspected medical diagnosis). If a physical sign or symptom is present, it is often a normal physiological sensation (e.g., orthostatic dizziness), a benign and self-limited dysfunction (e.g., transient tinnitus), or a bodily discomfort not generally considered indicative of disease (e.g., belching). If a diagnosable medical condition is present, the individual's anxiety and preoccupation are clearly excessive and disproportionate to the severity of the condition (Criterion B). Empirical evidence and existing literature pertain to previously defined DSM hypochondriasis, and it is unclear to what extent and how precisely they apply to the description of this new diagnosis.

The preoccupation with the idea that one is sick is accompanied by substantial anxiety about health and disease (Criterion C). Individuals with illness anxiety disorder are easily

alarmed about illness, such as by hearing about someone else falling ill or reading a health-related news story. Their concerns about undiagnosed disease do not respond to appropriate medical reassurance, negative diagnostic tests, or benign course. The physician's attempts at reassurance and symptom palliation generally do not alleviate the individual's concerns and may heighten them. Illness concerns assume a prominent place in the individual's life, affecting daily activities, and may even result in invalidism. Illness becomes a central feature of the individual's identity and self-image, a frequent topic of social discourse, and a characteristic response to stressful life events. Individuals with the disorder often examine themselves repeatedly (e.g., examining one's throat in the mirror) (Criterion D). They research their suspected disease excessively (e.g., on the Internet) and repeatedly seek reassurance from family, friends, or physicians. This incessant worrying often becomes frustrating for others and may result in considerable strain within the family. In some cases, the anxiety leads to maladaptive avoidance of situations (e.g., visiting sick family members) or activities (e.g., exercise) that these individuals fear might jeopardize their health.

Associated Features Supporting Diagnosis

Because they believe they are medically ill, individuals with illness anxiety disorder are encountered far more frequently in medical than in mental health settings. The majority of individuals with illness anxiety disorder have extensive yet unsatisfactory medical care, though some may be too anxious to seek medical attention. They generally have elevated rates of medical utilization but do not utilize mental health services more than the general population. They often consult multiple physicians for the same problem and obtain repeatedly negative diagnostic test results. At times, medical attention leads to a paradoxical exacerbation of anxiety or to iatrogenic complications from diagnostic tests and procedures. Individuals with the disorder are generally dissatisfied with their medical care and find it unhelpful, often feeling they are not being taken seriously by physicians. At times, these concerns may be justified, since physicians sometimes are dismissive or respond with frustration or hostility. This response can occasionally result in a failure to diagnose a medical condition that is present.

Prevalence

Prevalence estimates of illness anxiety disorder are based on estimates of the DSM-III and DSM-IV diagnosis *hypochondriasis*. The 1- to 2-year prevalence of health anxiety and/or disease conviction in community surveys and population-based samples ranges from 1.3% to 10%. In ambulatory medical populations, the 6-month/1-year prevalence rates are between 3% and 8%. The prevalence of the disorder is similar in males and females.

Development and Course

The development and course of illness anxiety disorder are unclear. Illness anxiety disorder is generally thought to be a chronic and relapsing condition with an age at onset in early and middle adulthood. In population-based samples, health-related anxiety increases with age, but the ages of individuals with high health anxiety in medical settings do not appear to differ from those of other patients in those settings. In older individuals, health-related anxiety often focuses on memory loss; the disorder is thought to be rare in children.

Risk and Prognostic Factors

Environmental. Illness anxiety disorder may sometimes be precipitated by a major life stress or a serious but ultimately benign threat to the individual's health. A history of child-

hood abuse or of a serious childhood illness may predispose to development of the disorder in adulthood.

Course modifiers. Approximately one-third to one-half of individuals with illness anxiety disorder have a transient form, which is associated with less psychiatric comorbidity, more medical comorbidity, and less severe illness anxiety disorder.

Culture-Related Diagnostic Issues

The diagnosis should be made with caution in individuals whose ideas about disease are congruent with widely held, culturally sanctioned beliefs. Little is known about the phenomenology of the disorder across cultures, although the prevalence appears to be similar across different countries with diverse cultures.

Functional Consequences of Illness Anxiety Disorder

Illness anxiety disorder causes substantial role impairment and decrements in physical function and health-related quality of life. Health concerns often interfere with interpersonal relationships, disrupt family life, and damage occupational performance.

Differential Diagnosis

Other medical conditions. The first differential diagnostic consideration is an underlying medical condition, including neurological or endocrine conditions, occult malignancies, and other diseases that affect multiple body systems. The presence of a medical condition does not rule out the possibility of coexisting illness anxiety disorder. If a medical condition is present, the health-related anxiety and disease concerns are clearly disproportionate to its seriousness. Transient preoccupations related to a medical condition do not constitute illness anxiety disorder.

Adjustment disorders. Health-related anxiety is a normal response to serious illness and is not a mental disorder. Such nonpathological health anxiety is clearly related to the medical condition and is typically time-limited. If the health anxiety is severe enough, an adjustment disorder may be diagnosed. However, only when the health anxiety is of sufficient duration, severity, and distress can illness anxiety disorder be diagnosed. Thus, the diagnosis requires the continuous persistence of disproportionate health-related anxiety for at least 6 months.

Somatic symptom disorder. Somatic symptom disorder is diagnosed when significant somatic symptoms are present. In contrast, individuals with illness anxiety disorder have minimal somatic symptoms and are primarily concerned with the idea they are ill.

Anxiety disorders. In generalized anxiety disorder, individuals worry about multiple events, situations, or activities, only one of which may involve health. In panic disorder, the individual may be concerned that the panic attacks reflect the presence of a medical illness; however, although these individuals may have health anxiety, their anxiety is typically very acute and episodic. In illness anxiety disorder, the health anxiety and fears are more persistent and enduring. Individuals with illness anxiety disorder may experience panic attacks that are triggered by their illness concerns.

Obsessive-compulsive and related disorders. Individuals with illness anxiety disorder may have intrusive thoughts about having a disease and also may have associated compulsive behaviors (e.g., seeking reassurance). However, in illness anxiety disorder, the preoccupations are usually focused on having a disease, whereas in obsessive-compulsive disorder (OCD), the thoughts are intrusive and are usually focused on fears of getting a disease in the future. Most individuals with OCD have obsessions or compulsions involving other concerns in addition to fears about contracting disease. In body dysmorphic dis-

order, concerns are limited to the individual's physical appearance, which is viewed as defective or flawed.

Major depressive disorder. Some individuals with a major depressive episode ruminate about their health and worry excessively about illness. A separate diagnosis of illness anxiety disorder is not made if these concerns occur only during major depressive episodes. However, if excessive illness worry persists after remission of an episode of major depressive disorder, the diagnosis of illness anxiety disorder should be considered.

Psychotic disorders. Individuals with illness anxiety disorder are not delusional and can acknowledge the possibility that the feared disease is not present. Their ideas do not attain the rigidity and intensity seen in the somatic delusions occurring in psychotic disorders (e.g., schizophrenia; delusional disorder, somatic type; major depressive disorder, with psychotic features). True somatic delusions are generally more bizarre (e.g., that an organ is rotting or dead) than the concerns seen in illness anxiety disorder. The concerns seen in illness anxiety disorder, though not founded in reality, are plausible.

Comorbidity

Because illness anxiety disorder is a new disorder, exact comorbidities are unknown. Hypochondriasis co-occurs with anxiety disorders (in particular, generalized anxiety disorder, panic disorder, and OCD) and depressive disorders. Approximately two-thirds of individuals with illness anxiety disorder are likely to have at least one other comorbid major mental disorder. Individuals with illness anxiety disorder may have an elevated risk for somatic symptom disorder and personality disorders.

Conversion Disorder (Functional Neurological Symptom Disorder)

Diagnostic Criteria

A. One or more symptoms of altered voluntary motor or sensory function.

B. Clinical findings provide evidence of incompatibility between the symptom and recognized neurological or medical conditions.

C. The symptom or deficit is not better explained by another medical or mental disorder.

D. The symptom or deficit causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or warrants medical evaluation.

Coding note: The ICD-9-CM code for conversion disorder is **300.11,** which is assigned regardless of the symptom type. The ICD-10-CM code depends on the symptom type (see below).

Specify symptom type:

(F44.4) With weakness or paralysis

(F44.4) With abnormal movement (e.g., tremor, dystonic movement, myoclonus, gait disorder)

(F44.4) With swallowing symptoms

(F44.4) With speech symptom (e.g., dysphonia, slurred speech)

(F44.5) With attacks or seizures

(F44.6) With anesthesia or sensory loss

(F44.6) With special sensory symptom (e.g., visual, olfactory, or hearing disturbance)

(F44.7) With mixed symptoms

Specify if:

Acute episode: Symptoms present for less than 6 months.

Persistent: Symptoms occurring for 6 months or more.

Specify if:

With psychological stressor *(specify stressor)*

Without psychological stressor

Diagnostic Features

Many clinicians use the alternative names of "functional" (referring to abnormal central nervous system functioning) or "psychogenic" (referring to an assumed etiology) to describe the symptoms of conversion disorder (functional neurological symptom disorder). In conversion disorder, there may be one or more symptoms of various types. Motor symptoms include weakness or paralysis; abnormal movements, such as tremor or dystonic movements; gait abnormalities; and abnormal limb posturing. Sensory symptoms include altered, reduced, or absent skin sensation, vision, or hearing. Episodes of abnormal generalized limb shaking with apparent impaired or loss of consciousness may resemble epileptic seizures (also called *psychogenic* or *non-epileptic seizures*). There may be episodes of unresponsiveness resembling syncope or coma. Other symptoms include reduced or absent speech volume (dysphonia/aphonia), altered articulation (dysarthria), a sensation of a lump in the throat (globus), and diplopia.

Although the diagnosis requires that the symptom is not explained by neurological disease, it should not be made simply because results from investigations are normal or because the symptom is "bizarre." There must be clinical findings that show clear evidence of incompatibility with neurological disease. Internal inconsistency at examination is one way to demonstrate incompatibility (i.e., demonstrating that physical signs elicited through one examination method are no longer positive when tested a different way). Examples of such examination findings include

- Hoover's sign, in which weakness of hip extension returns to normal strength with contralateral hip flexion against resistance.
- Marked weakness of ankle plantar-flexion when tested on the bed in an individual who is able to walk on tiptoes;
- Positive findings on the tremor entrainment test. On this test, a unilateral tremor may be identified as functional if the tremor changes when the individual is distracted away from it. This may be observed if the individual is asked to copy the examiner in making a rhythmical movement with their unaffected hand and this causes the functional tremor to change such that it copies or "entrains" to the rhythm of the unaffected hand or the functional tremor is suppressed, or no longer makes a simple rhythmical movement.
- In attacks resembling epilepsy or syncope ("psychogenic" non-epileptic attacks), the occurrence of closed eyes with resistance to opening or a normal simultaneous electroencephalogram (although this alone does not exclude all forms of epilepsy or syncope).
- For visual symptoms, a tubular visual field (i.e., tunnel vision).

It is important to note that the diagnosis of conversion disorder should be based on the overall clinical picture and not on a single clinical finding.

Associated Features Supporting Diagnosis

A number of associated features can support the diagnosis of conversion disorder. There may be a history of multiple similar somatic symptoms. Onset may be associated with stress or trauma, either psychological or physical in nature. The potential etiological rele-

vance of this stress or trauma may be suggested by a close temporal relationship. However, while assessment for stress and trauma is important, the diagnosis should not be withheld if none is found.

Conversion disorder is often associated with dissociative symptoms, such as depersonalization, derealization, and dissociative amnesia, particularly at symptom onset or during attacks.

The diagnosis of conversion disorder does not require the judgment that the symptoms are not intentionally produced (i.e., not feigned), as the definite absence of feigning may not be reliably discerned. The phenomenon of *la belle indifférence* (i.e., lack of concern about the nature or implications of the symptom) has been associated with conversion disorder but it is not specific for conversion disorder and should not be used to make the diagnosis. Similarly the concept of *secondary gain* (i.e., when individuals derive external benefits such as money or release from responsibilities) is also not specific to conversion disorder and particularly in the context of definite evidence for feigning, the diagnoses that should be considered instead would include factitious disorder or malingering (see the section "Differential Diagnosis" for this disorder).

Prevalence

Transient conversion symptoms are common, but the precise prevalence of the disorder is unknown. This is partly because the diagnosis usually requires assessment in secondary care, where it is found in approximately 5% of referrals to neurology clinics. The incidence of individual persistent conversion symptoms is estimated to be 2–5/100,000 per year.

Development and Course

Onset has been reported throughout the life course. The onset of non-epileptic attacks peaks in the third decade, and motor symptoms have their peak onset in the fourth decade. The symptoms can be transient or persistent. The prognosis may be better in younger children than in adolescents and adults.

Risk and Prognostic Factors

Temperamental. Maladaptive personality traits are commonly associated with conversion disorder.

Environmental. There may be a history of childhood abuse and neglect. Stressful life events are often, but not always, present.

Genetic and physiological. The presence of neurological disease that causes similar symptoms is a risk factor (e.g., non-epileptic seizures are more common in patients who also have epilepsy).

Course modifiers. Short duration of symptoms and acceptance of the diagnosis are positive prognostic factors. Maladaptive personality traits, the presence of comorbid physical disease, and the receipt of disability benefits may be negative prognostic factors.

Culture-Related Diagnostic Issues

Changes resembling conversion (and dissociative) symptoms are common in certain culturally sanctioned rituals. If the symptoms are fully explained within the particular cultural context and do not result in clinically significant distress or disability, then the diagnosis of conversion disorder is not made.

Gender-Related Diagnostic Issues

Conversion disorder is two to three times more common in females.

Functional Consequences of Conversion Disorder

Individuals with conversion symptoms may have substantial disability. The severity of disability can be similar to that experienced by individuals with comparable medical diseases.

Differential Diagnosis

If another mental disorder better explains the symptoms, that diagnosis should be made. However the diagnosis of conversion disorder may be made in the presence of another mental disorder.

Neurological disease. The main differential diagnosis is neurological disease that might better explain the symptoms. After a thorough neurological assessment, an unexpected neurological disease cause for the symptoms is rarely found at follow up. However, reassessment may be required if the symptoms appear to be progressive. Conversion disorder may coexist with neurological disease.

Somatic symptom disorder. Conversion disorder may be diagnosed in addition to somatic symptom disorder. Most of the somatic symptoms encountered in somatic symptom disorder cannot be demonstrated to be clearly incompatible with pathophysiology (e.g., pain, fatigue), whereas in conversion disorder, such incompatibility is required for the diagnosis. The excessive thoughts, feelings, and behaviors characterizing somatic symptom disorder are often absent in conversion disorder.

Factitious disorder and malingering. The diagnosis of conversion disorder does not require the judgment that the symptoms are *not* intentionally produced (i.e., not feigned), because assessment of conscious intention is unreliable. However definite evidence of feigning (e.g., clear evidence that loss of function is present during the examination but not at home) would suggest a diagnosis of factitious disorder if the individual's apparent aim is to assume the sick role or malingering if the aim is to obtain an incentive such as money.

Dissociative disorders. Dissociative symptoms are common in individuals with conversion disorder. If both conversion disorder and a dissociative disorder are present, both diagnoses should be made.

Body dysmorphic disorder. Individuals with body dysmorphic disorder are excessively concerned about a perceived defect in their physical features but do not complain of symptoms of sensory or motor functioning in the affected body part.

Depressive disorders. In depressive disorders, individuals may report general heaviness of their limbs, whereas the weakness of conversion disorder is more focal and prominent. Depressive disorders are also differentiated by the presence of core depressive symptoms.

Panic disorder. Episodic neurological symptoms (e.g., tremors and paresthesias) can occur in both conversion disorder and panic attacks. In panic attacks, the neurological symptoms are typically transient and acutely episodic with characteristic cardiorespiratory symptoms. Loss of awareness with amnesia for the attack and violent limb movements occur in non-epileptic attacks, but not in panic attacks.

Comorbidity

Anxiety disorders, especially panic disorder, and depressive disorders commonly co-occur with conversion disorder. Somatic symptom disorder may co-occur as well. Psychosis, substance use disorder, and alcohol misuse are uncommon. Personality disorders are more common in individuals with conversion disorder than in the general population. Neurological or other medical conditions commonly coexist with conversion disorder as well.

Psychological Factors Affecting Other Medical Conditions

Diagnostic Criteria **316 (F54)**

A. A medical symptom or condition (other than a mental disorder) is present.

B. Psychological or behavioral factors adversely affect the medical condition in one of the following ways:

1. The factors have influenced the course of the medical condition as shown by a close temporal association between the psychological factors and the development or exacerbation of, or delayed recovery from, the medical condition.
2. The factors interfere with the treatment of the medical condition (e.g., poor adherence).
3. The factors constitute additional well-established health risks for the individual.
4. The factors influence the underlying pathophysiology, precipitating or exacerbating symptoms or necessitating medical attention.

C. The psychological and behavioral factors in Criterion B are not better explained by another mental disorder (e.g., panic disorder, major depressive disorder, posttraumatic stress disorder).

Specify current severity:

Mild: Increases medical risk (e.g., inconsistent adherence with antihypertension treatment).

Moderate: Aggravates underlying medical condition (e.g., anxiety aggravating asthma).

Severe: Results in medical hospitalization or emergency room visit.

Extreme: Results in severe, life-threatening risk (e.g., ignoring heart attack symptoms).

Diagnostic Features

The essential feature of psychological factors affecting other medical conditions is the presence of one or more clinically significant psychological or behavioral factors that adversely affect a medical condition by increasing the risk for suffering, death, or disability (Criterion B). These factors can adversely affect the medical condition by influencing its course or treatment, by constituting an additional well-established health risk factor, or by influencing the underlying pathophysiology to precipitate or exacerbate symptoms or to necessitate medical attention.

Psychological or behavioral factors include psychological distress, patterns of interpersonal interaction, coping styles, and maladaptive health behaviors, such as denial of symptoms or poor adherence to medical recommendations. Common clinical examples are anxiety-exacerbating asthma, denial of need for treatment for acute chest pain, and manipulation of insulin by an individual with diabetes wishing to lose weight. Many different psychological factors have been demonstrated to adversely influence medical conditions—for example, symptoms of depression or anxiety, stressful life events, relationship style, personality traits, and coping styles. The adverse effects can range from acute, with immediate medical consequences (e.g., Takotsubo cardiomyopathy) to chronic, occurring over a long period of time (e.g., chronic occupational stress increasing risk for hypertension). Affected medical conditions can be those with clear pathophysiology (e.g., diabetes, cancer, coronary disease), functional syndromes (e.g., migraine, irritable bowel syndrome, fibromyalgia), or idiopathic medical symptoms (e.g., pain, fatigue, dizziness).

This diagnosis should be reserved for situations in which the effect of the psychological factor on the medical condition is evident and the psychological factor has clinically significant effects on the course or outcome of the medical condition. Abnormal psychological or behavioral symptoms that develop in response to a medical condition are more properly coded as an adjustment disorder (a clinically significant psychological response to an identifiable stressor). There must be reasonable evidence to suggest an association between the psychological factors and the medical condition, although it may often not be possible to demonstrate direct causality or the mechanisms underlying the relationship.

Prevalence

The prevalence of psychological factors affecting other medical conditions is unclear. In U.S. private insurance billing data, it is a more common diagnosis than somatic symptom disorders.

Development and Course

Psychological factors affecting other medical conditions can occur across the lifespan. Particularly with young children, corroborative history from parents or school can assist the diagnostic evaluation. Some conditions are characteristic of particular life stages (e.g., in older individuals, the stress associated with acting as a caregiver for an ill spouse or partner).

Culture-Related Diagnostic Issues

Many differences between cultures may influence psychological factors and their effects on medical conditions, such as those in language and communication style, explanatory models of illness, patterns of seeking health care, service availability and organization, doctor-patient relationships and other healing practices, family and gender roles, and attitudes toward pain and death. Psychological factors affecting other medical conditions must be differentiated from culturally specific behaviors such as using faith or spiritual healers or other variations in illness management that are acceptable within a culture and represent an attempt to help the medical condition rather than interfere with it. These local practices may complement rather than obstruct evidence-based interventions. If they do not adversely affect outcomes, they should not be pathologized as psychological factors affecting other medical conditions.

Functional Consequences of Psychological Factors Affecting Other Medical Conditions

Psychological and behavioral factors have been demonstrated to affect the course of many medical diseases.

Differential Diagnosis

Mental disorder due to another medical condition. A temporal association between symptoms of a mental disorder and those of a medical condition is also characteristic of a mental disorder due to another medical condition, but the presumed causality is in the opposite direction. In a mental disorder due to another medical condition, the medical condition is judged to be causing the mental disorder through a direct physiological mechanism. In psychological factors affecting other medical conditions, the psychological or behavioral factors are judged to affect the course of the medical condition.

Adjustment disorders. Abnormal psychological or behavioral symptoms that develop in response to a medical condition are more properly coded as an adjustment disorder (a clinically significant psychological response to an identifiable stressor). For example, an indi-

vidual with angina that is precipitated whenever he becomes enraged would be diagnosed as having psychological factors affecting other medical conditions, whereas an individual with angina who developed maladaptive anticipatory anxiety would be diagnosed as having an adjustment disorder with anxiety. In clinical practice, however, psychological factors and a medical condition are often mutually exacerbating (e.g., anxiety as both a precipitant and a consequence of angina), in which case the distinction is arbitrary. Other mental disorders frequently result in medical complications, most notably substance use disorders (e.g., alcohol use disorder, tobacco use disorder). If an individual has a coexisting major mental disorder that adversely affects or causes another medical condition, diagnoses of the mental disorder and the medical condition are usually sufficient. Psychological factors affecting other medical conditions is diagnosed when the psychological traits or behaviors do not meet criteria for a mental diagnosis.

Somatic symptom disorder. Somatic symptom disorder is characterized by a combination of distressing somatic symptoms and excessive or maladaptive thoughts, feelings, and behavior in response to these symptoms or associated health concerns. The individual may or may not have a diagnosable medical condition. In contrast, in psychological factors affecting other medical conditions, the psychological factors adversely affect a medical condition; the individual's thoughts, feelings, and behavior are not necessarily excessive. The difference is one of emphasis, rather than a clear-cut distinction. In psychological factors affecting other medical conditions, the emphasis is on the exacerbation of the medical condition (e.g., an individual with angina that is precipitated whenever he becomes anxious). In somatic symptom disorder, the emphasis is on maladaptive thoughts, feelings, and behavior (e.g., an individual with angina who worries constantly that she will have a heart attack, takes her blood pressure multiple times per day, and restricts her activities).

Illness anxiety disorder. Illness anxiety disorder is characterized by high illness anxiety that is distressing and/or disruptive to daily life with minimal somatic symptoms. The focus of clinical concern is the individual's worry about having a disease; in most cases, no serious disease is present. In psychological factors affecting other medical conditions, anxiety may be a relevant psychological factor affecting a medical condition, but the clinical concern is the adverse effects on the medical condition.

Comorbidity

By definition, the diagnosis of psychological factors affecting other medical conditions entails a relevant psychological or behavioral syndrome or trait and a comorbid medical condition.

Factitious Disorder

Diagnostic Criteria **300.19 (F68.10)**

Factitious Disorder Imposed on Self

A. Falsification of physical or psychological signs or symptoms, or induction of injury or disease, associated with identified deception.
B. The individual presents himself or herself to others as ill, impaired, or injured.
C. The deceptive behavior is evident even in the absence of obvious external rewards.
D. The behavior is not better explained by another mental disorder, such as delusional disorder or another psychotic disorder.

Specify:

Single episode

Recurrent episodes (two or more events of falsification of illness and/or induction of injury)

Factitious Disorder Imposed on Another (Previously Factitious Disorder by Proxy)

A. Falsification of physical or psychological signs or symptoms, or induction of injury or disease, in another, associated with identified deception.
B. The individual presents another individual (victim) to others as ill, impaired, or injured.
C. The deceptive behavior is evident even in the absence of obvious external rewards.
D. The behavior is not better explained by another mental disorder, such as delusional disorder or another psychotic disorder.

Note: The perpetrator, not the victim, receives this diagnosis.

Specify:

Single episode

Recurrent episodes (two or more events of falsification of illness and/or induction of injury)

Recording Procedures

When an individual falsifies illness in another (e.g., children, adults, pets), the diagnosis is factitious disorder imposed on another. The perpetrator, not the victim, is given the diagnosis. The victim may be given an abuse diagnosis (e.g., 995.54 [T74.12X]; see the chapter "Other Conditions That May Be a Focus of Clinical Attention").

Diagnostic Features

The essential feature of factitious disorder is the falsification of medical or psychological signs and symptoms in oneself or others that are associated with the identified deception. Individuals with factitious disorder can also seek treatment for themselves or another following induction of injury or disease. The diagnosis requires demonstrating that the individual is taking surreptitious actions to misrepresent, simulate, or cause signs or symptoms of illness or injury in the absence of obvious external rewards. Methods of illness falsification can include exaggeration, fabrication, simulation, and induction. While a preexisting medical condition may be present, the deceptive behavior or induction of injury associated with deception causes others to view such individuals (or another) as more ill or impaired, and this can lead to excessive clinical intervention. Individuals with factitious disorder might, for example, report feelings of depression and suicidality following the death of a spouse despite the death not being true or the individual's not having a spouse; deceptively report episodes of neurological symptoms (e.g., seizures, dizziness, or blacking out); manipulate a laboratory test (e.g., by adding blood to urine) to falsely indicate an abnormality; falsify medical records to indicate an illness; ingest a substance (e.g., insulin or warfarin) to induce an abnormal laboratory result or illness; or physically injure themselves or induce illness in themselves or another (e.g., by injecting fecal material to produce an abscess or to induce sepsis).

Associated Features Supporting Diagnosis

Individuals with factitious disorder imposed on self or factitious disorder imposed on another are at risk for experiencing great psychological distress or functional impairment by causing harm to themselves and others. Family, friends, and health care professionals are also often adversely affected by their behavior. Factitious disorders have similarities to substance use disorders, eating disorders, impulse-control disorders, pedophilic disorder, and some other established disorders related to both the persistence of the behavior and the intentional efforts to conceal the disordered behavior through deception. Whereas some aspects of factitious disorders might represent criminal behavior (e.g., factitious dis-

order imposed on another, in which the parent's actions represent abuse and maltreatment of a child), such criminal behavior and mental illness are not mutually exclusive. The diagnosis of factitious disorder emphasizes the objective identification of falsification of signs and symptoms of illness, rather than an inference about intent or possible underlying motivation. Moreover, such behaviors, including the induction of injury or disease, are associated with deception.

Prevalence

The prevalence of factitious disorder is unknown, likely because of the role of deception in this population. Among patients in hospital settings, it is estimated that about 1% of individuals have presentations that meet the criteria for factitious disorder.

Development and Course

The course of factitious disorder is usually one of intermittent episodes. Single episodes and episodes that are characterized as persistent and unremitting are both less common. Onset is usually in early adulthood, often after hospitalization for a medical condition or a mental disorder. When imposed on another, the disorder may begin after hospitalization of the individual's child or other dependent. In individuals with recurrent episodes of falsification of signs and symptoms of illness and/or induction of injury, this pattern of successive deceptive contact with medical personnel, including hospitalizations, may become lifelong.

Differential Diagnosis

Caregivers who lie about abuse injuries in dependents solely to protect themselves from liability are not diagnosed with factitious disorder imposed on another because protection from liability is an external reward (Criterion C, the deceptive behavior is evident even in the absence of obvious external rewards). Such caregivers who, upon observation, analysis of medical records, and/or interviews with others, are found to lie more extensively than needed for immediate self-protection are diagnosed with factitious disorder imposed on another.

Somatic symptom disorder. In somatic symptom disorder, there may be excessive attention and treatment seeking for perceived medical concerns, but there is no evidence that the individual is providing false information or behaving deceptively.

Malingering. Malingering is differentiated from factitious disorder by the intentional reporting of symptoms for personal gain (e.g., money, time off work). In contrast, the diagnosis of factitious disorder requires the absence of obvious rewards.

Conversion disorder (functional neurological symptom disorder). Conversion disorder is characterized by neurological symptoms that are inconsistent with neurological pathophysiology. Factitious disorder with neurological symptoms is distinguished from conversion disorder by evidence of deceptive falsification of symptoms.

Borderline personality disorder. Deliberate physical self-harm in the absence of suicidal intent can also occur in association with other mental disorders such as borderline personality disorder. Factitious disorder requires that the induction of injury occur in association with deception.

Medical condition or mental disorder not associated with intentional symptom falsification. Presentation of signs and symptoms of illness that do not conform to an identifiable medical condition or mental disorder increases the likelihood of the presence of a factitious disorder. However, the diagnosis of factitious disorder does not exclude the presence of true medical condition or mental disorder, as comorbid illness often occurs in the individual along with factitious disorder. For example, individuals who might manipulate blood sugar levels to produce symptoms may also have diabetes.

Other Specified Somatic Symptom and Related Disorder

300.89 (F45.8)

This category applies to presentations in which symptoms characteristic of a somatic symptom and related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the somatic symptom and related disorders diagnostic class.

Examples of presentations that can be specified using the "other specified" designation include the following:

1. **Brief somatic symptom disorder:** Duration of symptoms is less than 6 months.
2. **Brief illness anxiety disorder:** Duration of symptoms is less than 6 months.
3. **Illness anxiety disorder without excessive health-related behaviors:** Criterion D for illness anxiety disorder is not met.
4. **Pseudocyesis:** A false belief of being pregnant that is associated with objective signs and reported symptoms of pregnancy.

Unspecified Somatic Symptom and Related Disorder

300.82 (F45.9)

This category applies to presentations in which symptoms characteristic of a somatic symptom and related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the somatic symptom and related disorders diagnostic class. The unspecified somatic symptom and related disorder category should not be used unless there are decidedly unusual situations where there is insufficient information to make a more specific diagnosis.

Feeding and Eating Disorders

Feeding and eating disorders are characterized by a persistent disturbance of eating or eating-related behavior that results in the altered consumption or absorption of food and that significantly impairs physical health or psychosocial functioning. Diagnostic criteria are provided for pica, rumination disorder, avoidant/restrictive food intake disorder, anorexia nervosa, bulimia nervosa, and binge-eating disorder.

The diagnostic criteria for rumination disorder, avoidant/restrictive food intake disorder, anorexia nervosa, bulimia nervosa, and binge-eating disorder result in a classification scheme that is mutually exclusive, so that during a single episode, only one of these diagnoses can be assigned. The rationale for this approach is that, despite a number of common psychological and behavioral features, the disorders differ substantially in clinical course, outcome, and treatment needs. A diagnosis of pica, however, may be assigned in the presence of any other feeding and eating disorder.

Some individuals with disorders described in this chapter report eating-related symptoms resembling those typically endorsed by individuals with substance use disorders, such as craving and patterns of compulsive use. This resemblance may reflect the involvement of the same neural systems, including those implicated in regulatory self-control and reward, in both groups of disorders. However, the relative contributions of shared and distinct factors in the development and perpetuation of eating and substance use disorders remain insufficiently understood.

Finally, obesity is not included in DSM-5 as a mental disorder. Obesity (excess body fat) results from the long-term excess of energy intake relative to energy expenditure. A range of genetic, physiological, behavioral, and environmental factors that vary across individuals contributes to the development of obesity; thus, obesity is not considered a mental disorder. However, there are robust associations between obesity and a number of mental disorders (e.g., binge-eating disorder, depressive and bipolar disorders, schizophrenia). The side effects of some psychotropic medications contribute importantly to the development of obesity, and obesity may be a risk factor for the development of some mental disorders (e.g., depressive disorders).

Pica

Diagnostic Criteria

A. Persistent eating of nonnutritive, nonfood substances over a period of at least 1 month.
B. The eating of nonnutritive, nonfood substances is inappropriate to the developmental level of the individual.
C. The eating behavior is not part of a culturally supported or socially normative practice.
D. If the eating behavior occurs in the context of another mental disorder (e.g., intellectual disability [intellectual developmental disorder], autism spectrum disorder, schizophrenia) or medical condition (including pregnancy), it is sufficiently severe to warrant additional clinical attention.

Coding note: The ICD-9-CM code for pica is **307.52** and is used for children or adults. The ICD-10-CM codes for pica are **(F98.3)** in children and **(F50.8)** in adults.
Specify if:

In remission: After full criteria for pica were previously met, the criteria have not been met for a sustained period of time.

Diagnostic Features

The essential feature of pica is the eating of one or more nonnutritive, nonfood substances on a persistent basis over a period of at least 1 month (Criterion A) that is severe enough to warrant clinical attention. Typical substances ingested tend to vary with age and availability and might include paper, soap, cloth, hair, string, wool, soil, chalk, talcum powder, paint, gum, metal, pebbles, charcoal or coal, ash, clay, starch, or ice. The term *nonfood* is included because the diagnosis of pica does not apply to ingestion of diet products that have minimal nutritional content. There is typically no aversion to food in general. The eating of nonnutritive, nonfood substances must be developmentally inappropriate (Criterion B) and not part of a culturally supported or socially normative practice (Criterion C). A minimum age of 2 years is suggested for a pica diagnosis to exclude developmentally normal mouthing of objects by infants that results in ingestion. The eating of nonnutritive, nonfood substances can be an associated feature of other mental disorders (e.g., intellectual disability [intellectual developmental disorder], autism spectrum disorder, schizophrenia). If the eating behavior occurs exclusively in the context of another mental disorder, a separate diagnosis of pica should be made only if the eating behavior is sufficiently severe to warrant additional clinical attention (Criterion D).

Associated Features Supporting Diagnosis

Although deficiencies in vitamins or minerals (e.g., zinc, iron) have been reported in some instances, often no specific biological abnormalities are found. In some cases, pica comes to clinical attention only following general medical complications (e.g., mechanical bowel problems; intestinal obstruction, such as that resulting from a bezoar; intestinal perforation; infections such as toxoplasmosis and toxocariasis as a result of ingesting feces or dirt; poisoning, such as by ingestion of lead-based paint).

Prevalence

The prevalence of pica is unclear. Among individuals with intellectual disability, the prevalence of pica appears to increase with the severity of the condition.

Development and Course

Onset of pica can occur in childhood, adolescence, or adulthood, although childhood onset is most commonly reported. Pica can occur in otherwise normally developing children, whereas in adults, it appears more likely to occur in the context of intellectual disability or other mental disorders. The eating of nonnutritive, nonfood substances may also manifest in pregnancy, when specific cravings (e.g., chalk or ice) might occur. The diagnosis of pica during pregnancy is only appropriate if such cravings lead to the ingestion of nonnutritive, nonfood substances to the extent that the eating of these substances poses potential medical risks. The course of the disorder can be protracted and can result in medical emergencies (e.g., intestinal obstruction, acute weight loss, poisoning). The disorder can potentially be fatal depending on substances ingested.

Risk and Prognostic Factors

Environmental. Neglect, lack of supervision, and developmental delay can increase the risk for this condition.

Culture-Related Diagnostic Issues

In some populations, the eating of earth or other seemingly nonnutritive substances is believed to be of spiritual, medicinal, or other social value, or may be a culturally supported or socially normative practice. Such behavior does not warrant a diagnosis of pica (Criterion C).

Gender-Related Diagnostic Issues

Pica occurs in both males and females. It can occur in females during pregnancy; however, little is known about the course of pica in the postpartum period.

Diagnostic Markers

Abdominal flat plate radiography, ultrasound, and other scanning methods may reveal obstructions related to pica. Blood tests and other laboratory tests can be used to ascertain levels of poisoning or the nature of infection.

Functional Consequences of Pica

Pica can significantly impair physical functioning, but it is rarely the sole cause of impairment in social functioning. Pica often occurs with other disorders associated with impaired social functioning.

Differential Diagnosis

Eating of nonnutritive, nonfood substances may occur during the course of other mental disorders (e.g., autism spectrum disorder, schizophrenia) and in Kleine-Levin syndrome. In any such instance, an additional diagnosis of pica should be given only if the eating behavior is sufficiently persistent and severe to warrant additional clinical attention.

Anorexia nervosa. Pica can usually be distinguished from the other feeding and eating disorders by the consumption of nonnutritive, nonfood substances. It is important to note, however, that some presentations of anorexia nervosa include ingestion of nonnutritive, nonfood substances, such as paper tissues, as a means of attempting to control appetite. In such cases, when the eating of nonnutritive, nonfood substances is primarily used as a means of weight control, anorexia nervosa should be the primary diagnosis.

Factitious disorder. Some individuals with factitious disorder may intentionally ingest foreign objects as part of the pattern of falsification of physical symptoms. In such instances, there is an element of deception that is consistent with deliberate induction of injury or disease.

Nonsuicidal self-injury and nonsuicidal self-injury behaviors in personality disorders. Some individuals may swallow potentially harmful items (e.g., pins, needles, knives) in the context of maladaptive behavior patterns associated with personality disorders or nonsuicidal self-injury.

Comorbidity

Disorders most commonly comorbid with pica are autism spectrum disorder and intellectual disability (intellectual developmental disorder), and, to a lesser degree, schizophrenia and obsessive-compulsive disorder. Pica can be associated with trichotillomania (hair-pulling disorder) and excoriation (skin-picking) disorder. In comorbid presentations, the hair or skin is typically ingested. Pica can also be associated with avoidant/restrictive food intake disorder, particularly in individuals with a strong sensory component to their presentation. When an individual is known to have pica, assessment should include consideration of the possibility of gastrointestinal complications, poisoning, infection, and nutritional deficiency.

Rumination Disorder

Diagnostic Criteria **307.53 (F98.21)**

A. Repeated regurgitation of food over a period of at least 1 month. Regurgitated food may be re-chewed, re-swallowed, or spit out.

B. The repeated regurgitation is not attributable to an associated gastrointestinal or other medical condition (e.g., gastroesophageal reflux, pyloric stenosis).

C. The eating disturbance does not occur exclusively during the course of anorexia nervosa, bulimia nervosa, binge-eating disorder, or avoidant/restrictive food intake disorder.

D. If the symptoms occur in the context of another mental disorder (e.g., intellectual disability [intellectual developmental disorder] or another neurodevelopmental disorder), they are sufficiently severe to warrant additional clinical attention.

Specify if:

In remission: After full criteria for rumination disorder were previously met, the criteria have not been met for a sustained period of time.

Diagnostic Features

The essential feature of rumination disorder is the repeated regurgitation of food occurring after feeding or eating over a period of at least 1 month (Criterion A). Previously swallowed food that may be partially digested is brought up into the mouth without apparent nausea, involuntary retching, or disgust. The food may be re-chewed and then ejected from the mouth or re-swallowed. Regurgitation in rumination disorder should be frequent, occurring at least several times per week, typically daily. The behavior is not better explained by an associated gastrointestinal or other medical condition (e.g., gastroesophageal reflux, pyloric stenosis) (Criterion B) and does not occur exclusively during the course of anorexia nervosa, bulimia nervosa, binge-eating disorder, or avoidant/restrictive food intake disorder (Criterion C). If the symptoms occur in the context of another mental disorder (e.g., intellectual disability [intellectual developmental disorder], neurodevelopmental disorder), they must be sufficiently severe to warrant additional clinical attention (Criterion D) and should represent a primary aspect of the individual's presentation requiring intervention. The disorder may be diagnosed across the life span, particularly in individuals who also have intellectual disability. Many individuals with rumination disorder can be directly observed engaging in the behavior by the clinician. In other instances diagnosis can be made on the basis of self-report or corroborative information from parents or caregivers. Individuals may describe the behavior as habitual or outside of their control.

Associated Features Supporting Diagnosis

Infants with rumination disorder display a characteristic position of straining and arching the back with the head held back, making sucking movements with their tongue. They may give the impression of gaining satisfaction from the activity. They may be irritable and hungry between episodes of regurgitation. Weight loss and failure to make expected weight gains are common features in infants with rumination disorder. Malnutrition may occur despite the infant's apparent hunger and the ingestion of relatively large amounts of food, particularly in severe cases, when regurgitation immediately follows each feeding episode and regurgitated food is expelled. Malnutrition might also occur in older children and adults, particularly when the regurgitation is accompanied by restriction of intake. Adolescents and adults may attempt to disguise the regurgitation behavior by placing a

hand over the mouth or coughing. Some will avoid eating with others because of the acknowledged social undesirability of the behavior. This may extend to an avoidance of eating prior to social situations, such as work or school (e.g., avoiding breakfast because it may be followed by regurgitation).

Prevalence

Prevalence data for rumination disorder are inconclusive, but the disorder is commonly reported to be higher in certain groups, such as individuals with intellectual disability.

Development and Course

Onset of rumination disorder can occur in infancy, childhood, adolescence, or adulthood. The age at onset in infants is usually between ages 3 and 12 months. In infants, the disorder frequently remits spontaneously, but its course can be protracted and can result in medical emergencies (e.g., severe malnutrition). It can potentially be fatal, particularly in infancy. Rumination disorder can have an episodic course or occur continuously until treated. In infants, as well as in older individuals with intellectual disability (intellectual developmental disorder) or other neurodevelopmental disorders, the regurgitation and rumination behavior appears to have a self-soothing or self-stimulating function, similar to that of other repetitive motor behaviors such as head banging.

Risk and Prognostic Factors

Environmental. Psychosocial problems such as lack of stimulation, neglect, stressful life situations, and problems in the parent-child relationship may be predisposing factors in infants and young children.

Functional Consequences of Rumination Disorder

Malnutrition secondary to repeated regurgitation may be associated with growth delay and have a negative effect on development and learning potential. Some older individuals with rumination disorder deliberately restrict their food intake because of the social undesirability of regurgitation. They may therefore present with weight loss or low weight. In older children, adolescents, and adults, social functioning is more likely to be adversely affected.

Differential Diagnosis

Gastrointestinal conditions. It is important to differentiate regurgitation in rumination disorder from other conditions characterized by gastroesophageal reflux or vomiting. Conditions such as gastroparesis, pyloric stenosis, hiatal hernia, and Sandifer syndrome in infants should be ruled out by appropriate physical examinations and laboratory tests.

Anorexia nervosa and bulimia nervosa. Individuals with anorexia nervosa and bulimia nervosa may also engage in regurgitation with subsequent spitting out of food as a means of disposing of ingested calories because of concerns about weight gain.

Comorbidity

Regurgitation with associated rumination can occur in the context of a concurrent medical condition or another mental disorder (e.g., generalized anxiety disorder). When the regurgitation occurs in this context, a diagnosis of rumination disorder is appropriate only when the severity of the disturbance exceeds that routinely associated with such conditions or disorders and warrants additional clinical attention.

Avoidant/Restrictive Food Intake Disorder

Diagnostic Criteria **307.59 (F50.8)**

A. An eating or feeding disturbance (e.g., apparent lack of interest in eating or food; avoidance based on the sensory characteristics of food; concern about aversive consequences of eating) as manifested by persistent failure to meet appropriate nutritional and/or energy needs associated with one (or more) of the following:
   1. Significant weight loss (or failure to achieve expected weight gain or faltering growth in children).
   2. Significant nutritional deficiency.
   3. Dependence on enteral feeding or oral nutritional supplements.
   4. Marked interference with psychosocial functioning.

B. The disturbance is not better explained by lack of available food or by an associated culturally sanctioned practice.

C. The eating disturbance does not occur exclusively during the course of anorexia nervosa or bulimia nervosa, and there is no evidence of a disturbance in the way in which one's body weight or shape is experienced.

D. The eating disturbance is not attributable to a concurrent medical condition or not better explained by another mental disorder. When the eating disturbance occurs in the context of another condition or disorder, the severity of the eating disturbance exceeds that routinely associated with the condition or disorder and warrants additional clinical attention.

Specify if:

In remission: After full criteria for avoidant/restrictive food intake disorder were previously met, the criteria have not been met for a sustained period of time.

Diagnostic Features

Avoidant/restrictive food intake disorder replaces and extends the DSM-IV diagnosis of feeding disorder of infancy or early childhood. The main diagnostic feature of avoidant/restrictive food intake disorder is avoidance or restriction of food intake (Criterion A) manifested by clinically significant failure to meet requirements for nutrition or insufficient energy intake through oral intake of food. One or more of the following key features must be present: significant weight loss, significant nutritional deficiency (or related health impact), dependence on enteral feeding or oral nutritional supplements, or marked interference with psychosocial functioning. The determination of whether weight loss is significant (Criterion A1) is a clinical judgment; instead of losing weight, children and adolescents who have not completed growth may not maintain weight or height increases along their developmental trajectory.

Determination of significant nutritional deficiency (Criterion A2) is also based on clinical assessment (e.g., assessment of dietary intake, physical examination, and laboratory testing), and related impact on physical health can be of a similar severity to that seen in anorexia nervosa (e.g., hypothermia, bradycardia, anemia). In severe cases, particularly in infants, malnutrition can be life threatening. "Dependence" on enteral feeding or oral nutritional supplements (Criterion A3) means that supplementary feeding is required to sustain adequate intake. Examples of individuals requiring supplementary feeding include infants with failure to thrive who require nasogastric tube feeding, children with neurodevelopmental disorders who are dependent on nutritionally complete supplements, and individuals who rely on gastrostomy tube feeding or complete oral nutrition supplements in the absence of an underlying medical condition. Inability to participate in normal social

activities, such as eating with others, or to sustain relationships as a result of the disturbance would indicate marked interference with psychosocial functioning (Criterion A4).

Avoidant/restrictive food intake disorder does not include avoidance or restriction of food intake related to lack of availability of food or to cultural practices (e.g., religious fasting or normal dieting) (Criterion B), nor does it include developmentally normal behaviors (e.g., picky eating in toddlers, reduced intake in older adults). The disturbance is not better explained by excessive concern about body weight or shape (Criterion C) or by concurrent medical factors or mental disorders (Criterion D).

In some individuals, food avoidance or restriction may be based on the sensory characteristics of qualities of food, such as extreme sensitivity to appearance, color, smell, texture, temperature, or taste. Such behavior has been described as "restrictive eating," "selective eating," "choosy eating," "perseverant eating," "chronic food refusal," and "food neophobia" and may manifest as refusal to eat particular brands of foods or to tolerate the smell of food being eaten by others. Individuals with heightened sensory sensitivities associated with autism may show similar behaviors.

Food avoidance or restriction may also represent a conditioned negative response associated with food intake following, or in anticipation of, an aversive experience, such as choking; a traumatic investigation, usually involving the gastrointestinal tract (e.g., esophagoscopy); or repeated vomiting. The terms *functional dysphagia* and *globus hystericus* have also been used for such conditions.

Associated Features Supporting Diagnosis

Several features may be associated with food avoidance or reduced food intake, including a lack of interest in eating or food, leading to weight loss or faltering growth. Very young infants may present as being too sleepy, distressed, or agitated to feed. Infants and young children may not engage with the primary caregiver during feeding or communicate hunger in favor of other activities. In older children and adolescents, food avoidance or restriction may be associated with more generalized emotional difficulties that do not meet diagnostic criteria for an anxiety, depressive, or bipolar disorder, sometimes called "food avoidance emotional disorder."

Development and Course

Food avoidance or restriction associated with insufficient intake or lack of interest in eating most commonly develops in infancy or early childhood and may persist in adulthood. Likewise, avoidance based on sensory characteristics of food tends to arise in the first decade of life but may persist into adulthood. Avoidance related to aversive consequences can arise at any age. The scant literature regarding long-term outcomes suggests that food avoidance or restriction based on sensory aspects is relatively stable and long-standing, but when persisting into adulthood, such avoidance/restriction can be associated with relatively normal functioning. There is currently insufficient evidence directly linking avoidant/restrictive food intake disorder and subsequent onset of an eating disorder.

Infants with avoidant/restrictive food intake disorder may be irritable and difficult to console during feeding, or may appear apathetic and withdrawn. In some instances, parent-child interaction may contribute to the infant's feeding problem (e.g., presenting food inappropriately, or interpreting the infant's behavior as an act of aggression or rejection). Inadequate nutritional intake may exacerbate the associated features (e.g., irritability, developmental lags) and further contribute to feeding difficulties. Associated factors include infant temperament or developmental impairments that reduce an infant's responsiveness to feeding. Coexisting parental psychopathology, or child abuse or neglect, is suggested if feeding and weight improve in response to changing caregivers. In infants, children, and prepubertal adolescents, avoidant/restrictive food intake disorder may be associated with growth delay, and the resulting malnutrition negatively affects development and learning

potential. In older children, adolescents, and adults, social functioning tends to be adversely affected. Regardless of the age, family function may be affected, with heightened stress at mealtimes and in other feeding or eating contexts involving friends and relatives.

Avoidant/restrictive food intake disorder manifests more commonly in children than in adults, and there may be a long delay between onset and clinical presentation. Triggers for presentation vary considerably and include physical, social, and emotional difficulties.

Risk and Prognostic Factors

Temperamental. Anxiety disorders, autism spectrum disorder, obsessive-compulsive disorder, and attention-deficit/hyperactivity disorder may increase risk for avoidant or restrictive feeding or eating behavior characteristic of the disorder.

Environmental. Environmental risk factors for avoidant/restrictive food intake disorder include familial anxiety. Higher rates of feeding disturbances may occur in children of mothers with eating disorders.

Genetic and physiological. History of gastrointestinal conditions, gastroesophageal reflux disease, vomiting, and a range of other medical problems has been associated with feeding and eating behaviors characteristic of avoidant/restrictive food intake disorder.

Culture-Reiated Diagnostic Issues

Presentations similar to avoidant/restrictive food intake disorder occur in various populations, including in the United States, Canada, Australia, and Europe. Avoidant/restrictive food intake disorder should not be diagnosed when avoidance of food intake is solely related to specific religious or cultural practices.

Gender-Reiated Diagnostic Issues

Avoidant/restrictive food intake disorder is equally common in males and females in infancy and early childhood, but avoidant/restrictive food intake disorder comorbid with autism spectrum disorder has a male predominance. Food avoidance or restriction related to altered sensory sensitivities can occur in some physiological conditions, most notably pregnancy, but is not usually extreme and does not meet full criteria for the disorder.

Diagnostic Markers

Diagnostic markers include malnutrition, low weight, growth delay, and the need for artificial nutrition in the absence of any clear medical condition other than poor intake.

Functionai Consequences of Avoidant/Restrictive Food Intake Disorder

Associated developmental and functional limitations include impairment of physical development and social difficulties that can have a significant negative impact on family function.

Differentiai Diagnosis

Appetite loss preceding restricted intake is a nonspecific symptom that can accompany a number of mental diagnoses. Avoidant/restrictive food intake disorder can be diagnosed concurrently with the disorders below if all criteria are met, and the eating disturbance requires specific clinical attention.

Other medical conditions (e.g., gastrointestinal disease, food allergies and intolerances, occult malignancies). Restriction of food intake may occur in other medical condi-

tions, especially those with ongoing symptoms such as vomiting, loss of appetite, nausea, abdominal pain, or diarrhea. A diagnosis of avoidant/restrictive food intake disorder requires that the disturbance of intake is beyond that directly accounted for by physical symptoms consistent with a medical condition; the eating disturbance may also persist after being triggered by a medical condition and following resolution of the medical condition.

Underlying medical or comorbid mental conditions may complicate feeding and eating. Because older individuals, postsurgical patients, and individuals receiving chemotherapy often lose their appetite, an additional diagnosis of avoidant/restrictive food intake disorder requires that the eating disturbance is a primary focus for intervention.

Specific neurological/neuromuscular, structural, or congenital disorders and conditions associated with feeding difficulties. Feeding difficulties are common in a number of congenital and neurological conditions often related to problems with oral/esophageal/pharyngeal structure and function, such as hypotonia of musculature, tongue protrusion, and unsafe swallowing. Avoidant/restrictive food intake disorder can be diagnosed in individuals with such presentations as long as all diagnostic criteria are met.

Reactive attachment disorder. Some degree of withdrawal is characteristic of reactive attachment disorder and can lead to a disturbance in the caregiver-child relationship that can affect feeding and the child's intake. Avoidant/restrictive food intake disorder should be diagnosed concurrently only if all criteria are met for both disorders and the feeding disturbance is a primary focus for intervention.

Autism spectrum disorder. Individuals with autism spectrum disorder often present with rigid eating behaviors and heightened sensory sensitivities. However, these features do not always result in the level of impairment that would be required for a diagnosis of avoidant/restrictive food intake disorder. Avoidant/restrictive food intake disorder should be diagnosed concurrently only if all criteria are met for both disorders and when the eating disturbance requires specific treatment.

Specific phobia, social anxiety disorder (social phobia), and other anxiety disorders. Specific phobia, other type, specifies "situations that may lead to choking or vomiting" and can represent the primary trigger for the fear, anxiety, or avoidance required for diagnosis. Distinguishing specific phobia from avoidant/restrictive food intake disorder can be difficult when a fear of choking or vomiting has resulted in food avoidance. Although avoidance or restriction of food intake secondary to a pronounced fear of choking or vomiting can be conceptualized as specific phobia, in situations when the eating problem becomes the primary focus of clinical attention, avoidant/restrictive food intake disorder becomes the appropriate diagnosis. In social anxiety disorder, the individual may present with a fear of being observed by others while eating, which can also occur in avoidant/restrictive food intake disorder.

Anorexia nervosa. Restriction of energy intake relative to requirements leading to significantly low body weight is a core feature of anorexia nervosa. However, individuals with anorexia nervosa also display a fear of gaining weight or of becoming fat, or persistent behavior that interferes with weight gain, as well as specific disturbances in relation to perception and experience of their own body weight and shape. These features are not present in avoidant/restrictive food intake disorder, and the two disorders should not be diagnosed concurrently. Differential diagnosis between avoidant/restrictive food intake disorder and anorexia nervosa may be difficult, especially in late childhood and early adolescence, because these disorders may share a number of common symptoms (e.g., food avoidance, low weight). Differential diagnosis is also potentially difficult in individuals with anorexia nervosa who deny any fear of fatness but nonetheless engage in persistent behaviors that prevent weight gain and who do not recognize the medical seriousness of their low weight—a presentation sometimes termed "non-fat phobic anorexia nervosa." Full consideration of symptoms, course, and family history is advised, and diagnosis may

be best made in the context of a clinical relationship over time. In some individuals, avoidant/restrictive food intake disorder might precede the onset of anorexia nervosa.

Obsessive-compulsive disorder. Individuals with obsessive-compulsive disorder may present with avoidance or restriction of intake in relation to preoccupations with food or ritualized eating behavior. Avoidant/restrictive food intake disorder should be diagnosed concurrently only if all criteria are met for both disorders and when the aberrant eating is a major aspect of the clinical presentation requiring specific intervention.

Major depressive disorder. In major depressive disorder, appetite might be affected to such an extent that individuals present with significantly restricted food intake, usually in relation to overall energy intake and often associated with weight loss. Usually appetite loss and related reduction of intake abate with resolution of mood problems. Avoidant/restrictive food intake disorder should only be used concurrently if full criteria are met for both disorders and when the eating disturbance requires specific treatment.

Schizophrenia spectrum disorders. Individuals with schizophrenia, delusional disorder, or other psychotic disorders may exhibit odd eating behaviors, avoidance of specific foods because of delusional beliefs, or other manifestations of avoidant or restrictive intake. In some cases, delusional beliefs may contribute to a concern about negative consequences of ingesting certain foods. Avoidant/restrictive food intake disorder should be used concurrently only if all criteria are met for both disorders and when the eating disturbance requires specific treatment.

Factitious disorder or factitious disorder imposed on another. Avoidant/restrictive food intake disorder should be differentiated from factitious disorder or factitious disorder imposed on another. In order to assume the sick role, some individuals with factitious disorder may intentionally describe diets that are much more restrictive than those they are actually able to consume, as well as complications of such behavior, such as a need for enteral feedings or nutritional supplements, an inability to tolerate a normal range of foods, and/or an inability to participate normally in age-appropriate situations involving food. The presentation may be impressively dramatic and engaging, and the symptoms reported inconsistently. In factitious disorder imposed on another, the caregiver describes symptoms consistent with avoidant/restrictive food intake disorder and may induce physical symptoms such as failure to gain weight. As with any diagnosis of factitious disorder imposed on another, the caregiver receives the diagnosis rather than the affected individual, and diagnosis should be made only on the basis of a careful, comprehensive assessment of the affected individual, the caregiver, and their interaction.

Comorbidity

The most commonly observed disorders comorbid with avoidant/restrictive food intake disorder are anxiety disorders, obsessive-compulsive disorder, and neurodevelopmental disorders (specifically autism spectrum disorder, attention-deficit/hyperactivity disorder, and intellectual disability [intellectual developmental disorder]).

Anorexia Nervosa

Diagnostic Criteria

A. Restriction of energy intake relative to requirements, leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health. *Significantly low weight* is defined as a weight that is less than minimally normal or, for children and adolescents, less than that minimally expected.

B. Intense fear of gaining weight or of becoming fat, or persistent behavior that interferes with weight gain, even though at a significantly low weight.

C. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body weight.

Coding note: The ICD-9-CM code for anorexia nervosa is **307.1**, which is assigned regardless of the subtype. The ICD-10-CM code depends on the subtype (see below).

Specify whether:

(F50.01) Restricting type: During the last 3 months, the individual has not engaged in recurrent episodes of binge eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas). This subtype describes presentations in which weight loss is accomplished primarily through dieting, fasting, and/or excessive exercise.

(F50.02) Binge-eating/purging type: During the last 3 months, the individual has engaged in recurrent episodes of binge eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas).

Specify if:

In partial remission: After full criteria for anorexia nervosa were previously met, Criterion A (low body weight) has not been met for a sustained period, but either Criterion B (intense fear of gaining weight or becoming fat or behavior that interferes with weight gain) or Criterion C (disturbances in self-perception of weight and shape) is still met.

In full remission: After full criteria for anorexia nervosa were previously met, none of the criteria have been met for a sustained period of time.

Specify current severity:

The minimum level of severity is based, for adults, on current body mass index (BMI) (see below) or, for children and adolescents, on BMI percentile. The ranges below are derived from World Health Organization categories for thinness in adults; for children and adolescents, corresponding BMI percentiles should be used. The level of severity may be increased to reflect clinical symptoms, the degree of functional disability, and the need for supervision.

Mild: BMI $\geq$ 17 kg/m^2

Moderate: BMI 16–16.99 kg/m^2

Severe: BMI 15–15.99 kg/m^2

Extreme: BMI < 15 kg/m^2

Subtypes

Most individuals with the binge-eating/purging type of anorexia nervosa who binge eat also purge through self-induced vomiting or the misuse of laxatives, diuretics, or enemas. Some individuals with this subtype of anorexia nervosa do not binge eat but do regularly purge after the consumption of small amounts of food.

Crossover between the subtypes over the course of the disorder is not uncommon; therefore, subtype description should be used to describe current symptoms rather than longitudinal course.

Diagnostic Features

There are three essential features of anorexia nervosa: persistent energy intake restriction; intense fear of gaining weight or of becoming fat, or persistent behavior that interferes with weight gain; and a disturbance in self-perceived weight or shape. The individual maintains a body weight that is below a minimally normal level for age, sex, developmental trajectory, and physical health (Criterion A). Individuals' body weights frequently meet this criterion following a significant weight loss, but among children and adolescents, there may alternatively be failure to make expected weight gain or to maintain a normal developmental trajectory (i.e., while growing in height) instead of weight loss.

Criterion A requires that the individual's weight be significantly low (i.e., less than minimally normal or, for children and adolescents, less than that minimally expected). Weight assessment can be challenging because normal weight range differs among individuals, and different thresholds have been published defining thinness or underweight status. Body mass index (BMI; calculated as weight in kilograms/height in meters^2) is a useful measure to assess body weight for height. For adults, a BMI of 18.5 kg/m^2 has been employed by the Centers for Disease Control and Prevention (CDC) and the World Health Organization (WHO) as the lower limit of normal body weight. Therefore, most adults with a BMI greater than or equal to 18.5 kg/m^2 would not be considered to have a significantly low body weight. On the other hand, a BMI of lower than 17.0 kg/m^2 has been considered by the WHO to indicate moderate or severe thinness; therefore, an individual with a BMI less than 17.0 kg/m^2 would likely be considered to have a significantly low weight. An adult with a BMI between 17.0 and 18.5 kg/m^2, or even above 18.5 kg/m^2, might be considered to have a significantly low weight if clinical history or other physiological information supports this judgment.

For children and adolescents, determining a BMI-for-age percentile is useful (see, e.g., the CDC BMI percentile calculator for children and teenagers. As for adults, it is not possible to provide definitive standards for judging whether a child's or an adolescent's weight is significantly low, and variations in developmental trajectories among youth limit the utility of simple numerical guidelines. The CDC has used a BMI-for-age below the 5th percentile as suggesting underweight; however, children and adolescents with a BMI above this benchmark may be judged to be significantly underweight in light of failure to maintain their expected growth trajectory. In summary, in determining whether Criterion A is met, the clinician should consider available numerical guidelines, as well as the individual's body build, weight history, and any physiological disturbances.

Individuals with this disorder typically display an intense fear of gaining weight or of becoming fat (Criterion B). This intense fear of becoming fat is usually not alleviated by weight loss. In fact, concern about weight gain may increase even as weight falls. Younger individuals with anorexia nervosa, as well as some adults, may not recognize or acknowledge a fear of weight gain. In the absence of another explanation for the significantly low weight, clinician inference drawn from collateral history, observational data, physical and laboratory findings, or longitudinal course either indicating a fear of weight gain or supporting persistent behaviors that prevent it may be used to establish Criterion B.

The experience and significance of body weight and shape are distorted in these individuals (Criterion C). Some individuals feel globally overweight. Others realize that they are thin but are still concerned that certain body parts, particularly the abdomen, buttocks, and thighs, are "too fat." They may employ a variety of techniques to evaluate their body size or weight, including frequent weighing, obsessive measuring of body parts, and persistent use of a mirror to check for perceived areas of "fat." The self-esteem of individuals with anorexia nervosa is highly dependent on their perceptions of body shape and weight. Weight loss is often viewed as an impressive achievement and a sign of extraordinary self-discipline, whereas weight gain is perceived as an unacceptable failure of self-control. Although some individuals with this disorder may acknowledge being thin, they often do not recognize the serious medical implications of their malnourished state.

Often, the individual is brought to professional attention by family members after marked weight loss (or failure to make expected weight gains) has occurred. If individuals seek help on their own, it is usually because of distress over the somatic and psychological sequelae of starvation. It is rare for an individual with anorexia nervosa to complain of weight loss per se. In fact, individuals with anorexia nervosa frequently either lack insight into or deny the problem. It is therefore often important to obtain information from family members or other sources to evaluate the history of weight loss and other features of the illness.

Associated Features Supporting Diagnosis

The semi-starvation of anorexia nervosa, and the purging behaviors sometimes associated with it, can result in significant and potentially life-threatening medical conditions. The nutritional compromise associated with this disorder affects most major organ systems and can produce a variety of disturbances. Physiological disturbances, including amenorrhea and vital sign abnormalities, are common. While most of the physiological disturbances associated with malnutrition are reversible with nutritional rehabilitation, some, including loss of bone mineral density, are often not completely reversible. Behaviors such as self-induced vomiting and misuse of laxatives, diuretics, and enemas may cause a number of disturbances that lead to abnormal laboratory findings; however, some individuals with anorexia nervosa exhibit no laboratory abnormalities.

When seriously underweight, many individuals with anorexia nervosa have depressive signs and symptoms such as depressed mood, social withdrawal, irritability, insomnia, and diminished interest in sex. Because these features are also observed in individuals without anorexia nervosa who are significantly undernourished, many of the depressive features may be secondary to the physiological sequelae of semi-starvation, although they may also be sufficiently severe to warrant an additional diagnosis of major depressive disorder.

Obsessive-compulsive features, both related and unrelated to food, are often prominent. Most individuals with anorexia nervosa are preoccupied with thoughts of food. Some collect recipes or hoard food. Observations of behaviors associated with other forms of starvation suggest that obsessions and compulsions related to food may be exacerbated by undernutrition. When individuals with anorexia nervosa exhibit obsessions and compulsions that are not related to food, body shape, or weight, an additional diagnosis of obsessive-compulsive disorder (OCD) may be warranted.

Other features sometimes associated with anorexia nervosa include concerns about eating in public, feelings of ineffectiveness, a strong desire to control one's environment, inflexible thinking, limited social spontaneity, and overly restrained emotional expression. Compared with individuals with anorexia nervosa, restricting type, those with binge-eating/purging type have higher rates of impulsivity and are more likely to abuse alcohol and other drugs.

A subgroup of individuals with anorexia nervosa show excessive levels of physical activity. Increases in physical activity often precede onset of the disorder, and over the course of the disorder increased activity accelerates weight loss. During treatment, excessive activity may be difficult to control, thereby jeopardizing weight recovery.

Individuals with anorexia nervosa may misuse medications, such as by manipulating dosage, in order to achieve weight loss or avoid weight gain. Individuals with diabetes mellitus may omit or reduce insulin doses in order to minimize carbohydrate metabolism.

Prevalence

The 12-month prevalence of anorexia nervosa among young females is approximately 0.4%. Less is known about prevalence among males, but anorexia nervosa is far less common in males than in females, with clinical populations generally reflecting approximately a 10:1 female-to-male ratio.

Development and Course

Anorexia nervosa commonly begins during adolescence or young adulthood. It rarely begins before puberty or after age 40, but cases of both early and late onset have been described. The onset of this disorder is often associated with a stressful life event, such as leaving home for college. The course and outcome of anorexia nervosa are highly variable. Younger individuals may manifest atypical features, including denying "fear of fat." Older

individuals more likely have a longer duration of illness, and their clinical presentation may include more signs and symptoms of long-standing disorder. Clinicians should not exclude anorexia nervosa from the differential diagnosis solely on the basis of older age.

Many individuals have a period of changed eating behavior prior to full criteria for the disorder being met. Some individuals with anorexia nervosa recover fully after a single episode, with some exhibiting a fluctuating pattern of weight gain followed by relapse, and others experiencing a chronic course over many years. Hospitalization may be required to restore weight and to address medical complications. Most individuals with anorexia nervosa experience remission within 5 years of presentation. Among individuals admitted to hospitals, overall remission rates may be lower. The crude mortality rate (CMR) for anorexia nervosa is approximately 5% per decade. Death most commonly results from medical complications associated with the disorder itself or from suicide.

Risk and Prognostic Factors

Temperamental. Individuals who develop anxiety disorders or display obsessional traits in childhood are at increased risk of developing anorexia nervosa.

Environmental. Historical and cross-cultural variability in the prevalence of anorexia nervosa supports its association with cultures and settings in which thinness is valued. Occupations and avocations that encourage thinness, such as modeling and elite athletics, are also associated with increased risk.

Genetic and physiological. There is an increased risk of anorexia nervosa and bulimia nervosa among first-degree biological relatives of individuals with the disorder. An increased risk of bipolar and depressive disorders has also been found among first-degree relatives of individuals with anorexia nervosa, particularly relatives of individuals with the binge-eating/purging type. Concordance rates for anorexia nervosa in monozygotic twins are significantly higher than those for dizygotic twins. A range of brain abnormalities has been described in anorexia nervosa using functional imaging technologies (functional magnetic resonance imaging, positron emission tomography). The degree to which these findings reflect changes associated with malnutrition versus primary abnormalities associated with the disorder is unclear.

Culture-Related Diagnostic Issues

Anorexia nervosa occurs across culturally and socially diverse populations, although available evidence suggests cross-cultural variation in its occurrence and presentation. Anorexia nervosa is probably most prevalent in post-industrialized, high-income countries such as in the United States, many European countries, Australia, New Zealand, and Japan, but its incidence in most low- and middle-income countries is uncertain. Whereas the prevalence of anorexia nervosa appears comparatively low among Latinos, African Americans, and Asians in the United States, clinicians should be aware that mental health service utilization among individuals with an eating disorder is significantly lower in these ethnic groups and that the low rates may reflect an ascertainment bias. The presentation of weight concerns among individuals with eating and feeding disorders varies substantially across cultural contexts. The absence of an expressed intense fear of weight gain, sometimes referred to as "fat phobia," appears to be relatively more common in populations in Asia, where the rationale for dietary restriction is commonly related to a more culturally sanctioned complaint such as gastrointestinal discomfort. Within the United States, presentations without a stated intense fear of weight gain may be comparatively more common among Latino groups.

Diagnostic Markers

The following laboratory abnormalities may be observed in anorexia nervosa; their presence may serve to increase diagnostic confidence.

Hematology. Leukopenia is common, with the loss of all cell types but usually with apparent lymphocytosis. Mild anemia can occur, as well as thrombocytopenia and, rarely, bleeding problems.

Serum chemistry. Dehydration may be reflected by an elevated blood urea nitrogen level. Hypercholesterolemia is common. Hepatic enzyme levels may be elevated. Hypomagnesemia, hypozincemia, hypophosphatemia, and hyperamylasemia are occasionally observed. Self-induced vomiting may lead to metabolic alkalosis (elevated serum bicarbonate), hypochloremia, and hypokalemia; laxative abuse may cause a mild metabolic acidosis.

Endocrine. Serum thyroxine (T_4) levels are usually in the low-normal range; triiodothyronine (T_3) levels are decreased, while reverse T_3 levels are elevated. Females have low serum estrogen levels, whereas males have low levels of serum testosterone.

Electrocardiography. Sinus bradycardia is common, and, rarely, arrhythmias are noted. Significant prolongation of the QTc interval is observed in some individuals.

Bone mass. Low bone mineral density, with specific areas of osteopenia or osteoporosis, is often seen. The risk of fracture is significantly elevated.

Electroencephalography. Diffuse abnormalities, reflecting a metabolic encephalopathy, may result from significant fluid and electrolyte disturbances.

Resting energy expenditure. There is often a significant reduction in resting energy expenditure.

Physical signs and symptoms. Many of the physical signs and symptoms of anorexia nervosa are attributable to starvation. Amenorrhea is commonly present and appears to be an indicator of physiological dysfunction. If present, amenorrhea is usually a consequence of the weight loss, but in a minority of individuals it may actually precede the weight loss. In prepubertal females, menarche may be delayed. In addition to amenorrhea, there may be complaints of constipation, abdominal pain, cold intolerance, lethargy, and excess energy.

The most remarkable finding on physical examination is emaciation. Commonly, there is also significant hypotension, hypothermia, and bradycardia. Some individuals develop lanugo, a fine downy body hair. Some develop peripheral edema, especially during weight restoration or upon cessation of laxative and diuretic abuse. Rarely, petechiae or ecchymoses, usually on the extremities, may indicate a bleeding diathesis. Some individuals evidence a yellowing of the skin associated with hypercarotenemia. As may be seen in individuals with bulimia nervosa, individuals with anorexia nervosa who self-induce vomiting may have hypertrophy of the salivary glands, particularly the parotid glands, as well as dental enamel erosion. Some individuals may have scars or calluses on the dorsal surface of the hand from repeated contact with the teeth while inducing vomiting.

Suicide Risk

Suicide risk is elevated in anorexia nervosa, with rates reported as 12 per 100,000 per year. Comprehensive evaluation of individuals with anorexia nervosa should include assessment of suicide-related ideation and behaviors as well as other risk factors for suicide, including a history of suicide attempt(s).

Functional Consequences of Anorexia Nervosa

Individuals with anorexia nervosa may exhibit a range of functional limitations associated with the disorder. While some individuals remain active in social and professional functioning, others demonstrate significant social isolation and/or failure to fulfill academic or career potential.

Differential Diagnosis

Other possible causes of either significantly low body weight or significant weight loss should be considered in the differential diagnosis of anorexia nervosa, especially when the presenting features are atypical (e.g., onset after age 40 years).

Medical conditions (e.g., gastrointestinal disease, hyperthyroidism, occult malignancies, and acquired immunodeficiency syndrome [AIDS]). Serious weight loss may occur in medical conditions, but individuals with these disorders usually do not also manifest a disturbance in the way their body weight or shape is experienced or an intense fear of weight gain or persist in behaviors that interfere with appropriate weight gain. Acute weight loss associated with a medical condition can occasionally be followed by the onset or recurrence of anorexia nervosa, which can initially be masked by the comorbid medical condition. Rarely, anorexia nervosa develops after bariatric surgery for obesity.

Major depressive disorder. In major depressive disorder, severe weight loss may occur, but most individuals with major depressive disorder do not have either a desire for excessive weight loss or an intense fear of gaining weight.

Schizophrenia. Individuals with schizophrenia may exhibit odd eating behavior and occasionally experience significant weight loss, but they rarely show the fear of gaining weight and the body image disturbance required for a diagnosis of anorexia nervosa.

Substance use disorders. Individuals with substance use disorders may experience low weight due to poor nutritional intake but generally do not fear gaining weight and do not manifest body image disturbance. Individuals who abuse substances that reduce appetite (e.g., cocaine, stimulants) and who also endorse fear of weight gain should be carefully evaluated for the possibility of comorbid anorexia nervosa, given that the substance use may represent a persistent behavior that interferes with weight gain (Criterion B).

Social anxiety disorder (social phobia), obsessive-compulsive disorder, and body dysmorphic disorder. Some of the features of anorexia nervosa overlap with the criteria for social phobia, OCD, and body dysmorphic disorder. Specifically, individuals may feel humiliated or embarrassed to be seen eating in public, as in social phobia; may exhibit obsessions and compulsions related to food, as in OCD; or may be preoccupied with an imagined defect in bodily appearance, as in body dysmorphic disorder. If the individual with anorexia nervosa has social fears that are limited to eating behavior alone, the diagnosis of social phobia should not be made, but social fears unrelated to eating behavior (e.g., excessive fear of speaking in public) may warrant an additional diagnosis of social phobia. Similarly, an additional diagnosis of OCD should be considered only if the individual exhibits obsessions and compulsions unrelated to food (e.g., an excessive fear of contamination), and an additional diagnosis of body dysmorphic disorder should be considered only if the distortion is unrelated to body shape and size (e.g., preoccupation that one's nose is too big).

Bulimia nervosa. Individuals with bulimia nervosa exhibit recurrent episodes of binge eating, engage in inappropriate behavior to avoid weight gain (e.g., self-induced vomiting), and are overly concerned with body shape and weight. However, unlike individuals with anorexia nervosa, binge-eating/purging type, individuals with bulimia nervosa maintain body weight at or above a minimally normal level.

Avoidant/restrictive food intake disorder. Individuals with this disorder may exhibit significant weight loss or significant nutritional deficiency, but they do not have a fear of gaining weight or of becoming fat, nor do they have a disturbance in the way they experience their body shape and weight.

Comorbidity

Bipolar, depressive, and anxiety disorders commonly co-occur with anorexia nervosa. Many individuals with anorexia nervosa report the presence of either an anxiety disorder

or symptoms prior to onset of their eating disorder. OCD is described in some individuals with anorexia nervosa, especially those with the restricting type. Alcohol use disorder and other substance use disorders may also be comorbid with anorexia nervosa, especially among those with the binge-eating/purging type.

Bulimia Nervosa

Diagnostic Criteria **307.51 (F50.2)**

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
   1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than what most individuals would eat in a similar period of time under similar circumstances.
   2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).

B. Recurrent inappropriate compensatory behaviors in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, or other medications; fasting; or excessive exercise.

C. The binge eating and inappropriate compensatory behaviors both occur, on average, at least once a week for 3 months.

D. Self-evaluation is unduly influenced by body shape and weight.

E. The disturbance does not occur exclusively during episodes of anorexia nervosa.

Specify if:

In partial remission: After full criteria for bulimia nervosa were previously met, some, but not all, of the criteria have been met for a sustained period of time.

In full remission: After full criteria for bulimia nervosa were previously met, none of the criteria have been met for a sustained period of time.

Specify current severity:

The minimum level of severity is based on the frequency of inappropriate compensatory behaviors (see below). The level of severity may be increased to reflect other symptoms and the degree of functional disability.

Mild: An average of 1–3 episodes of inappropriate compensatory behaviors per week.

Moderate: An average of 4–7 episodes of inappropriate compensatory behaviors per week.

Severe: An average of 8–13 episodes of inappropriate compensatory behaviors per week.

Extreme: An average of 14 or more episodes of inappropriate compensatory behaviors per week.

Diagnostic Features

There are three essential features of bulimia nervosa: recurrent episodes of binge eating (Criterion A), recurrent inappropriate compensatory behaviors to prevent weight gain (Criterion B), and self-evaluation that is unduly influenced by body shape and weight (Criterion D). To qualify for the diagnosis, the binge eating and inappropriate compensatory behaviors must occur, on average, at least once per week for 3 months (Criterion C).

An "episode of binge eating" is defined as eating, in a discrete period of time, an amount of food that is definitely larger than most individuals would eat in a similar period of time under similar circumstances (Criterion A1). The context in which the eating occurs

may affect the clinician's estimation of whether the intake is excessive. For example, a quantity of food that might be regarded as excessive for a typical meal might be considered normal during a celebration or holiday meal. A "discrete period of time" refers to a limited period, usually less than 2 hours. A single episode of binge eating need not be restricted to one setting. For example, an individual may begin a binge in a restaurant and then continue to eat on returning home. Continual snacking on small amounts of food throughout the day would not be considered an eating binge.

An occurrence of excessive food consumption must be accompanied by a sense of lack of control (Criterion A2) to be considered an episode of binge eating. An indicator of loss of control is the inability to refrain from eating or to stop eating once started. Some individuals describe a dissociative quality during, or following, the binge-eating episodes. The impairment in control associated with binge eating may not be absolute; for example, an individual may continue binge eating while the telephone is ringing but will cease if a roommate or spouse unexpectedly enters the room. Some individuals report that their binge-eating episodes are no longer characterized by an acute feeling of loss of control but rather by a more generalized pattern of uncontrolled eating. If individuals report that they have abandoned efforts to control their eating, loss of control should be considered as present. Binge eating can also be planned in some instances.

The type of food consumed during binges varies both across individuals and for a given individual. Binge eating appears to be characterized more by an abnormality in the amount of food consumed than by a craving for a specific nutrient. However, during binges, individuals tend to eat foods they would otherwise avoid.

Individuals with bulimia nervosa are typically ashamed of their eating problems and attempt to conceal their symptoms. Binge eating usually occurs in secrecy or as inconspicuously as possible. The binge eating often continues until the individual is uncomfortably, or even painfully, full. The most common antecedent of binge eating is negative affect. Other triggers include interpersonal stressors; dietary restraint; negative feelings related to body weight, body shape, and food; and boredom. Binge eating may minimize or mitigate factors that precipitated the episode in the short-term, but negative self-evaluation and dysphoria often are the delayed consequences.

Another essential feature of bulimia nervosa is the recurrent use of inappropriate compensatory behaviors to prevent weight gain, collectively referred to as *purge behaviors* or *purging* (Criterion B). Many individuals with bulimia nervosa employ several methods to compensate for binge eating. Vomiting is the most common inappropriate compensatory behavior. The immediate effects of vomiting include relief from physical discomfort and reduction of fear of gaining weight. In some cases, vomiting becomes a goal in itself, and the individual will binge eat in order to vomit or will vomit after eating a small amount of food. Individuals with bulimia nervosa may use a variety of methods to induce vomiting, including the use of fingers or instruments to stimulate the gag reflex. Individuals generally become adept at inducing vomiting and are eventually able to vomit at will. Rarely, individuals consume syrup of ipecac to induce vomiting. Other purging behaviors include the misuse of laxatives and diuretics. A number of other compensatory methods may also be used in rare cases. Individuals with bulimia nervosa may misuse enemas following episodes of binge eating, but this is seldom the sole compensatory method employed. Individuals with this disorder may take thyroid hormone in an attempt to avoid weight gain. Individuals with diabetes mellitus and bulimia nervosa may omit or reduce insulin doses in order to reduce the metabolism of food consumed during eating binges. Individuals with bulimia nervosa may fast for a day or more or exercise excessively in an attempt to prevent weight gain. Exercise may be considered excessive when it significantly interferes with important activities, when it occurs at inappropriate times or in inappropriate settings, or when the individual continues to exercise despite injury or other medical complications.

Individuals with bulimia nervosa place an excessive emphasis on body shape or weight in their self-evaluation, and these factors are typically extremely important in determining

self-esteem (Criterion D). Individuals with this disorder may closely resemble those with anorexia nervosa in their fear of gaining weight, in their desire to lose weight, and in the level of dissatisfaction with their bodies. However, a diagnosis of bulimia nervosa should not be given when the disturbance occurs only during episodes of anorexia nervosa (Criterion E).

Associated Features Supporting Diagnosis

Individuals with bulimia nervosa typically are within the normal weight or overweight range (body mass index [BMI] ≥ 18.5 and < 30 in adults). The disorder occurs but is uncommon among obese individuals. Between eating binges, individuals with bulimia nervosa typically restrict their total caloric consumption and preferentially select low-calorie ("diet") foods while avoiding foods that they perceive to be fattening or likely to trigger a binge.

Menstrual irregularity or amenorrhea often occurs among females with bulimia nervosa; it is uncertain whether such disturbances are related to weight fluctuations, to nutritional deficiencies, or to emotional distress. The fluid and electrolyte disturbances resulting from the purging behavior are sometimes sufficiently severe to constitute medically serious problems. Rare but potentially fatal complications include esophageal tears, gastric rupture, and cardiac arrhythmias. Serious cardiac and skeletal myopathies have been reported among individuals following repeated use of syrup of ipecac to induce vomiting. Individuals who chronically abuse laxatives may become dependent on their use to stimulate bowel movements. Gastrointestinal symptoms are commonly associated with bulimia nervosa, and rectal prolapse has also been reported among individuals with this disorder.

Prevalence

Twelve-month prevalence of bulimia nervosa among young females is 1%–1.5%. Point prevalence is highest among young adults since the disorder peaks in older adolescence and young adulthood. Less is known about the point prevalence of bulimia nervosa in males, but bulimia nervosa is far less common in males than it is in females, with an approximately 10:1 female-to-male ratio.

Development and Course

Bulimia nervosa commonly begins in adolescence or young adulthood. Onset before puberty or after age 40 is uncommon. The binge eating frequently begins during or after an episode of dieting to lose weight. Experiencing multiple stressful life events also can precipitate onset of bulimia nervosa.

Disturbed eating behavior persists for at least several years in a high percentage of clinic samples. The course may be chronic or intermittent, with periods of remission alternating with recurrences of binge eating. However, over longer-term follow-up, the symptoms of many individuals appear to diminish with or without treatment, although treatment clearly impacts outcome. Periods of remission longer than 1 year are associated with better long-term outcome.

Significantly elevated risk for mortality (all-cause and suicide) has been reported for individuals with bulimia nervosa. The CMR (crude mortality rate) for bulimia nervosa is nearly 2% per decade.

Diagnostic cross-over from initial bulimia nervosa to anorexia nervosa occurs in a minority of cases (10%–15%). Individuals who do experience cross-over to anorexia nervosa commonly will revert back to bulimia nervosa or have multiple occurrences of cross-overs between these disorders. A subset of individuals with bulimia nervosa continue to binge eat but no longer engage in inappropriate compensatory behaviors, and therefore their

symptoms meet criteria for binge-eating disorder or other specified eating disorder. Diagnosis should be based on the current (i.e., past 3 months) clinical presentation.

Risk and Prognostic Factors

Temperamental. Weight concerns, low self-esteem, depressive symptoms, social anxiety disorder, and overanxious disorder of childhood are associated with increased risk for the development of bulimia nervosa.

Environmental. Internalization of a thin body ideal has been found to increase risk for developing weight concerns, which in turn increase risk for the development of bulimia nervosa. Individuals who experienced childhood sexual or physical abuse are at increased risk for developing bulimia nervosa.

Genetic and physiological. Childhood obesity and early pubertal maturation increase risk for bulimia nervosa. Familial transmission of bulimia nervosa may be present, as well as genetic vulnerabilities for the disorder.

Course modifiers. Severity of psychiatric comorbidity predicts worse long-term outcome of bulimia nervosa.

Culture-Related Diagnostic Issues

Bulimia nervosa has been reported to occur with roughly similar frequencies in most industrialized countries, including the United States, Canada, many European countries, Australia, Japan, New Zealand, and South Africa. In clinical studies of bulimia nervosa in the United States, individuals presenting with this disorder are primarily white. However, the disorder also occurs in other ethnic groups and with prevalence comparable to estimated prevalences observed in white samples.

Gender-Related Diagnostic Issues

Bulimia nervosa is far more common in females than in males. Males are especially underrepresented in treatment-seeking samples, for reasons that have not yet been systematically examined.

Diagnostic Markers

No specific diagnostic test for bulimia nervosa currently exists. However, several laboratory abnormalities may occur as a consequence of purging and may increase diagnostic certainty. These include fluid and electrolyte abnormalities, such as hypokalemia (which can provoke cardiac arrhythmias), hypochloremia, and hyponatremia. The loss of gastric acid through vomiting may produce a metabolic alkalosis (elevated serum bicarbonate), and the frequent induction of diarrhea or dehydration through laxative and diuretic abuse can cause metabolic acidosis. Some individuals with bulimia nervosa exhibit mildly elevated levels of serum amylase, probably reflecting an increase in the salivary isoenzyme.

Physical examination usually yields no physical findings. However, inspection of the mouth may reveal significant and permanent loss of dental enamel, especially from lingual surfaces of the front teeth due to recurrent vomiting. These teeth may become chipped and appear ragged and "moth-eaten." There may also be an increased frequency of dental caries. In some individuals, the salivary glands, particularly the parotid glands, may become notably enlarged. Individuals who induce vomiting by manually stimulating the gag reflex may develop calluses or scars on the dorsal surface of the hand from repeated contact with the teeth. Serious cardiac and skeletal myopathies have been reported among individuals following repeated use of syrup of ipecac to induce vomiting.

Suicide Risk

Suicide risk is elevated in bulimia nervosa. Comprehensive evaluation of individuals with this disorder should include assessment of suicide-related ideation and behaviors as well as other risk factors for suicide, including a history of suicide attempts.

Functional Consequences of Bulimia Nervosa

Individuals with bulimia nervosa may exhibit a range of functional limitations associated with the disorder. A minority of individuals report severe role impairment, with the social-life domain most likely to be adversely affected by bulimia nervosa.

Differential Diagnosis

Anorexia nervosa, binge-eating/purging type. Individuals whose binge-eating behavior occurs only during episodes of anorexia nervosa are given the diagnosis anorexia nervosa, binge-eating/purging type, and should not be given the additional diagnosis of bulimia nervosa. For individuals with an initial diagnosis of anorexia nervosa who binge and purge but whose presentation no longer meets the full criteria for anorexia nervosa, binge-eating/purging type (e.g., when weight is normal), a diagnosis of bulimia nervosa should be given only when all criteria for bulimia nervosa have been met for at least 3 months.

Binge-eating disorder. Some individuals binge eat but do not engage in regular inappropriate compensatory behaviors. In these cases, the diagnosis of binge-eating disorder should be considered.

Kleine-Levin syndrome. In certain neurological or other medical conditions, such as Kleine-Levin syndrome, there is disturbed eating behavior, but the characteristic psychological features of bulimia nervosa, such as overconcern with body shape and weight, are not present.

Major depressive disorder, with atypical features. Overeating is common in major depressive disorder, with atypical features, but individuals with this disorder do not engage in inappropriate compensatory behaviors and do not exhibit the excessive concern with body shape and weight characteristic of bulimia nervosa. If criteria for both disorders are met, both diagnoses should be given.

Borderline personality disorder. Binge-eating behavior is included in the impulsive behavior criterion that is part of the definition of borderline personality disorder. If the criteria for both borderline personality disorder and bulimia nervosa are met, both diagnoses should be given.

Comorbidity

Comorbidity with mental disorders is common in individuals with bulimia nervosa, with most experiencing at least one other mental disorder and many experiencing multiple comorbidities. Comorbidity is not limited to any particular subset but rather occurs across a wide range of mental disorders. There is an increased frequency of depressive symptoms (e.g., low self-esteem) and bipolar and depressive disorders (particularly depressive disorders) in individuals with bulimia nervosa. In many individuals, the mood disturbance begins at the same time as or following the development of bulimia nervosa, and individuals often ascribe their mood disturbances to the bulimia nervosa. However, in some individuals, the mood disturbance clearly precedes the development of bulimia nervosa. There may also be an increased frequency of anxiety symptoms (e.g., fear of social situations) or anxiety disorders. These mood and anxiety disturbances frequently remit follow-

ing effective treatment of the bulimia nervosa. The lifetime prevalence of substance use, particularly alcohol or stimulant use, is at least 30% among individuals with bulimia nervosa. Stimulant use often begins in an attempt to control appetite and weight. A substantial percentage of individuals with bulimia nervosa also have personality features that meet criteria for one or more personality disorders, most frequently borderline personality disorder.

Binge-Eating Disorder

Diagnostic Criteria **307.51 (F50.8)**

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
   1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than what most people would eat in a similar period of time under similar circumstances.
   2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).

B. The binge-eating episodes are associated with three (or more) of the following:
   1. Eating much more rapidly than normal.
   2. Eating until feeling uncomfortably full.
   3. Eating large amounts of food when not feeling physically hungry.
   4. Eating alone because of feeling embarrassed by how much one is eating.
   5. Feeling disgusted with oneself, depressed, or very guilty afterward.

C. Marked distress regarding binge eating is present.

D. The binge eating occurs, on average, at least once a week for 3 months.

E. The binge eating is not associated with the recurrent use of inappropriate compensatory behavior as in bulimia nervosa and does not occur exclusively during the course of bulimia nervosa or anorexia nervosa.

Specify if:

In partial remission: After full criteria for binge-eating disorder were previously met, binge eating occurs at an average frequency of less than one episode per week for a sustained period of time.

In full remission: After full criteria for binge-eating disorder were previously met, none of the criteria have been met for a sustained period of time.

Specify current severity:

The minimum level of severity is based on the frequency of episodes of binge eating (see below). The level of severity may be increased to reflect other symptoms and the degree of functional disability.

Mild: 1–3 binge-eating episodes per week.

Moderate: 4–7 binge-eating episodes per week.

Severe: 8–13 binge-eating episodes per week.

Extreme: 14 or more binge-eating episodes per week.

Diagnostic Features

The essential feature of binge-eating disorder is recurrent episodes binge eating that must occur, on average, at least once per week for 3 months (Criterion D). An "episode of binge eating" is defined as eating, in a discrete period of time, an amount of food that is defi-

nitely larger than most people would eat in a similar period of time under similar circumstances (Criterion A1). The context in which the eating occurs may affect the clinician's estimation of whether the intake is excessive. For example, a quantity of food that might be regarded as excessive for a typical meal might be considered normal during a celebration or holiday meal. A "discrete period of time" refers to a limited period, usually less than 2 hours. A single episode of binge eating need not be restricted to one setting. For example, an individual may begin a binge in a restaurant and then continue to eat on returning home. Continual snacking on small amounts of food throughout the day would not be considered an eating binge.

An occurrence of excessive food consumption must be accompanied by a sense of lack of control (Criterion A2) to be considered an episode of binge eating. An indicator of loss of control is the inability to refrain from eating or to stop eating once started. Some individuals describe a dissociative quality during, or following, the binge-eating episodes. The impairment in control associated with binge eating may not be absolute; for example, an individual may continue binge eating while the telephone is ringing but will cease if a roommate or spouse unexpectedly enters the room. Some individuals report that their binge-eating episodes are no longer characterized by an acute feeling of loss of control but rather by a more generalized pattern of uncontrolled eating. If individuals report that they have abandoned efforts to control their eating, loss of control may still be considered as present. Binge eating can also be planned in some instances.

The type of food consumed during binges varies both across individuals and for a given individual. Binge eating appears to be characterized more by an abnormality in the amount of food consumed than by a craving for a specific nutrient.

Binge eating must be characterized by marked distress (Criterion C) and at least three of the following features: eating much more rapidly than normal; eating until feeling uncomfortably full; eating large amounts of food when not feeling physically hungry; eating alone because of feeling embarrassed by how much one is eating; and feeling disgusted with oneself, depressed, or very guilty afterward (Criterion B).

Individuals with binge-eating disorder are typically ashamed of their eating problems and attempt to conceal their symptoms. Binge eating usually occurs in secrecy or as inconspicuously as possible. The most common antecedent of binge eating is negative affect. Other triggers include interpersonal stressors; dietary restraint; negative feelings related to body weight, body shape, and food; and boredom. Binge eating may minimize or mitigate factors that precipitated the episode in the short-term, but negative self-evaluation and dysphoria often are the delayed consequences.

Associated Features Supporting Diagnosis

Binge-eating disorder occurs in normal-weight/overweight and obese individuals. It is reliably associated with overweight and obesity in treatment-seeking individuals. Nevertheless, binge-eating disorder is distinct from obesity. Most obese individuals do not engage in recurrent binge eating. In addition, compared with weight-matched obese individuals without binge-eating disorder, those with the disorder consume more calories in laboratory studies of eating behavior and have greater functional impairment, lower quality of life, more subjective distress, and greater psychiatric comorbidity.

Prevalence

Twelve-month prevalence of binge-eating disorder among U.S. adult (age 18 or older) females and males is 1.6% and 0.8%, respectively. The gender ratio is far less skewed in binge-eating disorder than in bulimia nervosa. Binge-eating disorder is as prevalent among females from racial or ethnic minority groups as has been reported for white females. The disorder is more prevalent among individuals seeking weight-loss treatment than in the general population.

Development and Course

Little is known about the development of binge-eating disorder. Both binge eating and loss-of-control eating without objectively excessive consumption occur in children and are associated with increased body fat, weight gain, and increases in psychological symptoms. Binge eating is common in adolescent and college-age samples. Loss-of-control eating or episodic binge eating may represent a prodromal phase of eating disorders for some individuals.

Dieting follows the development of binge eating in many individuals with binge-eating disorder. (This is in contrast to bulimia nervosa, in which dysfunctional dieting usually precedes the onset of binge eating.) Binge-eating disorder typically begins in adolescence or young adulthood but can begin in later adulthood. Individuals with binge-eating disorder who seek treatment usually are older than individuals with either bulimia nervosa or anorexia nervosa who seek treatment.

Remission rates in both natural course and treatment outcome studies are higher for binge-eating disorder than for bulimia nervosa or anorexia nervosa. Binge-eating disorder appears to be relatively persistent, and the course is comparable to that of bulimia nervosa in terms of severity and duration. Crossover from binge-eating disorder to other eating disorders is uncommon.

Risk and Prognostic Factors

Genetic and physiological. Binge-eating disorder appears to run in families, which may reflect additive genetic influences.

Culture-Reiated Diagnostic Issues

Binge-eating disorder occurs with roughly similar frequencies in most industrialized countries, including the United States, Canada, many European countries, Australia, and New Zealand. In the United States, the prevalence of binge-eating disorder appears comparable among non-Latino whites, Latinos, Asians, and African Americans.

Functionai Consequences of Binge-Eating Disorder

Binge-eating disorder is associated with a range of functional consequences, including social role adjustment problems, impaired health-related quality of life and life satisfaction, increased medical morbidity and mortality, and associated increased health care utilization compared with body mass index (BMI)–matched control subjects. It may also be associated with an increased risk for weight gain and the development of obesity.

Differential Diagnosis

Bulimia nervosa. Binge-eating disorder has recurrent binge eating in common with bulimia nervosa but differs from the latter disorder in some fundamental respects. In terms of clinical presentation, the recurrent inappropriate compensatory behavior (e.g., purging, driven exercise) seen in bulimia nervosa is absent in binge-eating disorder. Unlike individuals with bulimia nervosa, individuals with binge-eating disorder typically do not show marked or sustained dietary restriction designed to influence body weight and shape between binge-eating episodes. They may, however, report frequent attempts at dieting. Binge-eating disorder also differs from bulimia nervosa in terms of response to treatment. Rates of improvement are consistently higher among individuals with binge-eating disorder than among those with bulimia nervosa.

Obesity. Binge-eating disorder is associated with overweight and obesity but has several key features that are distinct from obesity. First, levels of overvaluation of body

weight and shape are higher in obese individuals with the disorder than in those without the disorder. Second, rates of psychiatric comorbidity are significantly higher among obese individuals with the disorder compared with those without the disorder. Third, the long-term successful outcome of evidence-based psychological treatments for binge-eating disorder can be contrasted with the absence of effective long-term treatments for obesity.

Bipolar and depressive disorders. Increases in appetite and weight gain are included in the criteria for major depressive episode and in the atypical features specifiers for depressive and bipolar disorders. Increased eating in the context of a major depressive episode may or may not be associated with loss of control. If the full criteria for both disorders are met, both diagnoses can be given. Binge eating and other symptoms of disordered eating are seen in association with bipolar disorder. If the full criteria for both disorders are met, both diagnoses should be given.

Borderline personality disorder. Binge eating is included in the impulsive behavior criterion that is part of the definition of borderline personality disorder. If the full criteria for both disorders are met, both diagnoses should be given.

Comorbidity

Binge-eating disorder is associated with significant psychiatric comorbidity that is comparable to that of bulimia nervosa and anorexia nervosa. The most common comorbid disorders are bipolar disorders, depressive disorders, anxiety disorders, and, to a lesser degree, substance use disorders. The psychiatric comorbidity is linked to the severity of binge eating and not to the degree of obesity.

Other Specified Feeding or Eating Disorder

307.59 (F50.8)

This category applies to presentations in which symptoms characteristic of a feeding and eating disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the feeding and eating disorders diagnostic class. The other specified feeding or eating disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific feeding and eating disorder. This is done by recording "other specified feeding or eating disorder" followed by the specific reason (e.g., "bulimia nervosa of low frequency").

Examples of presentations that can be specified using the "other specified" designation include the following:

1. **Atypical anorexia nervosa:** All of the criteria for anorexia nervosa are met, except that despite significant weight loss, the individual's weight is within or above the normal range.
2. **Bulimia nervosa (of low frequency and/or limited duration):** All of the criteria for bulimia nervosa are met, except that the binge eating and inappropriate compensatory behaviors occur, on average, less than once a week and/or for less than 3 months.
3. **Binge-eating disorder (of low frequency and/or limited duration):** All of the criteria for binge-eating disorder are met, except that the binge eating occurs, on average, less than once a week and/or for less than 3 months.
4. **Purging disorder:** Recurrent purging behavior to influence weight or shape (e.g., self-induced vomiting; misuse of laxatives, diuretics, or other medications) in the absence of binge eating.

5. **Night eating syndrome:** Recurrent episodes of night eating, as manifested by eating after awakening from sleep or by excessive food consumption after the evening meal. There is awareness and recall of the eating. The night eating is not better explained by external influences such as changes in the individual's sleep-wake cycle or by local social norms. The night eating causes significant distress and/or impairment in functioning. The disordered pattern of eating is not better explained by binge-eating disorder or another mental disorder, including substance use, and is not attributable to another medical disorder or to an effect of medication.

Unspecified Feeding or Eating Disorder

307.50 (F50.9)

This category applies to presentations in which symptoms characteristic of a feeding and eating disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the feeding and eating disorders diagnostic class. The unspecified feeding and eating disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific feeding and eating disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Elimination Disorders

Elimination disorders all involve the inappropriate elimination of urine or feces and are usually first diagnosed in childhood or adolescence. This group of disorders includes *enuresis*, the repeated voiding of urine into inappropriate places, and *encopresis*, the repeated passage of feces into inappropriate places. Subtypes are provided to differentiate nocturnal from diurnal (i.e., during waking hours) voiding for enuresis and the presence or absence of constipation and overflow incontinence for encopresis. Although there are minimum age requirements for diagnosis of both disorders, these are based on developmental age and not solely on chronological age. Both disorders may be voluntary or involuntary. Although these disorders typically occur separately, co-occurrence may also be observed.

Enuresis

Diagnostic Criteria — 307.6 (F98.0)

A. Repeated voiding of urine into bed or clothes, whether involuntary or intentional.

B. The behavior is clinically significant as manifested by either a frequency of at least twice a week for at least 3 consecutive months or the presence of clinically significant distress or impairment in social, academic (occupational), or other important areas of functioning.

C. Chronological age is at least 5 years (or equivalent developmental level).

D. The behavior is not attributable to the physiological effects of a substance (e.g., a diuretic, an antipsychotic medication) or another medical condition (e.g., diabetes, spina bifida, a seizure disorder).

Specify whether:

Nocturnal only: Passage of urine only during nighttime sleep.

Diurnal only: Passage of urine during waking hours.

Nocturnal and diurnal: A combination of the two subtypes above.

Subtypes

The nocturnal-only subtype of enuresis, sometimes referred to as *monosymptomatic enuresis*, is the most common subtype and involves incontinence only during nighttime sleep, typically during the first one-third of the night. The diurnal-only subtype occurs in the absence of nocturnal enuresis and may be referred to simply as *urinary incontinence*. Individuals with this subtype can be divided into two groups. Individuals with "urge incontinence" have sudden urge symptoms and detrusor instability, whereas individuals with "voiding postponement" consciously defer micturition urges until incontinence results. The nocturnal-and-diurnal subtype is also known as *nonmonosymptomatic enuresis*.

Diagnostic Features

The essential feature of enuresis is repeated voiding of urine during the day or at night into bed or clothes (Criterion A). Most often the voiding is involuntary, but occasionally it may

be intentional. To qualify for a diagnosis of enuresis, the voiding of urine must occur at least twice a week for at least 3 consecutive months or must cause clinically significant distress or impairment in social, academic (occupational), or other important areas of functioning (Criterion B). The individual must have reached an age at which continence is expected (i.e., a chronological age of at least 5 years or, for children with developmental delays, a mental age of at least 5 years) (Criterion C). The urinary incontinence is not attributable to the physiological effects of a substance (e.g., a diuretic, an antipsychotic medication) or another medical condition (e.g., diabetes, spina bifida, a seizure disorder) (Criterion D).

Associated Features Supporting Diagnosis

During nocturnal enuresis, occasionally the voiding takes place during rapid eye movement (REM) sleep, and the child may recall a dream that involved the act of urinating. During daytime (diurnal) enuresis, the child defers voiding until incontinence occurs, sometimes because of a reluctance to use the toilet as a result of social anxiety or a preoccupation with school or play activity. The enuretic event most commonly occurs in the early afternoon on school days and may be associated with symptoms of disruptive behavior. The enuresis commonly persists after appropriate treatment of an associated infection.

Prevalence

The prevalence of enuresis is 5%–10% among 5-year-olds, 3%–5% among 10-year-olds, and around 1% among individuals 15 years or older.

Development and Course

Two types of course of enuresis have been described: a "primary" type, in which the individual has never established urinary continence, and a "secondary" type, in which the disturbance develops after a period of established urinary continence. There are no differences in prevalence of comorbid mental disorders between the two types. By definition, primary enuresis begins at age 5 years. The most common time for the onset of secondary enuresis is between ages 5 and 8 years, but it may occur at any time. After age 5 years, the rate of spontaneous remission is 5%–10% per year. Most children with the disorder become continent by adolescence, but in approximately 1% of cases the disorder continues into adulthood. Diurnal enuresis is uncommon after age 9 years. While occasional diurnal incontinence is not uncommon in middle childhood, it is substantially more common in those who also have persistent nocturnal enuresis. When enuresis persists into late childhood or adolescence, the frequency of incontinence may increase, whereas continence in early childhood is usually associated with a declining frequency of wet nights.

Risk and Prognostic Factors

Environmental. A number of predisposing factors for enuresis have been suggested, including delayed or lax toilet training and psychosocial stress.

Genetic and physiological. Enuresis has been associated with delays in the development of normal circadian rhythms of urine production, with resulting nocturnal polyuria or abnormalities of central vasopressin receptor sensitivity, and reduced functional bladder capacities with bladder hyperreactivity (unstable bladder syndrome). Nocturnal enuresis is a genetically heterogeneous disorder. Heritability has been shown in family, twin, and segregation analyses. Risk for childhood nocturnal enuresis is approximately 3.6 times higher in offspring of enuretic mothers and 10.1 times higher in the presence of paternal urinary incontinence. The risk magnitudes for nocturnal enuresis and diurnal incontinence are similar.

Culture-Related Diagnostic Issues

Enuresis has been reported in a variety of European, African, and Asian countries as well as in the United States. At a national level, prevalence rates are remarkably similar, and there is great similarity in the developmental trajectories found in different countries. There are very high rates of enuresis in orphanages and other residential institutions, likely related to the mode and environment in which toilet training occurs.

Gender-Related Diagnostic Issues

Nocturnal enuresis is more common in males. Diurnal incontinence is more common in females. The relative risk of having a child who develops enuresis is greater for previously enuretic fathers than for previously enuretic mothers.

Functional Consequences of Enuresis

The amount of impairment associated with enuresis is a function of the limitation on the child's social activities (e.g., ineligibility for sleep-away camp) or its effect on the child's self-esteem, the degree of social ostracism by peers, and the anger, punishment, and rejection on the part of caregivers.

Differential Diagnosis

Neurogenic bladder or another medical condition. The diagnosis of enuresis is not made in the presence of a neurogenic bladder or another medical condition that causes polyuria or urgency (e.g., untreated diabetes mellitus or diabetes insipidus) or during an acute urinary tract infection. However, a diagnosis is compatible with such conditions if urinary incontinence was regularly present prior to the development of another medical condition or if it persists after the institution of appropriate treatment of the medical condition.

Medication side effects. Enuresis may occur during treatment with antipsychotic medications, diuretics, or other medications that may induce incontinence. In this case, the diagnosis should not be made in isolation but may be noted as a medication side effect. However, a diagnosis of enuresis may be made if urinary incontinence was regularly present prior to treatment with the medication.

Comorbidity

Although most children with enuresis do not have a comorbid mental disorder, the prevalence of comorbid behavioral symptoms is higher in children with enuresis than in children without enuresis. Developmental delays, including speech, language, learning, and motor skills delays, are also present in a portion of children with enuresis. Encopresis, sleepwalking, and sleep terror disorder may be present. Urinary tract infections are more common in children with enuresis, especially the diurnal subtype, than in those who are continent.

Encopresis

Diagnostic Criteria **307.7 (F98.1)**

A. Repeated passage of feces into inappropriate places (e.g., clothing, floor), whether involuntary or intentional.

B. At least one such event occurs each month for at least 3 months.

C. Chronological age is at least 4 years (or equivalent developmental level).

D. The behavior is not attributable to the physiological effects of a substance (e.g., laxatives) or another medical condition except through a mechanism involving constipation.

Specify whether:

With constipation and overflow incontinence: There is evidence of constipation on physical examination or by history.

Without constipation and overflow incontinence: There is no evidence of constipation on physical examination or by history.

Subtypes

Feces in the with constipation and overflow incontinence subtype are characteristically (but not invariably) poorly formed, and leakage can be infrequent to continuous, occurring mostly during the day and rarely during sleep. Only part of the feces is passed during toileting, and the incontinence resolves after treatment of the constipation.

In the without constipation and overflow incontinence subtype, feces are likely to be of normal form and consistency, and soiling is intermittent. Feces may be deposited in a prominent location. This is usually associated with the presence of oppositional defiant disorder or conduct disorder or may be the consequence of anal masturbation. Soiling without constipation appears to be less common than soiling with constipation.

Diagnostic Features

The essential feature of encopresis is repeated passage of feces into inappropriate places (e.g., clothing or floor) (Criterion A). Most often the passage is involuntary but occasionally may be intentional. The event must occur at least once a month for at least 3 months (Criterion B), and the chronological age of the child must be at least 4 years (or for children with developmental delays, the mental age must be at least 4 years) (Criterion C). The fecal incontinence must not be exclusively attributable to the physiological effects of a substance (e.g., laxatives) or another medical condition except through a mechanism involving constipation (Criterion D).

When the passage of feces is involuntary rather than intentional, it is often related to constipation, impaction, and retention with subsequent overflow. The constipation may develop for psychological reasons (e.g., anxiety about defecating in a particular place, a more general pattern of anxious or oppositional behavior), leading to avoidance of defecation. Physiological predispositions to constipation include ineffectual straining or paradoxical defecation dynamics, with contraction rather than relaxation of the external sphincter or pelvic floor during straining for defecation. Dehydration associated with a febrile illness, hypothyroidism, or a medication side effect can also induce constipation. Once constipation has developed, it may be complicated by an anal fissure, painful defecation, and further fecal retention. The consistency of the stool may vary. In some individuals the stool may be of normal or near-normal consistency. In other individuals—such as those with overflow incontinence secondary to fecal retention—it may be liquid.

Associated Features Supporting Diagnosis

The child with encopresis often feels ashamed and may wish to avoid situations (e.g., camp, school) that might lead to embarrassment. The amount of impairment is a function of the effect on the child's self-esteem, the degree of social ostracism by peers, and the anger, punishment, and rejection on the part of caregivers. Smearing feces may be deliberate or accidental, resulting from the child's attempt to clean or hide feces that were passed involuntarily. When the incontinence is clearly deliberate, features of oppositional defiant disorder or conduct disorder may also be present. Many children with encopresis and chronic constipation also have enuresis symptoms and may have associated urinary reflux in the bladder or ureters that may lead to chronic urinary infections, the symptoms of which may remit with treatment of the constipation.

Prevalence

It is estimated that approximately 1% of 5-year-olds have encopresis, and the disorder is more common in males than in females.

Development and Course

Encopresis is not diagnosed until a child has reached a chronological age of at least 4 years (or for children with developmental delays, a mental age of at least 4 years). Inadequate, inconsistent toilet training and psychosocial stress (e.g., entering school, the birth of a sibling) may be predisposing factors. Two types of course have been described: a "primary" type, in which the individual has never established fecal continence, and a "secondary" type, in which the disturbance develops after a period of established fecal continence. Encopresis can persist, with intermittent exacerbations, for years.

Risk and Prognostic Factors

Genetic and physiological. Painful defecation can lead to constipation and a cycle of withholding behaviors that make encopresis more likely. Use of some medications (e.g., anticonvulsants, cough suppressants) may increase constipation and make encopresis more likely.

Diagnostic Markers

In addition to physical examination, gastrointestinal imaging (e.g., abdominal radiograph) may be informative to assess retained stool and gas in the colon. Additional tests, such as barium enema and anorectal manography, may be used to help exclude other medical conditions, such as Hirschsprung's disease.

Differential Diagnosis

A diagnosis of encopresis in the presence of another medical condition is appropriate only if the mechanism involves constipation that cannot be explained by other medical conditions. Fecal incontinence related to other medical conditions (e.g., chronic diarrhea, spina bifida, anal stenosis) would not warrant a DSM-5 diagnosis of encopresis.

Comorbidity

Urinary tract infections can be comorbid with encopresis and are more common in females.

Other Specified Elimination Disorder

This category applies to presentations in which symptoms characteristic of an elimination disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the elimination disorders diagnostic class. The other specified elimination disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific elimination disorder. This is done by recording "other specified elimination disorder" followed by the specific reason (e.g., "low-frequency enuresis").

Coding note: Code **788.39 (N39.498)** for other specified elimination disorder with urinary symptoms; **787.60 (R15.9)** for other specified elimination disorder with fecal symptoms.

Unspecified Elimination Disorder

This category applies to presentations in which symptoms characteristic of an elimination disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the elimination disorders diagnostic class. The unspecified elimination disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific elimination disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Coding note: Code **788.30 (R32)** for unspecified elimination disorder with urinary symptoms; **787.60 (R15.9)** for unspecified elimination disorder with fecal symptoms.

Sleep-Wake Disorders

The DSM-5 classification of sleep-wake disorders is intended for use by general mental health and medical clinicians (those caring for adult, geriatric, and pediatric patients). Sleep-wake disorders encompass 10 disorders or disorder groups: insomnia disorder, hypersomnolence disorder, narcolepsy, breathing-related sleep disorders, circadian rhythm sleep-wake disorders, non–rapid eye movement (NREM) sleep arousal disorders, nightmare disorder, rapid eye movement (REM) sleep behavior disorder, restless legs syndrome, and substance/medication-induced sleep disorder. Individuals with these disorders typically present with sleep-wake complaints of dissatisfaction regarding the quality, timing, and amount of sleep. Resulting daytime distress and impairment are core features shared by all of these sleep-wake disorders.

The organization of this chapter is designed to facilitate differential diagnosis of sleep-wake complaints and to clarify when referral to a sleep specialist is appropriate for further assessment and treatment planning. The DSM-5 sleep disorders nosology uses a simple, clinically useful approach, while also reflecting scientific advances in epidemiology, genetics, pathophysiology, assessment, and interventions research since DSM-IV. In some cases (e.g., insomnia disorder), a "lumping" approach has been adopted, whereas in others (e.g., narcolepsy), a "splitting" approach has been taken, reflecting the availability of validators derived from epidemiological, neurobiological, and interventions research.

Sleep disorders are often accompanied by depression, anxiety, and cognitive changes that must be addressed in treatment planning and management. Furthermore, persistent sleep disturbances (both insomnia and excessive sleepiness) are established risk factors for the subsequent development of mental illnesses and substance use disorders. They may also represent a prodromal expression of an episode of mental illness, allowing the possibility of early intervention to preempt or to attenuate a full-blown episode.

The differential diagnosis of sleep-wake complaints necessitates a multidimensional approach, with consideration of possibly coexisting medical and neurological conditions. Coexisting clinical conditions are the rule, not the exception. Sleep disturbances furnish a clinically useful indicator of medical and neurological conditions that often coexist with depression and other common mental disorders. Prominent among these comorbidities are breathing-related sleep disorders, disorders of the heart and lungs (e.g., congestive heart failure, chronic obstructive pulmonary disease), neurodegenerative disorders (e.g., Alzheimer's disease), and disorders of the musculoskeletal system (e.g., osteoarthritis). These disorders not only may disturb sleep but also may themselves be worsened during sleep (e.g., prolonged apneas or electrocardiographic arrhythmias during REM sleep; confusional arousals in patients with dementing illness; seizures in persons with complex partial seizures). REM sleep behavior disorder is often an early indicator of neurodegenerative disorders (alpha synucleinopathies) like Parkinson's disease. For all of these reasons—related to differential diagnosis, clinical comorbidity, and facilitation of treatment planning—sleep disorders are included in DSM-5.

The approach taken to the classification of sleep-wake disorders in DSM-5 can be understood within the context of "lumping versus splitting." DSM-IV represented an effort to simplify sleep-wake disorders classification and thus aggregated diagnoses under broader, less differentiated labels. At the other pole, the *International Classification of Sleep Disorders*,

2nd Edition (ICSD-2) elaborated numerous diagnostic subtypes. DSM-IV was prepared for use by mental health and general medical clinicians who are not experts in sleep medicine. ICSD-2 reflected the science and opinions of the sleep specialist community and was prepared for use by specialists.

The weight of available evidence supports the superior performance characteristics (interrater reliability, as well as convergent, discriminant, and face validity) of simpler, less-differentiated approaches to diagnosis of sleep-wake disorders. The text accompanying each set of diagnostic criteria provides linkages to the corresponding disorders included in ICSD-2. The DSM-5 sleep-wake disorders classification also specifies corresponding non-psychiatric listings (e.g., neurology codes) from the *International Classification of Diseases* (ICD).

The field of sleep disorders medicine has progressed in this direction since the publication of DSM-IV. The use of biological validators is now embodied in the DSM-5 classification of sleep-wake disorders, particularly for disorders of excessive sleepiness, such as narcolepsy; for breathing-related sleep disorders, for which formal sleep studies (i.e., polysomnography) are indicated; and for restless legs syndrome, which can often coexist with periodic limb movements during sleep, detectable via polysomnography.

Insomnia Disorder

Diagnostic Criteria **780.52 (G47.00)**

A. A predominant complaint of dissatisfaction with sleep quantity or quality, associated with one (or more) of the following symptoms:
   1. Difficulty initiating sleep. (In children, this may manifest as difficulty initiating sleep without caregiver intervention.)
   2. Difficulty maintaining sleep, characterized by frequent awakenings or problems returning to sleep after awakenings. (In children, this may manifest as difficulty returning to sleep without caregiver intervention.)
   3. Early-morning awakening with inability to return to sleep.

B. The sleep disturbance causes clinically significant distress or impairment in social, occupational, educational, academic, behavioral, or other important areas of functioning.

C. The sleep difficulty occurs at least 3 nights per week.

D. The sleep difficulty is present for at least 3 months.

E. The sleep difficulty occurs despite adequate opportunity for sleep.

F. The insomnia is not better explained by and does not occur exclusively during the course of another sleep-wake disorder (e.g., narcolepsy, a breathing-related sleep disorder, a circadian rhythm sleep-wake disorder, a parasomnia).

G. The insomnia is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication).

H. Coexisting mental disorders and medical conditions do not adequately explain the predominant complaint of insomnia.

Specify if:

With non–sleep disorder mental comorbidity, including substance use disorders

With other medical comorbidity

With other sleep disorder

Coding note: The code 780.52 (G47.00) applies to all three specifiers. Code also the relevant associated mental disorder, medical condition, or other sleep disorder immediately after the code for insomnia disorder in order to indicate the association.

Specify if:

Episodic: Symptoms last at least 1 month but less than 3 months.

Persistent: Symptoms last 3 months or longer.

Recurrent: Two (or more) episodes within the space of 1 year.

Note: Acute and short-term insomnia (i.e., symptoms lasting less than 3 months but otherwise meeting all criteria with regard to frequency, intensity, distress, and/or impairment) should be coded as an other specified insomnia disorder.

Note. The diagnosis of insomnia disorder is given whether it occurs as an independent condition or is comorbid with another mental disorder (e.g., major depressive disorder), medical condition (e.g., pain), or another sleep disorder (e.g., a breathing-related sleep disorder). For instance, insomnia may develop its own course with some anxiety and depressive features but in the absence of criteria being met for any one mental disorder. Insomnia may also manifest as a clinical feature of a more predominant mental disorder. Persistent insomnia may even be a risk factor for depression and is a common residual symptom after treatment for this condition. With comorbid insomnia and a mental disorder, treatment may also need to target both conditions. Given these different courses, it is often impossible to establish the precise nature of the relationship between these clinical entities, and this relationship may change over time. Therefore, in the presence of insomnia and a comorbid disorder, it is not necessary to make a causal attribution between the two conditions. Rather, the diagnosis of insomnia disorder is made with concurrent specification of the clinically comorbid conditions. A concurrent insomnia diagnosis should only be considered when the insomnia is sufficiently severe to warrant independent clinical attention; otherwise, no separate diagnosis is necessary.

Diagnostic Features

The essential feature of insomnia disorder is dissatisfaction with sleep quantity or quality with complaints of difficulty initiating or maintaining sleep. The sleep complaints are accompanied by clinically significant distress or impairment in social, occupational, or other important areas of functioning. The sleep disturbance may occur during the course of another mental disorder or medical condition, or it may occur independently.

Different manifestations of insomnia can occur at different times of the sleep period. *Sleep-onset insomnia* (or *initial insomnia*) involves difficulty initiating sleep at bedtime. *Sleep maintenance insomnia* (or *middle insomnia*) involves frequent or prolonged awakenings throughout the night. *Late insomnia* involves early-morning awakening with an inability to return to sleep. Difficulty maintaining sleep is the most common single symptom of insomnia, followed by difficulty falling asleep, while a combination of these symptoms is the most common presentation overall. The specific type of sleep complaint often varies over time. Individuals who complain of difficulty falling asleep at one time may later complain of difficulty maintaining sleep, and vice versa. Symptoms of difficulty falling asleep and difficulty maintaining sleep can be quantified by the individual's retrospective self-report, sleep diaries, or other methods, such as actigraphy or polysomnography, but the diagnosis of insomnia disorder is based on the individual's subjective perception of sleep or a caretaker's report.

Nonrestorative sleep, a complaint of poor sleep quality that does not leave the individual rested upon awakening despite adequate duration, is a common sleep complaint usually occurring in association with difficulty initiating or maintaining sleep, or less frequently in isolation. This complaint can also be reported in association with other sleep disorders (e.g., breathing-related sleep disorder). When a complaint of nonrestorative sleep occurs in isolation (i.e., in the absence of difficulty initiating and/or maintaining sleep) but all diagnostic criteria with regard to frequency, duration, and daytime distress and impairments are otherwise met, a diagnosis of other specified insomnia disorder or unspecified insomnia disorder is made.

Aside from the frequency and duration criteria required to make the diagnosis, additional criteria are useful to quantify insomnia severity. These quantitative criteria, while arbitrary, are provided for illustrative purpose only. For instance, difficulty initiating sleep is defined by a subjective sleep latency greater than 20–30 minutes, and difficulty maintaining sleep is defined by a subjective time awake after sleep onset greater than 20–30 minutes. Although there is no standard definition of early-morning awakening, this symptom involves awakening at least 30 minutes before the scheduled time and before total sleep time reaches 6½ hours. It is essential to take into account not only the final awakening time but also the bedtime on the previous evening. Awakening at 4:00 A.M. does not have the same clinical significance in those who go to bed at 9:00 P.M. as in those who go to bed at 11:00 P.M. Such a symptom may also reflect an age-dependent decrease in the ability to sustain sleep or an age-dependent shift in the timing of the main sleep period.

Insomnia disorder involves daytime impairments as well as nighttime sleep difficulties. These include fatigue or, less commonly, daytime sleepiness; the latter is more common among older individuals and when insomnia is comorbid with another medical condition (e.g., chronic pain) or sleep disorder (e.g., sleep apnea). Impairment in cognitive performance may include difficulties with attention, concentration and memory, and even with performing simple manual skills. Associated mood disturbances are typically described as irritability or mood lability and less commonly as depressive or anxiety symptoms. Not all individuals with nighttime sleep disturbances are distressed or have functional impairment. For example, sleep continuity is often interrupted in healthy older adults who nevertheless identify themselves as good sleepers. A diagnosis of insomnia disorder should be reserved for those individuals with significant daytime distress or impairment related to their nighttime sleep difficulties.

Associated Features Supporting Diagnosis

Insomnia is often associated with physiological and cognitive arousal and conditioning factors that interfere with sleep. A preoccupation with sleep and distress due to the inability to sleep may lead to a vicious cycle: the more the individual strives to sleep, the more frustration builds and further impairs sleep. Thus, excessive attention and efforts to sleep, which override normal sleep-onset mechanisms, may contribute to the development of insomnia. Individuals with persistent insomnia may also acquire maladaptive sleep habits (e.g., spending excessive time in bed; following an erratic sleep schedule; napping) and cognitions (e.g., fear of sleeplessness; apprehensions of daytime impairments; clock monitoring) during the course of the disorder. Engaging in such activities in an environment in which the individual has frequently spent sleepless nights may further compound the conditioned arousal and perpetuate sleep difficulties. Conversely, the individual may fall asleep more easily when not trying to do so. Some individuals also report better sleep when away from their own bedrooms and their usual routines.

Insomnia may be accompanied by a variety of daytime complaints and symptoms, including fatigue, decreased energy, and mood disturbances. Symptoms of anxiety or depression that do not meet criteria for a specific mental disorder may be present, as well as an excessive focus on the perceived effects of sleep loss on daytime functioning.

Individuals with insomnia may have elevated scores on self-report psychological or personality inventories with profiles indicating mild depression and anxiety, a worrisome cognitive style, an emotion-focused and internalizing style of conflict resolution, and a somatic focus. Patterns of neurocognitive impairment among individuals with insomnia disorder are inconsistent, although there may be impairments in performing tasks of higher complexity and those requiring frequent changes in performance strategy. Individuals with insomnia often require more effort to maintain cognitive performance.

Prevalence

Population-based estimates indicate that about one-third of adults report insomnia symptoms, 10%–15% experience associated daytime impairments, and 6%–10% have symptoms

that meet criteria for insomnia disorder. Insomnia disorder is the most prevalent of all sleep disorders. In primary care settings, approximately 10%–20% of individuals complain of significant insomnia symptoms. Insomnia is a more prevalent complaint among females than among males, with a gender ratio of about 1.44:1. Although insomnia can be a symptom or an independent disorder, it is most frequently observed as a comorbid condition with another medical condition or mental disorder. For instance, 40%–50% of individuals with insomnia also present with a comorbid mental disorder.

Development and Course

The onset of insomnia symptoms can occur at any time during life, but the first episode is more common in young adulthood. Less frequently, insomnia begins in childhood or adolescence. In women, new-onset insomnia may occur during menopause and persist even after other symptoms (e.g., hot flashes) have resolved. Insomnia may have a late-life onset, which is often associated with the onset of other health-related conditions.

Insomnia can be situational, persistent, or recurrent. Situational or acute insomnia usually lasts a few days or a few weeks and is often associated with life events or rapid changes in sleep schedules or environment. It usually resolves once the initial precipitating event subsides. For some individuals, perhaps those more vulnerable to sleep disturbances, insomnia may persist long after the initial triggering event, possibly because of conditioning factors and heightened arousal. The factors that precipitate insomnia may differ from those that perpetuate it. For example, an individual who is bedridden with a painful injury and has difficulty sleeping may then develop negative associations for sleep. Conditioned arousal may then persist and lead to persistent insomnia. A similar course may develop in the context of an acute psychological stress or a mental disorder. For instance, insomnia that occurs during an episode of major depressive disorder can become a focus of attention, with consequent negative conditioning, and persist even after resolution of the depressive episode. In some cases, insomnia may also have an insidious onset without any identifiable precipitating factor.

The course of insomnia may also be episodic, with recurrent episodes of sleep difficulties associated with the occurrence of stressful events. Chronicity rates range from 45% to 75% for follow-ups of 1–7 years. Even when the course of the insomnia has become chronic, there is night-to-night variability in sleep patterns, with an occasional restful night's sleep interspersed with several nights of poor sleep. The characteristics of insomnia may also change over time. Many individuals with insomnia have a history of "light" or easily disturbed sleep prior to onset of more persistent sleep problems.

Insomnia complaints are more prevalent among middle-age and older adults. The type of insomnia symptom changes as a function of age, with difficulties initiating sleep being more common among young adults and problems maintaining sleep occurring more frequently among middle-age and older individuals.

Difficulties initiating and maintaining sleep can also occur in children and adolescents, but there are more limited data on prevalence, risk factors, and comorbidity during these developmental phases of the lifespan. Sleep difficulties in childhood can result from conditioning factors (e.g., a child who does not learn to fall asleep or return to sleep without the presence of a parent) or from the absence of consistent sleep schedules and bedtime routines. Insomnia in adolescence is often triggered or exacerbated by irregular sleep schedules (e.g., phase delay). In both children and adolescents, psychological and medical factors can contribute to insomnia.

The increased prevalence of insomnia in older adults is partly explained by the higher incidence of physical health problems with aging. Changes in sleep patterns associated with the normal developmental process must be differentiated from those exceeding age-related changes. Although polysomnography is of limited value in the routine evaluation of insomnia, it may be more useful in the differential diagnosis among older adults because the etiologies of insomnia (e.g., sleep apnea) are more often identifiable in older individuals.

Risk and Prognostic Factors

While the risk and prognostic factors discussed in this section increase vulnerability to insomnia, sleep disturbances are more likely to occur when predisposed individuals are exposed to precipitating events, such as major life events (e.g., illness, separation) or less severe but more chronic daily stress. Most individuals resume normal sleep patterns after the initial triggering event has disappeared, but others—perhaps those more vulnerable to insomnia—continue experiencing persistent sleep difficulties. Perpetuating factors such as poor sleep habits, irregular sleep scheduling, and the fear of not sleeping feed into the insomnia problem and may contribute to a vicious cycle that may induce persistent insomnia.

Temperamental. Anxiety or worry-prone personality or cognitive styles, increased arousal predisposition, and tendency to repress emotions can increase vulnerability to insomnia.

Environmental. Noise, light, uncomfortably high or low temperature, and high altitude may also increase vulnerability to insomnia.

Genetic and physiological. Female gender and advancing age are associated with increased vulnerability to insomnia. Disrupted sleep and insomnia display a familial disposition. The prevalence of insomnia is higher among monozygotic twins relative to dizygotic twins; it is also higher in first-degree family members compared with the general population. The extent to which this link is inherited through a genetic predisposition, learned by observations of parental models, or established as a by-product of another psychopathology remains undetermined.

Course modifiers. Deleterious course modifiers include poor sleep hygiene practices (e.g., excessive caffeine use, irregular sleep schedules).

Gender-Related Diagnostic Issues

Insomnia is a more prevalent complaint among females than among males, with first onset often associated with the birth of a new child or with menopause. Despite higher prevalence among older females, polysomnographic studies suggest better preservation of sleep continuity and slow-wave sleep in older females than in older males.

Diagnostic Markers

Polysomnography usually shows impairments of sleep continuity (e.g., increased sleep latency and time awake after sleep onset and decreased sleep efficiency [percentage of time in bed asleep] and may show increased stage 1 sleep and decreased stages 3 and 4 sleep. The severity of these sleep impairments does not always match the individual's clinical presentation or subjective complaint of poor sleep, as individuals with insomnia often underestimate sleep duration and overestimate wakefulness relative to polysomnography. Quantitative electroencephalographic analyses may indicate that individuals with insomnia have greater high-frequency electroencephalography power relative to good sleepers both around the sleep onset period and during non–rapid eye movement sleep, a feature suggestive of increased cortical arousal. Individuals with insomnia disorder may have a lower sleep propensity and typically do not show increased daytime sleepiness on objective sleep laboratory measures compared with individuals without sleep disorders.

Other laboratory measures show evidence, although not consistently, of increased arousal and a generalized activation of the hypothalamic-pituitary-adrenal axis (e.g., increased cortisol levels, heart rate variability, reactivity to stress, metabolic rate). In general, findings are consistent with the hypothesis that increased physiological and cognitive arousal plays a significant role in insomnia disorder.

Individuals with insomnia disorder may appear either fatigued or haggard or, conversely, overaroused and "wired." However, there are no consistent or characteristic abnormalities on physical examination. There may be an increased incidence of stress-

related psychophysiological symptoms (e.g., tension headache, muscle tension or pain, gastrointestinal symptoms).

Functional Consequences of Insomnia Disorder

Interpersonal, social, and occupational problems may develop as a result of insomnia or excessive concern with sleep, increased daytime irritability, and poor concentration. Decreased attention and concentration are common and may be related to higher rates of accidents observed in insomnia. Persistent insomnia is also associated with long-term consequences, including increased risks of major depressive disorder, hypertension, and myocardial infarction; increased absenteeism and reduced productivity at work; reduced quality of life; and increased economic burden.

Differential Diagnosis

Normal sleep variations. Normal sleep duration varies considerably across individuals. Some individuals who require little sleep ("short sleepers") may be concerned about their sleep duration. Short sleepers differ from individuals with insomnia disorder by the lack of difficulty falling or staying asleep and by the absence of characteristic daytime symptoms (e.g., fatigue, concentration problems, irritability). However, some short sleepers may desire or attempt to sleep for a longer period of time and, by prolonging time in bed, may create an insomnia-like sleep pattern. Clinical insomnia also should be distinguished from normal, age-related sleep changes. Insomnia must also be distinguished from sleep deprivation due to inadequate opportunity or circumstance for sleep resulting, for example, from an emergency or from professional or family obligations forcing the individual to stay awake.

Situational/acute insomnia. *Situational/acute insomnia* is a condition lasting a few days to a few weeks, often associated with life events or with changes in sleep schedules. These acute or short-term insomnia symptoms may also produce significant distress and interfere with social, personal, and occupational functioning. When such symptoms are frequent enough and meet all other criteria except for the 3-month duration, a diagnosis of other specified insomnia disorder or unspecified insomnia disorder is made.

Delayed sleep phase and shift work types of circadian rhythm sleep-wake disorder. Individuals with the delayed sleep phase type of circadian rhythm sleep-wake disorder report sleep-onset insomnia only when they try to sleep at socially normal times, but they do not report difficulty falling asleep or staying asleep when their bed and rising times are delayed and coincide with their endogenous circadian rhythm. Shift work type differs from insomnia disorder by the history of recent shift work.

Restless legs syndrome. Restless legs syndrome often produces difficulties initiating and maintaining sleep. However, an urge to move the legs and any accompanying unpleasant leg sensations are features that differentiate this disorder from insomnia disorder.

Breathing-related sleep disorders. Most individuals with a breathing-related sleep disorder have a history of loud snoring, breathing pauses during sleep, and excessive daytime sleepiness. Nonetheless, as many as 50% of individuals with sleep apnea may also report insomnia symptoms, a feature that is more common among females and older adults.

Narcolepsy. Narcolepsy may cause insomnia complaints but is distinguished from insomnia disorder by the predominance of symptoms of excessive daytime sleepiness, cataplexy, sleep paralysis, and sleep-related hallucinations.

Parasomnias. Parasomnias are characterized by a complaint of unusual behavior or events during sleep that may lead to intermittent awakenings and difficulty resuming sleep. However, it is these behavioral events, rather than the insomnia per se, that dominate the clinical picture.

Substance/medication-induced sleep disorder, insomnia type. Substance/medication-induced sleep disorder, insomnia type, is distinguished from insomnia disorder by the fact that a substance (i.e., a drug of abuse, a medication, or exposure to a toxin) is judged to be etiologically related to the insomnia (see "Substance/Medication-Induced Sleep Disorder" later in this chapter). For example, insomnia occurring only in the context of heavy coffee consumption would be diagnosed as caffeine-induced sleep disorder, insomnia type, with onset during intoxication.

Comorbidity

Insomnia is a common comorbidity of many medical conditions, including diabetes, coronary heart disease, chronic obstructive pulmonary disease, arthritis, fibromyalgia, and other chronic pain conditions. The risk relationship appears to be bidirectional: insomnia increases the risk of medical conditions, and medical problems increase the risk of insomnia. The direction of the relationship is not always clear and may change over time; for this reason, comorbid insomnia is the preferred terminology in the presence of coexisting insomnia with another medical condition (or mental disorder).

Individuals with insomnia disorder frequently have a comorbid mental disorder, particularly bipolar, depressive, and anxiety disorders. Persistent insomnia represents a risk factor or an early symptom of subsequent bipolar, depressive, anxiety, and substance use disorders. Individuals with insomnia may misuse medications or alcohol to help with nighttime sleep, anxiolytics to combat tension or anxiety, and caffeine or other stimulants to combat excessive fatigue. In addition to worsening the insomnia, this type of substance use may in some cases progress to a substance use disorder.

Relationship to International Classification of Sleep Disorders

There are several distinct insomnia phenotypes relating to the perceived source of the insomnia that are recognized by the *International Classification of Sleep Disorders*, 2nd Edition (ICSD-2). These include *psychophysiological insomnia, idiopathic insomnia, sleep-state misperception,* and *inadequate sleep hygiene.* Despite their clinical appeal and heuristic value, there is limited evidence to support these distinct phenotypes.

Hypersomnolence Disorder

Diagnostic Criteria **780.54 (G47.10)**

A. Self-reported excessive sleepiness (hypersomnolence) despite a main sleep period lasting at least 7 hours, with at least one of the following symptoms:
   1. Recurrent periods of sleep or lapses into sleep within the same day.
   2. A prolonged main sleep episode of more than 9 hours per day that is nonrestorative (i.e., unrefreshing).
   3. Difficulty being fully awake after abrupt awakening.

B. The hypersomnolence occurs at least three times per week, for at least 3 months.

C. The hypersomnolence is accompanied by significant distress or impairment in cognitive, social, occupational, or other important areas of functioning.

D. The hypersomnolence is not better explained by and does not occur exclusively during the course of another sleep disorder (e.g., narcolepsy, breathing-related sleep disorder, circadian rhythm sleep-wake disorder, or a parasomnia).

E. The hypersomnolence is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication).

F. Coexisting mental and medical disorders do not adequately explain the predominant complaint of hypersomnolence.

Specify if:

With mental disorder, including substance use disorders

With medical condition

With another sleep disorder

Coding note: The code 780.54 (G47.10) applies to all three specifiers. Code also the relevant associated mental disorder, medical condition, or other sleep disorder immediately after the code for hypersomnolence disorder in order to indicate the association.

Specify if:

Acute: Duration of less than 1 month.

Subacute: Duration of 1–3 months.

Persistent: Duration of more than 3 months.

Specify current severity:

Specify severity based on degree of difficulty maintaining daytime alertness as manifested by the occurrence of multiple attacks of irresistible sleepiness within any given day occurring, for example, while sedentary, driving, visiting with friends, or working.

Mild: Difficulty maintaining daytime alertness 1–2 days/week.

Moderate: Difficulty maintaining daytime alertness 3–4 days/week.

Severe: Difficulty maintaining daytime alertness 5–7 days/week.

Diagnostic Features

Hypersomnolence is a broad diagnostic term and includes symptoms of excessive quantity of sleep (e.g., extended nocturnal sleep or involuntary daytime sleep), deteriorated quality of wakefulness (i.e., sleep propensity during wakefulness as shown by difficulty awakening or inability to remain awake when required), and sleep inertia (i.e., a period of impaired performance and reduced vigilance following awakening from the regular sleep episode or from a nap) (Criterion A). Individuals with this disorder fall asleep quickly and have a good sleep efficiency (>90%). They may have difficulty waking up in the morning, sometimes appearing confused, combative, or ataxic. This prolonged impairment of alertness at the sleep-wake transition is often referred to as *sleep inertia* (i.e., sleep drunkenness). It can also occur upon awakening from a daytime nap. During that period, the individual appears awake, but there is a decline in motor dexterity, behavior may be very inappropriate, and memory deficits, disorientation in time and space, and feelings of grogginess may occur. This period may last some minutes to hours.

The persistent need for sleep can lead to automatic behavior (usually of a very routine, low-complexity type) that the individual carries out with little or no subsequent recall. For example, individuals may find themselves having driven several miles from where they thought they were, unaware of the "automatic" driving they did in the preceding minutes. For some individuals with hypersomnolence disorder, the major sleep episode (for most individuals, nocturnal sleep) has a duration of 9 hours or more. However, the sleep is often nonrestorative and is followed by difficulty awakening in the morning. For other individuals with hypersomnolence disorder, the major sleep episode is of normal nocturnal sleep duration (6–9 hours). In these cases, the excessive sleepiness is characterized by several unintentional daytime naps. These daytime naps tend to be relatively long (often lasting 1 hour or more), are experienced as nonrestorative (i.e., unrefreshing), and do not lead to improved alertness. Individuals with hypersomnolence have daytime naps nearly everyday regardless of the nocturnal sleep duration. Subjective sleep quality may or may not be reported as good. Individuals typically feel sleepiness developing over a period of time, rather than

experiencing a sudden sleep "attack." Unintentional sleep episodes typically occur in low-stimulation and low-activity situations (e.g., while attending lectures, reading, watching television, or driving long distances), but in more severe cases they can manifest in high-attention situations such as at work, in meetings, or at social gatherings.

Associated Features Supporting Diagnosis

Nonrestorative sleep, automatic behavior, difficulties awakening in the morning, and sleep inertia, although common in hypersomnolence disorder, may also be seen in a variety of conditions, including narcolepsy. Approximately 80% of individuals with hypersomnolence report that their sleep is nonrestorative, and as many have difficulties awakening in the morning. Sleep inertia, though less common (i.e., observed in 36%–50% of individuals with hypersomnolence disorder), is highly specific to hypersomnolence. Short naps (i.e., duration of less than 30 minutes) are often unrefreshing. Individuals with hypersomnolence often appear sleepy and may even fall asleep in the clinician's waiting area.

A subset of individuals with hypersomnolence disorder have a family history of hypersomnolence and also have symptoms of autonomic nervous system dysfunction, including recurrent vascular-type headaches, reactivity of the peripheral vascular system (Raynaud's phenomenon), and fainting.

Prevalence

Approximately 5%–10% of individuals who consult in sleep disorders clinics with complaints of daytime sleepiness are diagnosed as having hypersomnolence disorder. It is estimated that about 1% of the European and U.S. general population has episodes of sleep inertia. Hypersomnolence occurs with relatively equal frequency in males and females.

Development and Course

Hypersomnolence disorder has a persistent course, with a progressive evolution in the severity of symptoms. In most extreme cases, sleep episodes can last up to 20 hours. However, the average nighttime sleep duration is around 9½ hours. While many individuals with hypersomnolence are able to reduce their sleep time during working days, weekend and holiday sleep is greatly increased (by up to 3 hours). Awakenings are very difficult and accompanied by sleep inertia episodes in nearly 40% of cases. Hypersomnolence fully manifests in most cases in late adolescence or early adulthood, with a mean age at onset of 17–24 years. Individuals with hypersomnolence disorder are diagnosed, on average, 10–15 years after the appearance of the first symptoms. Pediatric cases are rare.

Hypersomnolence has a progressive onset, with symptoms beginning between ages 15 and 25 years, with a gradual progression over weeks to months. For most individuals, the course is then persistent and stable, unless treatment is initiated. The development of other sleep disorders (e.g., breathing-related sleep disorder) may worsen the degree of sleepiness. Although hyperactivity may be one of the presenting signs of daytime sleepiness in children, voluntary napping increases with age. This normal phenomenon is distinct from hypersomnolence.

Risk and Prognostic Factors

Environmental. Hypersomnolence can be increased temporarily by psychological stress and alcohol use, but they have not been documented as environmental precipitating factors. Viral infections have been reported to have preceded or accompanied hypersomnolence in about 10% of cases. Viral infections, such as HIV pneumonia, infectious mononucleosis, and Guillain-Barré syndrome, can also evolve into hypersomnolence within

months after the infection. Hypersomnolence can also appear within 6–18 months following a head trauma.

Genetic and physiological. Hypersomnolence may be familial, with an autosomal-dominant mode of inheritance.

Diagnostic Markers

Nocturnal polysomnography demonstrates a normal to prolonged sleep duration, short sleep latency, and normal to increased sleep continuity. The distribution of rapid eye movement (REM) sleep is also normal. Sleep efficiency is mostly greater than 90%. Some individuals with hypersomnolence disorder have increased amounts of slow-wave sleep. The multiple sleep latency test documents sleep tendency, typically indicated by mean sleep latency values of less than 8 minutes. In hypersomnolence disorder, the mean sleep latency is typically less than 10 minutes and frequently 8 minutes or less. Sleep-onset REM periods (SOREMPs; i.e., the occurrence of REM sleep within 20 minutes of sleep onset) may be present but occur less than two times in four to five nap opportunities.

Functional Consequences of Hypersomnolence Disorder

The low level of alertness that occurs while an individual fights the need for sleep can lead to reduced efficiency, diminished concentration, and poor memory during daytime activities. Hypersomnolence can lead to significant distress and dysfunction in work and social relationships. Prolonged nocturnal sleep and difficulty awakening can result in difficulty in meeting morning obligations, such as arriving at work on time. Unintentional daytime sleep episodes can be embarrassing and even dangerous, if, for instance, the individual is driving or operating machinery when the episode occurs.

Differential Diagnosis

Normative variation in sleep. "Normal" sleep duration varies considerably in the general population. "Long sleepers" (i.e., individuals who require a greater than average amount of sleep) do not have excessive sleepiness, sleep inertia, or automatic behavior when they obtain their required amount of nocturnal sleep. Sleep is reported to be refreshing. If social or occupational demands lead to shorter nocturnal sleep, daytime symptoms may appear. In hypersomnolence disorder, by contrast, symptoms of excessive sleepiness occur regardless of nocturnal sleep duration. An inadequate amount of nocturnal sleep, or *behaviorally induced insufficient sleep syndrome*, can produce symptoms of daytime sleepiness very similar to those of hypersomnolence. An average sleep duration of fewer than 7 hours per night strongly suggests inadequate nocturnal sleep, and an average of more than 9–10 hours of sleep per 24-hour period suggests hypersomnolence. Individuals with inadequate nocturnal sleep typically "catch up" with longer sleep durations on days when they are free from social or occupational demands or on vacations. Unlike hypersomnolence, insufficient nocturnal sleep is unlikely to persist unabated for decades. A diagnosis of hypersomnolence disorder should not be made if there is a question regarding the adequacy of nocturnal sleep duration. A diagnostic and therapeutic trial of sleep extension for 10–14 days can often clarify the diagnosis.

Poor sleep quality and fatigue. Hypersomnolence disorder should be distinguished from excessive sleepiness related to insufficient sleep quantity or quality and fatigue (i.e., tiredness not necessarily relieved by increased sleep and unrelated to sleep quantity or quality). Excessive sleepiness and fatigue are difficult to differentiate and may overlap considerably.

Breathing-related sleep disorders. Individuals with hypersomnolence and breathing-related sleep disorders may have similar patterns of excessive sleepiness. Breathing-

related sleep disorders are suggested by a history of loud snoring, pauses in breathing during sleep, brain injury, or cardiovascular disease and by the presence of obesity, oropharyngeal anatomical abnormalities, hypertension, or heart failure on physical examination. Polysomnographic studies can confirm the presence of apneic events in breathing-related sleep disorder (and their absence in hypersomnolence disorder).

Circadian rhythm sleep-wake disorders. Circadian rhythm sleep-wake disorders are often characterized by daytime sleepiness. A history of an abnormal sleep-wake schedule (with shifted or irregular hours) is present in individuals with a circadian rhythm sleep-wake disorder.

Parasomnias. Parasomnias rarely produce the prolonged, undisturbed nocturnal sleep or daytime sleepiness characteristic of hypersomnolence disorder.

Other mental disorders. Hypersomnolence disorder must be distinguished from mental disorders that include hypersomnolence as an essential or associated feature. In particular, complaints of daytime sleepiness may occur in a major depressive episode, with atypical features, and in the depressed phase of bipolar disorder. Assessment for other mental disorders is essential before a diagnosis of hypersomnolence disorder is considered. A diagnosis of hypersomnolence disorder can be made in the presence of another current or past mental disorder.

Comorbidity

Hypersomnolence can be associated with depressive disorders, bipolar disorders (during a depressive episode), and major depressive disorder, with seasonal pattern. Many individuals with hypersomnolence disorder have symptoms of depression that may meet criteria for a depressive disorder. This presentation may be related to the psychosocial consequences of persistent increased sleep need. Individuals with hypersomnolence disorder are also at risk for substance-related disorders, particularly related to self-medication with stimulants. This general lack of specificity may contribute to very heterogeneous profiles among individuals whose symptoms meet the same diagnostic criteria for hypersomnolence disorder. Neurodegenerative conditions, such as Alzheimer's disease, Parkinson's disease, and multiple system atrophy, may also be associated with hypersomnolence.

Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders*, 2nd Edition (ICSD-2), differentiates nine subtypes of "hypersomnias of central origin," including recurrent hypersomnia (Kleine-Levin syndrome).

Narcolepsy

Diagnostic Criteria

A. Recurrent periods of an irrepressible need to sleep, lapsing into sleep, or napping occurring within the same day. These must have been occurring at least three times per week over the past 3 months.

B. The presence of at least one of the following:

   1. Episodes of cataplexy, defined as either (a) or (b), occurring at least a few times per month:

      a. In individuals with long-standing disease, brief (seconds to minutes) episodes of sudden bilateral loss of muscle tone with maintained consciousness that are precipitated by laughter or joking.

b. In children or in individuals within 6 months of onset, spontaneous grimaces or jaw-opening episodes with tongue thrusting or a global hypotonia, without any obvious emotional triggers.

2. Hypocretin deficiency, as measured using cerebrospinal fluid (CSF) hypocretin-1 immunoreactivity values (less than or equal to one-third of values obtained in healthy subjects tested using the same assay, or less than or equal to 110 pg/mL). Low CSF levels of hypocretin-1 must not be observed in the context of acute brain injury, inflammation, or infection.
3. Nocturnal sleep polysomnography showing rapid eye movement (REM) sleep latency less than or equal to 15 minutes, or a multiple sleep latency test showing a mean sleep latency less than or equal to 8 minutes and two or more sleep-onset REM periods.

Specify whether:

347.00 (G47.419) Narcolepsy without cataplexy but with hypocretin deficiency: Criterion B requirements of low CSF hypocretin-1 levels and positive polysomnography/multiple sleep latency test are met, but no cataplexy is present (Criterion B1 not met).

347.01 (G47.411) Narcolepsy with cataplexy but without hypocretin deficiency: In this rare subtype (less than 5% of narcolepsy cases), Criterion B requirements of cataplexy and positive polysomnography/multiple sleep latency test are met, but CSF hypocretin-1 levels are normal (Criterion B2 not met).

347.00 (G47.419) Autosomal dominant cerebellar ataxia, deafness, and narcolepsy: This subtype is caused by exon 21 DNA (cytosine-5)-methyltransferase-1 mutations and is characterized by late-onset (age 30–40 years) narcolepsy (with low or intermediate CSF hypocretin-1 levels), deafness, cerebellar ataxia, and eventually dementia.

347.00 (G47.419) Autosomal dominant narcolepsy, obesity, and type 2 diabetes: Narcolepsy, obesity, and type 2 diabetes and low CSF hypocretin-1 levels have been described in rare cases and are associated with a mutation in the myelin oligodendrocyte glycoprotein gene.

347.10 (G47.429) Narcolepsy secondary to another medical condition: This subtype is for narcolepsy that develops secondary to medical conditions that cause infectious (e.g., Whipple's disease, sarcoidosis), traumatic, or tumoral destruction of hypocretin neurons.

Coding note (for ICD-9-CM code 347.10 only): Code first the underlying medical condition (e.g., 040.2 Whipple's disease; 347.10 narcolepsy secondary to Whipple's disease).

Specify current severity:

Mild: Infrequent cataplexy (less than once per week), need for naps only once or twice per day, and less disturbed nocturnal sleep.

Moderate: Cataplexy once daily or every few days, disturbed nocturnal sleep, and need for multiple naps daily.

Severe: Drug-resistant cataplexy with multiple attacks daily, nearly constant sleepiness, and disturbed nocturnal sleep (i.e., movements, insomnia, and vivid dreaming).

Subtypes

In narcolepsy without cataplexy but with hypocretin deficiency, unclear "cataplexy-like" symptoms may be reported (e.g., the symptoms are not triggered by emotions and are unusually long lasting). In extremely rare cases, cerebrospinal fluid (CSF) levels of hypocretin-1 are low, and polysomnographic/multiple sleep latency test (MSLT) results are negative: repeating the test is advised before establishing the subtype diagnosis. In narco-

lepsy with cataplexy but without hypocretin deficiency, test results for human leukocyte antigen (HLA) DQB1*06:02 may be negative. Seizures, falls of other origin, and conversion disorder (functional neurological symptom disorder) should be excluded. In narcolepsy secondary to infectious (e.g., Whipple's disease, sarcoidosis), traumatic, or tumoral destruction of hypocretin neurons, test results for HLA DQB1*06:02 may be positive and may result from the insult triggering the autoimmune process. In other cases, the destruction of hypocretin neurons may be secondary to trauma or hypothalamic surgery. Head trauma or infections of the central nervous system can, however, produce transitory decreases in CSF hypocretin-1 levels without hypocretin cell loss, complicating the diagnosis.

Diagnostic Features

The essential features of sleepiness in narcolepsy are recurrent daytime naps or lapses into sleep. Sleepiness typically occurs daily but must occur at a minimum three times a week for at least 3 months (Criterion A). Narcolepsy generally produces cataplexy, which most commonly presents as brief episodes (seconds to minutes) of sudden, bilateral loss of muscle tone precipitated by emotions, typically laughing and joking. Muscles affected may include those of the neck, jaw, arms, legs, or whole body, resulting in head bobbing, jaw dropping, or complete falls. Individuals are awake and aware during cataplexy. To meet Criterion B1(a), cataplexy must be triggered by laughter or joking and must occur at least a few times per month when the condition is untreated or in the past.

Cataplexy should not be confused with "weakness" occurring in the context of athletic activities (physiological) or exclusively after unusual emotional triggers such as stress or anxiety (suggesting possible psychopathology). Episodes lasting hours or days, or those not triggered by emotions, are unlikely to be cataplexy, nor is rolling on the floor while laughing hysterically.

In children close to onset, genuine cataplexy can be atypical, affecting primarily the face, causing grimaces or jaw opening with tongue thrusting ("cataplectic faces"). Alternatively, cataplexy may present as low-grade continuous hypotonia, yielding a wobbling walk. In these cases, Criterion B1(b) can be met in children or in individuals within 6 months of a rapid onset.

Narcolepsy-cataplexy nearly always results from the loss of hypothalamic hypocretin (orexin)–producing cells, causing hypocretin deficiency (less than or equal to one-third of control values, or 110 pg/mL in most laboratories). Cell loss is likely autoimmune, and approximately 99% of affected individuals carry HLA-DQB1*06:02 (vs. 12%–38% of control subjects). Thus, checking for the presence of DQB1*06:02 prior to a lumbar puncture for evaluation of CSF hypocretin-1 immunoreactivity may be useful. Rarely, low CSF levels of hypocretin-1 occur without cataplexy, notably in youths who may develop cataplexy later. CSF hypocretin-1 measurement represents the gold standard, excepting associated severe conditions (neurological, inflammatory, infectious, trauma) that can interfere with the assay.

A nocturnal polysomnographic sleep study followed by an MSLT can also be used to confirm the diagnosis (Criterion B3). These tests must be performed after the individual has stopped all psychotropic medications, following 2 weeks of adequate sleep time (as documented with sleep diaries, actigraphy). Short rapid eye movement (REM) latency (sleep-onset REM period, REM latency less than or equal to 15 minutes) during polysomnography is sufficient to confirm the diagnosis and meets Criterion B3. Alternatively, the MSLT result must be positive, showing a mean sleep latency of less than or equal to 8 minutes and two or more sleep-onset REM periods in four to five naps.

Associated Features Supporting Diagnosis

When sleepiness is severe, automatic behaviors may occur, with the individual continuing his or her activities in a semi-automatic, hazelike fashion without memory or consciousness. Approximately 20%–60% of individuals experience vivid hypnagogic hallucinations

before or upon falling asleep or hypnopompic hallucinations just after awakening. These hallucinations are distinct from the less vivid, nonhallucinatory dreamlike mentation at sleep onset that occurs in normal sleepers. Nightmares and vivid dreaming are also frequent in narcolepsy, as is REM sleep behavior disorder. Approximately 20%–60% of individuals experience sleep paralysis upon falling asleep or awakening, leaving them awake but unable to move or speak. However, many normal sleepers also report sleep paralysis, especially with stress or sleep deprivation. Nocturnal eating may occur. Obesity is common. Nocturnal sleep disruption with frequent long or short awakenings is common and can be disabling.

Individuals may appear sleepy or fall asleep in the waiting area or during clinical examination. During cataplexy, individuals may slump in a chair and have slurred speech or drooping eyelids. If the clinician has time to check reflexes during cataplexy (most attacks are less than 10 seconds), reflexes are abolished—an important finding distinguishing genuine cataplexy from conversion disorder.

Prevalence

Narcolepsy-cataplexy affects 0.02%–0.04% of the general population in most countries. Narcolepsy affects both genders, with possibly a slight male preponderance.

Development and Course

Onset is typically in children and adolescents/young adults but rarely in older adults. Two peaks of onset are suggested, at ages 15–25 years and ages 30–35 years. Onset can be abrupt or progressive (over years). Severity is highest when onset is abrupt in children, and then decreases with age or with treatment, so that symptoms such as cataplexy can occasionally disappear. Abrupt onset in young, prepubescent children can be associated with obesity and premature puberty, a phenotype more frequently observed since 2009. In adolescents, onset is more difficult to pinpoint. Onset in adults is often unclear, with some individuals reporting having had excessive sleepiness since birth. Once the disorder has manifested, the course is persistent and lifelong.

In 90% of cases, the first symptom to manifest is sleepiness or increased sleep, followed by cataplexy (within 1 year in 50% of cases, within 3 years in 85%). Sleepiness, hypnagogic hallucinations, vivid dreaming, and REM sleep behavior disorder (excessive movements during REM sleep) are early symptoms. Excessive sleep rapidly progresses to an inability to stay awake during the day, and to maintain good sleep at night, without a clear increase in total 24-hour sleep needs. In the first months, cataplexy may be atypical, especially in children. Sleep paralysis usually develops around puberty in children with prepubertal onset. Exacerbations of symptoms suggest lack of compliance with medications or development of a concurrent sleep disorder, notably sleep apnea.

Young children and adolescents with narcolepsy often develop aggression or behavioral problems secondary to sleepiness and/or nighttime sleep disruption. Workload and social pressure increase through high school and college, reducing available sleep time at night. Pregnancy does not seem to modify symptoms consistently. After retirement, individuals typically have more opportunity for napping, reducing the need for stimulants. Maintaining a regular schedule benefits individuals at all ages.

Risk and Prognostic Factors

Temperamental. Parasomnias, such as sleepwalking, bruxism, REM sleep behavior disorder, and enuresis, may be more common in individuals who develop narcolepsy. Individuals commonly report that they need more sleep than other family members.

Environmental. Group A streptococcal throat infection, influenza (notably pandemic H1N1 2009), or other winter infections are likely triggers of the autoimmune process, pro-

ducing narcolepsy a few months later. Head trauma and abrupt changes in sleep-wake patterns (e.g., job changes, stress) may be additional triggers.

Genetic and physiological. Monozygotic twins are 25%–32% concordant for narcolepsy. The prevalence of narcolepsy is 1%–2% in first-degree relatives (a 10- to 40-fold increase overall). Narcolepsy is strongly associated with DQB1*06:02 (99% vs. 12%–38% in control subjects of various ethnic groups; 25% in the general U.S. population). DQB1*03:01 increases, while DQB1*05:01, DQB1*06:01, and DQB1*06:03 reduce risk in the presence of DQB1*06:02, but the effect is small. Polymorphisms within the T-cell receptor alpha gene and other immune modulating genes also modulate risk slightly.

Culture-Related Diagnostic Issues

Narcolepsy has been described in all ethnic groups and in many cultures. Among African Americans, more cases present without cataplexy or with atypical cataplexy, complicating diagnosis, especially in the presence of obesity and obstructive sleep apnea.

Diagnostic Markers

Functional imaging suggests impaired hypothalamic responses to humorous stimuli. Nocturnal polysomnography followed by an MSLT is used to confirm the diagnosis of narcolepsy, especially when the disorder is first being diagnosed and before treatment has begun, and if hypocretin deficiency has not been documented biochemically. The polysomnography/MSLT should be performed after the individual is no longer taking any psychotropic drugs and after regular sleep-wake patterns, without shift work or sleep deprivation, have been documented.

A sleep-onset REM period during the polysomnography (REM sleep latency less than or equal to 15 minutes) is highly specific (approximately 1% positive in control subjects) but moderately sensitive (approximately 50%). A positive MSLT result displays an average sleep latency of less than or equal to 8 minutes, and sleep-onset REM periods in two or more naps on a four- or five-nap test. The MSLT result is positive in 90%–95% of individuals with narcolepsy versus 2%–4% of control subjects or individuals with other sleep disorders. Additional polysomnographic findings often include frequent arousals, decreased sleep efficiency, and increased stage 1 sleep. Periodic limb movements (found in about 40% of individuals with narcolepsy) and sleep apnea are often noted.

Hypocretin deficiency is demonstrated by measuring CSF hypocretin-1 immunoreactivity. The test is particularly useful in individuals with suspected conversion disorder and those without typical cataplexy, or in treatment-refractory cases. The diagnostic value of the test is not affected by medications, sleep deprivation, or circadian time, but the findings are uninterpretable when the individual is severely ill with a concurrent infection or head trauma or is comatose. CSF cytology, protein, and glucose are within normal range even when sampled within weeks of rapid onset. CSF hypocretin-1 in these incipient cases is typically already very diminished or undetectable.

Functional Consequences of Narcolepsy

Driving and working are impaired, and individuals with narcolepsy should avoid jobs that place themselves (e.g., working with machinery) or others (e.g., bus driver, pilot) in danger. Once the narcolepsy is controlled with therapy, patients can usually drive, although rarely long distances alone. Untreated individuals are also at risk for social isolation and accidental injury to themselves or others. Social relations may suffer as these individuals strive to avert cataplexy by exerting control over emotions.

Differential Diagnosis

Other hypersomnias. Hypersomnolence and narcolepsy are similar with respect to the degree of daytime sleepiness, age at onset, and stable course over time but can be distin-

guished based on distinctive clinical and laboratory features. Individuals with hypersomnolence typically have longer and less disrupted nocturnal sleep, greater difficulty awakening, more persistent daytime sleepiness (as opposed to more discrete "sleep attacks" in narcolepsy), longer and less refreshing daytime sleep episodes, and little or no dreaming during daytime naps. By contrast, individuals with narcolepsy have cataplexy and recurrent intrusions of elements of REM sleep into the transition between sleep and wakefulness (e.g., sleep-related hallucinations and sleep paralysis). The MSLT typically demonstrates shorter sleep latencies (i.e., greater physiological sleepiness) as well as the presence of multiple sleep-onset REM periods in individuals with narcolepsy.

Sleep deprivation and insufficient nocturnal sleep. Sleep deprivation and insufficient nocturnal sleep are common in adolescents and shift workers. In adolescents, difficulties falling asleep at night are common, causing sleep deprivation. The MSLT result may be positive if conducted while the individual is sleep deprived or while his or her sleep is phase delayed.

Sleep apnea syndromes. Sleep apneas are especially likely in the presence of obesity. Because obstructive sleep apnea is more frequent than narcolepsy, cataplexy may be overlooked (or absent), and the individual is assumed to have obstructive sleep apnea unresponsive to usual therapies.

Major depressive disorder. Narcolepsy or hypersomnia may be associated or confused with depression. Cataplexy is not present in depression. The MSLT results are most often normal, and there is dissociation between subjective and objective sleepiness, as measured by the mean sleep latency during the MSLT.

Conversion disorder (functional neurological symptom disorder). Atypical features, such as long-lasting cataplexy or unusual triggers, may be present in conversion disorder (functional neurological symptom disorder). Individuals may report sleeping and dreaming, yet the MSLT does not show the characteristic sleep-onset REM period. Full-blown, long-lasting pseudocataplexy may occur during consultation, allowing the examining physician enough time to verify reflexes, which remain intact.

Attention-deficit/hyperactivity disorder or other behavioral problems. In children and adolescents, sleepiness can cause behavioral problems, including aggressiveness and inattention, leading to a misdiagnosis of attention-deficit/hyperactivity disorder.

Seizures. In young children, cataplexy can be misdiagnosed as seizures. Seizures are not commonly triggered by emotions, and when they are, the trigger is not usually laughing or joking. During a seizure, individuals are more likely to hurt themselves when falling. Seizures characterized by isolated atonia are rarely seen in isolation of other seizures, and they also have signatures on the electroencephalogram.

Chorea and movement disorders. In young children, cataplexy can be misdiagnosed as chorea or pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections, especially in the context of a strep throat infection and high antistreptolysin O antibody levels. Some children may have an overlapping movement disorder close to onset of the cataplexy.

Schizophrenia. In the presence of florid and vivid hypnagogic hallucinations, individuals may think these experiences are real—a feature that suggests schizophrenia. Similarly, with stimulant treatment, persecutory delusions may develop. If cataplexy is present, the clinician should first assume that these symptoms are secondary to narcolepsy before considering a co-occurring diagnosis of schizophrenia.

Comorbidity

Narcolepsy can co-occur with bipolar, depressive, and anxiety disorders, and in rare cases with schizophrenia. Narcolepsy is also associated with increased body mass index or obe-

sity, especially when the narcolepsy is untreated. Rapid weight gain is common in young children with a sudden disease onset. Comorbid sleep apnea should be considered if there is a sudden aggravation of preexisting narcolepsy.

Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders*, 2nd Edition (ICSD-2), differentiates five subtypes of narcolepsy.

Breathing-Related Sleep Disorders

The breathing-related sleep disorders category encompasses three relatively distinct disorders: obstructive sleep apnea hypopnea, central sleep apnea, and sleep-related hypoventilation.

Obstructive Sleep Apnea Hypopnea

Diagnostic Criteria **327.23 (G47.33)**

A. Either (1) or (2):

1. Evidence by polysomnography of at least five obstructive apneas or hypopneas per hour of sleep and either of the following sleep symptoms:
   a. Nocturnal breathing disturbances: snoring, snorting/gasping, or breathing pauses during sleep.
   b. Daytime sleepiness, fatigue, or unrefreshing sleep despite sufficient opportunities to sleep that is not better explained by another mental disorder (including a sleep disorder) and is not attributable to another medical condition.
2. Evidence by polysomnography of 15 or more obstructive apneas and/or hypopneas per hour of sleep regardless of accompanying symptoms.

Specify current severity:

Mild: Apnea hypopnea index is less than 15.

Moderate: Apnea hypopnea index is 15–30.

Severe: Apnea hypopnea index is greater than 30.

Specifiers

Disease severity is measured by a count of the number of apneas plus hypopneas per hour of sleep (apnea hypopnea index) using polysomnography or other overnight monitoring. Overall severity is also informed by levels of nocturnal desaturation and sleep fragmentation (measured by brain cortical arousal frequency and sleep stages) and degree of associated symptoms and daytime impairment. However, the exact number and thresholds may vary according to the specific measurement techniques used, and these numbers may change over time. Regardless of the apnea hypopnea index (count) per se, the disorder is considered to be more severe when apneas and hypopneas are accompanied by significant oxygen hemoglobin desaturation (e.g., when more than 10% of the sleep time is spent at desaturation levels of less than 90%) or when sleep is severely fragmented as shown by an

elevated arousal index (arousal index greater than 30) or reduced stages in deep sleep (e.g., percentage stage N3 [slow-wave sleep] less than 5%).

Diagnostic Features

Obstructive sleep apnea hypopnea is the most common breathing-related sleep disorder. It is characterized by repeated episodes of upper (pharyngeal) airway obstruction (apneas and hypopneas) during sleep. *Apnea* refers to the total absence of airflow, and *hypopnea* refers to a reduction in airflow. Each apnea or hypopnea represents a reduction in breathing of at least 10 seconds in duration in adults or two missed breaths in children and is typically associated with drops in oxygen saturation of 3% or greater and/or an electroencephalographic arousal. Both sleep-related (nocturnal) and wake-time symptoms are common. The cardinal symptoms of obstructive sleep apnea hypopnea are snoring and daytime sleepiness.

Obstructive sleep apnea hypopnea in adults is diagnosed on the basis of polysomnographic findings and symptoms. The diagnosis is based on symptoms of 1) nocturnal breathing disturbances (i.e., snoring, snorting/gasping, breathing pauses during sleep), or 2) daytime sleepiness, fatigue, or unrefreshing sleep despite sufficient opportunities to sleep that are not better explained by another mental disorder and not attributable to another medical condition, along with 3) evidence by polysomnography of five or more obstructive apneas or hypopneas per hour of sleep (Criterion A1). Diagnosis can be made in the absence of these symptoms if there is evidence by polysomnography of 15 or more obstructive apneas and/or hypopneas per hour of sleep (Criterion A2).

Specific attention to disturbed sleep occurring in association with snoring or breathing pauses and physical findings that increase risk of obstructive sleep apnea hypopnea (e.g., central obesity, crowded pharyngeal airway, elevated blood pressure) is needed to reduce the chance of misdiagnosing this treatable condition.

Associated Features Supporting Diagnosis

Because of the frequency of nocturnal awakenings that occur with obstructive sleep apnea hypopnea, individuals may report symptoms of insomnia. Other common, though nonspecific, symptoms of obstructive sleep apnea hypopnea are heartburn, nocturia, morning headaches, dry mouth, erectile dysfunction, and reduced libido. Rarely, individuals may complain of difficulty breathing while lying supine or sleeping. Hypertension may occur in more than 60% of individuals with obstructive sleep apnea hypopnea.

Prevalence

Obstructive sleep apnea hypopnea is a very common disorder, affecting at least 1%–2% of children, 2%–15% of middle-age adults, and more than 20% of older individuals. In the general community, prevalence rates of undiagnosed obstructive sleep apnea hypopnea may be very high in elderly individuals. Since the disorder is strongly associated with obesity, increases in obesity rates are likely to be accompanied by an increased prevalence of this disorder. Prevalence may be particularly high among males, older adults, and certain racial/ethnic groups. In adults, the male-to-female ratio of obstructive sleep apnea hypopnea ranges from 2:1 to 4:1. Gender differences decline in older age, possibly because of an increased prevalence in females after menopause. There is no gender difference among prepubertal children.

Deveiopment and Course

The age distribution of obstructive sleep apnea hypopnea likely follows a J-shaped distribution. There is a peak in children ages 3–8 years when the nasopharynx may be compromised by a relatively large mass of tonsillar tissue compared with the size of the upper

airway. With growth of the airway and regression of lymphoid tissue during later childhood, there is reduction in prevalence. Then, as obesity prevalence increases in midlife and females enter menopause, obstructive sleep apnea hypopnea again increases. The course in older age is unclear; the disorder may level off after age 65 years, but in other individuals, prevalence may increase with aging. Because there is some age dependency of the occurrence of apneas and hypopneas, polysomnographic results must be interpreted in light of other clinical data. In particular, significant clinical symptoms of insomnia or hypersomnia should be investigated regardless of the individual's age.

Obstructive sleep apnea hypopnea usually has an insidious onset, gradual progression, and persistent course. Typically the loud snoring has been present for many years, often since childhood, but an increase in its severity may lead the individual to seek evaluation. Weight gain may precipitate an increase in symptoms. Although obstructive sleep apnea hypopnea can occur at any age, it most commonly manifests among individuals ages 40–60 years. Over 4–5 years, the average apnea hypopnea index increases in adults and older individuals by approximately two apneas/hypopneas per hour. The apnea hypopnea index is increased and incident obstructive sleep apnea hypopnea is greater among individuals who are older, who are male, or who have a higher baseline body mass index (BMI) or increase their BMI over time. Spontaneous resolution of obstructive sleep apnea hypopnea has been reported with weight loss, particularly after bariatric surgery. In children, seasonal variation in obstructive sleep apnea hypopnea has been observed, as has improvement with overall growth.

In young children, the signs and symptoms of obstructive sleep apnea hypopnea may be more subtle than in adults, making diagnosis more difficult to establish. Polysomnography is useful in confirming diagnosis. Evidence of fragmentation of sleep on the polysomnogram may not be as apparent as in studies of older individuals, possibly because of the high homeostatic drive in young individuals. Symptoms such as snoring are usually parent-reported and thus have reduced sensitivity. Agitated arousals and unusual sleep postures, such as sleeping on the hands and knees, may occur. Nocturnal enuresis also may occur and should raise the suspicion of obstructive sleep apnea hypopnea if it recurs in a child who was previously dry at night. Children may also manifest excessive daytime sleepiness, although this is not as common or pronounced as in adults. Daytime mouth breathing, difficulty in swallowing, and poor speech articulation are also common features in children. Children younger than 5 years more often present with nighttime symptoms, such as observed apneas or labored breathing, than with behavioral symptoms (i.e., the nighttime symptoms are more noticeable and more often bring the child to clinical attention). In children older than 5 years, daytime symptoms such as sleepiness and behavioral problems (e.g., impulsivity and hyperactivity), attention-deficit/hyperactivity disorder, learning difficulties, and morning headaches are more often the focus of concern. Children with obstructive sleep apnea hypopnea also may present with failure to thrive and developmental delays. In young children, obesity is a less common risk factor, while delayed growth and "failure to thrive" may be present.

Risk and Prognostic Factors

Genetic and physiological. The major risk factors for obstructive sleep apnea hypopnea are obesity and male gender. Others include maxillary-mandibular retrognathia or micrognathia, positive family history of sleep apnea, genetic syndromes that reduce upper airway patency (e.g., Down's syndrome, Treacher Collin's syndrome), adenotonsillar hypertrophy (especially in young children), menopause (in females), and various endocrine syndromes (e.g., acromegaly). Compared with premenopausal females, males are at increased risk for obstructive sleep apnea hypopnea, possibly reflecting the influences of sex hormones on ventilatory control and body fat distribution, as well as because of gender differences in airway structure. Medications for mental disorders and medical conditions that tend to induce somnolence may worsen the course of apnea symptoms if these medications are not managed carefully.

Obstructive sleep apnea hypopnea has a strong genetic basis, as evidenced by the significant familial aggregation of the apnea hypopnea index. The prevalence of obstructive sleep apnea hypopnea is approximately twice as high among the first-degree relatives of probands with obstructive sleep apnea hypopnea as compared with members of control families. One-third of the variance in the apnea hypopnea index is explained by shared familial factors. Although genetic markers with diagnostic or prognostic value are not yet available for use, eliciting a family history of obstructive sleep apnea hypopnea should increase the clinical suspicion for the disorder.

Culture-Related Diagnostic Issues

There is a potential for sleepiness and fatigue to be reported differently across cultures. In some groups, snoring may be considered a sign of health and thus may not trigger concerns. Individuals of Asian ancestry may be at increased risk for obstructive sleep apnea hypopnea despite relatively low BMI, possibly reflecting the influence of craniofacial risk factors that narrow the nasopharynx.

Gender-Related Issues

Females may more commonly report fatigue rather than sleepiness and may underreport snoring.

Diagnostic Markers

Polysomnography provides quantitative data on frequency of sleep-related respiratory disturbances and associated changes in oxygen saturation and sleep continuity. Polysomnographic findings in children differ from those in adults in that children demonstrate labored breathing, partial obstructive hypoventilation with cyclical desaturations, hypercapnia and paradoxical movements. Apnea hypopnea index levels as low as 2 are used to define thresholds of abnormality in children.

Arterial blood gas measurements while the individual is awake are usually normal, but some individuals can have waking hypoxemia or hypercapnia. This pattern should alert the clinician to the possibility of coexisting lung disease or hypoventilation. Imaging procedures may reveal narrowing of the upper airway. Cardiac testing may show evidence of impaired ventricular function. Individuals with severe nocturnal oxygen desaturation may also have elevated hemoglobin or hematocrit values. Validated sleep measures (e.g., multiple sleep latency test [MSLT], maintenance of wakefulness test) may identify sleepiness.

Functional Consequences of Obstructive Sleep Apnea Hypopnea

More than 50% of individuals with moderate to severe obstructive sleep apnea hypopnea report symptoms of daytime sleepiness. A twofold increased risk of occupational accidents has been reported in association with symptoms of snoring and sleepiness. Motor vehicle crashes also have been reported to be as much as sevenfold higher among individuals with elevated apnea hypopnea index values. Clinicians should be cognizant of state government requirements for reporting this disorder, especially in relationship to commercial drivers. Reduced scores on measures of health-related quality of life are common in individuals with obstructive sleep apnea hypopnea, with the largest decrements observed in the physical and vitality subscales.

Differential Diagnosis

Primary snoring and other sleep disorders. Individuals with obstructive sleep apnea hypopnea must be differentiated from individuals with primary snoring (i.e., otherwise

asymptomatic individuals who snore and do not have abnormalities on overnight polysomnography). Individuals with obstructive sleep apnea hypopnea may additionally report nocturnal gasping and choking. The presence of sleepiness or other daytime symptoms not explained by other etiologies suggests the diagnosis of obstructive sleep apnea hypopnea, but this differentiation requires polysomnography. Definitive differential diagnosis between hypersomnia, central sleep apnea, sleep-related hypoventilation, and obstructive sleep apnea hypopnea also requires polysomnographic studies.

Obstructive sleep apnea hypopnea must be differentiated from other causes of sleepiness, such as narcolepsy, hypersomnia, and circadian rhythm sleep disorders. Obstructive sleep apnea hypopnea can be differentiated from narcolepsy by the absence of cataplexy, sleep-related hallucinations, and sleep paralysis and by the presence of loud snoring, gasping during sleep, or observed apneas in sleep. Daytime sleep episodes in narcolepsy are characteristically shorter, more refreshing, and more often associated with dreaming. Obstructive sleep apnea hypopnea shows characteristic apneas and hypopneas and oxygen desaturation during nocturnal polysomnographic studies. Narcolepsy results in multiple sleep-onset rapid eye movement (REM) periods during the MSLT. Narcolepsy, like obstructive sleep apnea hypopnea, may be associated with obesity, and some individuals have concurrent narcolepsy and obstructive sleep apnea hypopnea. A diagnosis of narcolepsy does not exclude the diagnosis of obstructive sleep apnea hypopnea, as the two conditions may co-occur.

Insomnia disorder. For individuals complaining of difficulty initiating or maintaining sleep or early-morning awakenings, insomnia disorder can be differentiated from obstructive sleep apnea hypopnea by the absence of snoring and the absence of the history, signs, and symptoms characteristic of the latter disorder. However, insomnia and obstructive sleep apnea hypopnea may coexist, and if so, both disorders may need to be addressed concurrently to improve sleep.

Panic attacks. Nocturnal panic attacks may include symptoms of gasping or choking during sleep that may be difficult to distinguish clinically from obstructive sleep apnea hypopnea. However, the lower frequency of episodes, intense autonomic arousal, and lack of excessive sleepiness differentiate nocturnal panic attacks from obstructive sleep apnea hypopnea. Polysomnography in individuals with nocturnal panic attacks does not reveal the typical pattern of apneas or oxygen desaturation characteristic of obstructive sleep apnea hypopnea. Individuals with obstructive sleep apnea hypopnea do not provide a history of daytime panic attacks.

Attention-deficit/hyperactivity disorder. Attention-deficit/hyperactivity disorder in children may include symptoms of inattention, academic impairment, hyperactivity, and internalizing behaviors, all of which may also be symptoms of childhood obstructive sleep apnea hypopnea. The presence of other symptoms and signs of childhood obstructive sleep apnea hypopnea (e.g., labored breathing or snoring during sleep and adenotonsillar hypertrophy) would suggest the presence of obstructive sleep apnea hypopnea. Obstructive sleep apnea hypopnea and attention-deficit/hyperactivity disorder may commonly co-occur, and there may be causal links between them; therefore, risk factors such as enlarged tonsils, obesity, or a family history of sleep apnea may help alert the clinician to their co-occurrence.

Substance/medication-induced insomnia or hypersomnia. Substance use and substance withdrawal (including medications) can produce insomnia or hypersomnia. A careful history is usually sufficient to identify the relevant substance/medication, and follow-up shows improvement of the sleep disturbance after discontinuation of the substance/medication. In other cases, the use of a substance/medication (e.g., alcohol, barbiturates, benzodiazepines, tobacco) has been shown to exacerbate obstructive sleep apnea hypopnea. An individual with symptoms and signs consistent with obstructive sleep apnea hypop-

nea should receive that diagnosis, even in the presence of concurrent substance use that is exacerbating the condition.

Comorbidity

Systemic hypertension, coronary artery disease, heart failure, stroke, diabetes, and increased mortality are consistently associated with obstructive sleep apnea hypopnea. Risk estimates vary from 30% to as much as 300% for moderate to severe obstructive sleep apnea hypopnea. Evidence of pulmonary hypertension and right heart failure (e.g., cor pulmonale, ankle edema, hepatic congestion) are rare in obstructive sleep apnea hypopnea and when present indicate either very severe disease or associated hypoventilation or cardiopulmonary comorbidities. Obstructive sleep apnea hypopnea also may occur with increased frequency in association with a number of medical or neurological conditions (e.g., cerebrovascular disease, Parkinson's disease). Physical findings reflect the co-occurrence of these conditions.

As many as one-third of individuals referred for evaluation of obstructive sleep apnea hypopnea report symptoms of depression, with as many of 10% having depression scores consistent with moderate to severe depression. Severity of obstructive sleep apnea hypopnea, as measured by the apnea hypopnea index, has been found to be correlated with severity of symptoms of depression. This association may be stronger in males than in females.

Reiationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders*, 2nd Edition (ICSD-2), differentiates 11 subtypes of "sleep-related breathing disorders," including primary central sleep apnea, obstructive sleep apnea, and sleep-related hypoventilation.

Central Sleep Apnea

Diagnostic Criteria

A. Evidence by polysomnography of five or more central apneas per hour of sleep.

B. The disorder is not better explained by another current sleep disorder.

Specify whether:

327.21 (G47.31) Idiopathic central sleep apnea: Characterized by repeated episodes of apneas and hypopneas during sleep caused by variability in respiratory effort but without evidence of airway obstruction.

786.04 (R06.3) Cheyne-Stokes breathing: A pattern of periodic crescendo-decrescendo variation in tidal volume that results in central apneas and hypopneas at a frequency of at least five events per hour, accompanied by frequent arousal.

780.57 (G47.37) Central sleep apnea comorbid with opioid use: The pathogenesis of this subtype is attributed to the effects of opioids on the respiratory rhythm generators in the medulla as well as the differential effects on hypoxic versus hypercapnic respiratory drive.

Coding note (for 780.57 [G47.37] code only): When an opioid use disorder is present, first code the opioid use disorder: 305.50 (F11.10) mild opioid use disorder or 304.00 (F11.20) moderate or severe opioid use disorder; then code 780.57 (G47.37) central sleep apnea comorbid with opioid use. When an opioid use disorder is not present (e.g., after a one-time heavy use of the substance), code only 780.57 (G47.37) central sleep apnea comorbid with opioid use.

Note: See the section "Diagnostic Features" in text.

Specify current severity:

Severity of central sleep apnea is graded according to the frequency of the breathing disturbances as well as the extent of associated oxygen desaturation and sleep fragmentation that occur as a consequence of repetitive respiratory disturbances.

Subtypes

Idiopathic central sleep apnea and Cheyne-Stokes breathing are characterized by increased gain of the ventilatory control system, also referred to as *high loop gain*, which leads to instability in ventilation and $PaCO_2$ levels. This instability is termed *periodic breathing* and can be recognized by hyperventilation alternating with hypoventilation. Individuals with these disorders typically have pCO_2 levels while awake that are slightly hypocapneic or normocapneic. Central sleep apnea may also manifest during initiation of treatment of obstructive sleep apnea hypopnea or may occur in association with obstructive sleep apnea hypopnea syndrome (termed *complex sleep apnea*). The occurrence of central sleep apnea in association with obstructive sleep apnea is also considered to be due to high loop gain. In contrast, the pathogenesis of central sleep apnea comorbid with opioid use has been attributed to the effects of opioids on the respiratory rhythm generators in the medulla as well as to its differential effects on hypoxic versus hypercapneic respiratory drive. These individuals may have elevated pCO_2 levels while awake. Individuals receiving chronic methadone maintenance therapy have been noted to have increased somnolence and depression, although the role of breathing disorders associated with opioid medication in causing these problems has not been studied.

Specifiers

An increase in the central apnea index (i.e., number of central apneas per hour of sleep) reflects an increase in severity of central sleep apnea. Sleep continuity and quality may be markedly impaired with reductions in restorative stages of non–rapid eye movement (REM) sleep (i.e., decreased slow-wave sleep [stage N3]). In individuals with severe Cheyne-Stokes breathing, the pattern can also be observed during resting wakefulness, a finding that is thought to be a poor prognostic marker for mortality.

Diagnostic Features

Central sleep apnea disorders are characterized by repeated episodes of apneas and hypopneas during sleep caused by variability in respiratory effort. These are disorders of ventilatory control in which respiratory events occur in a periodic or intermittent pattern. *Idiopathic central sleep apnea* is characterized by sleepiness, insomnia, and awakenings due to dyspnea in association with five or more central apneas per hour of sleep. Central sleep apnea occurring in individuals with heart failure, stroke, or renal failure typically have a breathing pattern called *Cheyne-Stokes breathing*, which is characterized by a pattern of periodic crescendo-decrescendo variation in tidal volume that results in central apneas and hypopneas occurring at a frequency of at least five events per hour that are accompanied by frequent arousals. Central and obstructive sleep apneas may coexist; the ratio of central to obstructive apneas/hypopneas may be used to identify which condition is predominant.

Alterations in neuromuscular control of breathing can occur in association with medications or substances used in individuals with mental health conditions, which can cause or exacerbate impairments of respiratory rhythm and ventilation. Individuals taking these medications have a sleep-related breathing disorder that could contribute to sleep disturbances and symptoms such as sleepiness, confusion, and depression. Specifically, *chronic*

use of long-acting opioid medications is often associated with impairment of respiratory control leading to central sleep apnea.

Associated Features Supporting Diagnosis

Individuals with central sleep apnea hypopneas can manifest with sleepiness or insomnia. There can be complaints of sleep fragmentation, including awakening with dyspnea. Some individuals are asymptomatic. Obstructive sleep apnea hypopnea can coexist with Cheyne-Stokes breathing, and thus snoring and abruptly terminating apneas may be observed during sleep.

Prevaience

The prevalence of idiopathic central sleep apnea is unknown but thought to be rare. The prevalence of Cheyne-Stokes breathing is high in individuals with depressed cardiac ventricular ejection fraction. In individuals with an ejection fraction of less than 45%, the prevalence has been reported to be 20% or higher. The male-to-female ratio for prevalence is even more highly skewed toward males than for obstructive sleep apnea hypopnea. Prevalence increases with age, and most patients are older than 60 years. Cheyne-Stokes breathing occurs in approximately 20% of individuals with acute stroke. Central sleep apnea comorbid with opioid use occurs in approximately 30% of individuals taking chronic opioids for nonmalignant pain and similarly in individuals receiving methadone maintenance therapy.

Development and Course

The onset of Cheyne-Stokes breathing appears tied to the development of heart failure. The Cheyne-Stokes breathing pattern is associated with oscillations in heart rate, blood pressure and oxygen desaturation, and elevated sympathetic nervous system activity that can promote progression of heart failure. The clinical significance of Cheyne-Stokes breathing in the setting of stroke is not known, but Cheyne-Stokes breathing may be a transient finding that resolves with time after acute stroke. Central sleep apnea comorbid with opioid use has been documented with chronic use (i.e., several months).

Risk and Prognostic Factors

Genetic and physiological. Cheyne-Stokes breathing is frequently present in individuals with heart failure. The coexistence of atrial fibrillation further increases risk, as do older age and male gender. Cheyne-Stokes breathing is also seen in association with acute stroke and possibly renal failure. The underlying ventilatory instability in the setting of heart failure has been attributed to increased ventilatory chemosensitivity and hyperventilation due to pulmonary vascular congestion and circulatory delay. Central sleep apnea is seen in individuals taking long-acting opioids.

Diagnostic Markers

Physical findings seen in individuals with a Cheyne-Stokes breathing pattern relate to its risk factors. Findings consistent with heart failure, such as jugular venous distension, S_3 heart sound, lung crackles, and lower extremity edema, may be present. Polysomnography is used to characterize the breathing characteristics of each breathing-related sleep disorder subtype. Central sleep apneas are recorded when periods of breathing cessation for longer than 10 seconds occur. Cheyne-Stokes breathing is characterized by a pattern of periodic crescendo-decrescendo variation in tidal volume that results in central apneas and hypopneas occurring at a frequency of at least five events per hour that are accompanied by frequent arousals. The cycle length of Cheyne-Stokes breathing (or time from end of one central apnea to the end of the next apnea) is about 60 seconds.

Functional Consequences of Central Sleep Apnea

Idiopathic central sleep apnea has been reported to cause symptoms of disrupted sleep, including insomnia and sleepiness. Cheyne-Stokes breathing with comorbid heart failure has been associated with excessive sleepiness, fatigue, and insomnia, although many individuals may be asymptomatic. Coexistence of heart failure and Cheyne-Stokes breathing may be associated with increased cardiac arrhythmias and increased mortality or cardiac transplantation. Individuals with central sleep apnea comorbid with opioid use may present with symptoms of sleepiness or insomnia.

Differential Diagnosis

Idiopathic central sleep apnea must be distinguished from other breathing-related sleep disorders, other sleep disorders, and medical conditions and mental disorders that cause sleep fragmentation, sleepiness, and fatigue. This is achieved using polysomnography.

Other breathing-related sleep disorders and sleep disorders. Central sleep apnea can be distinguished from obstructive sleep apnea hypopnea by the presence of at least five central apneas per hour of sleep. These conditions may co-occur, but central sleep apnea is considered to predominate when the ratio of central to obstructive respiratory events exceeds 50%.

Cheyne-Stokes breathing can be distinguished from other mental disorders, including other sleep disorders, and other medical conditions that cause sleep fragmentation, sleepiness, and fatigue based on the presence of a predisposing condition (e.g., heart failure or stroke) and signs and polysomnographic evidence of the characteristic breathing pattern. Polysomnographic respiratory findings can help distinguish Cheyne-Stokes breathing from insomnia due to other medical conditions. High-altitude periodic breathing has a pattern that resembles Cheyne-Stokes breathing but has a shorter cycle time, occurs only at high altitude, and is not associated with heart failure.

Central sleep apnea comorbid with opioid use can be differentiated from other types of breathing-related sleep disorders based on the use of long-acting opioid medications in conjunction with polysomnographic evidence of central apneas and periodic or ataxic breathing. It can be distinguished from insomnia due to drug or substance use based on polysomnographic evidence of central sleep apnea.

Comorbidity

Central sleep apnea disorders are frequently present in users of long-acting opioids, such as methadone. Individuals taking these medications have a sleep-related breathing disorder that could contribute to sleep disturbances and symptoms such as sleepiness, confusion, and depression. While the individual is asleep, breathing patterns such as central apneas, periodic apneas, and ataxic breathing may be observed. Obstructive sleep apnea hypopnea may coexist with central sleep apnea, and features consistent with this condition can also be present (see "Obstructive Sleep Apnea Hypopnea" earlier in this chapter). Cheyne-Stokes breathing is more commonly observed in association with conditions that include heart failure, stroke, and renal failure and is seen more frequently in individuals with atrial fibrillation. Individuals with Cheyne-Stokes breathing are more likely to be older, to be male, and to have lower weight than individuals with obstructive sleep apnea hypopnea.

Sleep-Related Hypoventilation

Diagnostic Criteria

A. Polysomnograpy demonstrates episodes of decreased respiration associated with elevated CO_2 levels. (**Note:** In the absence of objective measurement of CO_2, persistent low levels of hemoglobin oxygen saturation unassociated with apneic/hypopneic events may indicate hypoventilation.)

B. The disturbance is not better explained by another current sleep disorder.

Specify whether:

327.24 (G47.34) Idiopathic hypoventilation: This subtype is not attributable to any readily identified condition.

327.25 (G47.35) Congenital central alveolar hypoventilation: This subtype is a rare congenital disorder in which the individual typically presents in the perinatal period with shallow breathing, or cyanosis and apnea during sleep.

327.26 (G47.36) Comorbid sleep-related hypoventilation: This subtype occurs as a consequence of a medical condition, such as a pulmonary disorder (e.g., interstitial lung disease, chronic obstructive pulmonary disease) or a neuromuscular or chest wall disorder (e.g., muscular dystrophies, postpolio syndrome, cervical spinal cord injury, kyphoscoliosis), or medications (e.g., benzodiazepines, opiates). It also occurs with obesity (obesity hypoventilation disorder), where it reflects a combination of increased work of breathing due to reduced chest wall compliance and ventilation-perfusion mismatch and variably reduced ventilatory drive. Such individuals usually are characterized by body mass index of greater than 30 and hypercapnia during wakefulness (with a pCO_2 of greater than 45), without other evidence of hypoventilation.

Specify current severity:

Severity is graded according to the degree of hypoxemia and hypercarbia present during sleep and evidence of end organ impairment due to these abnormalities (e.g., right-sided heart failure). The presence of blood gas abnormalities during wakefulness is an indicator of greater severity.

Subtypes

Regarding obesity hypoventilation disorder, the prevalence of obesity hypoventilation in the general population is not known but is thought to be increasing in association with the increased prevalence of obesity and extreme obesity.

Diagnostic Features

Sleep-related hypoventilation can occur independently or, more frequently, comorbid with medical or neurological disorders, medication use, or substance use disorder. Although symptoms are not mandatory to make this diagnosis, individuals often report excessive daytime sleepiness, frequent arousals and awakenings during sleep, morning headaches, and insomnia complaints.

Associated Features Supporting Diagnosis

Individuals with sleep-related hypoventilation can present with sleep-related complaints of insomnia or sleepiness. Episodes of orthopnea can occur in individuals with diaphragm weakness. Headaches upon awakening may be present. During sleep, episodes of shallow breathing may be observed, and obstructive sleep apnea hypopnea or central sleep apnea may coexist. Consequences of ventilatory insufficiency, including pulmonary hypertension, cor pulmonale (right heart failure), polycythemia, and neurocognitive dysfunction,

can be present. With progression of ventilatory insufficiency, blood gas abnormalities extend into wakefulness. Features of the medical condition causing sleep-related hypoventilation can also be present. Episodes of hypoventilation may be associated with frequent arousals or bradytachycardia. Individuals may complain of excessive sleepiness and insomnia or morning headaches or may present with findings of neurocognitive dysfunction or depression. Hypoventilation may not be present during wakefulness.

Prevalence

Idiopathic sleep-related hypoventilation in adults is very uncommon. The prevalence of congenital central alveolar hypoventilation is unknown, but the disorder is rare. Comorbid sleep-related hypoventilation (i.e., hypoventilation comorbid with other conditions, such as chronic obstructive pulmonary disease [COPD], neuromuscular disorders, or obesity) is more common.

Development and Course

Idiopathic sleep-related hypoventilation is thought to be a slowly progressive disorder of respiratory impairment. When this disorder occurs comorbidly with other disorders (e.g., COPD, neuromuscular disorders, obesity), disease severity reflects the severity of the underlying condition, and the disorder progresses as the condition worsens. Complications such as pulmonary hypertension, cor pulmonale, cardiac dysrhythmias, polycythemia, neurocognitive dysfunction, and worsening respiratory failure can develop with increasing severity of blood gas abnormalities.

Congenital central alveolar hypoventilation usually manifests at birth with shallow, erratic, or absent breathing. This disorder can also manifest during infancy, childhood, and adulthood because of variable penetrance of the *PHOX2B* mutation. Children with congenital central alveolar hypoventilation are more likely to have disorders of the autonomic nervous system, Hirschsprung's disease, neural crest tumors, and characteristic box-shaped face (i.e., the face is short relative to its width).

Risk and Prognostic Factors

Environmental. Ventilatory drive can be reduced in individuals using central nervous system depressants, including benzodiazepines, opiates, and alcohol.

Genetic and physiological. Idiopathic sleep-related hypoventilation is associated with reduced ventilatory drive due to a blunted chemoresponsiveness to CO_2 (reduced respiratory drive; i.e., "won't breathe"), reflecting underlying neurological deficits in centers governing the control of ventilation. More commonly, sleep-related hypoventilation is comorbid with another medical condition, such as a pulmonary disorder, a neuromuscular or chest wall disorder, or hypothyroidism, or with use of medications (e.g., benzodiazepines, opiates). In these conditions, the hypoventilation may be a consequence of increased work of breathing and/or impairment of respiratory muscle function (i.e., "can't breathe") or reduced respiratory drive (i.e., "won't breathe").

Neuromuscular disorders influence breathing through impairment of respiratory motor innervation or respiratory muscle function. They include conditions such as amyotrophic lateral sclerosis, spinal cord injury, diaphragmatic paralysis, myasthenia gravis, Lambert-Eaton syndrome, toxic or metabolic myopathies, postpolio syndrome, and Charcot-Marie-Tooth syndrome.

Congenital central alveolar hypoventilation is a genetic disorder attributable to mutations of *PHOX2B*, a gene that is crucial for the development of the embryonic autonomic nervous system and neural crest derivatives. Children with congenital central alveolar hypoventilation show blunted ventilatory responses to hypercapnia, especially in non–rapid eye movement sleep.

Gender-Related Diagnostic Issues

Gender distributions for sleep-related hypoventilation occurring in association with comorbid conditions reflect the gender distributions of the comorbid conditions. For example, COPD is more frequently present in males and with increasing age.

Diagnostic Markers

Sleep-related hypoventilation is diagnosed using polysomnography showing sleep-related hypoxemia and hypercapnia that is not better explained by another breathing-related sleep disorder. The documentation of increased arterial pCO_2 levels to greater than 55 mmHg during sleep or a 10 mmHg or greater increase in pCO_2 levels (to a level that also exceeds 50 mmHg) during sleep in comparison to awake supine values, for 10 minutes or longer, is the gold standard for diagnosis. However, obtaining arterial blood gas determinations during sleep is impractical, and non-invasive measures of pCO_2 have not been adequately validated during sleep and are not widely used during polysomnography in adults. Prolonged and sustained decreases in oxygen saturation (oxygen saturation of less than 90% for more than 5 minutes with a nadir of at least 85%, or oxygen saturation of less than 90% for at least 30% of sleep time) in the absence of evidence of upper airway obstruction are often used as an indication of sleep-related hypoventilation; however, this finding is not specific, as there are other potential causes of hypoxemia, such as that due to lung disease.

Functional Consequences of Sleep-Related Hypoventilation

The consequences of sleep-related hypoventilation are related to the effects of chronic exposure to hypercapnia and hypoxemia. These blood gas derangements cause vasoconstriction of the pulmonary vasculature leading to pulmonary hypertension, which, if severe, can result in right-sided heart failure (cor pulmonale). Hypoxemia can lead to dysfunction of organs such as the brain, blood, and heart, leading to outcomes such as cognitive dysfunction, polycythemia, and cardiac arrhythmias. Hypercapnia can depress ventilatory drive, leading to progressive respiratory failure.

Differential Diagnosis

Other medical conditions affecting ventilation. In adults, the idiopathic variety of sleep-related hypoventilation is very uncommon and is determined by excluding the presence of lung diseases, skeletal malformations, neuromuscular disorders, and other medical and neurological disorders or medications that affect ventilation. Sleep-related hypoventilation must be distinguished from other causes of sleep-related hypoxemia, such as that due to lung disease.

Other breathing-related sleep disorders. Sleep-related hypoventilation can be distinguished from obstructive sleep apnea hypopnea and central sleep apnea based on clinical features and findings on polysomnography. Sleep-related hypoventilation typically shows more sustained periods of oxygen desaturation rather that the periodic episodes seen in obstructive sleep apnea hypopnea and central sleep apnea. Obstructive sleep apnea hypopnea and central sleep apnea also show a pattern of discrete episodes of repeated airflow decreases that can be absent in sleep-related hypoventilation.

Comorbidity

Sleep-related hypoventilation often occurs in association with a pulmonary disorder (e.g., interstitial lung disease, COPD), with a neuromuscular or chest wall disorder (e.g., muscular dystrophies, post-polio syndrome, cervical spinal cord injury, obesity, kyphoscoliosis), or,

most relevant to the mental health provider, with medication use (e.g., benzodiazepines, opiates). Congenital central alveolar hypoventilation often occurs in association with autonomic dysfunction and may occur in association with Hirschsprung's disease. COPD, a disorder of lower airway obstruction usually associated with cigarette smoking, can result in sleep-related hypoventilation and hypoxemia. The presence of coexisting obstructive sleep apnea hypopnea is thought to exacerbate hypoxemia and hypercapnia during sleep and wakefulness. The relationship between congenital central alveolar hypoventilation and idiopathic sleep-related hypoventilation is unclear; in some individuals, idiopathic sleep-related hypoventilation may represent cases of late-onset congenital central alveolar hypoventilation.

Relationship to Internationai Ciassification of Sleep Disorders

The *International Classification of Sleep Disorders,* 2nd Edition (ICSD-2), combines sleep-related hypoventilation and sleep-related hypoxemia under the category of sleep-related hypoventilation/hypoxemic syndromes. This approach to classification reflects the frequent co-occurrence of disorders that lead to hypoventilation and hypoxemia. In contrast, the classification used in DSM-5 reflects evidence that there are distinct sleep-related pathogenetic processes leading to hypoventilation.

Circadian Rhythm Sleep-Wake Disorders

Diagnostic Criteria

A. A persistent or recurrent pattern of sleep disruption that is primarily due to an alteration of the circadian system or to a misalignment between the endogenous circadian rhythm and the sleep–wake schedule required by an individual's physical environment or social or professional schedule.

B. The sleep disruption leads to excessive sleepiness or insomnia, or both.

C. The sleep disturbance causes clinically significant distress or impairment in social, occupational, and other important areas of functioning.

Coding note: For ICD-9-CM, code **307.45** for all subtypes. For ICD-10-CM, code is based on subtype.

Specify whether:

307.45 (G47.21) Delayed sleep phase type: A pattern of delayed sleep onset and awakening times, with an inability to fall asleep and awaken at a desired or conventionally acceptable earlier time.

Specify if:

Familial: A family history of delayed sleep phase is present.

Specify if:

Overlapping with non-24-hour sleep-wake type: Delayed sleep phase type may overlap with another circadian rhythm sleep-wake disorder, non-24-hour sleep-wake type.

307.45 (G47.22) Advanced sleep phase type: A pattern of advanced sleep onset and awakening times, with an inability to remain awake or asleep until the desired or conventionally acceptable later sleep or wake times.

Specify if:

Familial: A family history of advanced sleep phase is present.

307.45 (G47.23) Irregular sleep-wake type: A temporally disorganized sleep-wake pattern, such that the timing of sleep and wake periods is variable throughout the 24-hour period.

307.45 (G47.24) Non-24-hour sleep-wake type: A pattern of sleep-wake cycles that is not synchronized to the 24-hour environment, with a consistent daily drift (usually to later and later times) of sleep onset and wake times.

307.45 (G47.26) Shift work type: Insomnia during the major sleep period and/or excessive sleepiness (including inadvertent sleep) during the major awake period associated with a shift work schedule (i.e., requiring unconventional work hours).

307.45 (G47.20) Unspecified type

Specify if:

Episodic: Symptoms last at least 1 month but less than 3 months.

Persistent: Symptoms last 3 months or longer.

Recurrent: Two or more episodes occur within the space of 1 year.

Delayed Sleep Phase Type

Diagnostic Features

The delayed sleep phase type is based primarily on a history of a delay in the timing of the major sleep period (usually more than 2 hours) in relation to the desired sleep and wake-up time, resulting in symptoms of insomnia and excessive sleepiness. When allowed to set their own schedule, individuals with delayed sleep phase type exhibit normal sleep quality and duration for age. Symptoms of sleep-onset insomnia, difficulty waking in the morning, and excessive early day sleepiness are prominent.

Associated Features Supporting Diagnosis

Common associated features of delayed sleep phase type include a history of mental disorders or a concurrent mental disorder. Extreme and prolonged difficulty awakening with morning confusion is also common. Psychophysiological insomnia may develop as a result of maladaptive behaviors that impair sleep and increase arousal because of repeated attempts to fall asleep at an earlier time.

Prevaience

Prevalence of delayed sleep phase type in the general population is approximately 0.17% but appears to be greater than 7% in adolescents. Although the prevalence of familial delayed sleep phase type has not been established, a family history of delayed sleep phase is present in individuals with delayed sleep phase type.

Development and Course

Course is persistent, lasting longer than 3 months, with intermittent exacerbations throughout adulthood. Although age at onset is variable, symptoms begin typically in adolescence and early adulthood and persist for several months to years before diagnosis is established. Severity may decrease with age. Relapse of symptoms is common.

Clinical expression may vary across the lifespan depending on social, school, and work obligations. Exacerbation is usually triggered by a change in work or school schedule that requires an early rise time. Individuals who can alter their work schedules to accommodate the delayed circadian sleep and wake timing can experience remission of symptoms.

Increased prevalence in adolescence may be a consequence of both physiological and behavioral factors. Hormonal changes may be involved specifically, as delayed sleep phase is associated with the onset of puberty. Thus, delayed sleep phase type in adolescents should be differentiated from the common delay in the timing of circadian rhythms in this age group. In the familial form, the course is persistent and may not improve significantly with age.

Risk and Prognostic Factors

Genetic and physiological. Predisposing factors may include a longer than average circadian period, changes in light sensitivity, and impaired homeostatic sleep drive. Some individuals with delayed sleep phase type may be hypersensitive to evening light, which can serve as a delay signal to the circadian clock, or they may be hyposensitive to morning light such that its phase-advancing effects are reduced. Genetic factors may play a role in the pathogenesis of familial and sporadic forms of delayed sleep phase type, including mutations in circadian genes (e.g., *PER3, CKIe*).

Diagnostic Markers

Confirmation of the diagnosis includes a complete history and use of a sleep diary or actigraphy (i.e., a wrist-worn motion detector that monitors motor activity for prolonged periods and can be used as a proxy for sleep-wake patterns for at least 7 days). The period covered should include weekends, when social and occupational obligations are less strict, to ensure that the individual exhibits a consistently delayed sleep-wake pattern. Biomarkers such as salivary dim light melatonin onset should be obtained only when the diagnosis is unclear.

Functional Consequences of Delayed Sleep Phase Type

Excessive early day sleepiness is prominent. Extreme and prolonged difficulty awakening with morning confusion (i.e., sleep inertia) is also common. The severity of insomnia and excessive sleepiness symptoms varies substantially among individuals and largely depends on the occupational and social demands on the individual.

Differential Diagnosis

Normative variations in sleep. Delayed sleep phase type must be distinguished from "normal" sleep patterns in which an individual has a late schedule that does not cause personal, social, or occupational distress (most commonly seen in adolescents and young adults).

Other sleep disorders. Insomnia disorder and other circadian rhythm sleep-wake disorders should be included in the differential. Excessive sleepiness may also be caused by other sleep disturbances, such as breathing-related sleep disorders, insomnias, sleep-related movement disorders, and medical, neurological, and mental disorders. Overnight polysomnography may help in evaluating for other comorbid sleep disorders, such as sleep apnea. The circadian nature of delayed sleep phase type, however, should differentiate it from other disorders with similar complaints.

Comorbidity

Delayed sleep phase type is strongly associated with depression, personality disorder, and somatic symptom disorder or illness anxiety disorder. In addition, comorbid sleep disorders, such as insomnia disorder, restless legs syndrome, and sleep apnea, as well as depressive and bipolar disorders and anxiety disorders, can exacerbate symptoms of insomnia and excessive sleepiness. Delayed sleep phase type may overlap with another circadian rhythm sleep-wake disorder, non-24-hour sleep-wake type. Sighted individuals with non-24-hour sleep-wake type disorder commonly also have a history of delayed circadian sleep phase.

Advanced Sleep Phase Type

Specifiers

Advanced sleep phase type may be documented with the specified "familial." Although the prevalence of familial advanced sleep phase type has not been established, a family history of advanced sleep phase is present in individuals with advanced sleep phase type. In this type, specific mutations demonstrate an autosomal dominant mode of inheritance. In the familial form, onset of symptoms may occur earlier (during childhood and early adulthood), the course is persistent, and the severity of symptoms may increase with age.

Diagnostic Features

Advanced sleep phase type is characterized by sleep-wake times that are several hours earlier than desired or conventional times. Diagnosis is based primarily on a history of an advance in the timing of the major sleep period (usually more than 2 hours) in relation to the desired sleep and wake-up time, with symptoms of early morning insomnia and excessive daytime sleepiness. When allowed to set their schedule, individuals with advanced sleep phase type will exhibit normal sleep quality and duration for age.

Associated Features Supporting Diagnosis

Individuals with advanced sleep phase type are "morning types," having earlier sleep-wake times, with the timing of circadian biomarkers such as melatonin and core body temperature rhythms occurring 2–4 hours earlier than normal. When required to keep a conventional schedule requiring a delay of bedtime, these individuals will continue to have an early rise time, leading to persistent sleep deprivation and daytime sleepiness. Use of hypnotics or alcohol to combat sleep-maintenance insomnia and stimulants to reduce daytime sleepiness may lead to substance abuse in these individuals.

Prevalence

The estimated prevalence of advanced sleep phase type is approximately 1% in middle-age adults. Sleep-wake times and circadian phase advance in older individuals, probably accounting for increased prevalence in this population.

Development and Course

Onset is usually in late adulthood. In the familial form, onset can be earlier. The course is typically persistent, lasting more than 3 months, but the severity may increase depending on work and social schedules. The advanced sleep phase type is more common in older adults.

Clinical expression may vary across the lifespan depending on social, school, and work obligations. Individuals who can alter their work schedules to accommodate the advanced circadian sleep and wake timing can experience remission of symptoms. Increasing age tends to advance the sleep phase, however, it is unclear whether the common age-associated advanced sleep phase type is due solely to a change in circadian timing (as seen in the familial form) or also to age-related changes in the homeostatic regulation of sleep, resulting in earlier awakening. Severity, remission, and relapse of symptoms suggest lack of adherence to behavioral and environmental treatments designed to control sleep and wake structure and light exposure.

Risk and Prognostic Factors

Environmental. Decreased late afternoon/early evening exposure to light and/or exposure to early morning light due to early morning awakening can increase the risk of advanced sleep phase type by advancing circadian rhythms. By going to bed early, these individuals are not exposed to light in the phase delay region of the curve, resulting in perpetuation of advanced phase. In familial advanced sleep phase type, a shortening of the endogenous circadian period can result in an advanced sleep phase, although circadian period does not appear to systematically decrease with age.

Genetic and physiological. Advanced sleep phase type has demonstrated an autosomal dominant mode of inheritance, including a *PER2* gene mutation causing hypophosphorylation of the PER2 protein and a missense mutation in *CKI*.

Culture-Related Diagnostic Issues

African Americans may have a shorter circadian period and larger magnitude phase advances to light than do Caucasians, possibly increasing the risk for development of advanced sleep phase type in this population.

Diagnostic Markers

A sleep diary and actigraphy may be used as diagnostic markers, as described earlier for delayed sleep phase type.

Functional Consequences of Advanced Sleep Phase Type

Excessive sleepiness associated with advanced sleep phase can have a negative effect on cognitive performance, social interaction, and safety. Use of wake-promoting agents to combat sleepiness or sedatives for early morning awakening may increase potential for substance abuse.

Differential Diagnosis

Other sleep disorders. Behavioral factors such as irregular sleep schedules, voluntary early awakening, and exposure to light in the early morning should be considered, particularly in older adults. Careful attention should be paid to rule out other sleep-wake disorders, such as insomnia disorder, and other mental disorders and medical conditions that can cause early morning awakening.

Depressive and bipolar disorders. Because early morning awakening, fatigue, and sleepiness are prominent features of major depressive disorder, depressive and bipolar disorders must also be considered.

Comorbidity

Medical conditions and mental disorders with the symptom of early morning awakening, such as insomnia, can co-occur with the advance sleep phase type.

Irregular Sleep-Wake Type

Diagnostic Features

The diagnosis of irregular sleep-wake type is based primarily on a history of symptoms of insomnia at night (during the usual sleep period) and excessive sleepiness (napping) during the day. Irregular sleep-wake type is characterized by a lack of discernable sleep-wake

circadian rhythm. There is no major sleep period, and sleep is fragmented into at least three periods during the 24-hour day.

Associated Features Supporting Diagnosis

Individuals with irregular sleep-wake type typically present with insomnia or excessive sleepiness, depending on the time of day. Sleep and wake periods across 24 hours are fragmented, although the longest sleep period tends to occur between 2:00 A.M. and 6:00 A.M. and is usually less than 4 hours. A history of isolation or reclusion may occur in association with the disorder and contribute to the symptoms via a lack of external stimuli to help entrain a normal pattern. Individuals or their caregivers report frequent naps throughout the day. Irregular sleep-wake type is most commonly associated with neurodegenerative disorders, such as major neurocognitive disorder, and many neurodevelopmental disorders in children.

Prevalence

Prevalence of irregular sleep-wake type in the general population is unknown.

Deveiopment and Course

The course of irregular sleep-wake type is persistent. Age at onset is variable, but the disorder is more common in older adults.

Risk and Prognostic Factors

Temperamental. Neurodegenerative disorders, such as Alzheimer's disease, Parkinson's disease, and Huntington's disease, and neurodevelopmental disorders in children increase the risk for irregular sleep-wake type.

Environmental. Decreased exposure to environmental light and structured daytime activity can be associated with a low-amplitude circadian rhythm. Hospitalized individuals are especially prone to such weak external entraining stimuli, and even outside the hospital setting, individuals with major neurocognitive disorder (i.e., dementia) are exposed to significantly less bright light.

Diagnostic Markers

A detailed sleep history and a sleep diary (by a caregiver) or actigraphy help confirm the irregular sleep-wake pattern.

Functional Consequences of Irregular Sleep-Wake Type

Lack of a clearly discernible major sleep and wake period in irregular sleep-wake type results in insomnia or excessive sleepiness, depending on the time of day. Disruption of the caregiver's sleep also often occurs and is an important consideration.

Differential Diagnosis

Normative variations in sleep. Irregular sleep-wake type should be distinguished from a voluntary irregular sleep-wake schedule and poor sleep hygiene, which can result in insomnia and excessive sleepiness.

Other medical conditions and mental disorders. Other causes of insomnia and daytime sleepiness, including comorbid medical conditions and mental disorders or medication, should be considered.

Comorbidity

Irregular sleep-wake type is often comorbid with neurodegenerative and neurodevelopmental disorders, such as major neurocognitive disorder, intellectual disability (intellectual developmental disorder), and traumatic brain injury. It is also comorbid with other medical conditions and mental disorders in which there is social isolation and/or lack of light and structured activities.

Non-24-Hour Sleep-Wake Type

Diagnostic Features

The diagnosis of non-24-hour sleep-wake type is based primarily on a history of symptoms of insomnia or excessive sleepiness related to abnormal synchronization between the 24-hour light-dark cycle and the endogenous circadian rhythm. Individuals typically present with periods of insomnia, excessive sleepiness, or both, which alternate with short asymptomatic periods. Starting with the asymptomatic period, when the individual's sleep phase is aligned to the external environment, sleep latency will gradually increase and the individual will complain of sleep-onset insomnia. As the sleep phase continues to drift so that sleep time is now in the daytime, the individual will have trouble staying awake during the day and will complain of sleepiness. Because the circadian period is not aligned to the external 24-hour environment, symptoms will depend on when an individual tries to sleep in relation to the circadian rhythm of sleep propensity.

Associated Features Supporting Diagnosis

Non-24-hour sleep-wake type is most common among blind or visually impaired individuals who have decreased light perception. In sighted individuals, there is often a history of delayed sleep phase and of decreased exposure to light and structured social and physical activity. Sighted individuals with non-24-hour sleep-wake type also demonstrate increased sleep duration.

Prevalence

Prevalence of non-24-hour sleep-wake type in the general population is unclear, but the disorder appears rare in sighted individuals. The prevalence in blind individuals is estimated to be 50%.

Development and Course

Course of non-24-hour sleep-wake type is persistent, with intermittent remission and exacerbations due to changes in work and social schedules throughout the lifespan. Age at onset is variable, depending on the onset of visual impairment. In sighted individuals, because of the overlap with delayed sleep phase type, non-24-hour sleep-wake type may develop in adolescence or early adulthood. Remission and relapse of symptoms in blind and sighted individuals largely depend on adherence to treatments designed to control sleep and wake structure and light exposure.

Clinical expression may vary across the lifespan depending on social, school, and work obligations. In adolescents and adults, irregular sleep-wake schedules and exposure to light or lack of light at critical times of the day can exacerbate the effects of sleep loss and disrupt circadian entrainment. Consequently, symptoms of insomnia, daytime sleepiness, and school, professional, and interpersonal functioning may worsen.

Risk and Prognostic Factors

Environmental. In sighted individuals, decreased exposure or sensitivity to light and social and physical activity cues may contribute to a free-running circadian rhythm. With the

high frequency of mental disorders involving social isolation and cases of non-24-hour sleep-wake type developing after a change in sleep habits (e.g., night shift work, job loss), behavioral factors in combination with physiological tendency may precipitate and perpetuate this disorder in sighted individuals. Hospitalized individuals with neurological and psychiatric disorders can become insensitive to social cues, predisposing them to the development of non-24-hour sleep-wake type.

Genetic and physiological. Blindness is a risk factor for non-24-hour sleep-wake type. Non-24-hour sleep-wake type has been associated with traumatic brain injury.

Diagnostic Markers

Diagnosis is confirmed by history and sleep diary or actigraphy for an extended period. Sequential measurement of phase markers (e.g., melatonin) can help determine circadian phase in both sighted and blind individuals.

Functional Consequences of Non-24-Hour Sleep-Wake Type

Complaints of insomnia (sleep onset and sleep maintenance), excessive sleepiness, or both are prominent. The unpredictability of sleep and wake times (typically a daily delay drift) results in an inability to attend school or maintain a steady job and may increase potential for social isolation.

Differential Diagnosis

Circadian rhythm sleep-wake disorders. In sighted individuals, non-24-hour sleep-wake type should be differentiated from delayed sleep phase type, as individuals with delayed sleep phase type may display a similar progressive delay in sleep period for several days.

Depressive disorders. Depressive symptoms and depressive disorders may result in similar circadian dysregulation and symptoms.

Comorbidity

Blindness is often comorbid with non-24-hour sleep-wake type, as are depressive and bipolar disorders with social isolation.

Shift Work Type

Diagnostic Features

Diagnosis is primarily based on a history of the individual working outside of the normal 8:00 A.M. to 6:00 P.M. daytime window (particularly at night) on a regularly scheduled (i.e., non-overtime) basis. Symptoms of excessive sleepiness at work, and impaired sleep at home, on a persistent basis are prominent. Presence of both sets of symptoms are usually required for a diagnosis of shift work type. Typically, when the individual reverts to a day-work routine, symptoms resolve. Although the etiology is slightly different, individuals who travel across many time zones on a very frequent basis may experience effects similar to those experienced by individuals with shift work type who work rotating shifts.

Prevalence

The prevalence of shift work type is unclear, but the disorder is estimated to affect 5%–10% of the night worker population (16%–20% of the workforce). Prevalence rises with advancement into middle-age and beyond (Drake et al. 2004).

Development and Course

Shift work type can appear in individuals of any age but is more prevalent in individuals older than 50 years and typically worsens with the passage of time if the disruptive work hours persist. Although older adults may show similar rates of circadian phase adjustment to a change in routine as do younger adults, they appear to experience significantly more sleep disruption as a consequence of the circadian phase shift.

Risk and Prognostic Factors

Temperamental. Predisposing factors include a morning-type disposition, a need for long (i.e., more than 8 hours) sleep durations in order to feel well rested, and strong competing social and domestic needs (e.g., parents of young children). Individuals who are able to commit to a nocturnal lifestyle, with few competing day-oriented demands, appear at lower risk for shift work type.

Genetic and physiological. Because shift workers are more likely than day workers to be obese, obstructive sleep apnea may be present and may exacerbate the symptoms.

Diagnostic Markers

A history and sleep diary or actigraphy may be useful in diagnosis, as discussed earlier for delayed sleep phase type.

Functional Consequences of Shift Work Type

Individuals with shift work type not only may perform poorly at work but also appear to be at risk for accidents both at work and on the drive home. They may also be at risk for poor mental health (e.g., alcohol use disorder, substance use disorder, depression) and physical health (e.g., gastrointestinal disorders, cardiovascular disease, diabetes, cancer). Individuals with a history of bipolar disorder are particularly vulnerable to shift work type–related episodes of mania resulting from missed nights of sleep. Shift work type often results in interpersonal problems.

Differential Diagnosis

Normative variations in sleep with shift work. The diagnosis of shift work type, as opposed to the "normal" difficulties of shift work, must depend to some extent on the severity of symptoms and/or level of distress experienced by the individual. Presence of shift work type symptoms even when the individual is able to live on a day-oriented routine for several weeks at a time may suggest the presence of other sleep disorders, such as sleep apnea, insomnia, and narcolepsy, which should be ruled out.

Comorbidity

Shift work type has been associated with increased alcohol use disorder, other substance use disorders, and depression. A variety of physical health disorders (e.g., gastrointestinal disorders, cardiovascular disease, diabetes, cancer) have been found to be associated with prolonged exposure to shift work.

Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders*, 2nd Edition (ICSD-2), differentiates nine circadian rhythm sleep disorders, including jet lag type.

Parasomnias

Parasomnias are disorders characterized by abnormal behavioral, experiential, or physiological events occurring in association with sleep, specific sleep stages, or sleep-wake transitions. The most common parasomnias—non–rapid eye movement (NREM) sleep arousal disorders and rapid eye movement (REM) sleep behavior disorder—represent admixtures of wakefulness and NREM sleep and wakefulness and REM sleep, respectively. These conditions serve as a reminder that sleep and wakefulness are not mutually exclusive and that sleep is not necessarily a global, whole-brain phenomenon.

Non–Rapid Eye Movement Sleep Arousal Disorders

Diagnostic Criteria

A. Recurrent episodes of incomplete awakening from sleep, usually occurring during the first third of the major sleep episode, accompanied by either one of the following:

   1. **Sleepwalking:** Repeated episodes of rising from bed during sleep and walking about. While sleepwalking, the individual has a blank, staring face; is relatively unresponsive to the efforts of others to communicate with him or her; and can be awakened only with great difficulty.
   2. **Sleep terrors:** Recurrent episodes of abrupt terror arousals from sleep, usually beginning with a panicky scream. There is intense fear and signs of autonomic arousal, such as mydriasis, tachycardia, rapid breathing, and sweating, during each episode. There is relative unresponsiveness to efforts of others to comfort the individual during the episodes.

B. No or little (e.g., only a single visual scene) dream imagery is recalled.

C. Amnesia for the episodes is present.

D. The episodes cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

E. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication).

F. Coexisting mental and medical disorders do not explain the episodes of sleepwalking or sleep terrors.

Coding note: For ICD-9-CM, code **307.46** for all subtypes. For ICD-10-CM, code is based on subtype.

Specify whether:

307.46 (F51.3) Sleepwalking type

Specify if:

With sleep-related eating

With sleep-related sexual behavior (sexsomnia)

307.46 (F51.4) Sleep terror type

Diagnostic Features

The essential feature of non–rapid eye movement (NREM) sleep arousal disorders is the repeated occurrence of incomplete arousals, usually beginning during the first third of the major sleep episode (Criterion A), that typically are brief, lasting 1–10 minutes, but may be protracted, lasting up to 1 hour. The maximum duration of an event is unknown. The eyes are typically open during these events. Many individuals exhibit both subtypes of arousals on different occasions, which underscores the unitary underlying pathophysiology. The subtypes reflect varying degrees of simultaneous occurrence of wakefulness and NREM sleep, resulting in complex behaviors arising from sleep with varying degrees of conscious awareness, motor activity, and autonomic activation.

The essential feature of *sleepwalking* is repeated episodes of complex motor behavior initiated during sleep, including rising from bed and walking about (Criterion A1). Sleepwalking episodes begin during any stage of NREM sleep, most commonly during slow-wave sleep and therefore most often occurring during the first third of the night. During episodes, the individual has reduced alertness and responsiveness, a blank stare, and relative unresponsiveness to communication with others or efforts by others to awaken the individual. If awakened during the episode (or on awakening the following morning), the individual has limited recall for the episode. After the episode, there may initially be a brief period of confusion or difficulty orienting, followed by full recovery of cognitive function and appropriate behavior.

The essential feature of *sleep terrors* is the repeated occurrence of precipitous awakenings from sleep, usually beginning with a panicky scream or cry (Criterion A2). Sleep terrors usually begin during the first third of the major sleep episode and last 1–10 minutes, but they may last considerably longer, particularly in children. The episodes are accompanied by impressive autonomic arousal and behavioral manifestations of intense fear. During an episode, the individual is difficult to awaken or comfort. If the individual awakens after the sleep terror, little or none of the dream, or only fragmentary, single images, are recalled. During a typical episode of sleep terrors, the individual abruptly sits up in bed screaming or crying, with a frightened expression and autonomic signs of intense anxiety (e.g., tachycardia, rapid breathing, sweating, dilation of the pupils). The individual may be inconsolable and is usually unresponsive to the efforts of others to awaken or comfort him or her. Sleep terrors are also called "night terrors" or "pavor nocturnus."

Associated Features Supporting Diagnosis

Sleepwalking episodes can include a wide variety of behaviors. Episodes may begin with confusion: the individual may simply sit up in bed, look about, or pick at the blanket or sheet. This behavior then becomes progressively complex. The individual may actually leave the bed and walk into closets, out of the room, and even out of buildings. Individuals may use the bathroom, eat, talk, or engage in more complex behaviors. Running and frantic attempts to escape some apparent threat can also occur. Most behaviors during sleepwalking episodes are routine and of low complexity. However, cases of unlocking doors and even operating machinery (driving an automobile) have been reported. Sleepwalking can also include inappropriate behavior (e.g., commonly, urinating in a closet or wastebasket). Most episodes last for several minutes to a half hour but may be more protracted. Inasmuch as sleep is a state of relative analgesia, painful injuries sustained during sleepwalking may not be appreciated until awakening after the fact.

There are two "specialized" forms of sleepwalking: sleep-related eating behavior and sleep-related sexual behavior (sexsomnia or sleep sex). Individuals with *sleep-related eating* experience unwanted recurrent episodes of eating with varying degrees of amnesia, ranging from no awareness to full awareness without the ability to not eat. During these episodes, inappropriate foods may be ingested. Individuals with sleep-related eating disorder may find evidence of their eating only the next morning. In *sexsomnia*, varying degrees of

sexual activity (e.g., masturbation, fondling, groping, sexual intercourse) occur as complex behaviors arising from sleep without conscious awareness. This condition is more common in males and may result in serious interpersonal relationship problems or medicolegal consequences.

During a typical episode of sleep terrors, there is often a sense of overwhelming dread, with a compulsion to escape. Although fragmentary vivid dream images may occur, a story-like dream sequence (as in nightmares) is not reported. Most commonly, the individual does not awaken fully, but returns to sleep and has amnesia for the episode on awakening the next morning. Usually only one episode will occur on any one night. Occasionally several episodes may occur at intervals throughout the night. These events rarely arise during daytime naps.

Prevalence

Isolated or infrequent NREM sleep arousal disorders are very common in the general population. From 10% to 30% of children have had at least one episode of sleepwalking, and 2%–3% sleepwalk often. The prevalence of sleepwalking disorder, marked by repeated episodes and impairment or distress, is much lower, probably in the range of 1%–5%. The prevalence of sleepwalking episodes (not sleepwalking disorder) is 1.0%–7.0% among adults, with weekly to monthly episodes occurring in 0.5%–0.7%. The lifetime prevalence of sleepwalking in adults is 29.2%, with a past-year prevalence of sleepwalking of 3.6%.

The prevalence of sleep terrors in the general population is unknown. The prevalence of sleep terror episodes (as opposed to sleep terror disorder, in which there is recurrence and distress or impairment) is approximately 36.9% at 18 months of age, 19.7% at 30 months of age, and 2.2% in adults.

Development and Course

NREM sleep arousal disorders occur most commonly in childhood and diminish in frequency with increasing age. The onset of sleepwalking in adults with no prior history of sleepwalking as children should prompt a search for specific etiologies, such as obstructive sleep apnea, nocturnal seizures, or effect of medication.

Risk and Prognostic Factors

Environmental. Sedative use, sleep deprivation, sleep-wake schedule disruptions, fatigue, and physical or emotional stress increase the likelihood of episodes. Fever and sleep deprivation can produce an increased frequency of NREM sleep arousal disorders.

Genetic and physiological. A family history for sleepwalking or sleep terrors may occur in up to 80% of individuals who sleepwalk. The risk for sleepwalking is further increased (to as much as 60% of offspring) when both parents have a history of the disorder.

Individuals with sleep terrors frequently have a positive family history of either sleep terrors or sleepwalking, with as high as a 10-fold increase in the prevalence of the disorder among first-degree biological relatives. Sleep terrors are much more common in monozygotic twins as compared with dizygotic twins. The exact mode of inheritance is unknown.

Gender-Related Diagnostic Issues

Violent or sexual activity during sleepwalking episodes is more likely to occur in adults. Eating during sleepwalking episodes is more commonly seen in females. Sleepwalking occurs more often in females during childhood but more often in males during adulthood.

Older children and adults provide a more detailed recollection of fearful images associated with sleep terrors than do younger children, who are more likely to have complete amnesia or report only a vague sense of fear. Among children, sleep terrors are more common in males than in females. Among adults, the sex ratio is even.

Diagnostic Markers

NREM sleep arousal disorders arise from any stage of NREM sleep but most commonly from deep NREM sleep (slow-wave sleep). They are most likely to appear in the first third of the night and do not commonly occur during daytime naps. During the episode, the polysomnogram may be obscured with movement artifact. In the absence of such artifact, the electroencephalogram typically shows theta or alpha frequency activity during the episode, indicating partial or incomplete arousal.

Polysomnography in conjunction with audiovisual monitoring can be used to document episodes of sleepwalking. In the absence of actually capturing an event during a polysomnographic recording, there are no polysomnographic features that can serve as a marker for sleepwalking. Sleep deprivation may increase the likelihood of capturing an event. As a group, individuals who sleepwalk show instability of deep NREM sleep, but the overlap in findings with individuals who do not sleepwalk is great enough to preclude use of this indicator in establishing a diagnosis. Unlike arousals from REM sleep associated with nightmares, in which there is an increase in heart rate and respiration prior to the arousal, the NREM sleep arousals of sleep terrors begin precipitously from sleep, without anticipatory autonomic changes. The arousals are associated with impressive autonomic activity, with doubling or tripling of the heart rate. The pathophysiology is poorly understood, but there appears to be instability in the deeper stages of NREM sleep. Absent capturing an event during a formal sleep study, there are no reliable polysomnographic indicators of the tendency to experience sleep terrors.

Functional Consequences of Non-REM Sleep Arousal Disorders

For the diagnosis of a NREM sleep arousal disorder to be made, the individual or household members must experience clinically significant distress or impairment, although parasomnia symptoms may occur occasionally in nonclinical populations and would be subthreshold for the diagnosis. Embarrassment concerning the episodes can impair social relationships. Social isolation or occupational difficulties can result. The determination of a "disorder" depends on a number of factors, which may vary on an individual basis and will depend on the frequency of events, potential for violence or injurious behaviors, embarrassment, or disruption/distress of other household members. Severity determination is best made based on the nature or consequence of the behaviors rather than simply on frequency. Uncommonly, NREM sleep arousal disorders may result in serious injury to the individual or to someone trying to console the individual. Injuries to others are confined to those in close proximity; individuals are not "sought out." Typically, sleepwalking in both children and adults is not associated with significant mental disorders. For individuals with sleep-related eating behaviors, unknowingly preparing or eating food during the sleep period may create problems such as poor diabetes control, weight gain, injury (cuts and burns), or consequences of eating dangerous or toxic inedibles. NREM sleep arousal disorders may rarely result in violent or injurious behaviors with forensic implications.

Differential Diagnosis

Nightmare disorder. In contrast to individuals with NREM sleep arousal disorders, individuals with nightmare disorder typically awaken easily and completely, report vivid storylike dreams accompanying the episodes, and tend to have episodes later in the night. NREM sleep arousal disorders occur during NREM sleep, whereas nightmares usually occur during REM sleep. Parents of children with NREM sleep arousal disorders may misinterpret reports of fragmentary imagery as nightmares.

Breathing-related sleep disorders. Breathing disorders during sleep can also produce confusional arousals with subsequent amnesia. However, breathing-related sleep disorders are also characterized by characteristic symptoms of snoring, breathing pauses, and

daytime sleepiness. In some individuals, a breathing-related sleep disorder may precipitate episodes of sleepwalking.

REM sleep behavior disorder. REM sleep behavior disorder may be difficult to distinguish from NREM sleep arousal disorders. REM sleep behavior disorder is characterized by episodes of prominent, complex movements, often involving personal injury arising from sleep. In contrast to NREM sleep arousal disorders, REM sleep behavior disorder occurs during REM sleep. Individuals with REM sleep behavior disorder awaken easily and report more detailed and vivid dream content than do individuals with NREM sleep arousal disorders. They often report that they "act out dreams."

Parasomnia overlap syndrome. Parasomnia overlap syndrome consists of clinical and polysomnographic features of both sleepwalking and REM sleep behavior disorder.

Sleep-related seizures. Some types of seizures can produce episodes of very unusual behaviors that occur predominantly or exclusively during sleep. Nocturnal seizures may closely mimic NREM sleep arousal disorders but tend to be more stereotypic in nature, occur multiple times nightly, and be more likely to occur from daytime naps. The presence of sleep-related seizures does not preclude the presence of NREM sleep arousal disorders. Sleep-related seizures should be classified as a form of epilepsy.

Alcohol-induced blackouts. Alcohol-induced blackouts may be associated with extremely complex behaviors in the absence of other suggestions of intoxication. They do not involve the loss of consciousness but rather reflect an isolated disruption of memory for events during a drinking episode. By history, these behaviors may be indistinguishable from those seen in NREM sleep arousal disorders.

Dissociative amnesia, with dissociative fugue. Dissociative fugue may be extremely difficult to distinguish from sleepwalking. Unlike all other parasomnias, nocturnal dissociative fugue arises from a period of wakefulness during sleep, rather than precipitously from sleep without intervening wakefulness. A history of recurrent childhood physical or sexual abuse is usually present (but may be difficult to obtain).

Malingering or other voluntary behavior occurring during wakefulness. As with dissociative fugue, malingering or other voluntary behavior occurring during wakefulness arises from wakefulness.

Panic disorder. Panic attacks may also cause abrupt awakenings from deep NREM sleep accompanied by fearfulness, but these episodes produce rapid and complete awakening without the confusion, amnesia, or motor activity typical of NREM sleep arousal disorders.

Medication-induced complex behaviors. Behaviors similar to those in NREM sleep arousal disorders can be induced by use of, or withdrawal from, substances or medications (e.g., benzodiazepines, nonbenzodiazepine sedative-hypnotics, opiates, cocaine, nicotine, antipsychotics, tricyclic antidepressants, chloral hydrate). Such behaviors may arise from the sleep period and may be extremely complex. The underlying pathophysiology appears to be a relatively isolated amnesia. In such cases, substance/medication-induced sleep disorder, parasomnia type, should be diagnosed (see "Substance/Medication-Induced Sleep Disorder" later in this chapter).

Night eating syndrome. The sleep-related eating disorder form of sleepwalking is to be differentiated from night eating syndrome, in which there is a delay in the circadian rhythm of food ingestion and an association with insomnia and/or depression.

Comorbidity

In adults, there is an association between sleepwalking and major depressive episodes and obsessive-compulsive disorder. Children or adults with sleep terrors may have elevated scores for depression and anxiety on personality inventories.

Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders,* 2nd Edition, includes "confusional arousal" as a NREM sleep arousal disorder.

Nightmare Disorder

Diagnostic Criteria **307.47 (F51.5)**

A. Repeated occurrences of extended, extremely dysphoric, and well-remembered dreams that usually involve efforts to avoid threats to survival, security, or physical integrity and that generally occur during the second half of the major sleep episode.

B. On awakening from the dysphoric dreams, the individual rapidly becomes oriented and alert.

C. The sleep disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The nightmare symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication).

E. Coexisting mental and medical disorders do not adequately explain the predominant complaint of dysphoric dreams.

Specify if:

During sleep onset

Specify if:

With associated non–sleep disorder, including substance use disorders

With associated other medical condition

With associated other sleep disorder

Coding note: The code 307.47 (F51.5) applies to all three specifiers. Code also the relevant associated mental disorder, medical condition, or other sleep disorder immediately after the code for nightmare disorder in order to indicate the association.

Specify if:

Acute: Duration of period of nightmares is 1 month or less.

Subacute: Duration of period of nightmares is greater than 1 month but less than 6 months.

Persistent: Duration of period of nightmares is 6 months or greater.

Specify current severity:

Severity can be rated by the frequency with which the nightmares occur:

Mild: Less than one episode per week on average.

Moderate: One or more episodes per week but less than nightly.

Severe: Episodes nightly.

Diagnostic Features

Nightmares are typically lengthy, elaborate, storylike sequences of dream imagery that seem real and that incite anxiety, fear, or other dysphoric emotions. Nightmare content typically focuses on attempts to avoid or cope with imminent danger but may involve themes that evoke other negative emotions. Nightmares occurring after traumatic experiences may replicate the threatening situation ("replicative nightmares"), but most do not. On awakening, nightmares are well remembered and can be described in detail. They arise

almost exclusively during rapid eye movement (REM) sleep and can thus occur throughout sleep but are more likely in the second half of the major sleep episode when dreaming is longer and more intense. Factors that increase early-night REM intensity, such as sleep fragmentation or deprivation, jet lag, and REM-sensitive medications, might facilitate nightmares earlier in the night, including at sleep onset.

Nightmares usually terminate with awakening and rapid return of full alertness. However, the dysphoric emotions may persist into wakefulness and contribute to difficulty returning to sleep and lasting daytime distress. Some nightmares, known as "bad dreams," may not induce awakening and are recalled only later. If nightmares occur during sleep-onset REM periods (*hypnagogic*), the dysphoric emotion is frequently accompanied by a sense of being both awake and unable to move voluntarily (*isolated sleep paralysis*).

Associated Features Supporting Diagnosis

Mild autonomic arousal, including sweating, tachycardia, and tachypnea, may characterize nightmares. Body movements and vocalizations are not characteristic because of REM sleep–related loss of skeletal muscle tone, but such behaviors may occur under situations of emotional stress or sleep fragmentation and in posttraumatic stress disorder (PTSD). When talking or emoting occurs, it is typically a brief event terminating the nightmare.

Individuals with frequent nightmares are at substantially greater risk for suicidal ideation and suicide attempts, even when gender and mental illness are taken into account.

Prevalence

Prevalence of nightmares increases through childhood into adolescence. From 1.3% to 3.9% of parents report that their preschool children have nightmares "often" or "always". Prevalence increases from ages 10 to 13 for both males and females but continues to increase to ages 20–29 for females (while decreasing for males), when it can be twice as high for females as for males. Prevalence decreases steadily with age for both sexes, but the gender difference remains. Among adults, prevalence of nightmares at least monthly is 6%, whereas prevalence for frequent nightmares is 1%–2%. Estimates often combine idiopathic and posttraumatic nightmares indiscriminately.

Development and Course

Nightmares often begin between ages 3 and 6 years but reach a peak prevalence and severity in late adolescence or early adulthood. Nightmares most likely appear in children exposed to acute or chronic psychosocial stressors and thus may not resolve spontaneously. In a minority, frequent nightmares persist into adulthood, becoming virtually a lifelong disturbance. Although specific nightmare content may reflect the individual's age, the essential features of the disorder are the same across age groups.

Risk and Prognostic Factors

Temperamental. Individuals who experience nightmares report more frequent past adverse events, but not necessarily trauma, and often display personality disturbances or psychiatric diagnosis.

Environmental. Sleep deprivation or fragmentation, and irregular sleep-wake schedules that alter the timing, intensity, or quantity of REM sleep, can put individuals at risk for nightmares.

Genetic and physiological. Twin studies have identified genetic effects on the disposition to nightmares and their co-occurrence with other parasomnias (e.g., sleeptalking).

Course modifiers. Adaptive parental bedside behaviors, such as soothing the child following nightmares, may protect against developing chronic nightmares.

Culture-Related Diagnostic Issues

The significance attributed to nightmares may vary by culture, and sensitivity to such beliefs may facilitate disclosure.

Gender-Related Diagnostic Issues

Adult females report having nightmares more frequently than do adult males. Nightmare content differs by sex, with adult females tending to report themes of sexual harassment or of loved ones disappearing/dying, and adult males tending to report themes of physical aggression or war/terror.

Diagnostic Markers

Polysomnographic studies demonstrate abrupt awakenings from REM sleep, usually during the second half of the night, prior to report of a nightmare. Heart, respiratory, and eye movement rates may quicken or increase in variability before awakening. Nightmares following traumatic events may also arise during non-REM (NREM), particularly stage 2, sleep. The typical sleep of individuals with nightmares is mildly impaired (e.g., reduced efficiency, less slow-wave sleep, more awakenings), with more frequent periodic leg movements in sleep and relative sympathetic nervous system activation after REM sleep deprivation.

Functional Consequences of Nightmare Disorder

Nightmares cause more significant subjective distress than demonstrable social or occupational impairment. However, if awakenings are frequent or result in sleep avoidance, individuals may experience excessive daytime sleepiness, poor concentration, depression, anxiety, or irritability. Frequent childhood nightmares (e.g., several per week), may cause significant distress to parents and child.

Differential Diagnosis

Sleep terror disorder. Both nightmare disorder and sleep terror disorder include awakenings or partial awakenings with fearfulness and autonomic activation, but the two disorders are differentiable. Nightmares typically occur later in the night, during REM sleep, and produce vivid, storylike, and clearly recalled dreams; mild autonomic arousal; and complete awakenings. Sleep terrors typically arise in the first third of the night during stage 3 or 4 NREM sleep and produce either no dream recall or images without an elaborate storylike quality. The terrors lead to partial awakenings that leave the individual confused, disoriented, and only partially responsive and with substantial autonomic arousal. There is usually amnesia for the event in the morning.

REM sleep behavior disorder. The presence of complex motor activity during frightening dreams should prompt further evaluation for REM sleep behavior disorder, which occurs more typically among late middle-age males and, unlike nightmare disorder, is associated with often violent dream enactments and a history of nocturnal injuries. The dream disturbance of REM sleep behavior disorder is described by patients as nightmares but is controlled by appropriate medication.

Bereavement. Dysphoric dreams may occur during bereavement but typically involve loss and sadness and are followed by self-reflection and insight, rather than distress, on awakening.

Narcolepsy. Nightmares are a frequent complaint in narcolepsy, but the presence of excessive sleepiness and cataplexy differentiates this condition from nightmare disorder.

Nocturnal seizures. Seizures may rarely manifest as nightmares and should be evaluated with polysomnography and continuous video electroencephalography. Nocturnal seizures usually involve stereotypical motor activity. Associated nightmares, if recalled,

are often repetitive in nature or reflect epileptogenic features such as the content of diurnal auras (e.g., unmotivated dread), phosphenes, or ictal imagery. Disorders of arousal, especially confusional arousals, may also be present.

Breathing-related sleep disorders. Breathing-related sleep disorders can lead to awakenings with autonomic arousal, but these are not usually accompanied by recall of nightmares.

Panic disorder. Attacks arising during sleep can produce abrupt awakenings with autonomic arousal and fearfulness, but nightmares are typically not reported and symptoms are similar to panic attacks arising during wakefulness.

Sleep-related dissociative disorders. Individuals may recall actual physical or emotional trauma as a "dream" during electroencephalography-documented awakenings.

Medication or substance use. Numerous substances/medications can precipitate nightmares, including dopaminergics; beta-adrenergic antagonists and other antihypertensives; amphetamine, cocaine, and other stimulants; antidepressants; smoking cessation aids; and melatonin. Withdrawal of REM sleep–suppressant medications (e.g., antidepressants) and alcohol can produce REM sleep rebound accompanied by nightmares. If nightmares are sufficiently severe to warrant independent clinical attention, a diagnosis of substance/medication-induced sleep disorder should be considered.

Comorbidity

Nightmares may be comorbid with several medical conditions, including coronary heart disease, cancer, parkinsonism, and pain, and can accompany medical treatments, such as hemodialysis, or withdrawal from medications or substances of abuse. Nightmares frequently are comorbid with other mental disorders, including PTSD; insomnia disorder; schizophrenia; psychosis; mood, anxiety, adjustment, and personality disorders; and grief during bereavement. A concurrent nightmare disorder diagnosis should only be considered when independent clinical attention is warranted (i.e., Criteria A–C are met). Otherwise, no separate diagnosis is necessary. These conditions should be listed under the appropriate comorbid category specifier. However, nightmare disorder may be diagnosed as a separate disorder in individuals with PTSD if the nightmares are temporally unrelated to PTSD (i.e., preceding other PTSD symptoms or persisting after other PTSD symptoms have resolved).

Nightmares are normally characteristic of REM sleep behavior disorder, PTSD, and acute stress disorder, but nightmare disorder may be independently coded if nightmares preceded the condition and their frequency or severity necessitates independent clinical attention. The latter may be determined by asking whether nightmares were a problem before onset of the other disorder and whether they continued after other symptoms had remitted.

Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders*, 2nd Edition (ICSD-2), presents similar diagnostic criteria for nightmare disorder.

Rapid Eye Movement Sleep Behavior Disorder

Diagnostic Criteria **327.42 (G47.52)**

A. Repeated episodes of arousal during sleep associated with vocalization and/or complex motor behaviors.

B. These behaviors arise during rapid eye movement (REM) sleep and therefore usually occur more than 90 minutes after sleep onset, are more frequent during the later portions of the sleep period, and uncommonly occur during daytime naps.

C. Upon awakening from these episodes, the individual is completely awake, alert, and not confused or disoriented.
D. Either of the following:
   1. REM sleep without atonia on polysomnographic recording.
   2. A history suggestive of REM sleep behavior disorder and an established synucleinopathy diagnosis (e.g., Parkinson's disease, multiple system atrophy).
E. The behaviors cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (which may include injury to self or the bed partner).
F. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.
G. Coexisting mental and medical disorders do not explain the episodes.

Diagnostic Features

The essential feature of rapid eye movement (REM) sleep behavior disorder is repeated episodes of arousal, often associated with vocalizations and/or complex motor behaviors arising from REM sleep (Criterion A). These behaviors often reflect motor responses to the content of action-filled or violent dreams of being attacked or trying to escape from a threatening situation, which may be termed *dream enacting behaviors*. The vocalizations are often loud, emotion-filled, and profane. These behaviors may be very bothersome to the individual and the bed partner and may result in significant injury (e.g., falling, jumping, or flying out of bed; running, punching, thrusting, hitting, or kicking). Upon awakening, the individual is immediately awake, alert, and oriented (Criterion C) and is often able to recall dream mentation, which closely correlates with the observed behavior. The eyes typically remain closed during these events. The diagnosis of REM sleep behavior disorder requires clinically significant distress or impairment (Criterion E); this determination will depend on a number of factors, including the frequency of events, the potential for violence or injurious behaviors, embarrassment, and distress in other household members.

Associated Features Supporting Diagnosis

Severity determination is best made based on the nature or consequence of the behavior rather than simply on frequency. Although the behaviors are typically prominent and violent, lesser behaviors may also occur.

Prevalence

The prevalence of REM sleep behavior disorder is approximately 0.38%–0.5% in the general population. Prevalence in patients with psychiatric disorders may be greater, possibly related to medications prescribed for the psychiatric disorder.

Development and Course

The onset of REM sleep behavior disorder may be gradual or rapid, and the course is usually progressive. REM sleep behavior disorder associated with neurodegenerative disorders may improve as the underlying neurodegenerative disorder progresses. Because of the very high association with the later appearance of an underlying neurodegenerative disorder, most notably one of the synucleinopathies (Parkinson's disease, multiple system atrophy, or major or mild neurocognitive disorder with Lewy bodies), the neurological status of individuals with REM sleep behavior disorder should be closely monitored.

REM sleep behavior disorder overwhelmingly affects males older than 50 years, but increasingly this disorder is being identified in females and in younger individuals. Symp-

toms in young individuals, particularly young females, should raise the possibility of narcolepsy or medication-induced REM sleep behavior disorder.

Risk and Prognostic Factors

Genetic and physiological. Many widely prescribed medications, including tricyclic antidepressants, selective serotonin reuptake inhibitors, serotonin-norepinephrine reuptake inhibitors, and beta-blockers, may result in polysomnographic evidence of REM sleep without atonia and in frank REM sleep behavior disorder. It is not known whether the medications per se result in REM sleep behavior disorder or they unmask an underlying predisposition.

Diagnostic Markers

Associated laboratory findings from polysomnography indicate increased tonic and/or phasic electromyographic activity during REM sleep that is normally associated with muscle atonia. The increased muscle activity variably affects different muscle groups, mandating more extensive electromyographic monitoring than is employed in conventional sleep studies. For this reason, it is suggested that electromyographic monitoring include the submentalis, bilateral extensor digitorum, and bilateral anterior tibialis muscle groups. Continuous video monitoring is mandatory. Other polysomnographic findings may include very frequent periodic and aperiodic extremity electromyography activity during non-REM (NREM) sleep. This polysomnography observation, termed *REM sleep without atonia*, is present in virtually all cases of REM sleep behavior disorder but may also be an asymptomatic polysomnographic finding. Clinical dream-enacting behaviors coupled with the polysomnographic finding of REM without atonia is necessary for the diagnosis of REM sleep behavior disorder. REM sleep without atonia without a clinical history of dream-enacting behaviors is simply an asymptomatic polysomnographic observation. It is not known whether isolated REM sleep without atonia is a precursor to REM sleep behavior disorder.

Functional Consequences of Rapid Eye Movement Sleep Behavior Disorder

REM sleep behavior disorder may occur in isolated occasions in otherwise unaffected individuals. Embarrassment concerning the episodes can impair social relationships. Individuals may avoid situations in which others might become aware of the disturbance, visiting friends overnight, or sleeping with bed partners. Social isolation or occupational difficulties can result. Uncommonly, REM sleep behavior disorder may result in serious injury to the victim or to the bed partner.

Differential Diagnosis

Other parasomnias. Confusional arousals, sleepwalking, and sleep terrors can easily be confused with REM sleep behavior disorder. In general, these disorders occur in younger individuals. Unlike REM sleep behavior disorder, they arise from deep NREM sleep and therefore tend to occur in the early portion of the sleep period. Awakening from a confusional arousal is associated with confusion, disorientation, and incomplete recall of dream mentation accompanying the behavior. Polysomnographic monitoring in the disorders of arousal reveals normal REM atonia.

Nocturnal seizures. Nocturnal seizures may perfectly mimic REM sleep behavior disorder, but the behaviors are generally more stereotyped. Polysomnographic monitoring employing a full electroencephalographic seizure montage may differentiate the two. REM sleep without atonia is not present on polysomnographic monitoring.

Obstructive sleep apnea. Obstructive sleep apnea may result in behaviors indistinguishable from REM sleep behavior disorder. Polysomnographic monitoring is necessary to differentiate between the two. In this case, the symptoms resolve following effective treatment of the obstructive sleep apnea, and REM sleep without atonia is not present on polysomnography monitoring.

Other specified dissociative disorder (sleep-related psychogenic dissociative disorder). Unlike virtually all other parasomnias, which arise precipitously from NREM or REM sleep, psychogenic dissociative behaviors arise from a period of well-defined wakefulness during the sleep period. Unlike REM sleep behavior disorder, this condition is more prevalent in young females.

Malingering. Many cases of malingering in which the individual reports problematic sleep movements perfectly mimic the clinical features of REM sleep behavior disorder, and polysomnographic documentation is mandatory.

Comorbidity

REM sleep behavior disorder is present concurrently in approximately 30% of patients with narcolepsy. When it occurs in narcolepsy, the demographics reflect the younger age range of narcolepsy, with equal frequency in males and females. Based on findings from individuals presenting to sleep clinics, most individuals (>50%) with initially "idiopathic" REM sleep behavior disorder will eventually develop a neurodegenerative disease—most notably, one of the synucleinopathies (Parkinson's disease, multiple system atrophy, or major or mild neurocognitive disorder with Lewy bodies). REM sleep behavior disorder often predates any other sign of these disorders by many years (often more than a decade).

Relationship to International Classification of Sleep Disorders

REM sleep behavior disorder is virtually identical to REM sleep behavior disorder in the *International Classification of Sleep Disorders*, 2nd Edition (ICSD-2).

Restless Legs Syndrome

Diagnostic Criteria **333.94 (G25.81)**

A. An urge to move the legs, usually accompanied by or in response to uncomfortable and unpleasant sensations in the legs, characterized by all of the following:
   1. The urge to move the legs begins or worsens during periods of rest or inactivity.
   2. The urge to move the legs is partially or totally relieved by movement.
   3. The urge to move the legs is worse in the evening or at night than during the day, or occurs only in the evening or at night.

B. The symptoms in Criterion A occur at least three times per week and have persisted for at least 3 months.

C. The symptoms in Criterion A are accompanied by significant distress or impairment in social, occupational, educational, academic, behavioral, or other important areas of functioning.

D. The symptoms in Criterion A are not attributable to another mental disorder or medical condition (e.g., arthritis, leg edema, peripheral ischemia, leg cramps) and are not better explained by a behavioral condition (e.g., positional discomfort, habitual foot tapping).

E. The symptoms are not attributable to the physiological effects of a drug of abuse or medication (e.g., akathisia).

Diagnostic Features

Restless legs syndrome (RLS) is a sensorimotor, neurological sleep disorder characterized by a desire to move the legs or arms, usually associated with uncomfortable sensations typically described as creeping, crawling, tingling, burning, or itching (Criterion A). The diagnosis of RLS is based primarily on patient self-report and history. Symptoms are worse when the individual is at rest, and frequent movements of the legs occur in an effort to relieve the uncomfortable sensations. Symptoms are worse in the evening or night, and in some individuals they occur only in the evening or night. Evening worsening occurs independently of any differences in activity. It is important to differentiate RLS from other conditions such as positional discomfort and leg cramps (Criterion D).

The symptoms of RLS can delay sleep onset and awaken the individual from sleep and are associated with significant sleep fragmentation. The relief obtained from moving the legs may no longer be apparent in severe cases. RLS is associated with daytime sleepiness and is frequently accompanied by significant clinical distress or functional impairment.

Associated Features Supporting Diagnosis

Periodic leg movements in sleep (PLMS) can serve as corroborating evidence for RLS, with up to 90% of individuals diagnosed with RLS demonstrating PLMS when recordings are taken over multiple nights. Periodic leg movements during wakefulness are supportive of an RLS diagnosis. Reports of difficulty initiating and maintaining sleep and of excessive daytime sleepiness may also support the diagnosis of RLS. Additional supportive features include a family history of RLS among first-degree relatives and a reduction in symptoms, at least initially, with dopaminergic treatment.

Prevalence

Prevalence rates of RLS vary widely when broad criteria are utilized but range from 2% to 7.2% when more defined criteria are employed. When frequency of symptoms is at least three times per week with moderate or severe distress, the prevalence rate is 1.6%; when frequency of symptoms is a minimum of one time per week, the prevalence rate is 4.5%. Females are 1.5–2 times more likely than males to have RLS. RLS also increases with age. The prevalence of RLS may be lower in Asian populations.

Development and Course

The onset of RLS typically occurs in the second or third decade. Approximately 40% of individuals diagnosed with RLS during adulthood report having experienced symptoms before age 20 years, and 20% report having experienced symptoms before age 10 years. Prevalence rates of RLS increase steadily with age until about age 60 years, with symptoms remaining stable or decreasing slightly in older age groups. Compared with nonfamilial cases, familial RLS usually has a younger age at onset and a slower progressive course. The clinical course of RLS differs by age at onset. When onset occurs before age 45, there is often a slow progression of symptoms. In late-onset RLS, rapid progression is typical, and aggravating factors are common. Symptoms of RLS appear similar across the lifespan, remaining stable or decreasing slightly in older age groups.

Diagnosis of RLS in children can be difficult because of the self-report component. While Criterion A for adults assumes that the description of "urge to move" is by the patient, pediatric diagnosis requires a description in the child's own words rather than by a parent or caretaker. Typically children age 6 years or older are able to provide detailed, adequate descriptors of RLS. However, children rarely use or understand the word "urge," reporting instead that their legs "have to" or "got to" move. Also, potentially related to prolonged periods of sitting during class, two-thirds of children and adolescents report daytime leg sensations. Thus, for diagnostic Criterion A3, it is important to compare equal

duration of sitting or lying down in the day to sitting or lying down in the evening or night. Nocturnal worsening tends to persist even in the context of pediatric RLS. As with RLS in adults, there is a significant negative impact on sleep, mood, cognition, and function. Impairment in children and adolescents is manifested more often in behavioral and educational domains.

Risk and Prognostic Factors

Genetic and physiological. Predisposing factors include female gender, advancing age, genetic risk variants, and family history of RLS. Precipitating factors are often time-limited, such as iron deficiency, with most individuals resuming normal sleep patterns after the initial triggering event has disappeared. Genetic risk variants also play a role in RLS secondary to such disorders as uremia, suggesting that individuals with a genetic susceptibility develop RLS in the presence of further risk factors. RLS has a strong familial component.

There are defined pathophysiological pathways subserving RLS. Genome-wide association studies have found that RLS is significantly associated with common genetic variants in intronic or intergenic regions in *MEIS1*, *BTBD9*, and *MAP2K5* on chromosomes 2p, 6p, and 15q, respectively. The association of these three variants with RLS has been independently replicated. *BTBD9* confers a very large (80%) excessive risk when even a single allele is present. Because of the high frequency of this variant in individuals of European descent, the population attributable risk (PAR) approximates 50%. At-risk alleles associated with *MEIS1* and *BTBD9* are less common in individuals of African or Asian descent, perhaps suggesting lower risk for RLS in these populations.

Pathophysiological mechanisms in RLS also include disturbances in the central dopaminergic system and disturbances in iron metabolism. The endogenous opiate system may also be involved. Treatment effects of dopaminergic drugs (primarily D_2 and D_3 nonergot agonists) provide further support that RLS is grounded in dysfunctional central dopaminergic pathways. While the effective treatment of RLS has also been shown to significantly reduce depressive symptoms, serotonergic antidepressants can induce or aggravate RLS in some individuals.

Gender-Reiated Diagnostic Issues

Although RLS is more prevalent in females than in males, there are no diagnostic differences according to gender. However, the prevalence of RLS during pregnancy is two to three times greater than in the general population. RLS associated with pregnancy peaks during the third trimester and improves or resolves in most cases soon after delivery. The gender difference in prevalence of RLS is explained at least in part by parity, with nulliparous females being at the same risk of RLS as age-matched males.

Diagnostic Markers

Polysomnography demonstrates significant abnormalities in RLS, commonly increased latency to sleep, and higher arousal index. Polysomnography with a preceding immobilization test may provide an indicator of the motor sign of RLS, periodic limb movements, under standard conditions of sleep and during quiet resting, both of which can provoke RLS symptoms.

Functionai Consequences of Restless Legs Syndrome

Forms of RLS severe enough to significantly impair functioning or associated with mental disorders, including depression and anxiety, occur in approximately 2%–3% of the population.

Although the impact of milder symptoms is less well characterized, individuals with RLS complain of disruption in at least one activity of daily living, with up to 50% reporting

a negative impact on mood, and 47.6% reporting a lack of energy. The most common consequences of RLS are sleep disturbance, including reduced sleep time, sleep fragmentation, and overall disturbance; depression, generalized anxiety disorder, panic disorder, and posttraumatic stress disorder; and quality-of-life impairments. RLS can result in daytime sleepiness or fatigue and is frequently accompanied by significant distress or impairment in affective, social, occupational, educational, academic, behavioral, or cognitive functioning.

Differential Diagnosis

The most important conditions in the differential diagnosis of RLS are leg cramps, positional discomfort, arthralgias/arthritis, myalgias, positional ischemia (numbness), leg edema, peripheral neuropathy, radiculopathy, and habitual foot tapping. "Knotting" of the muscle (cramps), relief with a single postural shift, limitation to joints, soreness to palpation (myalgias), and other abnormalities on physical examination are not characteristic of RLS. Unlike RLS, nocturnal leg cramps do not typically present with the desire to move the limbs nor are there frequent limb movements. Less common conditions to be differentiated from RLS include neuroleptic-induced akathisia, myelopathy, symptomatic venous insufficiency, peripheral artery disease, eczema, other orthopedic problems, and anxiety-induced restlessness. Worsening at night and periodic limb movements are more common in RLS than in medication-induced akathisia or peripheral neuropathy.

While is it important that RLS symptoms not be solely accounted for by another medical or behavioral condition, it should also be appreciated that any of these similar conditions can occur in an individual with RLS. This necessitates a separate focus on each possible condition in the diagnostic process and when assessing impact. For cases in which the diagnosis of RLS is not certain, evaluation for the supportive features of RLS, particularly PLMS or a family history of RLS, may be helpful. Clinical features, such as response to a dopaminergic agent and positive family history for RLS, can help with the differential diagnosis.

Comorbidity

Depressive disorders, anxiety disorders, and attentional disorders are commonly comorbid with RLS and are discussed in the section "Functional Consequences of Restless Legs Syndrome." The main medical disorder comorbid with RLS is cardiovascular disease. There may be an association with numerous other medical disorders, including hypertension, narcolepsy, migraine, Parkinson's disease, multiple sclerosis, peripheral neuropathy, obstructive sleep apnea, diabetes mellitus, fibromyalgia, osteoporosis, obesity, thyroid disease, and cancer. Iron deficiency, pregnancy, and chronic renal failure are also comorbid with RLS.

Reiationship to Internationai Ciassification of Sieep Disorders

The *International Classification of Sleep Disorders*, 2nd Edition (ICSD-2), presents similar diagnostic criteria for RLS but does not contain a criterion specifying frequency or duration of symptoms.

Substance/Medication-Induced Sleep Disorder

Diagnostic Criteria

A. A prominent and severe disturbance in sleep.
B. There is evidence from the history, physical examination, or laboratory findings of both (1) and (2):

1. The symptoms in Criterion A developed during or soon after substance intoxication or after withdrawal from or exposure to a medication.
2. The involved substance/medication is capable of producing the symptoms in Criterion A.

C. The disturbance is not better explained by a sleep disorder that is not substance/medication-induced. Such evidence of an independent sleep disorder could include the following:

   The symptoms precede the onset of the substance/medication use; the symptoms persist for a substantial period of time (e.g., about 1 month) after the cessation of acute withdrawal or severe intoxication; or there is other evidence suggesting the existence of an independent non-substance/medication-induced sleep disorder (e.g., a history of recurrent non-substance/medication-related episodes).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Note: This diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and when they are sufficiently severe to warrant clinical attention.

Coding note: The ICD-9-CM and ICD-10-CM codes for the [specific substance/medication]-induced sleep disorders are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. If a mild substance use disorder is comorbid with the substance-induced sleep disorder, the 4th position character is "1," and the clinician should record "mild [substance] use disorder" before the substance-induced sleep disorder (e.g., "mild cocaine use disorder with cocaine-induced sleep disorder"). If a moderate or severe substance use disorder is comorbid with the substance-induced sleep disorder, the 4th position character is "2," and the clinician should record "moderate [substance] use disorder" or "severe [substance] use disorder," depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder (e.g., after a one-time heavy use of the substance), then the 4th position character is "9," and the clinician should record only the substance-induced sleep disorder. A moderate or severe tobacco use disorder is required in order to code a tobacco-induced sleep disorder; it is not permissible to code a comorbid mild tobacco use disorder or no tobacco use disorder with a tobacco-induced sleep disorder.

Specify whether:

Insomnia type: Characterized by difficulty falling asleep or maintaining sleep, frequent nocturnal awakenings, or nonrestorative sleep.

Daytime sleepiness type: Characterized by predominant complaint of excessive sleepiness/fatigue during waking hours or, less commonly, a long sleep period.

Parasomnia type: Characterized by abnormal behavioral events during sleep.

Mixed type: Characterized by a substance/medication-induced sleep problem characterized by multiple types of sleep symptoms, but no symptom clearly predominates.

Specify if (see Table 1 in the chapter "Substance-Related and Addictive Disorders" for diagnoses associated with substance class):

With onset during intoxication: This specifier should be used if criteria are met for intoxication with the substance/medication and symptoms developed during the intoxication period.

With onset during discontinuation/withdrawal: This specifier should be used if criteria are met for discontinuation/withdrawal from the substance/medication and symptoms developed during, or shortly after, discontinuation of the substance/medication.

		ICD-10-CM		
	ICD-9-CM	With use disorder, mild	With use disorder, moderate or severe	Without use disorder
Alcohol	291.82	F10.182	F10.282	F10.982
Caffeine	292.85	F15.182	F15.282	F15.982
Cannabis	292.85	F12.188	F12.288	F12.988
Opioid	292.85	F11.182	F11.282	F11.982
Sedative, hypnotic, or anxiolytic	292.85	F13.182	F13.282	F13.982
Amphetamine (or other stimulant)	292.85	F15.182	F15.282	F15.982
Cocaine	292.85	F14.182	F14.282	F14.982
Tobacco	292.85	NA	F17.208	NA
Other (or unknown) substance	292.85	F19.182	F19.282	F19.982

Recording Procedures

ICD-9-CM. The name of the substance/medication-induced sleep disorder begins with the specific substance (e.g., cocaine, bupropion) that is presumed to be causing the sleep disturbance. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class. For substances that do not fit into any of the classes (e.g., bupropion), the code for "other substance" should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category "unknown substance" should be used.

The name of the disorder is followed by the specification of onset (i.e., onset during intoxication, onset during discontinuation/withdrawal), followed by the subtype designation (i.e., insomnia type, daytime sleepiness type, parasomnia type, mixed type). Unlike the recording procedures for ICD-10-CM, which combine the substance-induced disorder and substance use disorder into a single code, for ICD-9-CM a separate diagnostic code is given for the substance use disorder. For example, in the case of insomnia occurring during withdrawal in a man with a severe lorazepam use disorder, the diagnosis is 292.85 lorazepam-induced sleep disorder, with onset during withdrawal, insomnia type. An additional diagnosis of 304.10 severe lorazepam use disorder is also given. When more than one substance is judged to play a significant role in the development of the sleep disturbance, each should be listed separately (e.g., 292.85 alcohol-induced sleep disorder, with onset during intoxication, insomnia type; 292.85 cocaine-induced sleep disorder, with onset during intoxication, insomnia type).

ICD-10-CM. The name of the substance/medication-induced sleep disorder begins with the specific substance (e.g., cocaine, bupropion) that is presumed to be causing the sleep disturbance. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class and presence or absence of a comorbid substance use disorder. For substances that do not fit into any of the classes (e.g., bupropion), the code for "other substance" should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category "unknown substance" should be used.

When recording the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by the word "with," followed by the name of the substance-induced sleep disorder, followed by the specification of onset (i.e., onset during intoxication, onset

during discontinuation/withdrawal), followed by the subtype designation (i.e., insomnia type, daytime sleepiness type, parasomnia type, mixed type). For example, in the case of insomnia occurring during withdrawal in a man with a severe lorazepam use disorder, the diagnosis is F13.282 severe lorazepam use disorder with lorazepam-induced sleep disorder, with onset during withdrawal, insomnia type. A separate diagnosis of the comorbid severe lorazepam use disorder is not given. If the substance-induced sleep disorder occurs without a comorbid substance use disorder (e.g., with medication use), no accompanying substance use disorder is noted (e.g., F19.982 bupropion-induced sleep disorder, with onset during medication use, insomnia type). When more than one substance is judged to play a significant role in the development of the sleep disturbance, each should be listed separately (e.g., F10.282 severe alcohol use disorder with alcohol-induced sleep disorder, with onset during intoxication, insomnia type; F14.282 severe cocaine use disorder with cocaine-induced sleep disorder, with onset during intoxication, insomnia type).

Diagnostic Features

The essential feature of substance/medication-induced sleep disorder is a prominent sleep disturbance that is sufficiently severe to warrant independent clinical attention (Criterion A) and that is judged to be primarily associated with the pharmacological effects of a substance (i.e., a drug of abuse, a medication, toxin exposure) (Criterion B). Depending on the substance involved, one of four types of sleep disturbances is reported. Insomnia type and daytime sleepiness type are most common, while parasomnia type is seen less often. The mixed type is noted when more than one type of sleep disturbance–related symptom is present and none predominates. The disturbance must not be better explained by another sleep disorder (Criterion C). A substance/medication-induced sleep disorder is distinguished from insomnia disorder or a disorder associated with excessive daytime sleepiness by considering onset and course. For drugs of abuse, there must be evidence of intoxication or withdrawal from the history, physical examination, or laboratory findings. Substance/medication-induced sleep disorder arises only in association with intoxication or discontinuation/withdrawal states, whereas other sleep disorders may precede the onset of substance use or occur during times of sustained abstinence. As discontinuation/withdrawal states for some substances can be protracted, onset of the sleep disturbance can occur 4 weeks after cessation of substance use, and the disturbance may have features atypical of other sleep disorders (e.g., atypical age at onset or course). The diagnosis is not made if the sleep disturbance occurs only during a delirium (Criterion D). The symptoms must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion E). This diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and when the symptoms warrant independent clinical attention.

Associated Features Supporting Diagnosis

During periods of substance/medication use, intoxication, or withdrawal, individuals frequently complain of dysphoric mood, including depression and anxiety, irritability, cognitive impairment, inability to concentrate, and fatigue.

Prominent and severe sleep disturbances can occur in association with intoxication with the following classes of substances: alcohol; caffeine; cannabis; opioids; sedatives, hypnotics, or anxiolytics; stimulants (including cocaine); and other (or unknown) substances. Prominent and severe sleep disturbances can occur in association with withdrawal from the following classes of substances: alcohol; caffeine; cannabis; opioids; sedatives, hypnotics, or anxiolytics; stimulant (including cocaine); tobacco; and other (or unknown) substances. Some medications that invoke sleep disturbances include adrenergic agonists and antagonists, dopamine agonists and antagonists, cholinergic agonists and antagonists, serotonergic agonists and antagonists, antihistamines, and corticosteroids.

Alcohol. Alcohol-induced sleep disorder typically occurs as insomnia type. During acute intoxication, alcohol produces an immediate sedative effect depending on dose, accompanied by increased stages 3 and 4 non–rapid eye movement (NREM) sleep and reduced rapid eye movement (REM) sleep. Following these initial effects, there may be increased wakefulness, restless sleep, and vivid and anxiety-laden dreams for the remaining sleep period. In parallel, stages 3 and 4 sleep are reduced, and wakefulness and REM sleep are increased. Alcohol can aggravate breathing-related sleep disorder. With habitual use, alcohol continues to show a short-lived sedative effect in the first half of the night, followed by sleep continuity disruption in the second half. During alcohol withdrawal, there is extremely disrupted sleep continuity, and an increased amount and intensity of REM sleep, associated frequently with vivid dreaming, which in extreme form, constitutes part of alcohol withdrawal delirium. After acute withdrawal, chronic alcohol users may continue to complain of light, fragmented sleep for weeks to years associated with a persistent deficit in slow-wave sleep.

Caffeine. Caffeine-induced sleep disorder produces insomnia in a dose-dependent manner, with some individuals presenting with daytime sleepiness related to withdrawal.

Cannabis. Acute administration of cannabis may shorten sleep latency, though arousing effects with increments in sleep latency also occur. Cannabis enhances slow-wave sleep and suppresses REM sleep after acute administration. In chronic users, tolerance to the sleep-inducing and slow-wave sleep–enhancing effects develops. Upon withdrawal, sleep difficulties and unpleasant dreams have been reported lasting for several weeks. Polysomnography studies demonstrate reduced slow-wave sleep and increased REM sleep during this phase.

Opioids. Opioids may produce an increase in sleepiness and in subjective depth of sleep, and reduced REM sleep, during acute short-term use. With continued administration, tolerance to the sedative effects of opioids develops and there are complaints of insomnia. Consistent with their respiratory depressant effects, opioids exacerbate sleep apnea.

Sedative, hypnotic, or anxiolytic substances. Sedatives, hypnotics, and anxiolytics (e.g., barbiturates, benzodiazepines receptor agonists, meprobamate, glutethimide, methyprylon) have similar effects as opioids on sleep. During acute intoxication, sedative-hypnotic drugs produce the expected increase in sleepiness and decrease in wakefulness. Chronic use (particularly of barbiturates and the older nonbarbiturate, nonbenzodiazepine drugs) may cause tolerance with subsequent return of insomnia. Daytime sleepiness may occur. Sedative-hypnotic drugs can increase the frequency and severity of obstructive sleep apnea events. Parasomnias are associated with use of benzodiazepine receptor agonists, especially when these medications are taken at higher doses and when they are combined with other sedative drugs. Abrupt discontinuation of chronic sedative, hypnotic, or anxiolytic use can lead to withdrawal but more commonly rebound insomnia, a condition of an exacerbation of insomnia upon drug discontinuation for 1–2 days reported to occur even with short-term use. Sedative, hypnotic, or anxiolytic drugs with short durations of action are most likely to produce complaints of rebound insomnia, whereas those with longer durations of action are more often associated with daytime sleepiness. Any sedative, hypnotic, or anxiolytic drug can potentially cause daytime sedation, withdrawal, or rebound insomnia.

Amphetamines and related substances and other stimulants. Sleep disorders induced by amphetamine and related substances and other stimulants are characterized by insomnia during intoxication and excessive sleepiness during withdrawal. During acute intoxication, stimulants reduce the total amount of sleep, increase sleep latency and sleep continuity disturbances, and decrease REM sleep. Slow-wave sleep tends to be reduced. During withdrawal from chronic stimulant use, there is both prolonged nocturnal sleep duration and excessive daytime sleepiness. Multiple sleep latency tests may show increased daytime sleepiness dur-

ing the withdrawal phase. Drugs like 3,4-methylenedioxymethamphetamine (MDMA; "ecstasy") and related substances lead to restless and disturbed sleep within 48 hours of intake; frequent use of these compounds is associated with persisting symptoms of anxiety, depression, and sleep disturbances, even during longer-term abstinence.

Tobacco. Chronic tobacco consumption is associated primarily with symptoms of insomnia, decreased slow-wave sleep with a reduction of sleep efficiency, and increased daytime sleepiness. Withdrawal from tobacco can lead to impaired sleep. Individuals who smoke heavily may experience regular nocturnal awakenings caused by tobacco craving.

Other or unknown substances/medications. Other substances/medications may produce sleep disturbances, particularly medications that affect the central or autonomic nervous systems (e.g., adrenergic agonists and antagonists, dopamine agonists and antagonists, cholinergic agonists and antagonists, serotonergic agonists and antagonists, antihistamines, corticosteroids).

Development and Course

Insomnia in children can be identified by either a parent or the child. Often the child has a clear sleep disturbance associated with initiation of a medication but may not report symptoms, although parents observe the sleep disturbances. The use of some illicit substances (e.g., cannabis, ecstasy) is prevalent in adolescence and early adulthood. Insomnia or any other sleep disturbance encountered in this age group should prompt careful consideration of whether the sleep disturbance is due to consumption of these substances. Help-seeking behavior for the sleep disturbance in these age groups is limited, and thus corroborative report may be elicited from a parent, caregiver, or teacher. Older individuals take more medications and are at increased risk for developing a substance/medication-induced sleep disorder. They may interpret sleep disturbance as part of normal aging and fail to report symptoms. Individuals with major neurocognitive disorder (e.g., dementia) are at risk for substance/medication-induced sleep disorders but may not report symptoms, making corroborative report from caregiver(s) particularly important.

Risk and Prognostic Factors

Risk and prognostic factors involved in substance abuse/dependence or medication use are normative for certain age groups. They are relevant for, and likely applicable to, the type of sleep disturbance encountered (see the chapter "Substance-Related and Addictive Disorders" for descriptions of respective substance use disorders).

Temperamental. Substance use generally precipitates or accompanies insomnia in vulnerable individuals. Thus, presence of insomnia in response to stress or change in sleep environment or timing can represent a risk for developing substance/medication-induced sleep disorder. A similar risk may be present for individuals with other sleep disorders (e.g., individuals with hypersomnia who use stimulants).

Culture-Related Diagnostic Issues

The consumption of substances, including prescribed medications, may depend in part on cultural background and specific local drug regulations.

Gender-Related Diagnostic Issues

Gender-specific prevalences (i.e., females affected more than males at a ratio of about 2:1) exist for patterns of consumption of some substances (e.g., alcohol). The same amount and duration of consumption of a given substance may lead to highly different sleep-related outcomes in males and females based on, for example, gender-specific differences in hepatic functioning.

Diagnostic Markers

Each of the substance/medication-induced sleep disorders produces electroencephalographic sleep patterns that are associated with, but cannot be considered diagnostic of, other disorders. The electroencephalographic sleep profile for each substance is related to the stage of use, whether intake/intoxication, chronic use, or withdrawal following discontinuation of the substance. All-night polysomnography can help define the severity of insomnia complaints, while the multiple sleep latency test provides information about the severity of daytime sleepiness. Monitoring of nocturnal respiration and periodic limb movements with polysomnography may verify a substance's impact on nocturnal breathing and motor behavior. Sleep diaries for 2 weeks and actigraphy are considered helpful in confirming the presence of substance/medication-induced sleep disorder. Drug screening can be of use when the individual is not aware or unwilling to relate information about substance intake.

Functional Consequences of Substance/Medication-Induced Sleep Disorder

While there are many functional consequences associated with sleep disorders, the only unique consequence for substance/medication-induced sleep disorder is increased risk for relapse. The degree of sleep disturbance during alcohol withdrawal (e.g., REM sleep rebound predicts risk of relapse of drinking). Monitoring of sleep quality and daytime sleepiness during and after withdrawal may provide clinically meaningful information on whether an individual is at increased risk for relapse.

Differential Diagnosis

Substance intoxication or substance withdrawal. Sleep disturbances are commonly encountered in the context of substance intoxication or substance discontinuation/withdrawal. A diagnosis of substance/medication-induced sleep disorder should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the sleep disturbance is predominant in the clinical picture and is sufficiently severe to warrant independent clinical attention.

Delirium. If the substance/medication-induced sleep disturbance occurs exclusively during the course of a delirium, it is not diagnosed separately.

Other sleep disorders. A substance/medication-induced sleep disorder is distinguished from another sleep disorder if a substance/medication is judged to be etiologically related to the symptoms. A substance/medication-induced sleep disorder attributed to a prescribed medication for a mental disorder or medical condition must have its onset while the individual is receiving the medication or during discontinuation, if there is a discontinuation/withdrawal syndrome associated with the medication. Once treatment is discontinued, the sleep disturbance will usually remit within days to several weeks. If symptoms persist beyond 4 weeks, other causes for the sleep disturbance–related symptoms should be considered. Not infrequently, individuals with another sleep disorder use medications or drugs of abuse to self-medicate their symptoms (e.g., alcohol for management of insomnia). If the substance/medication is judged to play a significant role in the exacerbation of the sleep disturbance, an additional diagnosis of a substance/medication-induced sleep disorder may be warranted.

Sleep disorder due to another medical condition. Substance/medication-induced sleep disorder and sleep disorder associated with another medical condition may produce similar symptoms of insomnia, daytime sleepiness, or a parasomnia. Many individuals with other medical conditions that cause sleep disturbance are treated with medications that may also cause sleep disturbances. The chronology of symptoms is the most important factor in distinguishing between these two sources of sleep symptoms. Difficulties with sleep that clearly preceded the use of any medication for treatment of a medical condition would

suggest a diagnosis of sleep disorder associated with another medical condition. Conversely, sleep symptoms that appear only after the initiation of a particular medication/substance suggest a substance/medication-induced sleep disorder. If the disturbance is comorbid with another medical condition and is also exacerbated by substance use, both diagnoses (i.e., sleep disorder associated with another medical condition and substance/medication-induced sleep disorder) are given. When there is insufficient evidence to determine whether the sleep disturbance is attributable to a substance/medication or to another medical condition or is primary (i.e., not due to either a substance/medication or another medical condition), a diagnosis of other specified sleep-wake disorder or unspecified sleep-wake disorder is indicated.

Comorbidity

See the "Comorbidity" sections for other sleep disorders in this chapter, including insomnia, hypersomnolence, central sleep apnea, sleep-related hypoventilation, and circadian rhythm sleep-wake disorders, shift work type.

Relationship to Internationai Ciassification of Sieep Disorders

The *International Classification of Sleep Disorders*, 2nd Edition (ICSD-2), lists sleep disorders "due to drug or substance" under their respective phenotypes (e.g., insomnia, hypersomnia).

Other Specified Insomnia Disorder

780.52 (G47.09)

This category applies to presentations in which symptoms characteristic of insomnia disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for insomnia disorder or any of the disorders in the sleep-wake disorders diagnostic class. The other specified insomnia disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for insomnia disorder or any specific sleep-wake disorder. This is done by recording "other specified insomnia disorder" followed by the specific reason (e.g., "brief insomnia disorder").

Examples of presentations that can be specified using the "other specified" designation include the following:

1. **Brief insomnia disorder:** Duration is less than 3 months.
2. **Restricted to nonrestorative sleep:** Predominant complaint is nonrestorative sleep unaccompanied by other sleep symptoms such as difficulty falling asleep or remaining asleep.

Unspecified Insomnia Disorder

780.52 (G47.00)

This category applies to presentations in which symptoms characteristic of insomnia disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for insomnia disorder or any of the disorders in the sleep-wake disorders diagnostic class. The unspecified

insomnia disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for insomnia disorder or a specific sleep-wake disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis.

Other Specified Hypersomnolence Disorder

780.54 (G47.19)

This category applies to presentations in which symptoms characteristic of hypersomnolence disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for hypersomnolence disorder or any of the disorders in the sleep-wake disorders diagnostic class. The other specified hypersomnolence disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for hypersomnolence disorder or any specific sleep-wake disorder. This is done by recording "other specified hypersomnolence disorder" followed by the specific reason (e.g., "brief-duration hypersomnolence," as in Kleine-Levin syndrome).

Unspecified Hypersomnolence Disorder

780.54 (G47.10)

This category applies to presentations in which symptoms characteristic of hypersomnolence disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for hypersomnolence disorder or any of the disorders in the sleep-wake disorders diagnostic class. The unspecified hypersomnolence disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for hypersomnolence disorder or a specific sleep-wake disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis.

Other Specified Sleep-Wake Disorder

780.59 (G47.8)

This category applies to presentations in which symptoms characteristic of a sleep-wake disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the sleep-wake disorders diagnostic class and do not qualify for a diagnosis of other specified insomnia disorder or other specified hypersomnolence disorder. The other specified sleep-wake disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific sleep-wake disorder. This is done by recording "other specified sleep-wake disorder" followed by the specific reason (e.g., "repeated arousals during rapid eye movement sleep without polysomnography or history of Parkinson's disease or other synucleinopathy").

Unspecified Sleep-Wake Disorder

780.59 (G47.9)

This category applies to presentations in which symptoms characteristic of a sleep-wake disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the sleep-wake disorders diagnostic class and do not qualify for a diagnosis of unspecified insomnia disorder or unspecified hypersomnolence disorder. The unspecified sleep-wake disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific sleep-wake disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis.

Sexual Dysfunctions

Sexual dysfunctions include delayed ejaculation, erectile disorder, female orgasmic disorder, female sexual interest/arousal disorder, genito-pelvic pain/penetration disorder, male hypoactive sexual desire disorder, premature (early) ejaculation, substance/medication-induced sexual dysfunction, other specified sexual dysfunction, and unspecified sexual dysfunction. Sexual dysfunctions are a heterogeneous group of disorders that are typically characterized by a clinically significant disturbance in a person's ability to respond sexually or to experience sexual pleasure. An individual may have several sexual dysfunctions at the same time. In such cases, all of the dysfunctions should be diagnosed.

Clinical judgment should be used to determine if the sexual difficulties are the result of inadequate sexual stimulation; in these cases, there may still be a need for care, but a diagnosis of a sexual dysfunction would not be made. These cases may include, but are not limited to, conditions in which lack of knowledge about effective stimulation prevents the experience of arousal or orgasm.

Subtypes are used to designate the onset of the difficulty. In many individuals with sexual dysfunctions, the time of onset may indicate different etiologies and interventions. *Lifelong* refers to a sexual problem that has been present from first sexual experiences, and *acquired* applies to sexual disorders that develop after a period of relatively normal sexual function. *Generalized* refers to sexual difficulties that are not limited to certain types of stimulation, situations, or partners, and *situational* refers to sexual difficulties that only occur with certain types of stimulation, situations, or partners.

In addition to the lifelong/acquired and generalized/situational subtypes, a number of factors must be considered during the assessment of sexual dysfunction, given that they may be relevant to etiology and/or treatment, and that may contribute, to varying degrees, across individuals: 1) partner factors (e.g., partner's sexual problems; partner's health status); 2) relationship factors (e.g., poor communication; discrepancies in desire for sexual activity); 3) individual vulnerability factors (e.g., poor body image; history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), or stressors (e.g., job loss, bereavement); 4) cultural or religious factors (e.g., inhibitions related to prohibitions against sexual activity or pleasure; attitudes toward sexuality); and 5) medical factors relevant to prognosis, course, or treatment.

Clinical judgment about the diagnosis of sexual dysfunction should take into consideration cultural factors that may influence expectations or engender prohibitions about the experience of sexual pleasure. Aging may be associated with a normative decrease in sexual response.

Sexual response has a requisite biological underpinning, yet is usually experienced in an intrapersonal, interpersonal, and cultural context. Thus, sexual function involves a complex interaction among biological, sociocultural, and psychological factors. In many clinical contexts, a precise understanding of the etiology of a sexual problem is unknown. Nonetheless, a sexual dysfunction diagnosis requires ruling out problems that are better explained by a nonsexual mental disorder, by the effects of a substance (e.g., drug or medication), by a medical condition (e.g., due to pelvic nerve damage), or by severe relationship distress, partner violence, or other stressors.

If the sexual dysfunction is mostly explainable by another nonsexual mental disorder (e.g., depressive or bipolar disorder, anxiety disorder, posttraumatic stress disorder, psychotic dis-

order), then only the other mental disorder diagnosis should be made. If the problem is thought to be better explained by the use/misuse or discontinuation of a drug or substance, it should be diagnosed accordingly as a substance/medication-induced sexual dysfunction. If the sexual dysfunction is attributable to another medical condition (e.g., peripheral neuropathy), the individual would not receive a psychiatric diagnosis. If severe relationship distress, partner violence, or significant stressors better explain the sexual difficulties, then a sexual dysfunction diagnosis is not made, but an appropriate V or Z code for the relationship problem or stressor may be listed. In many cases, a precise etiological relationship between another condition (e.g., a medical condition) and a sexual dysfunction cannot be established.

Delayed Ejaculation

Diagnostic Criteria **302.74 (F52.32)**

A. Either of the following symptoms must be experienced on almost all or all occasions (approximately 75%–100%) of partnered sexual activity (in identified situational contexts or, if generalized, in all contexts), and without the individual desiring delay:
   1. Marked delay in ejaculation.
   2. Marked infrequency or absence of ejaculation.

B. The symptoms in Criterion A have persisted for a minimum duration of approximately 6 months.

C. The symptoms in Criterion A cause clinically significant distress in the individual.

D. The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of severe relationship distress or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition.

Specify whether:

Lifelong: The disturbance has been present since the individual became sexually active.

Acquired: The disturbance began after a period of relatively normal sexual function.

Specify whether:

Generalized: Not limited to certain types of stimulation, situations, or partners.

Situational: Only occurs with certain types of stimulation, situations, or partners.

Specify current severity:

Mild: Evidence of mild distress over the symptoms in Criterion A.

Moderate: Evidence of moderate distress over the symptoms in Criterion A.

Severe: Evidence of severe or extreme distress over the symptoms in Criterion A.

Diagnostic Features

The distinguishing feature of delayed ejaculation is a marked delay in or inability to achieve ejaculation (Criterion A). The man reports difficulty or inability to ejaculate despite the presence of adequate sexual stimulation and the desire to ejaculate. The presenting complaint usually involves partnered sexual activity. In most cases, the diagnosis will be made by self-report of the individual. The definition of "delay" does not have precise boundaries, as there is noconsensus as to what constitutes a reasonable time to reach orgasm or what is unacceptably long for most men and their sexual partners.

Associated Features Supporting Diagnosis

The man and his partner may report prolonged thrusting to achieve orgasm to the point of exhaustion or genital discomfort and then ceasing efforts. Some men may report avoiding

sexual activity because of a repetitive pattern of difficulty ejaculating. Some sexual partners may report feeling less sexually attractive because their partner cannot ejaculate easily.

In addition to the subtypes "lifelong/acquired" and "generalized/situational," the following five factors must be considered during assessment and diagnosis of delayed ejaculation, given that they may be relevant to etiology and/or treatment: 1) partner factors (e.g., partner's sexual problems, partner's health status); 2) relationship factors (e.g., poor communication, discrepancies in desire for sexual activity); 3) individual vulnerability factors (e.g., poor body image; history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), or stressors (e.g., job loss, bereavement); 4) cultural/religious factors (e.g., inhibitions related to prohibitions against sexual activity; attitudes toward sexuality); and 5) medical factors relevant to prognosis, course, or treatment. Each of these factors may contribute differently to the presenting symptoms of different men with this disorder.

Prevalence

Prevalence is unclear because of the lack of a precise definition of this syndrome. It is the least common male sexual complaint. Only 75% of men report always ejaculating during sexual activity, and less than 1% of men will complain of problems with reaching ejaculation that last more than 6 months.

Development and Course

Lifelong delayed ejaculation begins with early sexual experiences and continues throughout life. By definition, acquired delayed ejaculation begins after a period of normal sexual function. There is minimal evidence concerning the course of acquired delayed ejaculation. The prevalence of delayed ejaculation appears to remain relatively constant until around age 50 years, when the incidence begins to increase significantly. Men in their 80s report twice as much difficulty ejaculating as men younger than 59 years.

Risk and Prognostic Factors

Genetic and physiological. Age-related loss of the fast-conducting peripheral sensory nerves and age-related decreased sex steroid secretion may be associated with the increase in delayed ejaculation in men older than 50 years.

Culture-Related Diagnostic Issues

Complaints of ejaculatory delay vary across countries and cultures. Such complaints are more common among men in Asian populations than in men living in Europe, Australia, or the United States. This variation may be attributable to cultural or genetic differences between cultures.

Functional Consequences of Delayed Ejaculation

Difficulty with ejaculation may contribute to difficulties in conception. Delayed ejaculation is often associated with considerable psychological distress in one or both partners.

Differential Diagnosis

Another medical condition. The major differential diagnosis is between delayed ejaculation fully explained by another medical illness or injury and delayed ejaculation with a psychogenic, idiopathic, or combined psychological and medical etiology. A situational aspect to the complaint is suggestive of a psychological basis for the problem (e.g., men who can ejaculate during sexual activity with one sex but not the other; men who can ejaculate with one partner but not another of the same sex; men with paraphilic arousal pat-

terns; men who require highly ritualized activity to ejaculate during partnered sexual activity). Another medical illness or injury may produce delays in ejaculation independent of psychological issues. For example, inability to ejaculate can be caused by interruption of the nerve supply to the genitals, such as can occur after traumatic surgical injury to the lumbar sympathetic ganglia, abdominoperitoneal surgery, or lumbar sympathectomy. Ejaculation is thought to be under autonomic nervous system control involving the hypogastric (sympathetic) and pudendal (parasympathetic) nerves. A number of neurodegenerative diseases, such as multiple sclerosis and diabetic and alcoholic neuropathy, can cause inability to ejaculate. Delayed ejaculation should also be differentiated from retrograde ejaculation (i.e., ejaculation into the bladder), which may follow transurethral prostatic resection.

Substance/medication use. A number of pharmacological agents, such as antidepressants, antipsychotics, alpha sympathetic drugs, and opioid drugs, can cause ejaculatory problems.

Dysfunction with orgasm. It is important in the history to ascertain whether the complaint concerns delayed ejaculation or the sensation of orgasm, or both. Ejaculation occurs in the genitals, whereas the experience of orgasm is believed to be primarily subjective. Ejaculation and orgasm usually occur together but not always. For example, a man with a normal ejaculatory pattern may complain of decreased pleasure (i.e., anhedonic ejaculation). Such a complaint would not be coded as delayed ejaculation but could be coded as other specified sexual dysfunction or unspecified sexual dysfunction.

Comorbidity

There is some evidence to suggest that delayed ejaculation may be more common in severe forms of major depressive disorder.

Erectile Disorder

Diagnostic Criteria **302.72 (F52.21)**

A. At least one of the three following symptoms must be experienced on almost all or all (approximately 75%–100%) occasions of sexual activity (in identified situational contexts or, if generalized, in all contexts):

   1. Marked difficulty in obtaining an erection during sexual activity.
   2. Marked difficulty in maintaining an erection until the completion of sexual activity.
   3. Marked decrease in erectile rigidity.

B. The symptoms in Criterion A have persisted for a minimum duration of approximately 6 months.

C. The symptoms in Criterion A cause clinically significant distress in the individual.

D. The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of severe relationship distress or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition.

Specify whether:

Lifelong: The disturbance has been present since the individual became sexually active.

Acquired: The disturbance began after a period of relatively normal sexual function.

Specify whether:

Generalized: Not limited to certain types of stimulation, situations, or partners.

Situational: Only occurs with certain types of stimulation, situations, or partners.

Specify current severity:

Mild: Evidence of mild distress over the symptoms in Criterion A.

Moderate: Evidence of moderate distress over the symptoms in Criterion A.

Severe: Evidence of severe or extreme distress over the symptoms in Criterion A.

Diagnostic Features

The essential feature of erectile disorder is the repeated failure to obtain or maintain erections during partnered sexual activities (Criterion A). A careful sexual history is necessary to ascertain that the problem has been present for a significant duration of time (i.e., at least approximately 6 months) and occurs on the majority of sexual occasions (i.e., at least 75% of the time). Symptoms may occur only in specific situations involving certain types of stimulation or partners, or they may occur in a generalized manner in all types of situations, stimulation, or partners.

Associated Features Supporting Diagnosis

Many men with erectile disorder may have low self-esteem, low self-confidence, and a decreased sense of masculinity, and may experience depressed affect. Fear and/or avoidance of future sexual encounters may occur. Decreased sexual satisfaction and reduced sexual desire in the individual's partner are common.

In addition to the subtypes "lifelong/acquired" and "generalized/situational," the following five factors must be considered during assessment and diagnosis of erectile disorder given that they may be relevant to etiology and/or treatment: 1) partner factors (e.g., partner's sexual problems, partner's health status); 2) relationship factors (e.g., poor communication, discrepancies in desire for sexual activity); 3) individual vulnerability factors (e.g., poor body image, history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), or stressors (e.g., job loss, bereavement); 4) cultural/religious factors (e.g., inhibitions related to prohibitions against sexual activity; attitudes toward sexuality); and 5) medical factors relevant to prognosis, course, or treatment. Each of these factors may contribute differently to the presenting symptoms of different men with this disorder.

Prevalence

The prevalence of lifelong versus acquired erectile disorder is unknown. There is a strong age-related increase in both prevalence and incidence of problems with erection, particularly after age 50 years. Approximately 13%–21% of men ages 40–80 years complain of occasional problems with erections. Approximately 2% of men younger than age 40–50 years complain of frequent problems with erections, whereas 40%–50% of men older than 60–70 years may have significant problems with erections. About 20% of men fear erectile problems on their first sexual experience, whereas approximately 8% experienced erectile problems that hindered penetration during their first sexual experience.

Development and Course

Erectile failure on first sexual attempt has been found to be related to having sex with a previously unknown partner, concomitant use of drugs or alcohol, not wanting to have sex, and peer pressure. There is minimal evi-2t most of these problems spontaneously remit without professional intervention, but some men may continue to have episodic problems. In contrast, acquired erectile disorder is often associated with biological factors such as diabetes and cardiovascular disease. Acquired erectile disorder is likely to be persistent in most men.

The natural history of lifelong erectile disorder is unknown. Clinical observation supports the association of lifelong erectile disorder with psychological factors that are self-

limiting or responsive to psychological interventions, whereas, as noted above, acquired erectile disorder is more likely to be related to biological factors and to be persistent. The incidence of erectile disorder increases with age. A minority of men diagnosed as having moderate erectile failure may experience spontaneous remission of symptoms without medical intervention. Distress associated with erectile disorder is lower in older men as compared with younger men.

Risk and Prognostic Factors

Temperamental. Neurotic personality traits may be associated with erectile problems in college students, and submissive personality traits may be associated with erectile problems in men age 40 years and older. *Alexithymia* (i.e., deficits in cognitive processing of emotions) is common in men diagnosed with "psychogenic" erectile dysfunction. Erectile problems are common in men diagnosed with depression and posttraumatic stress disorder.

Course modifiers. Risk factors for acquired erectile disorder include age, smoking tobacco, lack of physical exercise, diabetes, and decreased desire.

Culture-Reiated Diagnostic Issues

Complaints of erectile disorder have been found to vary across countries. It is unclear to what extent these differences represent differences in cultural expectations as opposed to genuine differences in the frequency of erectile failure.

Diagnostic Markers

Nocturnal penile tumescence testing and measured erectile turgidity during sleep can be employed to help differentiate organic from psychogenic erectile problems on the assumption that adequate erections during rapid eye movement sleep indicate a psychological etiology to the problem. A number of other diagnostic procedures may be employed depending on the clinician's assessment of their relevance given the individual's age, comorbid medical problems, and clinical presentation. Doppler ultrasonography and intravascular injection of vasoactive drugs, as well as invasive diagnostic procedures such as dynamic infusion cavernosography, can be used to assess vascular integrity. Pudendal nerve conduction studies, including somatosensory evoked potentials, can be employed when a peripheral neuropathy is suspected. In men also complaining of decreased sexual desire, serum bioavailable or free testosterone is frequently assessed to determine if the difficulty is secondary to endocrinological factors. Thyroid function may also be assessed. Determination of fasting serum glucose is useful to screen for the presence of diabetes mellitus. The assessment of serum lipids is important, as erectile disorder in men 40 years and older is predictive of the future risk of coronary artery disease.

Functionai Consequences of Erectiie Disorder

Erectile disorder can interfere with fertility and produce both individual and interpersonal distress. Fear and/or avoidance of sexual encounters may interfere with the ability to develop intimate relationships.

Differentiai Diagnosis

Nonsexual mental disorders. Major depressive disorder and erectile disorder are closely associated, and erectile disorder accompanying severe depressive disorder may occur.

Normal erectile function. The differential should include consideration of normal erectile function in men with excessive expectations.

Substance/medication use. Another major differential diagnosis is whether the erectile problem is secondary to substance/medication use. An onset that coincides with the beginning of substance/medication use and that dissipates with discontinuation of the substance/medication or dose reduction is suggestive of a substance/medication-induced sexual dysfunction.

Another medical condition. The most difficult aspect of the differential diagnosis of erectile disorder is ruling out erectile problems that are fully explained by medical factors. Such cases would not receive a diagnosis of a mental disorder. The distinction between erectile disorder as a mental disorder and erectile dysfunction as the result of another medical condition is usually unclear, and many cases will have complex, interactive biological and psychiatric etiologies. If the individual is older than 40–50 years and/or has concomitant medical problems, the differential diagnosis should include medical etiologies, especially vascular disease. The presence of an organic disease known to cause erectile problems does not confirm a causal relationship. For example, a man with diabetes mellitus can develop erectile disorder in response to psychological stress. In general, erectile dysfunction due to organic factors is generalized and gradual in onset. An exception would be erectile problems after traumatic injury to the nervous innervation of the genital organs (e.g., spinal cord injury). Erectile problems that are situational and inconsistent and that have an acute onset after a stressful life event are most often due to psychological events. An age of less than 40 years is also suggestive of a psychological etiology to the difficulty.

Other sexual dysfunctions. Erectile disorder may coexist with premature (early) ejaculation and male hypoactive sexual desire disorder.

Comorbidity

Erectile disorder can be comorbid with other sexual diagnoses, such as premature (early) ejaculation and male hypoactive sexual desire disorder, as well as with anxiety and depressive disorders. Erectile disorder is common in men with lower urinary tract symptoms related to prostatic hypertrophy. Erectile disorder may be comorbid with dyslipidemia, cardiovascular disease, hypogonadism, multiple sclerosis, diabetes mellitus, and other diseases that interfere with the vascular, neurological, or endocrine function necessary for normal erectile function.

Relationship to International Classification of Diseases

Erectile response is coded as failure of genital response in ICD-10 (F2.2).

Female Orgasmic Disorder

Diagnostic Criteria **302.73 (F52.31)**

A. Presence of either of the following symptoms and experienced on almost all or all (approximately 75%–100%) occasions of sexual activity (in identified situational contexts or, if generalized, in all contexts):

   1. Marked delay in, marked infrequency of, or absence of orgasm.
   2. Markedly reduced intensity of orgasmic sensations.

B. The symptoms in Criterion A have persisted for a minimum duration of approximately 6 months.

C. The symptoms in Criterion A cause clinically significant distress in the individual.

D. The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of severe relationship distress (e.g., partner violence) or other significant

stressors and is not attributable to the effects of a substance/medication or another medical condition.

Specify whether:

Lifelong: The disturbance has been present since the individual became sexually active.

Acquired: The disturbance began after a period of relatively normal sexual function.

Specify whether:

Generalized: Not limited to certain types of stimulation, situations, or partners.

Situational: Only occurs with certain types of stimulation, situations, or partners.

Specify if:

Never experienced an orgasm under any situation.

Specify current severity:

Mild: Evidence of mild distress over the symptoms in Criterion A.

Moderate: Evidence of moderate distress over the symptoms in Criterion A.

Severe: Evidence of severe or extreme distress over the symptoms in Criterion A.

Diagnostic Features

Female orgasmic disorder is characterized by difficulty experiencing orgasm and/or markedly reduced intensity of orgasmic sensations (Criterion A). Women show wide variability in the type or intensity of stimulation that elicits orgasm. Similarly, subjective descriptions of orgasm are extremely varied, suggesting that it is experienced in very different ways, both across women and on different occasions by the same woman. For a diagnosis of female orgasmic disorder, symptoms must be experienced on almost all or all (approximately 75%–100%) occasions of sexual activity (in identified situational contexts or, if generalized, in all contexts) and have a minimum duration of approximately 6 months. The use of the minimum severity and duration criteria is intended to distinguish transient orgasm difficulties from more persistent orgasmic dysfunction. The inclusion of "approximately" in Criterion B allows for clinician judgment in cases in which symptom duration does not meet the recommended 6-month threshold.

For a woman to have a diagnosis of female orgasmic disorder, clinically significant distress must accompany the symptoms (Criterion C). In many cases of orgasm problems, the causes are multifactorial or cannot be determined. If female orgasmic disorder is deemed to be better explained by another mental disorder, the effects of a substance/medication, or a medical condition, then a diagnosis of female orgasmic disorder would not be made. Finally, if interpersonal or significant contextual factors, such as severe relationship distress, intimate partner violence, or other significant stressors, are present, then a diagnosis of female orgasmic disorder would not be made.

Many women require clitoral stimulation to reach orgasm, and a relatively small proportion of women report that they always experience orgasm during penile-vaginal intercourse. Thus, a woman's experiencing orgasm through clitoral stimulation but not during intercourse does not meet criteria for a clinical diagnosis of female orgasmic disorder. It is also important to consider whether orgasmic difficulties are the result of inadequate sexual stimulation; in these cases, there may still be a need for care, but a diagnosis of female orgasmic disorder would not be made.

Associated Features Supporting Diagnosis

Associations between specific patterns of personality traits or psychopathology and orgasmic dysfunction have generally not been supported. Compared with women without the disorder, some women with female orgasmic disorder may have greater difficulty communicating about sexual issues. Overall sexual satisfaction, however, is not strongly correlated with orgasmic experience. Many women report high levels of sexual satisfaction

despite rarely or never experiencing orgasm. Orgasmic difficulties in women often co-occur with problems related to sexual interest and arousal.

In addition to the subtypes "lifelong/acquired" and "generalized/situational," the following five factors must be considered during assessment and diagnosis of female orgasmic disorder given that they may be relevant to etiology and/or treatment: 1) partner factors (e.g., partner's sexual problems, partner's health status); 2) relationship factors (e.g., poor communication, discrepancies in desire for sexual activity); 3) individual vulnerability factors (e.g., poor body image, history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), or stressors (e.g., job loss, bereavement); (4) cultural/religious factors (e.g., inhibitions related to prohibitions against sexual activity; attitudes toward sexuality); and 5) medical factors relevant to prognosis, course, or treatment. Each of these factors may contribute differently to the presenting symptoms of different women with this disorder.

Prevalence

Reported prevalence rates for female orgasmic problems in women vary widely, from 10% to 42%, depending on multiple factors (e.g., age, culture, duration, and severity of symptoms); however, these estimates do not take into account the presence of distress. Only a proportion of women experiencing orgasm difficulties also report associated distress. Variation in how symptoms are assessed (e.g., the duration of symptoms and the recall period) also influence prevalence rates. Approximately 10% of women do not experience orgasm throughout their lifetime.

Development and Course

By definition, lifelong female orgasmic disorder indicates that the orgasmic difficulties have always been present, whereas the acquired subtype would be assigned if the woman's orgasmic difficulties developed after a period of normal orgasmic functioning.

A woman's first experience of orgasm can occur any time from the prepubertal period to well into adulthood. Women show a more variable pattern in age at first orgasm than do men, and women's reports of having experienced orgasm increase with age. Many women learn to experience orgasm as they experience a wide variety of stimulation and acquire more knowledge about their bodies. Women's rates of orgasm consistency (defined as "usually or always" experiencing orgasm) are higher during masturbation than during sexual activity with a partner.

Risk and Prognostic Factors

Temperamental. A wide range of psychological factors, such as anxiety and concerns about pregnancy, can potentially interfere with a woman's ability to experience orgasm.

Environmental. There is a strong association between relationship problems, physical health, and mental health and orgasm difficulties in women. Sociocultural factors (e.g., gender role expectations and religious norms) are also important influences on the experience of orgasmic difficulties.

Genetic and physiological. Many physiological factors may influence a woman's experience of orgasm, including medical conditions and medications. Conditions such as multiple sclerosis, pelvic nerve damage from radical hysterectomy, and spinal cord injury can all influence orgasmic functioning in women. Selective serotonin reuptake inhibitors are known to delay or inhibit orgasm in women. Women with vulvovaginal atrophy (characterized by symptoms such as vaginal dryness, itching, and pain) are significantly more likely to report orgasm difficulties than are women without this condition. Menopausal status is not consistently associated with the likelihood of orgasm difficulties. There may be a significant genetic contribution to variation in female orgasmic function. However,

psychological, sociocultural, and physiological factors likely interact in complex ways to influence women's experience of orgasm and of orgasm difficulties.

Culture-Related Diagnostic Issues

The degree to which lack of orgasm in women is regarded as a problem that requires treatment may vary depending on cultural context. In addition, women differ in how important orgasm is to their sexual satisfaction. There may be marked sociocultural and generational differences in women's orgasmic ability. For example, the prevalence of inability to reach orgasm has ranged from 17.7% (in Northern Europe) to 42.2% (in Southeast Asia).

Diagnostic Markers

Although measurable physiological changes occur during female orgasm, including changes in hormones, pelvic floor musculature, and brain activation, there is significant variability in these indicators of orgasm across women. In clinical situations, the diagnosis of female orgasmic disorder is based on a woman's self-report.

Functional Consequences of Female Orgasmic Disorder

The functional consequences of female orgasmic disorder are unclear. Although there is a strong association between relationship problems and orgasmic difficulties in women, it is unclear whether relationship factors are risk factors for orgasmic difficulties or are consequences of those difficulties.

Differential Diagnosis

Nonsexual mental disorders. Nonsexual mental disorders, such as major depressive disorder, which is characterized by markedly diminished interest or pleasure in all, or almost all, activities, may explain female orgasmic disorder. If the orgasmic difficulties are better explained by another mental disorder, then a diagnosis of female orgasmic disorder would not be made.

Substance/medication-induced sexual dysfunction. Substance/medication use may explain the orgasmic difficulties.

Another medical condition. If the disorder is due to another medical condition (e.g., multiple sclerosis, spinal cord injury), then a diagnosis of female orgasmic disorder would not be made.

Interpersonal factors. If interpersonal or significant contextual factors, such as severe relationship distress, intimate partner violence, or other significant stressors, are associated with the orgasmic difficulties, then a diagnosis of female orgasmic disorder would not be made.

Other sexual dysfunctions. Female orgasmic disorder may occur in association with other sexual dysfunctions (e.g., female sexual interest/arousal disorder). The presence of another sexual dysfunction does not rule out a diagnosis of female orgasmic disorder. Occasional orgasmic difficulties that are short-term or infrequent and are not accompanied by clinically significant distress or impairment are not diagnosed as female orgasmic disorder. A diagnosis is also not appropriate if the problems are the result of inadequate sexual stimulation.

Comorbidity

Women with female orgasmic disorder may have co-occurring sexual interest/arousal difficulties. Women with diagnoses of other nonsexual mental disorders, such as major depressive disorder, may experience lower sexual interest/arousal, and this may indirectly increase the likelihood of orgasmic difficulties.

Female Sexual Interest/Arousal Disorder

Diagnostic Criteria 302.72 (F52.22)

A. Lack of, or significantly reduced, sexual interest/arousal, as manifested by at least three of the following:
   1. Absent/reduced interest in sexual activity.
   2. Absent/reduced sexual/erotic thoughts or fantasies.
   3. No/reduced initiation of sexual activity, and typically unreceptive to a partner's attempts to initiate.
   4. Absent/reduced sexual excitement/pleasure during sexual activity in almost all or all (approximately 75%–100%) sexual encounters (in identified situational contexts or, if generalized, in all contexts).
   5. Absent/reduced sexual interest/arousal in response to any internal or external sexual/erotic cues (e.g., written, verbal, visual).
   6. Absent/reduced genital or nongenital sensations during sexual activity in almost all or all (approximately 75%–100%) sexual encounters (in identified situational contexts or, if generalized, in all contexts).

B. The symptoms in Criterion A have persisted for a minimum duration of approximately 6 months.

C. The symptoms in Criterion A cause clinically significant distress in the individual.

D. The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of severe relationship distress (e.g., partner violence) or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition.

Specify whether:

Lifelong: The disturbance has been present since the individual became sexually active.

Acquired: The disturbance began after a period of relatively normal sexual function.

Specify whether:

Generalized: Not limited to certain types of stimulation, situations, or partners.

Situational: Only occurs with certain types of stimulation, situations, or partners.

Specify current severity:

Mild: Evidence of mild distress over the symptoms in Criterion A.

Moderate: Evidence of moderate distress over the symptoms in Criterion A.

Severe: Evidence of severe or extreme distress over the symptoms in Criterion A.

Diagnostic Features

In assessing female sexual interest/arousal disorder, interpersonal context must be taken into account. A "desire discrepancy," in which a woman has lower desire for sexual activity than her partner, is not sufficient to diagnose female sexual interest/arousal disorder. In order for the criteria for the disorder to be met, there must be absence or reduced frequency or intensity of at least three of six indicators (Criterion A) for a minimum duration of approximately 6 months (Criterion B). There may be different symptom profiles across women, as well as variability in how sexual interest and arousal are expressed. For example, in one woman, sexual interest/arousal disorder may be expressed as a lack of interest in sexual activity, an absence of erotic or sexual thoughts, and reluctance to initiate sexual activity and respond to a partner's sexual invitations. In another woman, an inability to become sexually excited, to respond to sexual stimuli with sexual desire, and a correspond-

ing lack of signs of physical sexual arousal may be the primary features. Because sexual desire and arousal frequently coexist and are elicited in response to adequate sexual cues, the criteria for female sexual interest/arousal disorder take into account that difficulties in desire and arousal often simultaneously characterize the complaints of women with this disorder. Short-term changes in sexual interest or arousal are common and may be adaptive responses to events in a woman's life and do not represent a sexual dysfunction. Diagnosis of female sexual interest/arousal disorder requires a minimum duration of symptoms of approximately 6 months as a reflection that the symptoms must be a persistent problem. The estimation of persistence may be determined by clinical judgment when a duration of 6 months cannot be ascertained precisely.

There may be absent or reduced frequency or intensity of interest in sexual activity (Criterion A1), which was previously termed *hypoactive sexual desire disorder*. The frequency or intensity of sexual and erotic thoughts or fantasies may be absent or reduced (Criterion A2). The expression of fantasies varies widely across women and may include memories of past sexual experiences. The normative decline in sexual thoughts with age should be taken into account when this criterion is being assessed. Absence or reduced frequency of initiating sexual activity and of receptivity to a partner's sexual invitations (Criterion A3) is a behaviorally focused criterion. A couple's beliefs and preferences for sexual initiation patterns are highly relevant to the assessment of this criterion. There may be absent or reduced sexual excitement or pleasure during sexual activity in almost all or all (approximately 75%–100%) sexual encounters (Criterion A4). Lack of pleasure is a common presenting clinical complaint in women with low desire. Among women who report low sexual desire, there are fewer sexual or erotic cues that elicit sexual interest or arousal (i.e., there is a lack of "responsive desire"). Assessment of the adequacy of sexual stimuli will assist in determining if there is a difficulty with responsive sexual desire (Criterion A5). Frequency or intensity of genital or nongenital sensations during sexual activity may be reduced or absent (Criterion A6). This may include reduced vaginal lubrication/vasocongestion, but because physiological measures of genital sexual response do not differentiate women who report sexual arousal concerns from those who do not, the self-report of reduced or absent genital or nongenital sensations is sufficient.

For a diagnosis of female sexual interest/arousal disorder to be made, clinically significant distress must accompany the symptoms in Criterion A. Distress may be experienced as a result of the lack of sexual interest/arousal or as a result of significant interference in a woman's life and well-being. If a lifelong lack of sexual desire is better explained by one's self-identification as "asexual," then a diagnosis of female sexual interest/arousal disorder would not be made.

Associated Features Supporting Diagnosis

Female sexual interest/arousal disorder is frequently associated with problems in experiencing orgasm, pain experienced during sexual activity, infrequent sexual activity, and couple-level discrepancies in desire. Relationship difficulties and mood disorders are also frequently associated features of female sexual interest/arousal disorder. Unrealistic expectations and norms regarding the "appropriate" level of sexual interest or arousal, along with poor sexual techniques and lack of information about sexuality, may also be evident in women diagnosed with female sexual interest/arousal disorder. The latter, as well as normative beliefs about gender roles, are important factors to consider.

In addition to the subtypes "lifelong/acquired" and "generalized/situational," the following five factors must be considered during assessment and diagnosis of female sexual interest/arousal disorder given that they may be relevant to etiology and/or treatment: 1) partner factors (e.g., partner's sexual problems, partner's health status); 2) relationship factors (e.g., poor communication, discrepancies in desire for sexual activity); 3) individual vulnerability factors (e.g., poor body image, history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), or stressors (e.g., job loss, bereavement); 4) cultural/religious factors (e.g., inhibitions related to prohibitions against sexual activity; attitudes toward sexuality); and

5) medical factors relevant to prognosis, course, or treatment. Note that each of these factors may contribute differently to the presenting symptoms of different women with this disorder.

Prevalence

The prevalence of female sexual interest/arousal disorder, as defined in this manual, is unknown. The prevalence of low sexual desire and of problems with sexual arousal (with and without associated distress), as defined by DSM-IV or ICD-10, may vary markedly in relation to age, cultural setting, duration of symptoms, and presence of distress. Regarding duration of symptoms, there are striking differences in prevalence estimates between short-term and persistent problems related to lack of sexual interest. When distress about sexual functioning is required, prevalence estimates are markedly lower. Some older women report less distress about low sexual desire than younger women, although sexual desire may decrease with age.

Development and Course

By definition, lifelong female sexual interest/arousal disorder suggests that the lack of sexual interest or arousal has been present for the woman's entire sexual life. For Criteria A3, A4, and A6, which assess functioning during sexual activity, a subtype of lifelong would mean presence of symptoms since the individual's first sexual experiences. The acquired subtype would be assigned if the difficulties with sexual interest or arousal developed after a period of nonproblematic sexual functioning. Adaptive and normative changes in sexual functioning may result from partner-related, interpersonal, or personal events and may be transient in nature. However, persistence of symptoms for approximately 6 months or more would constitute a sexual dysfunction.

There are normative changes in sexual interest and arousal across the life span. Furthermore, women in relationships of longer duration are more likely to report engaging in sex despite no obvious feelings of sexual desire at the outset of a sexual encounter compared with women in shorter-duration relationships. Vaginal dryness in older women is related to age and menopausal status.

Risk and Prognostic Factors

Temperamental. Temperamental factors include negative cognitions and attitudes about sexuality and past history of mental disorders. Differences in propensity for sexual excitation and sexual inhibition may also predict the likelihood of developing sexual problems.

Environmental. Environmental factors include relationship difficulties, partner sexual functioning, and developmental history, such as early relationships with caregivers and childhood stressors.

Genetic and physiological. Some medical conditions (e.g., diabetes mellitus, thyroid dysfunction) can be risk factors for female sexual interest/arousal disorder. There appears to be a strong influence of genetic factors on vulnerability to sexual problems in women. Psychophysiological research using vaginal photoplethysmography has not found differences between women with and without perceived lack of genital arousal.

Culture-Related Diagnostic Issues

There is marked variability in prevalence rates of low desire across cultures. Lower rates of sexual desire may be more common among East Asian women compared with Euro-Canadian women. Although the lower levels of sexual desire and arousal found in men and women from East Asian countries compared with Euro-American groups may reflect less interest in sex in those cultures, the possibility remains that such group differences are an artifact of the measures used to quantify desire. A judgment about whether low sexual

desire reported by a woman from a certain ethnocultural group meets criteria for female sexual interest/arousal disorder must take into account the fact that different cultures may pathologize some behaviors and not others.

Gender-Related Diagnostic Issues

By definition, the diagnosis of female sexual interest/arousal disorder is only given to women. Distressing difficulties with sexual desire in men would be considered under male hypoactive sexual desire disorder.

Functional Consequences of Female Sexual Interest/Arousal Disorder

Difficulties in sexual interest/arousal are often associated with decreased relationship satisfaction.

Differential Diagnosis

Nonsexual mental disorders. Nonsexual mental disorders, such as major depressive disorder, in which there is "markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day," may explain the lack of sexual interest/arousal. If the lack of interest or arousal is completely attributable to another mental disorder, then a diagnosis of female sexual interest/arousal disorder would not be made.

Substance/medication use. Substance or medication use may explain the lack of interest/arousal.

Another medical condition. If the sexual symptoms are considered to be almost exclusively associated with the effects of another medical condition (e.g., diabetes mellitus, endothelial disease, thyroid dysfunction, central nervous system disease), then a diagnosis of female sexual interest/arousal disorder would not be made.

Interpersonal factors. If interpersonal or significant contextual factors, such as severe relationship distress, intimate partner violence, or other significant stressors, explain the sexual interest/arousal symptoms, then a diagnosis of female sexual interest/arousal disorder would not be made.

Other sexual dysfunctions. The presence of another sexual dysfunction does not rule out a diagnosis of female sexual interest/arousal disorder. It is common for women to experience more than one sexual dysfunction. For example, the presence of chronic genital pain may lead to a lack of desire for the (painful) sexual activity. Lack of interest and arousal during sexual activity may impair orgasmic ability. For some women, all aspects of the sexual response may be unsatisfying and distressing.

Inadequate or absent sexual stimuli. When differential diagnoses are being considered, it is important to assess the adequacy of sexual stimuli within the woman's sexual experience. In cases where inadequate or absent sexual stimuli are contributing to the clinical picture, there may be evidence for clinical care, but a sexual dysfunction diagnosis would not be made. Similarly, transient and adaptive alterations in sexual functioning that are secondary to a significant life or personal event must be considered in the differential diagnosis.

Comorbidity

Comorbidity between sexual interest/arousal problems and other sexual difficulties is extremely common. Sexual distress and dissatisfaction with sex life are also highly correlated in women with low sexual desire. Distressing low desire is associated with depression, thyroid problems, anxiety, urinary incontinence, and other medical factors. Arthritis and inflammatory or irritable bowel disease are also associated with sexual arousal prob-

lems. Low desire appears to be comorbid with depression, sexual and physical abuse in adulthood, global mental functioning, and use of alcohol.

Genito-Pelvic Pain/Penetration Disorder

Diagnostic Criteria **302.76 (F52.6)**

A. Persistent or recurrent difficulties with one (or more) of the following:
   1. Vaginal penetration during intercourse.
   2. Marked vulvovaginal or pelvic pain during vaginal intercourse or penetration attempts.
   3. Marked fear or anxiety about vulvovaginal or pelvic pain in anticipation of, during, or as a result of vaginal penetration.
   4. Marked tensing or tightening of the pelvic floor muscles during attempted vaginal penetration.

B. The symptoms in Criterion A have persisted for a minimum duration of approximately 6 months.

C. The symptoms in Criterion A cause clinically significant distress in the individual.

D. The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of a severe relationship distress (e.g., partner violence) or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition.

Specify whether:

Lifelong: The disturbance has been present since the individual became sexually active.

Acquired: The disturbance began after a period of relatively normal sexual function.

Specify current severity:

Mild: Evidence of mild distress over the symptoms in Criterion A.

Moderate: Evidence of moderate distress over the symptoms in Criterion A.

Severe: Evidence of severe or extreme distress over the symptoms in Criterion A.

Diagnostic Features

Genito-pelvic pain/penetration disorder refers to four commonly comorbid symptom dimensions: 1) difficulty having intercourse, 2) genito-pelvic pain, 3) fear of pain or vaginal penetration, and 4) tension of the pelvic floor muscles (Criterion A). Because major difficulty in any one of these symptom dimensions is often sufficient to cause clinically significant distress, a diagnosis can be made on the basis of marked difficulty in only one symptom dimension. However, all four symptom dimensions should be assessed even if a diagnosis can be made on the basis of only one symptom dimension.

Marked difficulty having vaginal intercourse/penetration (Criterion A1) can vary from a total inability to experience vaginal penetration in any situation (e.g., intercourse, gynecological examinations, tampon insertion) to the ability to easily experience penetration in one situation and but not in another. Although the most common clinical situation is when a woman is unable to experience intercourse or penetration with a partner, difficulties in undergoing required gynecological examinations may also be present. *Marked vulvovaginal or pelvic pain during vaginal intercourse or penetration attempts* (Criterion A2) refers to pain occurring in different locations in the genito-pelvic area. Location of pain as well as intensity should be assessed. Typically, pain can be characterized as superficial (vulvovaginal or occurring during penetration) or deep (pelvic; i.e., not felt until deeper penetration). The intensity of the pain is often not linearly related to distress or interference with sexual intercourse or other sexual activities. Some genito-pelvic pain only occurs when provoked (i.e., by intercourse or mechanical stim-

ulation); other genito-pelvic pain may be spontaneous as well as provoked. Genito-pelvic pain can also be usefully characterized qualitatively (e.g., "burning," "cutting," "shooting," "throbbing"). The pain may persist for a period after intercourse is completed and may also occur during urination. Typically, the pain experienced during sexual intercourse can be reproduced during a gynecological examination.

Marked fear or anxiety about vulvovaginal or pelvic pain either in anticipation of, or during, or as a result of vaginal penetration (Criterion A3) is commonly reported by women who have regularly experienced pain during sexual intercourse. This "normal" reaction may lead to avoidance of sexual/intimate situations. In other cases, this marked fear does not appear to be closely related to the experience of pain but nonetheless leads to avoidance of intercourse and vaginal penetration situations. Some have described this as similar to a phobic reaction except that the phobic object may be vaginal penetration or the fear of pain.

Marked tensing or tightening of the pelvic floor muscles during attempted vaginal penetration (Criterion A4) can vary from reflexive-like spasm of the pelvic floor in response to attempted vaginal entry to "normal/voluntary" muscle guarding in response to the anticipated or the repeated experience of pain or to fear or anxiety. In the case of "normal/guarding" reactions, penetration may be possible under circumstances of relaxation. The characterization and assessment of pelvic floor dysfunction is often best undertaken by a specialist gynecologist or by a pelvic floor physical therapist.

Associated Features Supporting Diagnosis

Genito-pelvic pain/penetration disorder is frequently associated with other sexual dysfunctions, particularly reduced sexual desire and interest (female sexual interest/arousal disorder). Sometimes desire and interest are preserved in sexual situations that are not painful or do not require penetration. Even when individuals with genito-pelvic pain/penetration disorder report sexual interest/motivation, there is often behavioral avoidance of sexual situations and opportunities. Avoidance of gynecological examinations despite medical recommendations is also frequent. The pattern of avoidance is similar to that seen in phobic disorders. It is common for women who have not succeeded in having sexual intercourse to come for treatment only when they wish to conceive. Many women with genito-pelvic pain/penetration disorder will experience associated relationship/marital problems; they also often report that the symptoms significantly diminish their feelings of femininity.

In addition to the subtype "lifelong/acquired," five factors should be considered during assessment and diagnosis of genito-pelvic pain/penetration disorder because they may be relevant to etiology and/or treatment: 1) partner factors (e.g., partner's sexual problems, partner's health status); 2) relationship factors (e.g., poor communication, discrepancies in desire for sexual activity); 3) individual vulnerability factors (e.g., poor body image, history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), or stressors (e.g., job loss, bereavement); 4) cultural/religious factors (e.g., inhibitions related to prohibitions against sexual activity; attitudes toward sexuality); and 5) medical factors relevant to prognosis, course, or treatment. Each of these factors may contribute differently to the presenting symptoms of different women with this disorder.

There are no valid physiological measures of any of the component symptom dimensions of genito-pelvic pain/penetration disorder. Validated psychometric inventories may be used to formally assess the pain and anxiety components related to genito-pelvic pain/penetration disorder.

Prevalence

The prevalence of genito-pelvic pain/penetration disorder is unknown. However, approximately 15% of women in North America report recurrent pain during intercourse. Difficulties having intercourse appear to be a frequent referral to sexual dysfunction clinics and to specialist clinicians.

Development and Course

The development and course of genito-pelvic pain/penetration disorder is unclear. Because women generally do not seek treatment until they experience problems in sexual functioning, it can, in general, be difficult to characterize genito-pelvic pain/penetration disorder as lifelong (primary) or acquired (secondary). Although women typically come to clinical attention after the initiation of sexual activity, there are often earlier clinical signs. For example, difficulty with or the avoidance of use of tampons is an important predictor of later problems. Difficulties with vaginal penetration (inability or fear or pain) may not be obvious until sexual intercourse is attempted. Even once intercourse is attempted, the frequency of attempts may not be significant or regular. In cases where it is difficult to establish whether symptomatology is lifelong or acquired, it is useful to determine the presence of any consistent period of successful pain-, fear-, and tension-free intercourse. If the experience of such a period can be established, then genito-pelvic pain/penetration disorder can be characterized as acquired. Once symptomatology is well established for a period of approximately 6 months, the probability of spontaneous and significant symptomatic remission appears to diminish.

Complaints related to genito-pelvic pain peak during early adulthood and in the peri- and postmenopausal period. Women with complaints about difficulty having intercourse appear to be primarily premenopausal. There may also be an increase in genito-pelvic pain–related symptoms in the postpartum period.

Risk and Prognostic Factors

Environmental. Sexual and/or physical abuse have often been cited as predictors of the DSM-IV-defined sexual pain disorders dyspareunia and vaginismus. This is a matter of controversy in the current literature.

Genetic and physiological. Women experiencing superficial pain during sexual intercourse often report the onset of the pain after a history of vaginal infections. Even after the infections have resolved and there are no known residual physical findings, the pain persists. Pain during tampon insertion or the inability to insert tampons before any sexual contact has been attempted is an important risk factor for genito-pelvic pain/penetration disorder.

Culture-Related Diagnostic Issues

In the past, inadequate sexual education and religious orthodoxy have often been considered to be culturally related predisposing factors to the DSM-IV diagnosis of vaginismus. This perception appears to be confirmed by recent reports from Turkey, a primarily Muslim country, indicating a strikingly high prevalence for the disorder. However, most available research, although limited in scope, does not support this notion (Lahaie et al. 2010).

Gender-Related Diagnostic Issues

By definition, the diagnosis of genito-pelvic pain/penetration disorder is only given to women. There is relatively new research concerning urological chronic pelvic pain syndrome in men, suggesting that men may experience some similar problems. The research and clinical experience are not sufficiently developed yet to justify the application of this diagnosis to men. Other specified sexual dysfunction or unspecified sexual dysfunction may be diagnosed in men appearing to fit this pattern.

Functional Consequences of Genito-Pelvic Pain/Penetration Disorder

Functional difficulties in genito-pelvic pain/penetration disorder are often associated with interference in relationship satisfaction and sometimes with the ability to conceive via penile/vaginal intercourse.

Differential Diagnosis

Another medical condition. In many instances, women with genito-pelvic pain/penetration disorder will also be diagnosed with another medical condition (e.g., lichen sclerosus, endometriosis, pelvic inflammatory disease, vulvovaginal atrophy). In some cases, treating the medical condition may alleviate the genito-pelvic pain/penetration disorder. Much of the time, this is not the case. There are no reliable tools or diagnostic methods to allow clinicians to know whether the medical condition or genito-pelvic pain/penetration disorder is primary. Often, the associated medical conditions are difficult to diagnose and treat. For example, the increased incidence of postmenopausal pain during intercourse may sometimes be attributable to vaginal dryness or vulvovaginal atrophy associated with declining estrogen levels. The relationship, however, between vulvovaginal atrophy/dryness, estrogen, and pain is not well understood.

Somatic symptom and related disorders. Some women with genito-pelvic pain/penetration disorder may also be diagnosable with somatic symptom disorder. Since both genito-pelvic pain/penetration disorder and the somatic symptom and related disorders are new diagnoses, it is not yet clear whether they can be reliably differentiated. Some women diagnosed with genito-pelvic pain/penetration disorder will also be diagnosed with a specific phobia.

Inadequate sexual stimuli. It is important that the clinician, in considering differential diagnoses, assess the adequacy of sexual stimuli within the woman's sexual experience. Sexual situations in which there is inadequate foreplay or arousal may lead to difficulties in penetration, pain, or avoidance. Erectile dysfunction or premature ejaculation in the male partner may result in difficulties with penetration. These conditions should be carefully assessed. In some situations, a diagnosis of genito-pelvic pain/penetration disorder may not be appropriate.

Comorbidity

Comorbidity between genito-pelvic pain/penetration disorder and other sexual difficulties appears to be common. Comorbidity with relationship distress is also common. This is not surprising, since in Western cultures the inability to have (pain-free) intercourse with a desired partner and the avoidance of sexual opportunities may be either a contributing factor to or the result of other sexual or relationship problems. Because pelvic floor symptoms are implicated in the diagnosis of genito-pelvic pain/penetration disorder, there is likely to be a higher prevalence of other disorders related to the pelvic floor or reproductive organs (e.g., interstitial cystitis, constipation, vaginal infection, endometriosis, irritable bowel syndrome).

Male Hypoactive Sexual Desire Disorder

Diagnostic Criteria **302.71 (F52.0)**

A. Persistently or recurrently deficient (or absent) sexual/erotic thoughts or fantasies and desire for sexual activity. The judgment of deficiency is made by the clinician, taking into account factors that affect sexual functioning, such as age and general and sociocultural contexts of the individual's life.

B. The symptoms in Criterion A have persisted for a minimum duration of approximately 6 months.

C. The symptoms in Criterion A cause clinically significant distress in the individual.

D. The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of severe relationship distress or other significant stressors and is not attributable to thes effects of a substance/medication or another medical condition.

Specify whether:

Lifelong: The disturbance has been present since the individual became sexually active.

Acquired: The disturbance began after a period of relatively normal sexual function.

Specify whether:

Generalized: Not limited to certain types of stimulation, situations, or partners.

Situational: Only occurs with certain types of stimulation, situations, or partners.

Specify current severity:

Mild: Evidence of mild distress over the symptoms in Criterion A.

Moderate: Evidence of moderate distress over the symptoms in Criterion A.

Severe: Evidence of severe or extreme distress over the symptoms in Criterion A.

Diagnostic Features

When an assessment for male hypoactive sexual desire disorder is being made, interpersonal context must be taken into account. A "desire discrepancy," in which a man has lower desire for sexual activity than his partner, is not sufficient to diagnose male hypoactive sexual desire disorder. Both low/absent desire for sex and deficient/absent sexual thoughts or fantasies are required for a diagnosis of the disorder. There may be variation across men in how sexual desire is expressed.

The lack of desire for sex and deficient/absent erotic thoughts or fantasies must be persistent or recurrent and must occur for a minimum duration of approximately 6 months. The inclusion of this duration criterion is meant to safeguard against making a diagnosis in cases in which a man's low sexual desire may represent an adaptive response to adverse life conditions (e.g., concern about a partner's pregnancy when the man is considering terminating the relationship). The introduction of "approximately" in Criterion B allows for clinician judgment in cases in which symptom duration does not meet the recommended 6-month threshold.

Associated Features Supporting Diagnosis

Male hypoactive sexual desire disorder is sometimes associated with erectile and/or ejaculatory concerns. For example, persistent difficulties obtaining an erection may lead a man to lose interest in sexual activity. Men with hypoactive sexual desire disorder often report that they no longer initiate sexual activity and that they are minimally receptive to a partner's attempt to initiate. Sexual activities (e.g., masturbation or partnered sexual activity) may sometimes occur even in the presence of low sexual desire. Relationship-specific preferences regarding patterns of sexual initiation must be taken into account when making a diagnosis of male hypoactive sexual desire disorder. Although men are more likely to initiate sexual activity, and thus low desire may be characterized by a pattern of non-initiation, many men may prefer to have their partner initiate sexual activity. In such situations, the man's lack of receptivity to a partner's initiation should be considered when evaluating low desire.

In addition to the subtypes "lifelong/acquired" and "generalized/situational," the following five factors must be considered during assessment and diagnosis of male hypoactive sexual desire disorder given that they may be relevant to etiology and/or treatment: 1) partner factors (e.g., partner's sexual problems, partner's health status); 2) relationship factors (e.g., poor communication, discrepancies in desire for sexual activity); 3) individual vulnerability factors (e.g., poor body image, history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), or stressors (e.g., job loss, bereavement); 4) cultural/religious factors (e.g., inhibitions related to prohibitions against sexual activity; attitudes toward sexuality); and 5) medical factors relevant to prognosis, course, or treat-

ment. Each of these factors may contribute differently to the presenting symptoms of different men with this disorder.

Prevalence

The prevalence of male hypoactive sexual desire disorder varies depending on country of origin and method of assessment. Approximately 6% of younger men (ages 18–24 years) and 41% of older men (ages 66–74 years) have problems with sexual desire. However, a persistent lack of interest in sex, lasting 6 months or more, affects only a small proportion of men ages 16–44 (1.8%).

Development and Course

By definition, lifelong male hypoactive sexual desire disorder indicates that low or no sexual desire has always been present, whereas the acquired subtype would be assigned if the man's low desire developed after a period of normal sexual desire. There is a requirement that low desire persist for approximately 6 months or more; thus, short-term changes in sexual desire should not be diagnosed as male hypoactive sexual desire disorder.

There is a normative age-related decline in sexual desire. Like women, men identify a variety of triggers for their sexual desire, and they describe a wide range of reasons that they choose to engage in sexual activity. Although erotic visual cues may be more potent elicitors of desire in younger men, the potency of sexual cues may decrease with age and must be considered when evaluating men for hypoactive sexual desire disorder.

Risk and Prognostic Factors

Temperamental. Mood and anxiety symptoms appear to be strong predictors of low desire in men. Up to half of men with a past history of psychiatric symptoms may have moderate or severe loss of desire, compared with only 15% of those without such a history. A man's feelings about himself, his perception of his partner's sexual desire toward him, feelings of being emotionally connected, and contextual variables may all negatively (as well as positively) affect sexual desire.

Environmental. Alcohol use may increase the occurrence of low desire. Among gay men, self-directed homophobia, interpersonal problems, attitudes, lack of adequate sex education, and trauma resulting from early life experiences must be taken into account in explaining the low desire. Social and cultural contextual factors should also be considered.

Genetic and physiological. Endocrine disorders such as hyperprolactinemia significantly affect sexual desire in men. Age is a significant risk factor for low desire in men. It is unclear whether or not men with low desire also have abnormally low levels of testosterone; however, among hypogonadal men, low desire is common. There also may be a critical threshold below which testosterone will affect sexual desire in men and above which there is little effect of testosterone on men's desire.

Culture-Related Diagnostic Issues

There is marked variability in prevalence rates of low desire across cultures, ranging from 12.5% in Northern European men to 28% in Southeast Asian men ages 40–80 years. Just as there are higher rates of low desire among East Asian subgroups of women, men of East Asian ancestry also have higher rates of low desire. Guilt about sex may mediate this association between East Asian ethnicity and sexual desire in men.

Gender-Related Diagnostic Issues

In contrast to the classification of sexual disorders in women, desire and arousal disorders have been retained as separate constructs in men. Despite some similarities in the experi-

ence of desire across men and women, and the fact that desire fluctuates over time and is dependent on contextual factors, men do report a significantly higher intensity and frequency of sexual desire compared with women.

Differential Diagnosis

Nonsexual mental disorders. Nonsexual mental disorders, such as major depressive disorder, which is characterized by "markedly diminished interest or pleasure in all, or almost all, activities," may explain the lack of sexual desire. If the lack of desire is better explained by another mental disorder, then a diagnosis of male hypoactive sexual desire disorder would not be made.

Substance/medication use. Substance/medication use may explain the lack of sexual desire.

Another medical condition. If the low/absent desire and deficient/absent erotic thoughts or fantasies are better explained by the effects of another medical condition (e.g., hypogonadism, diabetes mellitus, thyroid dysfunction, central nervous system disease), then a diagnosis of male hypoactive sexual desire disorder would not be made.

Interpersonal factors. If interpersonal or significant contextual factors, such as severe relationship distress or other significant stressors, are associated with the loss of desire in the man, then a diagnosis of male hypoactive sexual desire disorder would not be made.

Other sexual dysfunctions. The presence of another sexual dysfunction does not rule out a diagnosis of male hypoactive sexual desire disorder; there is some evidence that up to one-half of men with low sexual desire also have erectile difficulties, and slightly fewer may also have early ejaculation difficulties. If the man's low desire is explained by self-identification as an asexual, then a diagnosis of male hypoactive sexual desire disorder is not made.

Comorbidity

Depression and other mental disorders, as well as endocrinological factors, are often comorbid with male hypoactive sexual desire disorder.

Premature (Early) Ejaculation

Diagnostic Criteria **302.75 (F52.4)**

A. A persistent or recurrent pattern of ejaculation occurring during partnered sexual activity within approximately 1 minute following vaginal penetration and before the individual wishes it.

   Note: Although the diagnosis of premature (early) ejaculation may be applied to individuals engaged in nonvaginal sexual activities, specific duration criteria have not been established for these activities.

B. The symptom in Criterion A must have been present for at least 6 months and must be experienced on almost all or all (approximately 75%–100%) occasions of sexual activity (in identified situational contexts or, if generalized, in all contexts).

C. The symptom in Criterion A causes clinically significant distress in the individual.

D. The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of severe relationship distress or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition.

Specify whether:

Lifelong: The disturbance has been present since the individual became sexually active.

Acquired: The disturbance began after a period of relatively normal sexual function.

Specify whether:

Generalized: Not limited to certain types of stimulation, situations, or partners.

Situational: Only occurs with certain types of stimulation, situations, or partners.

Specify current severity:

Mild: Ejaculation occurring within approximately 30 seconds to 1 minute of vaginal penetration.

Moderate: Ejaculation occurring within approximately 15–30 seconds of vaginal penetration.

Severe: Ejaculation occurring prior to sexual activity, at the start of sexual activity, or within approximately 15 seconds of vaginal penetration.

Diagnostic Features

Premature (early) ejaculation is manifested by ejaculation that occurs prior to or shortly after vaginal penetration, operationalized by an individual's estimate of ejaculatory latency (i.e., elapsed time before ejaculation) after vaginal penetration. Estimated and measured intravaginal ejaculatory latencies are highly correlated as long as the ejaculatory latency is of short duration; therefore, self-reported estimates of ejaculatory latency are sufficient for diagnostic purposes. A 60-second intravaginal ejaculatory latency time is an appropriate cutoff for the diagnosis of lifelong premature (early) ejaculation in heterosexual men. There are insufficient data to determine if this duration criterion can be applied to acquired premature (early) ejaculation. The durational definition may apply to males of varying sexual orientations, since ejaculatory latencies appear to be similar across men of different sexual orientations and across different sexual activities.

Associated Features Supporting Diagnosis

Many males with premature (early) ejaculation complain of a sense of lack of control over ejaculation and report apprehension about their anticipated inability to delay ejaculation on future sexual encounters.

The following factors may be relevant in the evaluation of any sexual dysfunction: 1) partner factors (e.g., partner's sexual problems, partner's health status); 2) relationship factors (e.g., poor communication, discrepancies in desire for sexual activity); 3) individual vulnerability factors (e.g., poor body image, history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), and stressors (e.g., job loss, bereavement); 4) cultural/religious factors (e.g., inhibitions related to prohibitions against sexual activity; attitudes toward sexuality); and 5) medical factors relevant to prognosis, course, or treatment.

Prevaience

Estimates of the prevalence of premature (early) ejaculation vary widely depending on the definition utilized. Internationally, more than 20%–30% of men ages 18–70 years report concern about how rapidly they ejaculate. With the new definition of premature (early) ejaculation (i.e., ejaculation occurring within approximately 1 minute of vaginal penetration), only 1%–3% of men would be diagnosed with the disorder. Prevalence of premature (early) ejaculation may increase with age.

Deveiopment and Course

By definition, lifelong premature (early) ejaculation starts during a male's initial sexual experiences and persists thereafter. Some men may experience premature (early) ejaculation during their initial sexual encounters but gain ejaculatory control over time. It is the persistence of ejaculatory problems for longer than 6 months that determines the diagnosis of premature (early) ejaculation. In contrast, some men develop the disorder after a period of

having a normal ejaculatory latency, known as *acquired premature (early) ejaculation.* There is far less known about acquired premature (early) ejaculation than about lifelong premature (early) ejaculation. The acquired form likely has a later onset, usually appearing during or after the fourth decade of life. Lifelong is relatively stable throughout life. Little is known about the course of acquired premature (early) ejaculation. Reversal of medical conditions such as hyperthyroidism and prostatitis appears to restore ejaculatory latencies to baseline values. Lifelong premature (early) ejaculation begins with early sexual experiences and persists throughout an individual's life. In approximately 20% of men with premature (early) ejaculation, ejaculatory latencies decrease further with age. Age and relationship length have been found to be negatively associated with prevalence of premature (early) ejaculation.

Risk and Prognostic Factors

Temperamental. Premature (early) ejaculation may be more common in men with anxiety disorders, especially social anxiety disorder (social phobia).

Genetic and physiological. There is a moderate genetic contribution to lifelong premature (early) ejaculation. Premature (early) ejaculation may be associated with dopamine transporter gene polymorphism or serotonin transporter gene polymorphism. Thyroid disease, prostatitis, and drug withdrawal are associated with acquired premature (early) ejaculation. Positron emission tomography measures of regional cerebral blood flow during ejaculation have shown primary activation in the mesocephalic transition zone, including the ventral tegmental area.

Culture-Related Diagnostic Issues

Perception of what constitutes a normal ejaculatory latency is different in many cultures. Measured ejaculatory latencies may differ in some countries. Such differences may be explained by cultural or religious factors as well as genetic differences between populations.

Gender-Related Diagnostic Issues

Premature (early) ejaculation is a sexual disorder in males. Males and their sexual partners may differ in their perception of what constitutes an acceptable ejaculatory latency. There may be increasing concerns in females about early ejaculation in their sexual partners, which may be a reflection of changing societal attitudes concerning female sexual activity.

Diagnostic Markers

Ejaculatory latency is usually monitored in research settings by the sexual partner utilizing a timing device (e.g., stopwatch), though this is not ideal in real-life sexual situations. For vaginal intercourse, the time between intravaginal penetration and ejaculation is measured.

Functional Consequences of Premature (Early) Ejaculation

A pattern of premature (early) ejaculation may be associated with decreased self-esteem, a sense of lack of control, and adverse consequences for partner relationships. It may also cause personal distress in the sexual partner and decreased sexual satisfaction in the sexual partner. Ejaculation prior to penetration may be associated with difficulties in conception.

Differential Diagnosis

Substance/medication-induced sexual dysfunction. When problems with premature ejaculation are due exclusively to substance use, intoxication, or withdrawal, substance/medication-induced sexual dysfunction should be diagnosed.

Ejaculatory concerns that do not meet diagnostic criteria. It is necessary to identify males with normal ejaculatory latencies who desire longer ejaculatory latencies and males who have episodic premature (early) ejaculation (e.g., during the first sexual encounter with a new partner when a short ejaculatory latency may be common or normative). Neither of these situations would lead to a diagnosis of premature (early) ejaculation, even though these situations may be distressing to some males.

Comorbidity

Premature (early) ejaculation may be associated with erectile problems. In many cases, it may be difficult to determine which difficulty preceded the other. Lifelong premature (early) ejaculation may be associated with certain anxiety disorders. Acquired premature (early) ejaculation may be associated with prostatitis, thyroid disease, or drug withdrawal (e.g., during opioid withdrawal).

Substance/Medication-Induced Sexual Dysfunction

Diagnostic Criteria

A. A clinically significant disturbance in sexual function is predominant in the clinical picture.

B. There is evidence from the history, physical examination, or laboratory findings of both (1) and (2):

   1. The symptoms in Criterion A developed during or soon after substance intoxication or withdrawal or after exposure to a medication.
   2. The involved substance/medication is capable of producing the symptoms in Criterion A.

C. The disturbance is not better explained by a sexual dysfunction that is not substance/medication-induced. Such evidence of an independent sexual dysfunction could include the following:

   The symptoms precede the onset of the substance/medication use; the symptoms persist for a substantial period of time (e.g., about 1 month) after the cessation of acute withdrawal or severe intoxication; or there is other evidence suggesting the existence of an independent non-substance/medication-induced sexual dysfunction (e.g., a history of recurrent non-substance/medication-related episodes).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress in the individual.

Note: This diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and are sufficiently severe to warrant clinical attention.

Coding note: The ICD-9-CM and ICD-10-CM codes for the [specific substance/medication]-induced sexual dysfunctions are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. If a mild substance use disorder is comorbid with the substance-induced sexual dysfunction, the 4th position character is "1," and the clinician should record "mild [substance] use disorder" before the substance-induced sexual dysfunction (e.g., "mild cocaine use disorder with cocaine-induced sexual dysfunction"). If a moderate or severe substance use disorder is comorbid with the substance-induced sexual dysfunction, the 4th position character is "2," and the clinician should record "moderate [substance] use disorder" or "severe [substance] use disorder," depending on the severity of the comorbid substance

use disorder. If there is no comorbid substance use disorder (e.g., after a one-time heavy use of the substance), then the 4th position character is "9," and the clinician should record only the substance-induced sexual dysfunction.

		ICD-10-CM		
	ICD-9-CM	With use disorder, mild	With use disorder, moderate or severe	Without use disorder
Alcohol	291.89	F10.181	F10.281	F10.981
Opioid	292.89	F11.181	F11.281	F11.981
Sedative, hypnotic, or anxiolytic	292.89	F13.181	F13.281	F13.981
Amphetamine (or other stimulant)	292.89	F15.181	F15.281	F15.981
Cocaine	292.89	F14.181	F14.281	F14.981
Other (or unknown) substance	292.89	F19.181	F19.281	F19.981

Specify if (see Table 1 in the chapter "Substance-Related and Addictive Disorders" for diagnoses associated with substance class):

With onset during intoxication: If the criteria are met for intoxication with the substance and the symptoms develop during intoxication.

With onset during withdrawal: If criteria are met for withdrawal from the substance and the symptoms develop during, or shortly after, withdrawal.

With onset after medication use: Symptoms may appear either at initiation of medication or after a modification or change in use.

Specify current severity:

Mild: Occurs on 25%–50% of occasions of sexual activity.

Moderate: Occurs on 50%–75% of occasions of sexual activity.

Severe: Occurs on 75% or more of occasions of sexual activity.

Recording Procedures

ICD-9-CM. The name of the substance/medication-induced sexual dysfunction begins with the specific substance (e.g., alcohol, fluoxetine) that is presumed to be causing the sexual dysfunction. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class. For substances that do not fit into any of the classes (e.g., fluoxetine), the code for "other substance" should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category "unknown substance" should be used.

The name of the disorder is followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal, with onset after medication use), followed by the severity specifier (e.g., mild, moderate, severe). Unlike the recording procedures for ICD-10-CM, which combine the substance-induced disorder and substance use disorder into a single code, for ICD-9-CM a separate diagnostic code is given for the substance use disorder. For example, in the case of erectile dysfunction occurring during intoxication in a man with a severe alcohol use disorder, the diagnosis is 291.89 alcohol-induced sexual dysfunction, with onset during intoxication, moderate. An additional diagnosis of 303.90 severe alcohol use disorder is also given. When more than one substance is judged to play a sig-

nificant role in the development of the sexual dysfunction, each should be listed separately (e.g., 292.89 cocaine-induced sexual dysfunction with onset during intoxication, moderate; 292.89 fluoxetine-induced sexual dysfunction, with onset after medication use).

ICD-10-CM. The name of the substance/medication-induced sexual dysfunction begins with the specific substance (e.g., alcohol, fluoxetine) that is presumed to be causing the sexual dysfunction. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class and presence or absence of a comorbid substance use disorder. For substances that do not fit into any of the classes (e.g., fluoxetine), the code for "other substance" should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category "unknown substance" should be used.

When recording the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by the word "with," followed by the name of the substance-induced sexual dysfunction, followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal, with onset after medication use), followed by the severity specifier (e.g., mild, moderate, severe). For example, in the case of erectile dysfunction occurring during intoxication in a man with a severe alcohol use disorder, the diagnosis is F10.281 moderate alcohol use disorder with alcohol-induced sexual dysfunction, with onset during intoxication, moderate. A separate diagnosis of the comorbid severe alcohol use disorder is not given. If the substance-induced sexual dysfunction occurs without a comorbid substance use disorder (e.g., after a one-time heavy use of the substance), no accompanying substance use disorder is noted (e.g., F15.981 amphetamine-induced sexual dysfunction, with onset during intoxication). When more than one substance is judged to play a significant role in the development of the sexual dysfunction, each should be listed separately (e.g., F14.181 mild cocaine use disorder with cocaine-induced sexual dysfunction, with onset during intoxication, moderate; F19.981 fluoxetine-induced sexual dysfunction, with onset after medication use, moderate).

Diagnostic Features

The major feature is a disturbance in sexual function that has a temporal relationship with substance/medication initiation, dose increase, or substance/medication discontinuation.

Associated Features Supporting Diagnosis

Sexual dysfunctions can occur in association with intoxication with the following classes of substances: alcohol; opioids; sedatives, hypnotics, or anxiolytics; stimulants (including cocaine); and other (or unknown) substances. Sexual dysfunctions can occur in association with withdrawal from the following classes of substances: alcohol; opioids; sedatives, hypnotics, or anxiolytics; and other (or unknown) substances. Medications that can induce sexual dysfunctions include antidepressants, antipsychotics, and hormonal contraceptives.

The most commonly reported side effect of antidepressant drugs is difficulty with orgasm or ejaculation. Problems with desire and erection are less frequent. Approximately 30% of sexual complaints are clinically significant. Certain agents, such as bupropion and mirtazapine, appear not to be associated with sexual side effects.

The sexual problems associated with antipsychotic drugs, including problems with sexual desire, erection, lubrication, ejaculation, or orgasm, have occurred with typical as well as atypical agents. However, problems are less common with prolactin-sparing antipsychotics than with agents that cause significant prolactin elevation.

Although the effects of mood stabilizers on sexual function are unclear, it is possible that lithium and anticonvulsants, with the possible exception of lamotrigine, have adverse effects on sexual desire. Problems with orgasm may occur with gabapentin. Similarly, there may be a higher prevalence of erectile and orgasmic problems associated with benzodiazepines. There have not been such reports with buspirone.

Many nonpsychiatric medications, such as cardiovascular, cytotoxic, gastrointestinal, and hormonal agents, are associated with disturbances in sexual function. Illicit substance use is associated with decreased sexual desire, erectile dysfunction, and difficulty reaching orgasm. Sexual dysfunctions are also seen in individuals receiving methadone but are seldom reported by patients receiving buprenorphine. Chronic alcohol abuse and chronic nicotine abuse are associated with erectile problems.

Prevalence

The prevalence and the incidence of substance/medication-induced sexual dysfunction are unclear, likely because of underreporting of treatment-emergent sexual side effects. Data on substance/medication-induced sexual dysfunction typically concern the effects of antidepressant drugs. The prevalence of antidepressant-induced sexual dysfunction varies in part depending on the specific agent. Approximately 25%–80% of individuals taking monoamine oxidase inhibitors, tricyclic antidepressants, serotonergic antidepressants, and combined serotonergic-adrenergic antidepressants report sexual side effects. There are differences in the incidence of sexual side effects between some serotonergic and combined adrenergic-serotonergic antidepressants, although it is unclear if these differences are clinically significant.

Approximately 50% of individuals taking antipsychotic medications will experience adverse sexual side effects, including problems with sexual desire, erection, lubrication, ejaculation, or orgasm. The incidence of these side effects among different antipsychotic agents is unclear.

Exact prevalence and incidence of sexual dysfunctions among users of nonpsychiatric medications such as cardiovascular, cytotoxic, gastrointestinal, and hormonal agents are unknown. Elevated rates of sexual dysfunction have been reported with methadone or high-dose opioid drugs for pain. There are increased rates of decreased sexual desire, erectile dysfunction, and difficulty reaching orgasm associated with illicit substance use. The prevalence of sexual problems appears related to chronic drug abuse and appears higher in individuals who abuse heroin (approximately 60%–70%) than in individuals who abuse amphetamines or 3,4-methylenedioxymethamphetamine (i.e., MDMA, ecstasy). Elevated rates of sexual dysfunction are also seen in individuals receiving methadone but are seldom reported by patients receiving buprenorphine. Chronic alcohol abuse and chronic nicotine abuse are related to higher rates of erectile problems.

Development and Course

The onset of antidepressant-induced sexual dysfunction may be as early as 8 days after the agent is first taken. Approximately 30% of individuals with mild to moderate orgasm delay will experience spontaneous remission of the dysfunction within 6 months. In some cases, serotonin reuptake inhibitor–induced sexual dysfunction may persist after the agent is discontinued. The time to onset of sexual dysfunction after initiation of antipsychotic drugs or drugs of abuse is unknown. It is probable that the adverse effects of nicotine and alcohol may not appear until after years of use. Premature (early) ejaculation can sometimes occur after cessation of opioid use. There is some evidence that disturbances in sexual function related to substance/medication use increase with age.

Culture-Related Diagnostic Issues

There may be an interaction among cultural factors, the influence of medications on sexual functioning, and the response of the individual to those changes.

Gender-Related Diagnostic Issues

Some gender differences in sexual side effects may exist.

Functional Consequences of Substance/Medication-Induced Sexual Dysfunction

Medication-induced sexual dysfunction may result in medication noncompliance.

Differential Diagnosis

Non-substance/medication-induced sexual dysfunctions. Many mental conditions, such as depressive, bipolar, anxiety, and psychotic disorders, are associated with disturbances of sexual function. Thus, differentiating a substance/medication-induced sexual dysfunction from a manifestation of the underlying mental disorder can be quite difficult. The diagnosis is usually established if a close relationship between substance/medication initiation or discontinuation is observed. A clear diagnosis can be established if the problem occurs after substance/medication initiation, dissipates with substance/medication discontinuation, and recurs with introduction of the same agent. Most substance/medication-induced side effects occur shortly after initiation or discontinuation. Sexual side effects that only occur after chronic use of a substance/medication may be extremely difficult to diagnose with certainty.

Other Specified Sexual Dysfunction

302.79 (F52.8)

This category applies to presentations in which symptoms characteristic of a sexual dysfunction that cause clinically significant distress in the individual predominate but do not meet the full criteria for any of the disorders in the sexual dysfunctions diagnostic class. The other specified sexual dysfunction category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific sexual dysfunction. This is done by recording "other specified sexual dysfunction" followed by the specific reason (e.g., "sexual aversion").

Unspecified Sexual Dysfunction

302.70 (F52.9)

This category applies to presentations in which symptoms characteristic of a sexual dysfunction that cause clinically significant distress in the individual predominate but do not meet the full criteria for any of the disorders in the sexual dysfunctions diagnostic class. The unspecified sexual dysfunction category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific sexual dysfunction, and includes presentations for which there is insufficient information to make a more specific diagnosis.

Gender Dysphoria

In this chapter, there is one overarching diagnosis of gender dysphoria, with separate developmentally appropriate criteria sets for children and for adolescents and adults. The area of sex and gender is highly controversial and has led to a proliferation of terms whose meanings vary over time and within and between disciplines. An additional source of confusion is that in English "sex" connotes both male/female and sexuality. This chapter employs constructs and terms as they are widely used by clinicians from various disciplines with specialization in this area. In this chapter, *sex* and *sexual* refer to the biological indicators of male and female (understood in the context of reproductive capacity), such as in sex chromosomes, gonads, sex hormones, and nonambiguous internal and external genitalia. Disorders of sex development denote conditions of inborn somatic deviations of the reproductive tract from the norm and/or discrepancies among the biological indicators of male and female. *Cross-sex* hormone treatment denotes the use of feminizing hormones in an individual assigned male at birth based on traditional biological indicators or the use of masculinizing hormones in an individual assigned female at birth.

The need to introduce the term *gender* arose with the realization that for individuals with conflicting or ambiguous biological indicators of sex (i.e., "intersex"), the lived role in society and/or the identification as male or female could not be uniformly associated with or predicted from the biological indicators and, later, that some individuals develop an identity as female or male at variance with their uniform set of classical biological indicators. Thus, *gender* is used to denote the public (and usually legally recognized) lived role as boy or girl, man or woman, but, in contrast to certain social constructionist theories, biological factors are seen as contributing, in interaction with social and psychological factors, to gender development. *Gender assignment* refers to the initial assignment as male or female. This occurs usually at birth and, thereby, yields the "natal gender." *Gender-atypical* refers to somatic features or behaviors that are not typical (in a statistical sense) of individuals with the same assigned gender in a given society and historical era; for behavior, *gender-nonconforming* is an alternative descriptive term. *Gender reassignment* denotes an official (and usually legal) change of gender. *Gender identity* is a category of social identity and refers to an individual's identification as male, female, or, occasionally, some category other than male or female. *Gender dysphoria* as a general descriptive term refers to an individual's affective/cognitive discontent with the assigned gender but is more specifically defined when used as a diagnostic category. *Transgender* refers to the broad spectrum of individuals who transiently or persistently identify with a gender different from their natal gender. *Transsexual* denotes an individual who seeks, or has undergone, a social transition from male to female or female to male, which in many, but not all, cases also involves a somatic transition by cross-sex hormone treatment and genital surgery (*sex reassignment surgery*).

Gender dysphoria refers to the distress that may accompany the incongruence between one's experienced or expressed gender and one's assigned gender. Although not all individuals will experience distress as a result of such incongruence, many are distressed if the desired physical interventions by means of hormones and/or surgery are not available. The current term is more descriptive than the previous DSM-IV term *gender identity disorder* and focuses on dysphoria as the clinical problem, not identity per se.

Gender Dysphoria

Diagnostic Criteria

Gender Dysphoria in Children 302.6 (F64.2)

A. A marked incongruence between one's experienced/expressed gender and assigned gender, of at least 6 months' duration, as manifested by at least six of the following (one of which must be Criterion A1):

1. A strong desire to be of the other gender or an insistence that one is the other gender (or some alternative gender different from one's assigned gender).
2. In boys (assigned gender), a strong preference for cross-dressing or simulating female attire; or in girls (assigned gender), a strong preference for wearing only typical masculine clothing and a strong resistance to the wearing of typical feminine clothing.
3. A strong preference for cross-gender roles in make-believe play or fantasy play.
4. A strong preference for the toys, games, or activities stereotypically used or engaged in by the other gender.
5. A strong preference for playmates of the other gender.
6. In boys (assigned gender), a strong rejection of typically masculine toys, games, and activities and a strong avoidance of rough-and-tumble play; or in girls (assigned gender), a strong rejection of typically feminine toys, games, and activities.
7. A strong dislike of one's sexual anatomy.
8. A strong desire for the primary and/or secondary sex characteristics that match one's experienced gender.

B. The condition is associated with clinically significant distress or impairment in social, school, or other important areas of functioning.

Specify if:

With a disorder of sex development (e.g., a congenital adrenogenital disorder such as 255.2 [E25.0] congenital adrenal hyperplasia or 259.50 [E34.50] androgen insensitivity syndrome).

Coding note: Code the disorder of sex development as well as gender dysphoria.

Gender Dysphoria in Adolescents and Adults 302.85 (F64.1)

A. A marked incongruence between one's experienced/expressed gender and assigned gender, of at least 6 months' duration, as manifested by at least two of the following:

1. A marked incongruence between one's experienced/expressed gender and primary and/or secondary sex characteristics (or in young adolescents, the anticipated secondary sex characteristics).
2. A strong desire to be rid of one's primary and/or secondary sex characteristics because of a marked incongruence with one's experienced/expressed gender (or in young adolescents, a desire to prevent the development of the anticipated secondary sex characteristics).
3. A strong desire for the primary and/or secondary sex characteristics of the other gender.
4. A strong desire to be of the other gender (or some alternative gender different from one's assigned gender).
5. A strong desire to be treated as the other gender (or some alternative gender different from one's assigned gender).
6. A strong conviction that one has the typical feelings and reactions of the other gender (or some alternative gender different from one's assigned gender).

B. The condition is associated with clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if:

With a disorder of sex development (e.g., a congenital adrenogenital disorder such as 255.2 [E25.0] congenital adrenal hyperplasia or 259.50 [E34.50] androgen insensitivity syndrome).

Coding note: Code the disorder of sex development as well as gender dysphoria.

Specify if:

Posttransition: The individual has transitioned to full-time living in the desired gender (with or without legalization of gender change) and has undergone (or is preparing to have) at least one cross-sex medical procedure or treatment regimen—namely, regular cross-sex hormone treatment or gender reassignment surgery confirming the desired gender (e.g., penectomy, vaginoplasty in a natal male; mastectomy or phalloplasty in a natal female).

Specifiers

The posttransition specifier may be used in the context of continuing treatment procedures that serve to support the new gender assignment.

Diagnostic Features

Individuals with gender dysphoria have a marked incongruence between the gender they have been assigned to (usually at birth, referred to as *natal gender*) and their experienced/expressed gender. This discrepancy is the core component of the diagnosis. There must also be evidence of distress about this incongruence. Experienced gender may include alternative gender identities beyond binary stereotypes. Consequently, the distress is not limited to a desire to simply be of the other gender, but may include a desire to be of an alternative gender, provided that it differs from the individual's assigned gender.

Gender dysphoria manifests itself differently in different age groups. Prepubertal natal girls with gender dysphoria may express the wish to be a boy, assert they are a boy, or assert they will grow up to be a man. They prefer boys' clothing and hairstyles, are often perceived by strangers as boys, and may ask to be called by a boy's name. Usually, they display intense negative reactions to parental attempts to have them wear dresses or other feminine attire. Some may refuse to attend school or social events where such clothes are required. These girls may demonstrate marked cross-gender identification in role-playing, dreams, and fantasies. Contact sports, rough-and-tumble play, traditional boyhood games, and boys as playmates are most often preferred. They show little interest in stereotypically feminine toys (e.g., dolls) or activities (e.g., feminine dress-up or role-play). Occasionally, they refuse to urinate in a sitting position. Some natal girls may express a desire to have a penis or claim to have a penis or that they will grow one when older. They may also state that they do not want to develop breasts or menstruate.

Prepubertal natal boys with gender dysphoria may express the wish to be a girl or assert they are a girl or that they will grow up to be a woman. They have a preference for dressing in girls' or women's clothes or may improvise clothing from available materials (e.g., using towels, aprons, and scarves for long hair or skirts). These children may role-play female figures (e.g., playing "mother") and often are intensely interested in female fantasy figures. Traditional feminine activities, stereotypical games, and pastimes (e.g., "playing house"; drawing feminine pictures; watching television or videos of favorite female characters) are most often preferred. Stereotypical female-type dolls (e.g., Barbie) are often favorite toys, and girls are their preferred playmates. They avoid rough-and-tumble play and competitive sports and have little interest in stereotypically masculine toys (e.g., cars, trucks). Some may pretend not to have a penis and insist on sitting to urinate. More

rarely, they may state that they find their penis or testes disgusting, that they wish them removed, or that they have, or wish to have, a vagina.

In young adolescents with gender dysphoria, clinical features may resemble those of children or adults with the condition, depending on developmental level. As secondary sex characteristics of young adolescents are not yet fully developed, these individuals may not state dislike of them, but they are concerned about imminent physical changes.

In adults with gender dysphoria, the discrepancy between experienced gender and physical sex characteristics is often, but not always, accompanied by a desire to be rid of primary and/or secondary sex characteristics and/or a strong desire to acquire some primary and/or secondary sex characteristics of the other gender. To varying degrees, adults with gender dysphoria may adopt the behavior, clothing, and mannerisms of the experienced gender. They feel uncomfortable being regarded by others, or functioning in society, as members of their assigned gender. Some adults may have a strong desire to be of a different gender and treated as such, and they may have an inner certainty to feel and respond as the experienced gender without seeking medical treatment to alter body characteristics. They may find other ways to resolve the incongruence between experienced/expressed and assigned gender by partially living in the desired role or by adopting a gender role neither conventionally male nor conventionally female.

Associated Features Supporting Diagnosis

When visible signs of puberty develop, natal boys may shave their legs at the first signs of hair growth. They sometimes bind their genitals to make erections less visible. Girls may bind their breasts, walk with a stoop, or use loose sweaters to make breasts less visible. Increasingly, adolescents request, or may obtain without medical prescription and supervision, hormonal suppressors ("blockers") of gonadal steroids (e.g., gonadotropin-releasing hormone [GnRH] analog, spironolactone). Clinically referred adolescents often want hormone treatment and many also wish for gender reassignment surgery. Adolescents living in an accepting environment may openly express the desire to be and be treated as the experienced gender and dress partly or completely as the experienced gender, have a hairstyle typical of the experienced gender, preferentially seek friendships with peers of the other gender, and/or adopt a new first name consistent with the experienced gender. Older adolescents, when sexually active, usually do not show or allow partners to touch their sexual organs. For adults with an aversion toward their genitals, sexual activity is constrained by the preference that their genitals not be seen or touched by their partners. Some adults may seek hormone treatment (sometimes without medical prescription and supervision) and gender reassignment surgery. Others are satisfied with either hormone treatment or surgery alone.

Adolescents and adults with gender dysphoria before gender reassignment are at increased risk for suicidal ideation, suicide attempts, and suicides. After gender reassignment, adjustment may vary, and suicide risk may persist.

Prevaience

For natal adult males, prevalence ranges from 0.005% to 0.014%, and for natal females, from 0.002% to 0.003%. Since not all adults seeking hormone treatment and surgical reassignment attend specialty clinics, these rates are likely modest underestimates. Sex differences in rate of referrals to specialty clinics vary by age group. In children, sex ratios of natal boys to girls range from 2:1 to 4.5:1. In adolescents, the sex ratio is close to parity; in adults, the sex ratio favors natal males, with ratios ranging from 1:1 to 6.1:1. In two countries, the sex ratio appears to favor natal females (Japan: 2.2:1; Poland: 3.4:1).

Development and Course

Because expression of gender dysphoria varies with age, there are separate criteria sets for children versus adolescents and adults. Criteria for children are defined in a more con-

crete, behavioral manner than those for adolescents and adults. Many of the core criteria draw on well-documented behavioral gender differences between typically developing boys and girls. Young children are less likely than older children, adolescents, and adults to express extreme and persistent anatomic dysphoria. In adolescents and adults, incongruence between experienced gender and somatic sex is a central feature of the diagnosis. Factors related to distress and impairment also vary with age. A very young child may show signs of distress (e.g., intense crying) only when parents tell the child that he or she is "really" not a member of the other gender but only "desires" to be. Distress may not be manifest in social environments supportive of the child's desire to live in the role of the other gender and may emerge only if the desire is interfered with. In adolescents and adults, distress may manifest because of strong incongruence between experienced gender and somatic sex. Such distress may, however, be mitigated by supportive environments and knowledge that biomedical treatments exist to reduce incongruence. Impairment (e.g., school refusal, development of depression, anxiety, and substance abuse) may be a consequence of gender dysphoria.

Gender dysphoria without a disorder of sex development. For clinic-referred children, onset of cross-gender behaviors is usually between ages 2 and 4 years. This corresponds to the developmental time period in which most typically developing children begin expressing gendered behaviors and interests. For some preschool-age children, both pervasive cross-gender behaviors and the expressed desire to be the other gender may be present, or, more rarely, labeling oneself as a member of the other gender may occur. In some cases, the expressed desire to be the other gender appears later, usually at entry into elementary school. A small minority of children express discomfort with their sexual anatomy or will state the desire to have a sexual anatomy corresponding to the experienced gender ("anatomic dysphoria"). Expressions of anatomic dysphoria become more common as children with gender dysphoria approach and anticipate puberty.

Rates of persistence of gender dysphoria from childhood into adolescence or adulthood vary. In natal males, persistence has ranged from 2.2% to 30%. In natal females, persistence has ranged from 12% to 50%. Persistence of gender dysphoria is modestly correlated with dimensional measures of severity ascertained at the time of a childhood baseline assessment. In one sample of natal males, lower socioeconomic background was also modestly correlated with persistence. It is unclear if particular therapeutic approaches to gender dysphoria in children are related to rates of long-term persistence. Extant follow-up samples consisted of children receiving no formal therapeutic intervention or receiving therapeutic interventions of various types, ranging from active efforts to reduce gender dysphoria to a more neutral, "watchful waiting" approach. It is unclear if children "encouraged" or supported to live socially in the desired gender will show higher rates of persistence, since such children have not yet been followed longitudinally in a systematic manner. For both natal male and female children showing persistence, almost all are sexually attracted to individuals of their natal sex. For natal male children whose gender dysphoria does not persist, the majority are *androphilic* (sexually attracted to males) and often self-identify as gay or homosexual (ranging from 63% to 100%). In natal female children whose gender dysphoria does not persist, the percentage who are *gynephilic* (sexually attracted to females) and self-identify as lesbian is lower (ranging from 32% to 50%).

In both adolescent and adult natal males, there are two broad trajectories for development of gender dysphoria: early onset and late onset. *Early-onset gender dysphoria* starts in childhood and continues into adolescence and adulthood; or, there is an intermittent period in which the gender dysphoria desists and these individuals self-identify as gay or homosexual, followed by recurrence of gender dysphoria. *Late-onset gender dysphoria* occurs around puberty or much later in life. Some of these individuals report having had a desire to be of the other gender in childhood that was not expressed verbally to others. Others do not recall any signs of childhood gender dysphoria. For adolescent males with late-onset gender dysphoria, parents often report surprise because they did not see signs of gender

dysphoria during childhood. Expressions of anatomic dysphoria are more common and salient in adolescents and adults once secondary sex characteristics have developed.

Adolescent and adult natal males with early-onset gender dysphoria are almost always sexually attracted to men (androphilic). Adolescents and adults with late-onset gender dysphoria frequently engage in transvestic behavior with sexual excitement. The majority of these individuals are gynephilic or sexually attracted to other posttransition natal males with late-onset gender dysphoria. A substantial percentage of adult males with late-onset gender dysphoria cohabit with or are married to natal females. After gender transition, many self-identify as lesbian. Among adult natal males with gender dysphoria, the early-onset group seeks out clinical care for hormone treatment and reassignment surgery at an earlier age than does the late-onset group. The late-onset group may have more fluctuations in the degree of gender dysphoria and be more ambivalent about and less likely satisfied after gender reassignment surgery.

In both adolescent and adult natal females, the most common course is the early-onset form of gender dysphoria. The late-onset form is much less common in natal females compared with natal males. As in natal males with gender dysphoria, there may have been a period in which the gender dysphoria desisted and these individuals self-identified as lesbian; however, with recurrence of gender dysphoria, clinical consultation is sought, often with the desire for hormone treatment and reassignment surgery. Parents of natal adolescent females with the late-onset form also report surprise, as no signs of childhood gender dysphoria were evident. Expressions of anatomic dysphoria are much more common and salient in adolescents and adults than in children.

Adolescent and adult natal females with early-onset gender dysphoria are almost always gynephilic. Adolescents and adults with the late-onset form of gender dysphoria are usually androphilic and after gender transition self-identify as gay men. Natal females with the late-onset form do not have co-occurring transvestic behavior with sexual excitement.

Gender dysphoria in association with a disorder of sex development. Most individuals with a disorder of sex development who develop gender dysphoria have already come to medical attention at an early age. For many, starting at birth, issues of gender assignment were raised by physicians and parents. Moreover, as infertility is quite common for this group, physicians are more willing to perform cross-sex hormone treatments and genital surgery before adulthood.

Disorders of sex development in general are frequently associated with gender-atypical behavior starting in early childhood. However, in the majority of cases, this does not lead to gender dysphoria. As individuals with a disorder of sex development become aware of their medical history and condition, many experience uncertainty about their gender, as opposed to developing a firm conviction that they are another gender. However, most do not progress to gender transition. Gender dysphoria and gender transition may vary considerably as a function of a disorder of sex development, its severity, and assigned gender.

Risk and Prognostic Factors

Temperamental. For individuals with gender dysphoria without a disorder of sex development, atypical gender behavior among individuals with early-onset gender dysphoria develops in early preschool age, and it is possible that a high degree of atypicality makes the development of gender dysphoria and its persistence into adolescence and adulthood more likely.

Environmental. Among individuals with gender dysphoria without a disorder of sex development, males with gender dysphoria (in both childhood and adolescence) more commonly have older brothers than do males without the condition. Additional predisposing

factors under consideration, especially in individuals with late-onset gender dysphoria (adolescence, adulthood), include habitual fetishistic transvestism developing into autogynephilia (i.e., sexual arousal associated with the thought or image of oneself as a woman) and other forms of more general social, psychological, or developmental problems.

Genetic and physiological. For individuals with gender dysphoria without a disorder of sex development, some genetic contribution is suggested by evidence for (weak) familiality of transsexualism among nontwin siblings, increased concordance for transsexualism in monozygotic compared with dizygotic same-sex twins, and some degree of heritability of gender dysphoria. As to endocrine findings, no endogenous systemic abnormalities in sex-hormone levels have been found in 46,XY individuals, whereas there appear to be increased androgen levels (in the range found in hirsute women but far below normal male levels) in 46,XX individuals. Overall, current evidence is insufficient to label gender dysphoria without a disorder of sex development as a form of intersexuality limited to the central nervous system.

In gender dysphoria associated with a disorder of sex development, the likelihood of later gender dysphoria is increased if prenatal production and utilization (via receptor sensitivity) of androgens are grossly atypical relative to what is usually seen in individuals with the same assigned gender. Examples include 46,XY individuals with a history of normal male prenatal hormone milieu but inborn nonhormonal genital defects (as in cloacal bladder exstrophy or penile agenesis) and who have been assigned to the female gender. The likelihood of gender dysphoria is further enhanced by additional, prolonged, highly gender-atypical postnatal androgen exposure with somatic virilization as may occur in female-raised and noncastrated 46,XY individuals with 5-alpha reductase-2 deficiency or 17-beta-hydroxysteroid dehydrogenase-3 deficiency or in female-raised 46,XX individuals with classical congenital adrenal hyperplasia with prolonged periods of non-adherence to glucocorticoid replacement therapy. However, the prenatal androgen milieu is more closely related to gendered behavior than to gender identity. Many individuals with disorders of sex development and markedly gender-atypical behavior do not develop gender dysphoria. Thus, gender-atypical behavior by itself should not be interpreted as an indicator of current or future gender dysphoria. There appears to be a higher rate of gender dysphoria and patient-initiated gender change from assigned female to male than from assigned male to female in 46,XY individuals with a disorder of sex development.

Culture-Related Diagnostic Issues

Individuals with gender dysphoria have been reported across many countries and cultures. The equivalent of gender dysphoria has also been reported in individuals living in cultures with institutionalized gender categories other than male or female. It is unclear whether with these individuals the diagnostic criteria for gender dysphoria would be met.

Diagnostic Markers

Individuals with a somatic disorder of sex development show some correlation of final gender identity outcome with the degree of prenatal androgen production and utilization. However, the correlation is not robust enough for the biological factor, where ascertainable, to replace a detailed and comprehensive diagnostic interview evaluation for gender dysphoria.

Functional Consequences of Gender Dysphoria

Preoccupation with cross-gender wishes may develop at all ages after the first 2–3 years of childhood and often interfere with daily activities. In older children, failure to develop age-typical same-sex peer relationships and skills may lead to isolation from peer groups and to distress. Some children may refuse to attend school because of teasing and harass-

ment or pressure to dress in attire associated with their assigned sex. Also in adolescents and adults, preoccupation with cross-gender wishes often interferes with daily activities. Relationship difficulties, including sexual relationship problems, are common, and functioning at school or at work may be impaired. Gender dysphoria, along with atypical gender expression, is associated with high levels of stigmatization, discrimination, and victimization, leading to negative self-concept, increased rates of mental disorder comorbidity, school dropout, and economic marginalization, including unemployment, with attendant social and mental health risks, especially in individuals from resource-poor family backgrounds. In addition, these individuals' access to health services and mental health services may be impeded by structural barriers, such as institutional discomfort or inexperience in working with this patient population.

Differential Diagnosis

Nonconformity to gender roles. Gender dysphoria should be distinguished from simple nonconformity to stereotypical gender role behavior by the strong desire to be of another gender than the assigned one and by the extent and pervasiveness of gender-variant activities and interests. The diagnosis is not meant to merely describe nonconformity to stereotypical gender role behavior (e.g., "tomboyism" in girls, "girly-boy" behavior in boys, occasional cross-dressing in adult men). Given the increased openness of atypical gender expressions by individuals across the entire range of the transgender spectrum, it is important that the clinical diagnosis be limited to those individuals whose distress and impairment meet the specified criteria.

Transvestic disorder. Transvestic disorder occurs in heterosexual (or bisexual) adolescent and adult males (rarely in females) for whom cross-dressing behavior generates sexual excitement and causes distress and/or impairment without drawing their primary gender into question. It is occasionally accompanied by gender dysphoria. An individual with transvestic disorder who also has clinically significant gender dysphoria can be given both diagnoses. In many cases of late-onset gender dysphoria in gynephilic natal males, transvestic behavior with sexual excitement is a precursor.

Body dysmorphic disorder. An individual with body dysmorphic disorder focuses on the alteration or removal of a specific body part because it is perceived as abnormally formed, not because it represents a repudiated assigned gender. When an individual's presentation meets criteria for both gender dysphoria and body dysmorphic disorder, both diagnoses can be given. Individuals wishing to have a healthy limb amputated (termed by some *body integrity identity disorder*) because it makes them feel more "complete" usually do not wish to change gender, but rather desire to live as an amputee or a disabled person.

Schizophrenia and other psychotic disorders. In schizophrenia, there may rarely be delusions of belonging to some other gender. In the absence of psychotic symptoms, insistence by an individual with gender dysphoria that he or she is of some other gender is not considered a delusion. Schizophrenia (or other psychotic disorders) and gender dysphoria may co-occur.

Other clinical presentations. Some individuals with an emasculinization desire who develop an alternative, nonmale/nonfemale gender identity do have a presentation that meets criteria for gender dysphoria. However, some males seek castration and/or penectomy for aesthetic reasons or to remove psychological effects of androgens without changing male identity; in these cases, the criteria for gender dysphoria are not met.

Comorbidity

Clinically referred children with gender dysphoria show elevated levels of emotional and behavioral problems—most commonly, anxiety, disruptive and impulse-control, and de-

pressive disorders. In prepubertal children, increasing age is associated with having more behavioral or emotional problems; this is related to the increasing non-acceptance of gender-variant behavior by others. In older children, gender-variant behavior often leads to peer ostracism, which may lead to more behavioral problems. The prevalence of mental health problems differs among cultures; these differences may also be related to differences in attitudes toward gender variance in children. However, also in some non-Western cultures, anxiety has been found to be relatively common in individuals with gender dysphoria, even in cultures with accepting attitudes toward gender-variant behavior. Autism spectrum disorder is more prevalent in clinically referred children with gender dysphoria than in the general population. Clinically referred adolescents with gender dysphoria appear to have comorbid mental disorders, with anxiety and depressive disorders being the most common. As in children, autism spectrum disorder is more prevalent in clinically referred adolescents with gender dysphoria than in the general population. Clinically referred adults with gender dysphoria may have coexisting mental health problems, most commonly anxiety and depressive disorders.

Other Specified Gender Dysphoria

302.6 (F64.8)

This category applies to presentations in which symptoms characteristic of gender dysphoria that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for gender dysphoria. The other specified gender dysphoria category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for gender dysphoria. This is done by recording "other specified gender dysphoria" followed by the specific reason (e.g., "brief gender dysphoria").

An example of a presentation that can be specified using the "other specified" designation is the following:

The current disturbance meets symptom criteria for gender dysphoria, but the duration is less than 6 months.

Unspecified Gender Dysphoria

302.6 (F64.9)

This category applies to presentations in which symptoms characteristic of gender dysphoria that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for gender dysphoria. The unspecified gender dysphoria category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for gender dysphoria, and includes presentations in which there is insufficient information to make a more specific diagnosis.

Disruptive, Impulse-Control, and Conduct Disorders

Disruptive, impulse-control, and conduct disorders include conditions involving problems in the self-control of emotions and behaviors. While other disorders in DSM-5 may also involve problems in emotional and/or behavioral regulation, the disorders in this chapter are unique in that these problems are manifested in behaviors that violate the rights of others (e.g., aggression, destruction of property) and/or that bring the individual into significant conflict with societal norms or authority figures. The underlying causes of the problems in the self-control of emotions and behaviors can vary greatly across the disorders in this chapter and among individuals within a given diagnostic category.

The chapter includes oppositional defiant disorder, intermittent explosive disorder, conduct disorder, antisocial personality disorder (which is described in the chapter "Personality Disorders"), pyromania, kleptomania, and other specified and unspecified disruptive, impulse-control, and conduct disorders. Although all the disorders in the chapter involve problems in both emotional and behavioral regulation, the source of variation among the disorders is the relative emphasis on problems in the two types of self-control. For example, the criteria for conduct disorder focus largely on poorly controlled behaviors that violate the rights of others or that violate major societal norms. Many of the behavioral symptoms (e.g., aggression) can be a result of poorly controlled emotions such as anger. At the other extreme, the criteria for intermittent explosive disorder focus largely on such poorly controlled emotion, outbursts of anger that are disproportionate to the interpersonal or other provocation or to other psychosocial stressors. Intermediate in impact to these two disorders is oppositional defiant disorder, in which the criteria are more evenly distributed between emotions (anger and irritation) and behaviors (argumentativeness and defiance). Pyromania and kleptomania are less commonly used diagnoses characterized by poor impulse control related to specific behaviors (fire setting or stealing) that relieve internal tension. Other specified disruptive, impulse-control, and conduct disorder is a category for conditions in which there are symptoms of conduct disorder, oppositional defiant disorder, or other disruptive, impulse-control, and conduct disorders, but the number of symptoms does not meet the diagnostic threshold for any of the disorders in this chapter, even though there is evidence of clinically significant impairment associated with the symptoms.

The disruptive, impulse-control, and conduct disorders all tend to be more common in males than in females, although the relative degree of male predominance may differ both across disorders and within a disorder at different ages. The disorders in this chapter tend to have first onset in childhood or adolescence. In fact, it is very rare for either conduct disorder or oppositional defiant disorder to first emerge in adulthood. There is a developmental relationship between oppositional defiant disorder and conduct disorder, in that most cases of conduct disorder previously would have met criteria for oppositional defiant disorder, at least in those cases in which conduct disorder emerges prior to adolescence. However, most children with oppositional defiant disorder do not eventually develop conduct disorder. Furthermore, children with oppositional defiant disorder are at risk for eventually developing other problems besides conduct disorder, including anxiety and depressive disorders.

Many of the symptoms that define the disruptive, impulse-control, and conduct disorders are behaviors that can occur to some degree in typically developing individuals. Thus, it is critical that the frequency, persistence, pervasiveness across situations, and im-

pairment associated with the behaviors indicative of the diagnosis be considered relative to what is normative for a person's age, gender, and culture when determining if they are symptomatic of a disorder.

The disruptive, impulse-control, and conduct disorders have been linked to a common externalizing spectrum associated with the personality dimensions labeled as *disinhibition* and (inversely) *constraint* and, to a lesser extent, negative emotionality. These shared personality dimensions could account for the high level of comorbidity among these disorders and their frequent comorbidity with substance use disorders and antisocial personality disorder. However, the specific nature of the shared diathesis that constitutes the externalizing spectrum remains unknown.

Oppositional Defiant Disorder

Diagnostic Criteria **313.81 (F91.3)**

A. A pattern of angry/irritable mood, argumentative/defiant behavior, or vindictiveness lasting at least 6 months as evidenced by at least four symptoms from any of the following categories, and exhibited during interaction with at least one individual who is not a sibling.

Angry/Irritable Mood

1. Often loses temper.
2. Is often touchy or easily annoyed.
3. Is often angry and resentful.

Argumentative/Defiant Behavior

4. Often argues with authority figures or, for children and adolescents, with adults.
5. Often actively defies or refuses to comply with requests from authority figures or with rules.
6. Often deliberately annoys others.
7. Often blames others for his or her mistakes or misbehavior.

Vindictiveness

8. Has been spiteful or vindictive at least twice within the past 6 months.

Note: The persistence and frequency of these behaviors should be used to distinguish a behavior that is within normal limits from a behavior that is symptomatic. For children younger than 5 years, the behavior should occur on most days for a period of at least 6 months unless otherwise noted (Criterion A8). For individuals 5 years or older, the behavior should occur at least once per week for at least 6 months, unless otherwise noted (Criterion A8). While these frequency criteria provide guidance on a minimal level of frequency to define symptoms, other factors should also be considered, such as whether the frequency and intensity of the behaviors are outside a range that is normative for the individual's developmental level, gender, and culture.

B. The disturbance in behavior is associated with distress in the individual or others in his or her immediate social context (e.g., family, peer group, work colleagues), or it impacts negatively on social, educational, occupational, or other important areas of functioning.

C. The behaviors do not occur exclusively during the course of a psychotic, substance use, depressive, or bipolar disorder. Also, the criteria are not met for disruptive mood dysregulation disorder.

Specify current severity:

Mild: Symptoms are confined to only one setting (e.g., at home, at school, at work, with peers).

Moderate: Some symptoms are present in at least two settings.
Severe: Some symptoms are present in three or more settings.

Specifiers

It is not uncommon for individuals with oppositional defiant disorder to show symptoms only at home and only with family members. However, the pervasiveness of the symptoms is an indicator of the severity of the disorder.

Diagnostic Features

The essential feature of oppositional defiant disorder is a frequent and persistent pattern of angry/irritable mood, argumentative/defiant behavior, or vindictiveness (Criterion A). It is not unusual for individuals with oppositional defiant disorder to show the behavioral features of the disorder without problems of negative mood. However, individuals with the disorder who show the angry/irritable mood symptoms typically show the behavioral features as well.

The symptoms of oppositional defiant disorder may be confined to only one setting, and this is most frequently the home. Individuals who show enough symptoms to meet the diagnostic threshold, even if it is only at home, may be significantly impaired in their social functioning. However, in more severe cases, the symptoms of the disorder are present in multiple settings. Given that the pervasiveness of symptoms is an indicator of the severity of the disorder, it is critical that the individual's behavior be assessed across multiple settings and relationships. Because these behaviors are common among siblings, they must be observed during interactions with persons other than siblings. Also, because symptoms of the disorder are typically more evident in interactions with adults or peers whom the individual knows well, they may not be apparent during a clinical examination.

The symptoms of oppositional defiant disorder can occur to some degree in individuals without this disorder. There are several key considerations for determining if the behaviors are symptomatic of oppositional defiant disorder. First, the diagnostic threshold of four or more symptoms within the preceding 6 months must be met. Second, the persistence and frequency of the symptoms should exceed what is normative for an individual's age, gender, and culture. For example, it is not unusual for preschool children to show temper tantrums on a weekly basis. Temper outbursts for a preschool child would be considered a symptom of oppositional defiant disorder only if they occurred on most days for the preceding 6 months, if they occurred with at least three other symptoms of the disorder, and if the temper outbursts contributed to the significant impairment associated with the disorder (e.g., led to destruction of property during outbursts, resulted in the child being asked to leave a preschool).

The symptoms of the disorder often are part of a pattern of problematic interactions with others. Furthermore, individuals with this disorder typically do not regard themselves as angry, oppositional, or defiant. Instead, they often justify their behavior as a response to unreasonable demands or circumstances. Thus, it can be difficult to disentangle the relative contribution of the individual with the disorder to the problematic interactions he or she experiences. For example, children with oppositional defiant disorder may have experienced a history of hostile parenting, and it is often impossible to determine if the child's behavior caused the parents to act in a more hostile manner toward the child, if the parents' hostility led to the child's problematic behavior, or if there was some combination of both. Whether or not the clinician can separate the relative contributions of potential causal factors should not influence whether or not the diagnosis is made. In the event that the child may be living in particularly poor conditions where neglect or mistreatment may occur (e.g., in institutional settings), clinical attention to reducing the contribution of the environment may be helpful.

Associated Features Supporting Diagnosis

In children and adolescents, oppositional defiant disorder is more prevalent in families in which child care is disrupted by a succession of different caregivers or in families in which harsh, inconsistent, or neglectful child-rearing practices are common. Two of the most common co-occurring conditions with oppositional defiant disorder are attention-deficit/hyperactivity disorder (ADHD) and conduct disorder (see the section "Comorbidity" for this disorder). Oppositional defiant disorder has been associated with increased risk for suicide attempts, even after comorbid disorders are controlled for.

Prevalence

The prevalence of oppositional defiant disorder ranges from 1% to 11%, with an average prevalence estimate of around 3.3%. The rate of oppositional defiant disorder may vary depending on the age and gender of the child. The disorder appears to be somewhat more prevalent in males than in females (1.4:1) prior to adolescence. This male predominance is not consistently found in samples of adolescents or adults.

Development and Course

The first symptoms of oppositional defiant disorder usually appear during the preschool years and rarely later than early adolescence. Oppositional defiant disorder often precedes the development of conduct disorder, especially for those with the childhood-onset type of conduct disorder. However, many children and adolescents with oppositional defiant disorder do not subsequently develop conduct disorder. Oppositional defiant disorder also conveys risk for the development of anxiety disorders and major depressive disorder, even in the absence of conduct disorder. The defiant, argumentative, and vindictive symptoms carry most of the risk for conduct disorder, whereas the angry-irritable mood symptoms carry most of the risk for emotional disorders.

Manifestations of the disorder across development appear consistent. Children and adolescents with oppositional defiant disorder are at increased risk for a number of problems in adjustment as adults, including antisocial behavior, impulse-control problems, substance abuse, anxiety, and depression.

Many of the behaviors associated with oppositional defiant disorder increase in frequency during the preschool period and in adolescence. Thus, it is especially critical during these development periods that the frequency and intensity of these behaviors be evaluated against normative levels before it is decided that they are symptoms of oppositional defiant disorder.

Risk and Prognostic Features

Temperamental. Temperamental factors related to problems in emotional regulation (e.g., high levels of emotional reactivity, poor frustration tolerance) have been predictive of the disorder.

Environmental. Harsh, inconsistent, or neglectful child-rearing practices are common in families of children and adolescents with oppositional defiant disorder, and these parenting practices play an important role in many causal theories of the disorder.

Genetic and physiological. A number of neurobiological markers (e.g., lower heart rate and skin conductance reactivity; reduced basal cortisol reactivity; abnormalities in the prefrontal cortex and amygdala) have been associated with oppositional defiant disorder. However, the vast majority of studies have not separated children with oppositional defiant disorder from those with conduct disorder. Thus, it is unclear whether there are markers specific to oppositional defiant disorder.

Culture-Related Diagnostic Issues

The prevalence of the disorder in children and adolescents is relatively consistent across countries that differ in race and ethnicity.

Functional Consequences of Oppositional Defiant Disorder

When oppositional defiant disorder is persistent throughout development, individuals with the disorder experience frequent conflicts with parents, teachers, supervisors, peers, and romantic partners. Such problems often result in significant impairments in the individual's emotional, social, academic, and occupational adjustment.

Differential Diagnosis

Conduct disorder. Conduct disorder and oppositional defiant disorder are both related to conduct problems that bring the individual in conflict with adults and other authority figures (e.g., teachers, work supervisors). The behaviors of oppositional defiant disorder are typically of a less severe nature than those of conduct disorder and do not include aggression toward people or animals, destruction of property, or a pattern of theft or deceit. Furthermore, oppositional defiant disorder includes problems of emotional dysregulation (i.e., angry and irritable mood) that are not included in the definition of conduct disorder.

Attention-deficit/hyperactivity disorder. ADHD is often comorbid with oppositional defiant disorder. To make the additional diagnosis of oppositional defiant disorder, it is important to determine that the individual's failure to conform to requests of others is not solely in situations that demand sustained effort and attention or demand that the individual sit still.

Depressive and bipolar disorders. Depressive and bipolar disorders often involve negative affect and irritability. As a result, a diagnosis of oppositional defiant disorder should not be made if the symptoms occur exclusively during the course of a mood disorder.

Disruptive mood dysregulation disorder. Oppositional defiant disorder shares with disruptive mood dysregulation disorder the symptoms of chronic negative mood and temper outbursts. However, the severity, frequency, and chronicity of temper outbursts are more severe in individuals with disruptive mood dysregulation disorder than in those with oppositional defiant disorder. Thus, only a minority of children and adolescents whose symptoms meet criteria for oppositional defiant disorder would also be diagnosed with disruptive mood dysregulation disorder. When the mood disturbance is severe enough to meet criteria for disruptive mood dysregulation disorder, a diagnosis of oppositional defiant disorder is not given, even if all criteria for oppositional defiant disorder are met.

Intermittent explosive disorder. Intermittent explosive disorder also involves high rates of anger. However, individuals with this disorder show serious aggression toward others that is not part of the definition of oppositional defiant disorder.

Intellectual disability (intellectual developmental disorder). In individuals with intellectual disability, a diagnosis of oppositional defiant disorder is given only if the oppositional behavior is markedly greater than is commonly observed among individuals of comparable mental age and with comparable severity of intellectual disability.

Language disorder. Oppositional defiant disorder must also be distinguished from a failure to follow directions that is the result of impaired language comprehension (e.g., hearing loss).

Social anxiety disorder (social phobia). Oppositional defiant disorder must also be distinguished from defiance due to fear of negative evaluation associated with social anxiety disorder.

Comorbidity

Rates of oppositional defiant disorder are much higher in samples of children, adolescents, and adults with ADHD, and this may be the result of shared temperamental risk factors. Also, oppositional defiant disorder often precedes conduct disorder, although this appears to be most common in children with the childhood-onset subtype. Individuals with oppositional defiant disorder are also at increased risk for anxiety disorders and major depressive disorder, and this seems largely attributable to the presence of the angry-irritable mood symptoms. Adolescents and adults with oppositional defiant disorder also show a higher rate of substance use disorders, although it is unclear if this association is independent of the comorbidity with conduct disorder.

Intermittent Explosive Disorder

Diagnostic Criteria **312.34 (F63.81)**

A. Recurrent behavioral outbursts representing a failure to control aggressive impulses as manifested by either of the following:
   1. Verbal aggression (e.g., temper tantrums, tirades, verbal arguments or fights) or physical aggression toward property, animals, or other individuals, occurring twice weekly, on average, for a period of 3 months. The physical aggression does not result in damage or destruction of property and does not result in physical injury to animals or other individuals.
   2. Three behavioral outbursts involving damage or destruction of property and/or physical assault involving physical injury against animals or other individuals occurring within a 12-month period.

B. The magnitude of aggressiveness expressed during the recurrent outbursts is grossly out of proportion to the provocation or to any precipitating psychosocial stressors.

C. The recurrent aggressive outbursts are not premeditated (i.e., they are impulsive and/or anger-based) and are not committed to achieve some tangible objective (e.g., money, power, intimidation).

D. The recurrent aggressive outbursts cause either marked distress in the individual or impairment in occupational or interpersonal functioning, or are associated with financial or legal consequences.

E. Chronological age is at least 6 years (or equivalent developmental level).

F. The recurrent aggressive outbursts are not better explained by another mental disorder (e.g., major depressive disorder, bipolar disorder, disruptive mood dysregulation disorder, a psychotic disorder, antisocial personality disorder, borderline personality disorder) and are not attributable to another medical condition (e.g., head trauma, Alzheimer's disease) or to the physiological effects of a substance (e.g., a drug of abuse, a medication). For children ages 6–18 years, aggressive behavior that occurs as part of an adjustment disorder should not be considered for this diagnosis.

Note: This diagnosis can be made in addition to the diagnosis of attention-deficit/hyperactivity disorder, conduct disorder, oppositional defiant disorder, or autism spectrum disorder when recurrent impulsive aggressive outbursts are in excess of those usually seen in these disorders and warrant independent clinical attention.

Diagnostic Features

The impulsive (or anger-based) aggressive outbursts in intermittent explosive disorder have a rapid onset and, typically, little or no prodromal period. Outbursts typically last for less