DSM-5 to DSM-5-TR Modifications to Attention-Deficit/Hyperactivity Disorder (ADHD) Chapter [APA], 2022		
Sub-Section of Criteria	**DSM-5 Wording/Removed Text** Reprinted with permission from the Diagnostic and Statistical Manual of Mental Disorders: DSM-5-TR, p. 68-70	**DSM-5-TR Updated Wording/Added Text**
Diagnostic Codes	Combined Presentation: 314.01 (F90.2) Predominantly Inattentive: 314.00 (F90.0) Predominantly Hyperactive/ Impulsive: 314.01 (F90.1)	Combined Presentation: F90.2 Predominantly Inattentive: F90.0 Predominantly Hyperactive/Impulsive: F90.1
Diagnostic Features	Lack of persistence	Failing to follow through on instructions or finishing work or chores
		ADHD cannot be diagnosed in the absence of any symptoms prior to age 12. When symptoms of what appear to be ADHD first occur after age 13, they are more likely to be explained by another mental disorder or to represent the cognitive effects of substance use.
Associated Features Supporting Diagnosis	*Mild* delays in language, motor, or social development are not specific to ADHD but often co-occur.	Delays in language, motor, or social development are not specific to ADHD but often co-occur.
	Associated features may include low frustration tolerance, irritability, or mood lability.	Emotional dysregulation or emotional impulsivity commonly occurs in children and adults with ADHD. Individuals with ADHD self-report and are described by others as being quick to anger, easily frustrated, and overactive emotionally.
	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.	Individuals with ADHD may exhibit neurocognitive deficits in a variety of areas, including working memory, set shifting, reaction time variability, response inhibition, vigilance, and planning/ organization.
	By early adulthood, ADHD is associated with an increased risk of suicide attempt, primarily when comorbid with mood, conduct, or substance use disorder. No biological marker is diagnostic for ADHD. As a group, compared with peers, children with ADHD display increased slow wave electroencephalogram, 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.	Although ADHD is not associated with specific physical features, rates of minor physical anomalies (e.g., hypertelorism, highly arched palate, low-set ears) may be 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].) Children with neurodevelopmental disorders with a known cause (e.g., fragile X syndrome, 22q11 deletion syndrome) may often also have symptoms of inattention and impulsivity/hyperactivity; they should receive an ADHD diagnosis if their symptoms meet the full criteria for the disorder.

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Prevalence	Population surveys suggest that ADHD occurs in most cultures in about 5% of children and about 2.5% of adults.	Population surveys suggest that ADHD occurs worldwide in about 7.2% of children; however, cross-national prevalence ranges widely, from 0.1% to 10.2% of children and adolescents. Prevalence is higher in special populations such as foster children or correctional settings. In a cross-national meta-analysis, ADHD occurred in 2.5% of adults.
Environmental	Very low birth weight (less than 1,500 grams) conveys a two- to three-fold 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.	Very low birth weight and degree of prematurity convey a greater risk for ADHD; *the more extreme the low weight, the greater the risk. Prenatal exposure to smoking is associated with ADHD even after controlling for parental psychiatric history and socioeconomic status.*
	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.	Neurotoxin exposure (e.g., lead), infections (e.g., encephalitis), and alcohol exposure in utero have 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.	The heritability of ADHD is approximately 74%. Large-scale genome-wide association studies (GWAS) have identified a number of loci enriched in evolutionarily constrained genomic regions and loss-of-function genes as well as around brain-expressed regulatory regions. There is no single gene for ADHD.
	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].)	ADHD is elevated in individuals with idiopathic epilepsy.

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Culture-Related Diagnostic Features	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.	Differences in ADHD prevalence across regions appear attributable mainly to different diagnostic procedures and methodological practices, including using different diagnostic interviews and differences in whether functional impairment was required and, if so, how it was defined. Prevalence is also affected by cultural variation in attitudes toward behavioral norms and expectations of children and youth in different social contexts, as well as cultural differences in interpretations of children's behaviors by parents and teachers, including differences by gender.
	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.	White populations. Under detection may result from mislabeling of ADHD symptoms as oppositional or disruptive in socially oppressed ethnic or racialized groups because of explicit or implicit clinician bias, leading to overdiagnosis of disruptive disorders. Higher prevalence in non-Latinx White youth may also be influenced by greater parental demand for diagnosis of behaviors seen as ADHD-related. Informant symptom ratings may be influenced by the cultural background of the child and the informant, suggesting that culturally competent diagnostic practices are relevant in assessing ADHD.
Gender-Related Diagnostic Issues	Gender-Related Diagnostic Issues	Sex- and Gender Related Diagnostic Issues
Sex- and Gender Related Diagnostic Issues		Sex differences in ADHD symptom severity may be due to differing genetic and cognitive liabilities between sexes.

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Added "Diagnostic Markers"		No biological marker is diagnostic for ADHD. Although ADHD has been associated with elevated power of slow waves (4–7 Hz "theta") as well as decreased power of fast waves (14–30 Hz "beta"), a later review found no differences in theta or beta power in either children or adults with ADHD relative to control subjects. Although some neuroimaging studies have shown differences in children with ADHD compared with control subjects, meta- analysis of all neuroimaging studies do not show differences between individuals with ADHD and control subjects. This likely is due to differences in diagnostic criteria, sample size, task used, and technical aspects of the neuroimaging technique. Until these issues are resolved, no form of neuroimaging can be used for diagnosis of ADHD.
Added "Association with Suicidal Thoughts or Behavior"		ADHD is a risk factor for suicidal ideation and behavior in children. Similarly, in adulthood, ADHD is associated with an increased risk of suicide attempt, when comorbid with mood, conduct, or substance use disorders, even after controlling for comorbidity. Suicidal thoughts are also more common in ADHD populations than in non- ADHD control subjects. ADHD predicted persistence of suicidal thoughts in U.S. Army soldiers.
Functional Consequences of Attention- Deficit/ Hyperactivity Disorder	Social rejection removed from association	
		Elevated likelihood of hypertension among individuals with ADHD
		Individuals with ADHD have lower self- esteem relative to peers without ADHD.
Specific Learning Disorder	However, inattention in individuals with a specific learning disorder who do not have ADHD is not impairing outside of academic work. (removed)	
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	ADHD shares symptoms of inattention with anxiety disorders. Individuals with ADHD are inattentive because of their *preferential engagement with novel and stimulating activities or preoccupation with enjoyable activities.*

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Posttraumatic Stress Disorder (added)		Concentration difficulties associated with posttraumatic stress disorder (PTSD) may be misdiagnosed in children as ADHD. Children younger than 6 years often manifest PTSD in nonspecific symptoms such as restlessness, irritability, inattention, and poor concentration, which can mimic ADHD. Parents may also minimize their children's trauma-related symptoms, and teachers and other caregivers are often unaware of the child's exposure to traumatic events. A comprehensive assessment of past exposure to traumatic events can rule out PTSD.
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*	Individuals with bipolar disorder may have increased activity, poor concentration, and increased impulsivity, but these features are episodic, *unlike ADHD, in which the symptoms are persistent.*
	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.	Children with ADHD may show significant changes in mood within the same day; such lability is distinct from a manic or hypomanic episode, which must last 4 or more days to be a clinical indicator of bipolar disorder, even in children.
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.	While impairment in complex attention may be one of the affected cognitive domains in a neurocognitive disorder, it must represent a decline from a previous level of performance to justify a diagnosis of major or mild neurocognitive disorder. Moreover, major or mild neurocognitive disorder typically has its onset in adulthood. In contrast, the inattention in ADHD must have been present prior to age 12 and does not represent a decline from previous functioning.

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Comorbidity	In clinical settings, comorbid disorders are frequent in individuals whose symptoms meet criteria for ADHD.	Although ADHD is more common in males, females with ADHD have higher rates of a number of comorbid disorders, particularly oppositional defiant disorder, autism spectrum disorder, and personality and substance use disorders.
	Specific learning disorder commonly co- occurs with ADHD. Intermittent explosive disorder occurs in a minority of adults with ADHD, but at rates above population levels.	
	Other disorders that may co-occur with ADHD include obsessive-compulsive disorder, tic disorders, and autism spectrum disorder.	ADHD may co-occur in variable symptom profiles with other neurodevelopmental disorders, including specific learning disorder, autism spectrum disorder, intellectual developmental disorder, language disorders, developmental coordination disorder, and tic disorders.
		Comorbid sleep disorders in ADHD are associated with daytime impairments in cognition (e.g., inattention). Many individuals with ADHD report daytime sleepiness that may meet criteria for hypersomnolence disorder. One quarter to one-half of individuals with ADHD report sleep difficulties; studies have shown an association of ADHD with insomnia, circadian rhythm sleep-wake disorder, sleep- disordered breathing, and restless legs syndrome. Individuals with ADHD have been found to have elevated rates of a number of medical conditions, particularly allergy and autoimmune disorders, as well as epilepsy

References for Appendix 1.1: DSM-5 to DSM-5-TR Modifications to Attention-Deficit/Hyperactivity Disorder (ADHD) Chapter [APA], 2022

American Psychiatric Association (Ed.). (2013). *Diagnostic and statistical manual of mental disorders:* DSM-5 (Fifth edition). American Psychiatric Association Publishing.

American Psychiatric Association (Ed.). (2022). *Diagnostic and statistical manual of mental disorders: DSM-5-TR* (Fifth edition, text revision). American Psychiatric Association Publishing.