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Adult ADHD: Underdiagnosis of a Treatable Condition

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Abstract
Attention-deficit/hyperactivity disorder is a common neurodevelopmental disorder, well-characterized within the pediatric population and representing one of the most prevalent mental conditions among children and adolescents. Once diagnosed, pharmacologic and psychosocial therapies can reduce symptomatic expression and functional impairment. Traditionally thought to resolve by young adulthood, it is now recognized that ADHD persists into adulthood. Many of these individuals were never evaluated for ADHD as children and remain undiagnosed as adults, continuing to struggle with symptoms. It is common to develop compensatory strategies geared at mitigating the disruptive effects of ADHD. In this article, we provide an overview of adult ADHD, review changes to DSM-5 diagnostic criteria meant to facilitate diagnosis of ADHD during adulthood, and discuss various factors that serve as barriers to accurate detection of adult ADHD.

Keywords Adult ADHD · DSM-5 ADHD · ADHD compensatory strategies

Clinical Vignette

Carlos, a 39-year-old IT systems analyst with no psychiatric history, was referred for mental health assessment by his PCP during the COVID-19 pandemic due to complaints of anxiety, poor focus, trouble staying on task, and increased difficulty in keeping up with his workload. His PCP was contemplating initiating antidepressant treatment, concerned that the stress of the pandemic was causing emotional symptoms that were beginning to impact his ability to work, but wanted a comprehensive mental health assessment prior to initiating any therapy.

Carlos reported to be in his usual state of health until shortly after the pandemic started. He described stressors and concerns that were experienced by many during that time, primarily centered around maintaining a safe environment for himself, his family, and his elderly parents. He was also concerned regarding the impact the pandemic would have on his job and the family's financial status. Carlos reported overseeing IT operations for a large healthcare organization (as it would turn out, activity at work remained steady and constant throughout the pandemic and the family never experienced financial hardship). Early into the pandemic, Carlos transitioned to working remotely from home, which made him feel more at ease regarding exposure to COVID-19. Neither he, his wife nor his two children had contracted COVID-19 during that time.

Carlos' medical history indicated borderline hypercholesterolemia, controlled with diet and exercise. Current medications were vitamins and fish oil. He denied tobacco, excessive alcohol, or recreational drug use. Psychiatric history was negative for episodes of anxiety, depression, or mania/hypomania, and he denied prior psychotropic or psychotherapeutic treatment history. Family medical and psychiatric history indicated cerebrovascular disease in father and ADHD in son; he denied any incidence of familial mood or anxiety disorders. Psychosocial history revealed that Carlos had been born and raised in the US to parents who had emigrated here from Latin America. He obtained a master's degree in information systems and had been with the same healthcare organization for 12 years. He had been married for 10 years and lived with his wife and two children, ages 8 and 6.

In obtaining detailed history regarding onset and emergence of symptoms, Carlos described that shortly after he transitioned to working from home, he began having difficulty with focus, distractibility, and staying on task. If interrupted while completing a specific task, he had difficulty resuming the initial the task and it would be left undone. He found himself procrastinating with increased frequency. After missing two deadlines, he had a meeting with the company's CFO who questioned whether he was under excessive stress and warned

that he might be placed on probation and some of his projects reassigned to a different team. He became increasingly anxious and mildly depressed regarding his job security.

In reviewing his remote history, Carlos admitted that this was not the first time that he had difficulty managing the demands of work or school. Although there were no objective signs of significant academic or occupational dysfunction throughout his life, Carlos described a chronic pattern of "struggling to stay on top" of his assignments. He had been placed in the gifted program while in middle school, but Carlos believed that he always underperformed academically (despite never receiving less than a B in school) and described having had to work harder than his classmates to obtain his grades. As a child, he had been restless, hyperkinetic, and tended to talk excessively, both at home and in the classroom. These hyperactive symptoms seemed to have waned and gradually abated as he entered his late teens. Throughout college and graduate school, he described a pattern of difficulty staying focused and on task, a tendency of cramming for exams the night before ("pulling many all-nighters"), and procrastinating until the last moment to complete assignments. At work, he credits his energy, creativity, people skills, and ability to problem-solve as having allowed him to move up in the organization, but he states his team is aware of his difficulty with focus, organization, multi-tasking, and meeting deadlines—and often "covers" for him. Over the years, he had developed various strategies and mechanisms to compensate for these shortcomings. Although his symptoms never produced frank impairment, it is clear that they reduced the quality and efficiency of his work.

Following a comprehensive assessment, Carlos was diagnosed with attention-deficit/hyperactive disorder, predominantly inattentive presentation, mild range of severity. He had never been evaluated for ADHD as a child, even though retrospective review indicated that he met criteria for combined presentation during that time. As is often the case, the hyperactive and impulsive behaviors waned during teenage neurodevelopment, with only the inattentive symptoms remaining active. While he had learned to compensate at work over the years, the transition to working from home introduced increased opportunities for distraction, removed his support system, and overwhelmed his traditional compensatory strategies, which caused the disorder to become more prominent. Following our consultation and psychoeducational interventions, he was referred for medication evaluation and was started on stimulant treatment (the same medication that his son had been prescribed and had shown a therapeutic response); he has demonstrated a dramatic reduction in symptoms and increased efficiency in work performance.

Background and Challenges

Attention-deficit/hyperactivity disorder (ADHD) is one of the most common neurobehavioral disorders of childhood and can profoundly affect children's academic achievement, interpersonal interactions, and sense of self and well-being (Wolraich et al., 2019). Population surveys suggest that ADHD occurs worldwide in about 7.2% of children; however, cross-national prevalence rates demonstrate wide variability, from 0.1% to 10.2% of children and adolescents, possibly due to differences in diagnostic or impairment criteria (American Psychiatric Association [APA] 2022; Turgay et al., 2012). The syndrome was traditionally thought to resolve along the gradual trajectory of brain development during adolescence and into young adulthood (Kasparek et al., 2015; Zalsman & Shilton, 2016). However, it is now generally accepted that ADHD persists into adulthood. Approximately one-half to two-thirds of children with ADHD continue to manifest symptoms of the disorder into adulthood (APA, 2022; Turgay et al., 2012). When accurately diagnosed, ADHD is a highly treatable disorder; however, ADHD frequently goes undetected in adults, exerting adverse impact on psychosocial functioning and serving as a risk factor for the development of other disorders, such as anxiety, depression, and substance use disorders.

Many adults with ADHD report struggling throughout life with a sense of underachievement and failure to realize their full potential. It is common to report developing compensatory strategies that prove effective in mitigating psychosocial disruption but paradoxically mask the presence of ADHD symptoms. Oftentimes, these adults seek mental health treatment due to depression or anxiety secondary to ADHD-related psychosocial struggles, or during transition periods when external changes overwhelm their typical compensatory mechanisms and they experience a decrement in their psychosocial efficiency. Interestingly, some adults present for evaluation of ADHD after their own children have gone through assessment for possible ADHD and they realize that they themselves have endured lifelong struggles with similar symptomatology. The purpose of this paper is to increase awareness and understanding of adult ADHD, review changes in our current diagnostic classification meant to increase recognition of ADHD in adulthood, and discuss some of the factors that serve to impede accurate detection.

Overview of ADHD

ADHD is a clinically heterogeneous disorder characterized by a persistent pattern of inattention-distractibility and/or hyperactivity-impulsivity that interferes with functioning or development (APA, 2022). Advances in the fields of genetics and neuroimaging have allowed for greater understanding of the etiology and pathophysiology of the disorder. Results from family and twin studies have demonstrated ADHD's high risk of inheritability. Pooled data from 20 extant twin studies estimate the heritability of ADHD to be 0.76 (Faraone et al., 2005), indicating

that approximately 76% of the etiologic contribution of the disorder is genetic (compared with depression [0.39 heritability] and generalized anxiety disorder [0.32 heritability]; Spencer et al., 2002). The genetic architecture of ADHD is believed to be multifactorial, with no less than seven genes having shown statistically significant evidence of contribution to the development of the disorder through mediation of basic neurodevelopmental processes (cell division, adhesion, neuronal migration, and plasticity) along specific neuroanatomical tracts and within specific neurotransmitter systems (Faraone et al., 2005; Franke et al., 2009; Purper-Ouakil et al., 2011).

The neurobiology of ADHD is complex and involves developmental, structural, and functional alterations across multiple anatomical sites. Several comprehensive reviews exist, which summarize the results from genetic, imaging, and neurobiologic studies (see Bonvicini et al., 2016; Kasparek et al., 2015; Purper-Ouakil et al., 2011; Rege, 2022; Tripp & Wickens, 2009). ADHD has traditionally been characterized as dysfunction in the frontostriatal pathway, particularly the prefrontal cortex (PFC), the last region of the brain to mature (Arnsten , 2009); however, studies have indicated a broader range of alterations extending beyond the frontal cortex and the corpus striatum of the basal ganglia. The most consistent findings in ADHD are an overall reduction in total brain size and in morphologic dimensions of several specific neuroanatomical regions, such as the caudate nucleus, the PFC white matter, the parietal cortex, the anterior cingulate, the splenium of the corpus callosum and the cerebellar vermis (Kasparek et al., 2015; Tripp & Wickens, 2009). Imaging studies have indicated that many of these alterations can persist into adulthood (see Cortese et al., 2012), although there may be normalization in specific regions (e.g., the caudate nucleus), which may underlie the characteristic resolution of hyperkinesis demonstrated by some individuals with ADHD as they enter into adulthood (Kasparek et al., 2015). Giedd & Rapoport (2010) have made the point that children with ADHD who demonstrate clinical remission during adolescence and do not go on to manifest adult ADHD may show a developmental brain trajectory that has gradually converged with that of typical development.

In addition to neuroanatomical alterations, the neurotransmitters dopamine (DA) and norepinephrine (NE) have also been implicated in the pathophysiology of ADHD. The PFC is highly dependent on these two catecholamines for optimal functioning. Several of the candidate genes that have been identified in ADHD mediate the expression of DA and NE receptor density and functionality (Bonvicini et al., 2016; Rege, 2022). Arnsten (2009) noted that the regulatory functions of the PFC are highly dependent on its neurochemical state, requiring the correct balance of DA and NE to produce maximal PFC activation; neurotransmitter levels that are either insufficiently low or excessively elevated can both result in dysfunction in attention, alertness, and capacity for inhibition. Therapeutic doses of various classes of ADHD medications, most notably the stimulants agents, can facilitate optimal DA and NE tone in the PFC, thereby enhancing neurotransmission and producing clinical improvement. Moreover, there is data indicating that stimulant treatment has a normalizing influence on cortical, subcortical, thalamic and white matter development, although it remains unclear if this normalization represents direct medication-effect on neuronal plasticity or the result of enhanced interaction with the environment produced by efficacious treatment (Giedd & Rapoport, 2010).

The neuropsychological impact of anomalies within these various regions and systems is compromised executive functioning (EF), which is felt to underlie the characteristic clinical manifestations of ADHD (Tripp & Wickens, 2009). EF refers to a set of cognitive processes mediating self-regulation and interaction with the environment, and includes components such as behavioral inhibition; working memory; regulation of alertness and attention; and sustaining and shifting attention to tasks (Barkley & Brown, 2008). Overall, EF involves the capability to organize, initiate, and maintain goal-directed behavior (Barkley & Brown, 2008). Motivation—specifically as it relates to reinforcement, delay of gratification, and perseverance of behavior under adverse conditions—is oftentimes included within ADHD's profile of compromise (Tripp & Wickens, 2009). Results from functional imaging studies have furthered our understanding of how select pathways are responsible for various components of EF. The PFC is critical for EF and mediates organization, attention, planning for the future, and sustained concentration under conditions perceived as "boring" (Arnsten, 2009). The right inferior PFC is specialized for behavioral inhibition via projections to the motor and premotor cortices, basal ganglia, and cerebellum, while the ventromedial PFC regulates emotions and inhibition of aggressive impulses through projections to the amygdala, hypothalamus, and nucleus accumbens (Rege, 2022). Compromised EF not only impairs psychosocial functioning in environments where attention, behavioral inhibition, and task initiation and maintenance are critical for success (e.g., work and school settings), but also increases the risk for development of mood, anxiety, impulse control, and substance use disorders. Observations that the anatomical and functional dysregulation that compromises EF can persist across the lifespan and produce ongoing psychosocial impairment has prompted the notion that ADHD should be viewed as a chronic condition for many individuals (Turgay et al., 2012).

Diagnostic Criteria and DSM Changes

As with the majority of DSM disorders, ADHD is a clinical diagnosis that is made by gathering history to document the presence and duration of symptoms, determine the degree

of psychosocial distress or impairment, and exclude other conditions (e.g., medical, substance-related, or psychiatric) that may be producing the individual's clinical presentation. To date, there are no genetic, biochemical, or psychometric tests that can diagnose ADHD. Initial assessment of ADHD may include collaboration with medical staff to pursue a thorough work-up, including blood work and possibly an electroencephalogram, to detect a medical condition that might be contributing to the individual's presenting symptoms. With regard to the use of psychometric instruments, the point has been made that standardized cognitive tests may be most useful in the diagnostic assessment of ADHD when integrated into a broader clinical assessment of the individual's symptomatology and family history (Nikolas et al., 2019). In the diagnostic assessment of ADHD, psychometric examination may be helpful in providing an overall level of intellectual functioning; detecting the presence of a learning disability; and documenting any objective signs of attentional, organization, or executive dysfunction—but to be clear, many individuals that have a negative psychometric study will nonetheless go on to be accurately diagnosed with ADHD by virtue of their clinical history.

In many ways, the evolution of the ADHD construct into its current characterization in DSM-5 reflects more than two decades of increased awareness that ADHD is not limited to childhood and adolescence. ADHD is certainly not a new clinical entity. As historical perspective, the first formal medical description of a disorder similar to ADHD appeared in the 1798 work of Scottish physician Sir Alexander Crichton "Attention and its Diseases" (Lange et al., 2010). In 1902, British physician Sir Frederick Still is credited with describing the "scientific starting point" of ADHD when he described a condition involving an "abnormal defect of moral control" in children (Lange et al., 2010). Observations of children following the 1917 encephalitis epidemic popularized the view that attentional and disruptive behavioral symptoms were the result of neurologic insult, a notion reflected in the emergence of the term "minimal brain dysfunction" to describe ADHD (Lange et al., 2010; Romeo, 2021). In 1968, the ADHD construct first appeared in the DSM's second edition as "hyperkinetic reaction of childhood," which featured the disorder's excessive locomotor activity (there was no mention of an ADHD syndrome in the first edition of the DSM published in 1952). For the third edition of DSM (DSM-III) in 1980, the criteria were markedly reconceptualized to capture attention and impulsivity, and the disorder was renamed "attention deficit disorder with and without hyperactivity" (Epstein & Loren, 2013). The current appellation, attention-deficit/hyperactivity disorder, first appeared in the 1987 revision to DSM-III. The designation of three subtypes (predominantly inattentive type, predominantly hyperactive-impulsive type, and combined type) were added in the fourth edition of DSM in 1994, as were the 18 symptoms (9 of inattention and 9 of hyperactivity/impulsivity), which remain unchanged in the DSM-5.

The representation of ADHD in DSM-5, while not fundamentally changing the concept of the disorder, more accurately describes the phenomenology of affected adults (Zalsman & Shilton, 2016). Prior to DSM-5, criteria focused on detection of ADHD in children and adolescents; field trials employed to develop criteria included only children and adolescents up to age 17 (Lahey et al., 1994), bringing into question the utility of the criteria in diagnosing adults (Fischer & Barkley, 2007). Consequently, the following changes in DSM-5 reflect how the criteria have been adapted with the explicit objective of facilitating application to adults presenting with ADHD symptomatology (Ginsberg et al., 2014; Zalsman & Shilton, 2016):

1. ADHD now appears in the DSM-5 Neurodevelopmental Disorders section (rather than its previous listing under Disorders Usually Diagnosed in Infancy, Childhood or Adolescence).
2. While the 18 core symptoms divided into two symptom domains (inattention and hyperactivity/impulsivity) remain the same as in DSM-IV, the symptom threshold for either domain has now been lowered when assessing individuals aged 17 and older, requiring only 5 of 9 criteria (rather than 6 of 9, which was retained when applied to children and adolescents 16 and younger).
3. While the wording of the 18 core symptoms remains unchanged from DSM-IV, DSM-5 has included illustrative examples of how each particular item may manifest itself in adults in order to facilitate application across the lifespan.
4. The age-of-onset and degree of impairment in Criteria B were changed, with symptoms no longer needing to cause impairment but merely needing to be present. Age-of-onset was changed based on observations that children who had symptoms at age 7 demonstrated persistence of symptoms at age 12, and that only 50% of adults recall onset of symptoms by age 7 versus 95% recall onset of symptoms by age 12 (Barkley & Brown, 2008; Zalsman & Shilton, 2016).
5. The softening in degree of impairment is again noted in a change to Criteria D. This change most likely reflects observations that some individuals with ADHD can utilize strategies and support to offset disruption and avoid frank impairment.
6. A subtle change in nomenclature from inattentive or hyperactive/impulsive *type* to inattentive or hyperactive/impulsive *presentation* reflects evidence that symptomatic manifestation may be fluid across the lifespan, rather than stable traits (Epstein & Loren, 2013). For example, an individual may have met criteria for combined presentation as a child but meets criteria for predominantly inattentive presentation as an adult.

7. Severity and remission specifiers are now available in DSM-5. The severity specifier allows for a rating from mild (indicating that few, if any, symptoms required to make the diagnosis are present and result in no more than minor impairment in functioning) to more severe presentations (indicating the presence of excess symptoms above the threshold that are producing moderate to marked impairment). The new partial remission specifier allows for the diagnosis to be given when full criteria were previously met, but symptoms have fallen below the criteria threshold for at least 6 months yet are still producing some degree of psychosocial impairment. We believe these specifiers will serve to increase awareness of ADHD existing along a continuum of severity and demonstrating some fluidity across the life span, which may enhance detection of milder or subthreshold presentations in adulthood.

These changes to ADHD in DSM-5 are perhaps the most substantial since 1980 and are in response to the overwhelming literature that supports the potentially lifelong persistence of the disorder, as well as fluctuations in expression of ADHD across the lifespan in response to changing developmental tasks and external demands. Data indicate that individuals tend to demonstrate fewer ADHD symptoms with age, and the core symptoms—impulsivity, hyperactivity, and inattention—are manifested differently in adulthood than in childhood and generally expressed in more subtle ways (Spencer et al., 2002; Zalsman & Shilton, 2016). The general consensus within the field is that the manner in which criteria were developed and characterized prior to what we now have in DSM-5 criteria was too stringent and would miss many adults with ADHD (Barkley & Brown, 2008).

Finally, a point needs to be made regarding the controversial notion of adult-onset ADHD. Based on observations from at least two studies in which some adults meeting criteria for ADHD failed to demonstrate a childhood history of the disorder (see Caye et al., 2016; Moffitt et al., 2015), these researchers suggests that there may be a separate variant of adult ADHD that is not merely a continuation of neurodevelopment ADHD into adulthood. We agree with Faraone & Biederman (2016) that this conclusion seems premature, and that these results may reflect methodological issues involving data collection or the potential for recall bias on the part of individuals in these samples (it is well established that adult recollection of childhood symptoms tends to be unreliable; APA, 2022). Certainly, we have all encountered adults in clinical practice presenting with cognitive, behavioral, and psychomotor symptoms (e.g., inattention, poor concentration, restlessness, agitation, impulsivity, low frustration tolerance) that may mimic ADHD, but whose recent onset and lack of childhood symptoms following thorough and comprehensive review of early history (ideally including history obtained from a collateral informant) indicate that this presentation may be due to another disorder. Should these individuals be misdiagnosed with ADHD, it is unclear how they will respond to traditional ADHD treatment. Based on our clinical experience and our review of the literature, we agree with the position expressed in DSM-5: True ADHD begins in childhood and should not be diagnosed in the absence of any symptoms prior to age 12. When symptoms of what appears to be ADHD first occur after age 13, they are more likely explained by another mental disorder or represent the cognitive effects of a substance (APA, 2022).

Adult ADHD: Clinical Presentation and Underdetection

With growing awareness of the persistence of ADHD into adulthood, attention has shifted to seeking greater understanding of possible differences in clinical presentation of adult ADHD and identifying factors that may result in its underdetection. ADHD is a highly treatable disorder, demonstrating a robust therapeutic response to several classes of pharmacologic agents and various psychotherapeutic treatment modalities (see Boland et al., 2020; Fullen et al., 2020; Knouse et al., 2017). The availability of effective treatments for symptomatic reduction and enhanced functional outcomes should bring a greater sense of urgency to the risk of missing this disorder in clinical practice.

It is estimated that 2.5% to 4.4% of adults meet criteria for ADHD (APA, 2022; Kessler et al., 2006). However, fewer than 20% of ADHD adults are accurately diagnosed and treated (Barkley & Brown, 2008; Ginsberg et al., 2014), leaving these individuals vulnerable to experience ongoing social, academic, and occupational difficulties, as well as at risk for developing comorbid anxiety, depression, or substance use disorders. As noted above, the neurobiological anomalies identified in children with ADHD have also been seen in adults with ADHD, implying that the persistent psychosocial difficulties are most likely mediated by ongoing EF compromise.

Faraone & Biederman (2016) posited that some of these individuals may have been subthreshold for full criteria of ADHD while younger, possibly due to insufficient number of symptoms or ambiguity with application of impairment criteria. This discrepancy is consistent with our experience of some ADHD adults who deny overt signs of objective academic impairment when younger, but report having had to put in greater effort than their classmates or having received external support from parents and/or teachers in order to attain academic success. In these subthreshold cases, onset of emergence of symptoms and the point at which impairment threshold is met might be separated by years, particularly among bright individuals with supportive

and well-structured environments. This intellectual and social scaffolding—which essentially constitutes an external auxiliary EF system—would help youth compensate for ADHD symptoms early in life but leave them at risk for decompensation once that scaffolding is removed (Faraone & Biederman, 2016). The point should also be made that subthreshold conditions should not be viewed as benign; it is well established that subthreshold presentations involving other disorders such as depression and anxiety, which would technically receive unspecified or otherwise specified designation in our diagnostic rendering (APA, 2022), can nonetheless produce significant distress and psychosocial impairment.

In addition to subthreshold presentations impeding accurate detection of adult ADHD, it is also widely accepted that the clinical presentation of ADHD changes over time into adulthood (possibly reflecting normalization of development within specific neuroanatomical pathways). Overt signs of hyperactivity/impulsivity—typically thought of as the more visible and troubling aspects of ADHD—decline with age, while the constellation of inattentive symptoms continues largely unchanged (Turgay et al., 2012). Of the three ADHD subtypes, predominantly inattentive type is felt to be the most prevalent among adults, accounting for nearly half of all cases (Zalsman & Shilton, 2016). Consequently, while the more acute hyperactive and impulsive symptoms that may prompt those with ADHD to seek intervention abate with time, the more enduring symptoms of inattention are easier to offset via compensation.

Studies have shown that many adults with ADHD develop a variety of compensatory strategies to minimize the disruptive effects of inattention; these strategies often begin in childhood and are utilized independently of whether the individual has ever been diagnosed with ADHD (Canela et al., 2017; Kysow et al., 2016). A compensatory strategy includes three prerequisites: (a) a mismatch that occurs between a skill and an environment, (b) the individual must be aware of the mismatch, and (c) the individual makes an explicit decision to compensate for this mismatch (Merkt et al., 2015). Compensatory strategies typically reported by adults with ADHD can be grouped into various categories, such as social, organizational, attentional, motor, avoidance, external support, and psychopharmacologic (see Canela et al., 2017; Kysow et al., 2016); many individuals with ADHD report use of multiple strategies. In social settings, ADHD individuals report interpersonal difficulties due to inattention in conversation, fidgeting, forgetfulness, and/or impulsive behaviors. In order to adjust for this mismatch of skills and environment, these individuals report striving to be overly punctual, selecting social settings with larger groups to avoid standing out, and declining to participate in social activity requiring a high degree of commitment and frequent meetings. To compensate for organizational deficits, strategies include relying on electronic devices for reminders and timers, creating rigid structures that augment organization, and delegating tasks to others to minimize feeling overwhelmed.

Individuals report compensating for attentional difficulties by structuring their work environment to restrict extraneous stimuli either physically (e.g., working in an isolated room) or opting to work or study at night when there are fewer distractions. To compensate for motor deficits of restlessness or fidgeting, the most common strategy is to engage in physical activity (e.g., exercising prior to work, taking scheduled breaks at work to walk around) or to structure the work environment in a fashion that allows them to fidget (e.g., moving a foot or a leg) without causing distraction for others. Some individuals have consciously developed strategies of simply avoiding a situation where ADHD symptoms will be more pronounced (e.g., waiting in a long line, going to a movie theatre) or having actively recruited the assistance of others to provide support via reminders or to inhibit them in social situations. Unfortunately, some individuals report pharmacologic compensation by use of various agents (e.g., tobacco, alcohol, cannabis, cocaine, excessive caffeine) that may otherwise have long-term deleterious health consequences. Although compensatory strategies provide scaffolding to offset disruption from compromised EF and augment psychosocial functioning, they nonetheless can mask detection of ADHD symptoms and delay appropriate treatment.

While life transitions can prove challenging for many of us, they are of particular clinical relevance to individuals with ADHD, specifically as they relate to external scaffolding and compensatory strategies. The significant transitional periods in life—from childhood to adolescence and then to adulthood—carry increased social, academic, and occupational demands, all of which represent more complex challenges for individuals with ADHD as they negotiate their interaction with the environment. Scaffolding and compensatory strategies that were present and effective at one stage of life may be dismantled or rendered ineffective during a transition to a more demanding stage (Faraone & Biederman, 2016). Aside from maturational and developmental transitions across the life span, academic and vocational transitions also represent potential periods of symptomatic exposure and functional derailment. It is not uncommon to assess a college freshman who is having "new-onset" academic difficulties, but whose careful review of history indicates that there was scaffolding provided by either parents or teachers in high school that provided structure that controlled for ADHD symptoms.

Similarly, we have assessed adults with undiagnosed ADHD who present following a promotion at work and then demonstrate "first-time" difficulties with time and task management. In-depth history may reveal that the

promotion requires increased EF demands for multi-tasking and self-regulation that overwhelmed the well-established compensatory strategies the individual had employed with reasonable success in their less challenging prior occupational role. The ADHD Life Transition Model (Turgay et al., 2012) represents a framework that was developed in response to the awareness of ADHD persistence into adulthood and recommends adopting a chronic illness approach to ADHD. Its objectives are to increase detection of adulthood ADHD by underscoring the changing symptom manifestations across the lifespan and to increase practitioners' acumen and diligence in recognition and management of ADHD individuals as they negotiate transition periods across the lifespan.

As challenging as transitions can be, they also represent opportunities for detection and intervention as they are the catalysts that prompt many adults with ADHD to seek treatment. However, various factors exist at this point that can mask accurate detection. Practitioners may not be well-versed as to the persistence and presentation of ADHD in adulthood, particularly subthreshold and mildly severe cases, and may not conduct a sufficiently in-depth review for presence of early symptoms. For example, we have heard practitioners base their exclusion of an ADHD diagnosis on the absence of objective academic impairment during childhood and adolescence (e.g., failing a class or a grade), rather than reviewing for "softer" signs of ADHD, such as the individual's sense of academic underachievement, inefficient studying habits, excessive time and effort expended to complete assignments, procrastination followed by "cramming," and the presence of scaffolding or accommodations. Weiss & Weiss (2004) suggested that a comprehensive assessment of ADHD may involve moving beyond strict adherence to the phrasing of the 18 items in DSM and posing questions such as the following: Did parents and/or teachers complain you were difficult? Were you ever labeled as having a learning disability? Did you need special help at school? Were you an underachiever? Was your performance at school variable or unpredictable? Do you have problems with rage attacks?

Conversely, even when ADHD adults are asked about their childhood, many of these individuals have limited retrospective recall of specifics regarding their school-age behavior, study habits, and classroom performance (Ginsberg et al., 2014; Miller et al., 2010). Given the high degree of symptom overlap between ADHD and other disorders, it is easy to understand how practitioners may misattribute ADHD behaviors to either mood or anxiety disorders. The most obvious is ascribing what appears to be new-onset concentration difficulties or restlessness to major depression or generalized anxiety disorder. Although mood dysregulation is not a formal component of DSM-5 ADHD criteria, many adults with ADHD report feeling overwhelmed, impatient, and experiencing difficulty in controlling intense emotions (Barkley & Brown, 2008); this may prompt the practitioner to start thinking bipolar disorder, particularly if the individual describes a pattern of impulsivity, or disorganized or poorly thought-out behavior.

Aside from symptom overlap, the issue of actual comorbidity is indeed a significant factor in preventing accurate detection of adulthood ADHD. ADHD is highly comorbid with major depressive disorder, anxiety disorder, bipolar disorder, and substance use disorders; the overall prevalence for ADHD was found to be 2- to sevenfold higher in individuals meeting criteria for those other DSM conditions (Kessler et al., 2006). The prevalence of comorbidity underscores the importance of assuming a broad approach when collecting history of present illness data and routinely screening all individuals for a wide range of psychiatric disorders. Oftentimes, the nature of the comorbid condition (e.g., tearfulness, suicidality, panic, mania, psychosocial dysfunction secondary to active substance abuse) may represent greater acuity than the ADHD symptoms and will then become the focus of assessment, potentially overshadowing the ADHD component.

Individuals presenting with psychiatric disorders that are comorbid with underlying ADHD report a higher degree of distress and psychosocial impairment and may not achieve remission of their mood, anxiety, or substance abuse symptoms until the ADHD is accurately detected and effectively managed (Newcorn et al., 2007; Weiss & Weiss, 2004). For individuals with ADHD and comorbid anxiety and/or depression, it is good practice to assess in detail to determine whether the anxiety and depression are independent of the ADHD symptoms (and may require specific treatment) or resultant from the social, academic, or occupational difficulties caused by ADHD. Many individuals experience worry, tension, irritability, sadness, apathy, guilt, and low self-esteem due to ADHD-related inefficiency, impairment, or under-achievement. In these cases, the improvement in performance and functioning produced by efficacious treatment of the ADHD can lead to amelioration of anxiety and depression without having to dispense additional, possibly unnecessary treatment. In any case, the point has been made that, given the high degree of symptom overlap and comorbidity with other DSM disorders, practitioners should be diligent in considering and screening for ADHD in every diagnostic assessment (Barkley & Brown, 2008).

Conclusion

As beneficial as the transition event can be in prompting the individual to seek assessment and treatment, it can paradoxically act as an impediment to detection of ADHD by misleading the practitioner to view the presenting symptoms as an adjustment reaction

to the stress inherent in the transition. We experienced this phenomenon frequently during the pandemic and recently reported on a small series of individuals, such as our case example presented in the opening, who were referred to our clinic by their primary care physicians due to cognitive and behavioral complaints that emerged following the COVID-19 outbreak presumably due to "pandemic-related stress." All patients reported onset of difficulties after the pandemic prompted a transition to working remotely from home (we excluded individuals who previously tested positive for COVID-19 to eliminate confounding effects from long-haul phenomena). As a group, their cognitive complaints included poor focus, distractibility, increased procrastination, difficulty staying on task, decreased capacity for multi-tasking, and difficulty resuming initial task if interrupted. All reported decreased efficiency and reduction from their typical work productivity, which led some patients to develop mild distress (e.g., anxiety, increased worry, some dysphoria, and reduced sleep).

A key clinical characteristic in our sample was that cognitive symptoms seemed to pre-date the emergence of emotional symptoms. These individuals described long-term use of various compensatory strategies geared at off-setting their ADHD symptoms; upon transitioning to working remotely from home, all described a break-down or difficulty in engaging in their typical strategies (Rivas-Vazquez et al., 2022). As a group, they reported the transition overwhelmed their ability to compensate for symptoms and maintain their pre-pandemic level of performance, as evidenced by an increase in careless mistakes, decreased productivity, or missed deadlines. Reasons for this breakdown included high levels of extraneous distractions in the home environment, inability to derive external support (e.g., from an assistant), difficulty in modifying the environment to reduce distractions, and inability to engage in a routine they had developed to minimize boredom or waning attention over a period of time. Once detected and accurately diagnosed, treatment of ADHD was initiated and patients reported reduced symptoms and improved psychosocial functioning (Rivas-Vazquez et al., 2022). We would encourage practitioners to be cognizant of and routinely screen for the presence of ADHD during the course of conducting their diagnostic assessments with all adults.

Key Clinical Takeaways

1. Practitioners should be aware of how compensatory strategies can mask detection of adult ADHD, and how transition events across the lifespan often serve as the precipitating event for functional disruption that prompts the individual to seek treatment.
2. Practitioners should thoroughly review childhood and adolescent history, including that provided by a collateral informant, to confirm the presence of several inattentive or hyperactive-impulsive symptoms prior to age 12 years.
3. Practitioners should be cognizant of and routinely screen for the presence of ADHD during the course of conducting their diagnostic assessments with all adults.

Declarations

Conflicts of Interest We have no known conflicts of interest, external funding or other disclosures to make. No previous publications.

References

American Psychiatric Association. (2022). *Diagnostic and statistical manual of mental disorders* (5th ed., text rev.). https://doi.org/10.1176/appi.books.9780890425787

Arnsten, A. F. (2009). The emerging neurobiology of attention deficit hyperactivity disorder: the key role of the prefrontal association cortex. *The Journal of pediatrics, 154*(5), I.

Barkley, R. A., & Brown, T. E. (2008). Unrecognized attention-deficit/hyperactivity disorder in adults presenting with other psychiatric disorders. *CNS spectrums, 13*(11), 977-984.

Boland, H., DiSalvo, M., Fried, R., Woodworth, K. Y., Wilens, T., Faraone, S. V., & Biederman, J. (2020). A literature review and meta-analysis on the effects of ADHD medications on functional outcomes. *Journal of Psychiatric Research, 123*, 21-30.

Bonvicini, C., Faraone, S. V., & Scassellati, C. (2016). Attention-deficit hyperactivity disorder in adults: a systematic review and meta-analysis of genetic, pharmacogenetic and biochemical studies. *Molecular psychiatry, 21*(7), 872-884.

Canela, C., Buadze, A., Dube, A., Eich, D., & Liebrenz, M. (2017). Skills and compensation strategies in adult ADHD–A qualitative study. *PloS one, 12*(9), e0184964.

Caye, A., Rocha, T. B. M., Anselmi, L., Murray, J., Menezes, A. M., Barros, F. C., ... & Rohde, L. A. (2016). Attention-deficit/hyperactivity disorder trajectories from childhood to young adulthood: evidence from a birth cohort supporting a late-onset syndrome. *JAMA psychiatry, 73*(7), 705–712.

Cortese, S., Kelly, C., Chabernaud, C., Proal, E., Di Martino, A., Milham, M. P., & Castellanos, F. X. (2012). Toward systems neuroscience of ADHD: a meta-analysis of 55 fMRI studies. *American Journal of Psychiatry, 169*(10), 1038-1055.

Epstein, J. N., & Loren, R. E. (2013). Changes in the definition of ADHD in DSM-5: subtle but important. *Neuropsychiatry, 3*(5), 455.

Faraone, S. V., & Biederman, J. (2016). Can attention-deficit/hyperactivity disorder onset occur in adulthood? *JAMA psychiatry*, *73*(7), 655-656.

Faraone, S. V., Perlis, R. H., Doyle, A. E., Smoller, J. W., Goralnick, J. J., Holmgren, M. A., & Sklar, P. (2005). Molecular genetics of attention-deficit/hyperactivity disorder. *Biological psychiatry, 57*(11), 1313-1323.

Fischer, M., & Barkley, R. A. (2007). The persistence of ADHD into adulthood: (once again) it depends on whom you ask. *The ADHD report, 15*(4), 7-16.

Franke, B., Neale, B. M., & Faraone, S. V. (2009). Genome-wide association studies in ADHD. *Human genetics, 126*(1), 13-50.

Fullen, T., Jones, S. L., Emerson, L. M., & Adamou, M. (2020). Psychological treatments in adult ADHD: a systematic review. *Journal of Psychopathology and Behavioral Assessment, 42*(3), 500-518.

Giedd, J. N., & Rapoport, J. L. (2010). Structural MRI of pediatric brain development: what have we learned and where are we going*? Neuron, 67*(5), 728-734.

Ginsberg, Y., Quintero, J., Anand, E., Casillas, M., & Upadhyaya, H. P. (2014). Underdiagnosis of attention-deficit/hyperactivity disorder in adult patients: a review of the literature. *The primary care companion for CNS disorders*, *16*(3), 23591.

Kasparek, T., Theiner, P., & Filova, A. (2015). Neurobiology of ADHD from childhood to adulthood: findings of imaging methods. *Journal of Attention Disorders*, *19*(11), 931-943.

Kessler, R. C., Adler, L., Barkley, R., Biederman, J., Conners, C. K., Demler, O., ... & Zaslavsky, A. M. (2006). The prevalence and correlates of adult ADHD in the United States: results from the National Comorbidity Survey Replication. *American Journal of psychiatry*, *163*(4), 716–723.

Knouse, L. E., Teller, J., & Brooks, M. A. (2017). Meta-analysis of cognitive–behavioral treatments for adult ADHD. *Journal of consulting and clinical psychology*, *85*(7), 737.

Kysow, K., Park, J., & Johnston, C. (2016). The use of compensatory strategies in adults with ADHD symptoms. *ADHD Attention Deficit and Hyperactivity Disorders*, *9*(2), 73-88.

Lahey, B. B., Applegate, B., McBurnett, K., Biederman, J., Greenhill, L., Hynd, G. W., Barkley, R. A., Newcorn, J., Jensen, P., & Richters, J. (1994). DSM-IV field trials for attention deficit hyperactivity disorder in children and adolescents. *The American Journal of Psychiatry , 151* (11), 1673–1685. https://doi.org/10.1176/ajp.151.11.1673

Lange, K. W., Reichl, S., Lange, K. M., Tucha, L., & Tucha, O. (2010). The history of attention deficit hyperactivity disorder. *ADHD Attention Deficit and Hyperactivity Disorders*, *2*(4), 241-255.

Merkt, J., Reinelt, T., & Petermann, F. (2015). A framework of psychological compensation in attention deficit hyperactivity disorder. *Frontiers in psychology*, *6*, 1580.

Miller, C. J., Newcorn, J. H., & Halperin, J. M. (2010). Fading memories: retrospective recall inaccuracies in ADHD. *Journal of attention disorders*, *14*(1), 7-14.

Moffitt, T. E., Houts, R., Asherson, P., Belsky, D. W., Corcoran, D. L., Hammerle, M., ... & Caspi, A. (2015). Is adult ADHD a childhood-onset neurodevelopmental disorder? Evidence from a four-decade longitudinal cohort study. *American Journal of Psychiatry*, *172*(10), 967–977.

Newcorn, J. H., Weiss, M., & Stein, M. A. (2007). The complexity of ADHD: diagnosis and treatment of the adult patient with comorbidities. *CNS spectrums*, *12*(S12), 1-16.

Nikolas, M.A., Marshall, P.S., & Hoelzle, J.B. (2019). The role of neurocognitive tests in the assessment of adult attention-deficit/hyperactivity disorder. *Psychological Assessment, 31*(5), 685-698.

Purper-Ouakil, D., Ramoz, N., Lepagnol-Bestel, A. M., Gorwood, P., & Simonneau, M. (2011). Neurobiology of attention deficit/hyperactivity disorder. *Pediatric research*, *69*(8), 69-76.

Rege, S. (2022). *Neurobiology of attention deficit hyperactivity disorder (ADHD) - A Primer*. Psych Scene Hub. Retrieved October 7, 2022, from https://psychscenehub.com/psychinsights/neurobiology-of-adhd/

Rivas-Vazquez, R. A., Ramirez-Mejia, C., & Diaz, S. (2022). Undiagnosed adult attention-deficit/hyperactivity disorder detected during the COVID-19 pandemic. *Prim Care Companion CNS Disord. 24*(5):22br03356. https://doi.org/10.4088/PCC.22br03356

Romeo, J. (2021, July 20). *ADHD: The History of a Diagnosis*. JSTOR Daily. https://daily.jstor.org/adhd-the-history-of-a-diagnosis/

Spencer, T. J., Biederman, J., Wilens, T. E., & Faraone, S. V. (2002). Overview and neurobiology of attention-deficit/hyperactivity disorder. *Journal of Clinical Psychiatry*, *63*, 3-9.

Tripp, G., & Wickens, J. R. (2009). Neurobiology of ADHD. *Neuropharmacology*, *57*(7-8), 579-589.

Turgay, A., Goodman, D. W., Asherson, P., Lasser, R. A., Babcock, T. F., Pucci, M. L., ... & ADHD Transition Phase Model Working Group. (2012). Lifespan persistence of ADHD: the life transition model and its application. *The Journal of clinical psychiatry*, *73*(2), 10337.

Weiss, M. D., & Weiss, J. R. (2004). A guide to the treatment of adults with ADHD. *Journal of Clinical Psychiatry*, *65*, 27-37.

Wolraich, M. L., Hagan Jr, J. F., Allan, C., Chan, E., Davison, D., Earls, M., ... & Zurhellen, W. (2019). Subcommittee on Children and Adolescents with Attention-Deficit/Hyperactive Disorder. Clinical practice guideline for the diagnosis, evaluation, and treatment of attention-deficit/hyperactivity disorder in children and adolescents. *Pediatrics*, *144*(4), e20192528.

Zalsman, G., & Shilton, T. (2016). Adult ADHD: a new disease? *International journal of psychiatry in clinical practice*, *20*(2), 70-76.

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