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### Authors

Ahmad, Shaikh I  
Owens, Elizabeth B  
Hinshaw, Stephen P

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## Little Evidence for Late-onset ADHD in a Longitudinal Sample of Women

Shaikh I. Ahmad, Elizabeth B. Owens, and Stephen P. Hinshaw

University of California, Berkeley

### Abstract

**Objective:** Individuals with late-onset symptoms of attention-deficit/hyperactivity disorder (ADHD) are presenting to providers at increasing rates. Recent birth-cohort studies reveal evidence for late-onset ADHD, but conclusions are challenged by measurement methods as well as presence of participant impairment and psychiatric comorbidities. We examined the occurrence of late-onset ADHD in a small but thoroughly investigated group of diverse (47% white) women followed from childhood to adulthood.

**Method:** From a larger, 16-year longitudinal study, a subsample of young women without childhood ADHD ( $N = 87$ ) was assessed at four time points between childhood and adulthood via a multi-method, multi-informant approach. We used a stepped diagnostic procedure to identify those who initially met symptom criteria for ADHD after childhood and then evaluated them for remaining DSM ADHD diagnostic criteria, including impairment, cross-situational symptoms, and comorbid diagnoses.

**Results:** Of 87 participants, 17 met ADHD symptom criteria after childhood. Fifteen showed no evidence of childhood onset, ten showed clear evidence of impairment, and nine had cross-situational symptoms. Of these nine, all but one showed clinically significant co-occurring or pre-existing psychiatric diagnoses and/or substance use that might account for ADHD symptoms.

**Conclusions:** Although 19.5% of women from our subsample without childhood ADHD met symptom criteria for ADHD during adolescence/adulthood, only one showed the needed combination of impairment and cross-situational symptoms without significant co-occurring mental health problems. It is possible that uncomplicated cases of adult ADHD do arise, yet we find little supporting evidence herein.

### Keywords

ADHD; late-onset; longitudinal; comorbidity; internalizing

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Attention-deficit/hyperactivity disorder (ADHD) carries significant individual, public health, and economic consequences. These include risk for family, social, and academic problems, employment instability, high divorce rates, criminal behavior, substance abuse, self-harm, and psychiatric comorbidities (Erskine et al., 2016; Kessler et al., 2006; Wilens, Faraone, &

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Corresponding Author: Shaikh I. Ahmad, Department of Psychology, University of California, Berkeley, 2121 Berkeley Way, Room 2160, Berkeley, CA 94720-1650. shaikhahmad@berkeley.edu.

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Biederman, 2004). Numerous prospective longitudinal studies have shown that a substantial percentage of children diagnosed with ADHD either continue to meet diagnostic criteria in adulthood or continue to have elevated symptoms and experience significant cross-domain impairment. An area of recent investigation and controversy, however, concerns late-onset ADHD. Birth-cohort studies have suggested a group of individuals who first present with ADHD symptom onset only in adolescence or adulthood—who therefore did *not* have ADHD in childhood (Agnew-Blais et al., 2016; Caye et al., 2016; Moffitt et al., 2015). These findings raise the issue that either (a) ADHD is not solely a childhood-onset neurodevelopmental disorder or (b) there is another, later-onset syndrome that appears similar to ADHD but with a different etiology and developmental course. Understanding this issue has vital implications for diagnosis and treatment, particularly given recent increases in adult ADHD diagnoses (Oehrlein, Burcu, Safer, & Zito, 2016; Olfson, Blanco, Wang, Laje, & Correll, 2014; see also Sibley et al., 2018) and stimulant medication use/misuse, especially among young adults and women (Anderson et al., 2018; Benson, Flory, Humphreys, & Lee, 2015).

Commentaries (Barkley, 2016; Faraone & Biederman, 2016; Solanto, 2017) suggest that at least three issues should be considered when assessing the validity of late-onset ADHD: (a) the measures/methodology used to diagnose ADHD across time need to be consistent, (b) rates of diagnosis and persistence vary significantly across informants, with problematic reliance on self-report only, and (c) comorbid or alternative diagnoses, with symptoms that are similar to those of ADHD, must be taken into consideration. Indeed, a large longitudinal study of young adults both with and without childhood ADHD (Sibley et al., 2018) revealed scant evidence of late-onset ADHD that was not better accounted for by complex psychiatric history or heavy substance use. However, this study (like most on the topic) employed a predominantly male sample. As women are more likely to receive a first-time diagnosis later in life (Nussbaum, 2012), the question of late-onset ADHD is particularly salient for women.

Our aim is to replicate the procedures of Sibley and colleagues (2018) and extend them via an all-female sample. We examined the potential for late-onset ADHD in an ethnically diverse (47% white), closely followed, and highly retained comparison group of the Berkeley Girls with ADHD Longitudinal Study (BGALS; Hinshaw, 2002; Owens, Zalecki, Gillette, & Hinshaw, 2017). This comparison subsample of young women did not meet ADHD diagnostic criteria at baseline and received three subsequent, thorough assessments over a 16-year period, between adolescence and adulthood. Each evaluation featured multiple informants and structured clinician interviews, assessing key domains of functioning and impairment. Utilizing a step-wise clinical diagnostic approach similar to that of Sibley and colleagues (2018), we assessed late-onset ADHD, taking into account key methodological issues noted above.

## Method

### Participants and Procedures

Comparison girls from BGALS ( $N = 88$ ) were demographically similar to the girls with childhood ADHD diagnoses, the focus of our 16-year prospective longitudinal study

(Hinshaw et al., 2012; Owens et al., 2017). The sample was ethnically diverse (47% white), with a median household income of \$60,000-\$70,000 and a median maternal education level of 16 years – and generally representative of the local area. By design, this subsample could not meet ADHD diagnostic criteria at baseline, but subthreshold symptoms and other common diagnoses were allowed. Mean age (in years) at each assessment point was as follows: Wave 1 (baseline)  $M = 9.4$  (range 6–12); Wave 2  $M = 14.2$  (range 11–18); Wave 3  $M = 19.6$  (range 17–24); Wave 4  $M = 25.4$  (range 21–29). Each assessment spanned 1–2 days and included structured and unstructured interviews, objective testing, and multi-informant ratings. This study was approved by the UC Berkeley’s Committee for Protection of Human Subjects. Written informed consent/verbal assent was obtained from participants’ parents and/or participants, depending on age.

### Measures and Analytic Plan

For detailed information on measures, please refer to previous publications (Owens, Zalecki, & Hinshaw, 2016; Owens et al., 2017). *ADHD symptoms* were measured at all four waves using the Swanson, Nolan and Pelham rating scale (Swanson, 1992). At W1 and W2, parents and teachers were informants; at W3 and W4 participants and parents were informants. A symptom was considered present if it received a score of 2 or 3 (on the 0–3 metric) from either informant. *Impairment* was assessed using the parent-reported Columbia Impairment Scale (CIS; Bird, 1999) at W2 and W3, and the clinician-rated Global Assessment of Functioning scale (GAF; American Psychiatric Association, 2013) at W3 and W4. The presence of *other mental disorders or psychopathology* was assessed with the Diagnostic Interview Schedule for Children at W2/W3 (DISC; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000), and the Structured Clinical Interview for DSM-5 at W4 (SCID; First, Williams, Karg, & Spitzer, 2015). We also utilized the Achenbach system of scales (CBCL, TRF, YSR) to identify participants who exhibited clinically significant Internalizing Problems (Ach-Int), indicated by a T score  $> 70$ . *Substance use* (SU) was assessed with the DISC/SCID and via the Substance Use Questionnaire (Molina & Pelham, 2003).

All W1 comparison participants ( $N = 88$ ) were considered for analysis, but one was excluded due to lack of follow-up data. Following Sibley et al. (2018), we utilized a step-wise diagnostic procedure to determine whether any of these participants met full criteria for ADHD during late adolescence or adulthood, which was not better explained by other psychiatric problems, in an attempt to increase both sensitivity and specificity for ADHD, as follows:

*Step 1–Symptom inclusion criteria.* To reduce false negatives, we utilized a more inclusive, best-estimate “or” algorithm regarding ADHD symptoms. That is, if *either* informant endorsed a symptom at the item-level on the SNAP, it was considered present. A participant was retained for further analysis if ADHD symptom level met or DSM-5 symptom threshold (6+ symptoms for ages 13–16, 5+ symptoms for age 17+).

*Rule-out criteria:* If a participant met these ADHD symptom criteria at W2, W3, or W4, the following DSM-5 criteria were used to rule out false-positives in assessing late-onset ADHD:

*Step 2–Age of onset:* Symptom onset must begin after age 11 for late-onset ADHD.

*Step 3–Impairment:* Either parent (at W2/W3 on the CIS) or study clinician (at W3/W4 on the GAF) must have endorsed impairment (at home, with peers, with academics, or at school) during the assessment point at which the participant met ADHD symptom criteria. For the CIS, if a parent endorsed at least one domain related to ADHD (“getting along with peers,” “behavior at school,” “behavior at home,” or “school work”) with a score of 3 or higher on the 0–4 metric (i.e., the situation causes more than “some problem”), impairment was considered present. Similarly, if the clinician recorded a GAF score below 70 (i.e., at least *some* difficulty was present in multiple domains of functioning), impairment was considered present.

*Step 4–Cross-situational symptoms:* To assess presence of ADHD symptoms in two or more settings, we utilized the cross-situational symptom criteria provided by the DISC from parent and participant interviews at W2/W3, and from information obtained from participants during clinician interviews regarding family, peers, romantic relationships, school, recreation, and employment functioning at W4.

*Step 5–Substance use and other mental disorders:* Remaining cases were assessed to determine whether ADHD symptoms might be better accounted for by co-occurring heavy substance use or a substance use disorder (SU), or by another mental disorder that could plausibly account for ADHD symptoms and related impairments (DSM-5 Criterion E). We also assessed whether the comorbid symptoms were present *before* ADHD symptoms were present (i.e., at an earlier wave).

## Results

Regarding trajectories of inattention symptoms for the final nine cases, refer to Figure 1. Note that only two cases had elevated hyperactive/impulsive symptoms (Case E had 8 self-reported symptoms at W3 and 6 at W4, but met diagnostic criteria only at W3; Case G had 4 self-reported symptoms at both W3 and W4). For comorbid diagnoses and psychopathology, refer to Table 1.

*Step 1–Symptom inclusion criteria:* Of the 87 comparison cases with follow up data, 17 (19.5%) surpassed DSM-5 ADHD symptom threshold criteria during at least one follow-up assessment. Of these, six first met criteria during adolescence (at or before age 17) and 11 first met criteria in adulthood (age 18+), with an overall *M* age of onset of 19.1 years.

*Step 2–Age of onset:* Of these 17 cases, two did not meet onset criteria. One was 11.8 years old at W2 and met symptom criteria at that time; one had 6 teacher-endorsed symptoms of inattention at W1. Both were thus considered to have child-onset ADHD.

*Step 3–Impairment:* Of the remaining 15 cases, five did not meet impairment criteria. Ten were deemed to be significantly impaired: parent CIS ranged from 1–3 domains impaired, mean = 1.6 domains. Clinician GAF scores ranged from 51–67, mean = 56.

*Step 4–Cross-situational symptoms:* Nine of these 10 cases were deemed to have symptoms across multiple settings, based on structured clinical interview (DISC) at W2/W3 or clinician interview at W4.

*Step 5–Substance use and other mental disorders:* Of these nine cases, all but one (Case B) had either co-occurring or pre-existing substance use and/or another mental disorder or clinically significant psychopathology that could potentially account for ADHD symptoms (for details on comorbidities, refer to Table 1). For five (Cases C, E, G, H, and I), internalizing/externalizing psychopathology or SU preceded late-onset ADHD diagnostic criteria. For these five, it is plausible that other symptoms or disorders better accounted for ADHD-related symptoms, as they were reported prior to ADHD symptoms. Of the remaining four cases, three (Cases A, D, and F) had late-onset ADHD that was comorbid with either SU, or internalizing/externalizing disorders/psychopathology. For these, it is less clear which symptoms/disorders started first. For the last case (Case B), late-onset ADHD preceded later SU.

Note that six out of nine cases met full criteria for ADHD only at a single time point (see Table 1); comorbidities often persisted across multiple time points. Also, seven out of nine cases had comorbid substance use and six had internalizing psychopathology. Finally, for seven of these nine cases, there were no parent/teacher reported inattentive ADHD symptoms at baseline.

## Discussion

We leveraged a closely followed comparison subsample from the BGALS investigation to assess rates of late-onset ADHD. We utilized a step-wise diagnostic procedure similar to that of Sibley et al. (2018) in an attempt to extend their findings in a sample of young women. We utilized a sensitive symptom-based diagnostic procedure to identify girls without initial childhood ADHD diagnoses who had significant ADHD symptoms in adolescence or adulthood. Then, our step-wise diagnostic strategy emphasized specificity in order to minimize false positives.

After considering the full range of diagnostic criteria (age of onset, impairment, cross-setting symptoms, and co-occurring psychiatric presentation), we found little evidence for late-onset ADHD within this sample of young women, similar to results from Sibley and colleagues. Seventeen cases without childhood ADHD (19.5%) initially met ADHD symptom criteria post-childhood. Yet after a thorough consideration of age of onset, impairment, and cross-situational symptoms, nine cases remained. Of these, all but one (Case B) met diagnostic criteria for multiple preceding or comorbid mental disorders, either before or during the period in which elevated ADHD symptoms were reported. These preceding and/or correlated symptoms may help to explain the supposed onset of ADHD-related symptoms. Thus, in only 1 of 87 cases was there clear evidence for late-onset ADHD, and for this case symptoms significantly abated by the next visit. In five of these cases, other disorders or psychopathology clearly preceded ADHD symptoms, and might better account for these symptoms. In the remaining three, it was less clear which symptoms manifested first.

It is certainly possible that ADHD-related symptoms may intensify or become more apparent later in adolescence, especially for females and higher-achieving youth, such as during the transition to middle or high school. Yet considerable research indicates that such children would be likely to have displayed at least *some* symptoms in childhood, given the neurodevelopmental nature of ADHD. We note that for each of the final nine cases, all had either zero or one symptom of both inattention and hyperactivity across *both* parent and teacher report at baseline (W1) – suggesting that their later-presenting ADHD symptoms might be due to another mental illness or other factors and not ADHD per se. Our findings also support existing research that comorbid internalizing problems might better explain apparently late-onset ADHD among young women, as six out of nine cases met criteria for an internalizing disorder, and seven had significant substance use.

Study limitations are noteworthy. First, only 4 assessment points were utilized, with each roughly 5 years apart. If ADHD and other comorbid symptoms were initially reported at the same time point, assessing which symptoms developed first was not possible. Three cases without a clear order of symptom onset could possibly represent late-onset ADHD. Also of note, seven of the final nine cases reported either heavy substance use or an SUD. It is possible that some of these cases actually started *using* substances before reporting doing so, which could account for ADHD-related symptoms. Second, although our findings are similar to those of Sibley and colleagues, our participants were not treatment-seeking women—who might well be more likely to receive a diagnosis in adulthood. In addition, although there were no indications of cognitive or academic impairment at baseline, we did not fully assess other potential neurological deficits or other health impairments at each time point – some of which could potentially manifest as ADHD symptoms. Last, it is possible that some of these final cases actually had childhood-onset ADHD but were not detected as such. Relevant factors here could include both individual (high academic achievement, resilience) and environmental (e.g., home/school environments, such as lack of parental/teacher monitoring). Future longitudinal studies would benefit from utilizing a larger sample size in order to increase generalizability—yet might do so at the cost of less time-intensive and rigorous assessments.

Implications emphasize the importance of utilizing multiple informants, conducting a thorough developmental history, establishing impairment, and assessing for cross-domain symptoms when diagnosing ADHD in adults. Findings also underscore the crucial importance of assessing the role of comorbid psychiatric diagnoses and substance use. Difficulty concentrating, paying attention, and feelings of restlessness/agitation could relate to a primary ADHD diagnosis or to an internalizing disorder—or could result from substance use, requiring careful and thorough clinical assessment.

Diagnosing and treating ADHD in adults presents a clinical challenge. Still, national surveys indicate that the vast majority of adults with ADHD do *not* receive treatment, with potentially sizeable implications for personal, family, and societal burden (Kessler et al., 2006). Caution is indicated when assessing adults for ADHD for the first time after childhood, given a combination of recent (a) increases in ADHD diagnoses; (b) increased stimulant medication prescriptions, especially among women; and (c) misuse of stimulant medication, especially among college populations (Anderson et al., 2018; Benson et al.,



2015). Our data suggest that some (or even most) individuals newly presenting with ADHD symptoms as adolescents or adults, with no history of childhood symptoms, might not in fact have ADHD. Non-specialists would have a particularly difficult time making a differential diagnosis (Olfson et al., 2014), so consultation with appropriate mental health professionals is strongly recommended. A thorough developmental history and a careful review of psychiatric and substance use problems is warranted to ensure accurate diagnoses and proper treatment.

Finally, we highlight that the clear majority of young women who met symptom criteria for late-onset ADHD also met criteria for an internalizing disorder. Evidence-based treatments for depression/anxiety may optimally serve these women. Use of screening tools by clinicians for substance use and other mental disorders when assessing for late-onset ADHD is certainly indicated, as is corroboration of symptoms with an informant who knows the individual well.

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**Public Health Statement:**

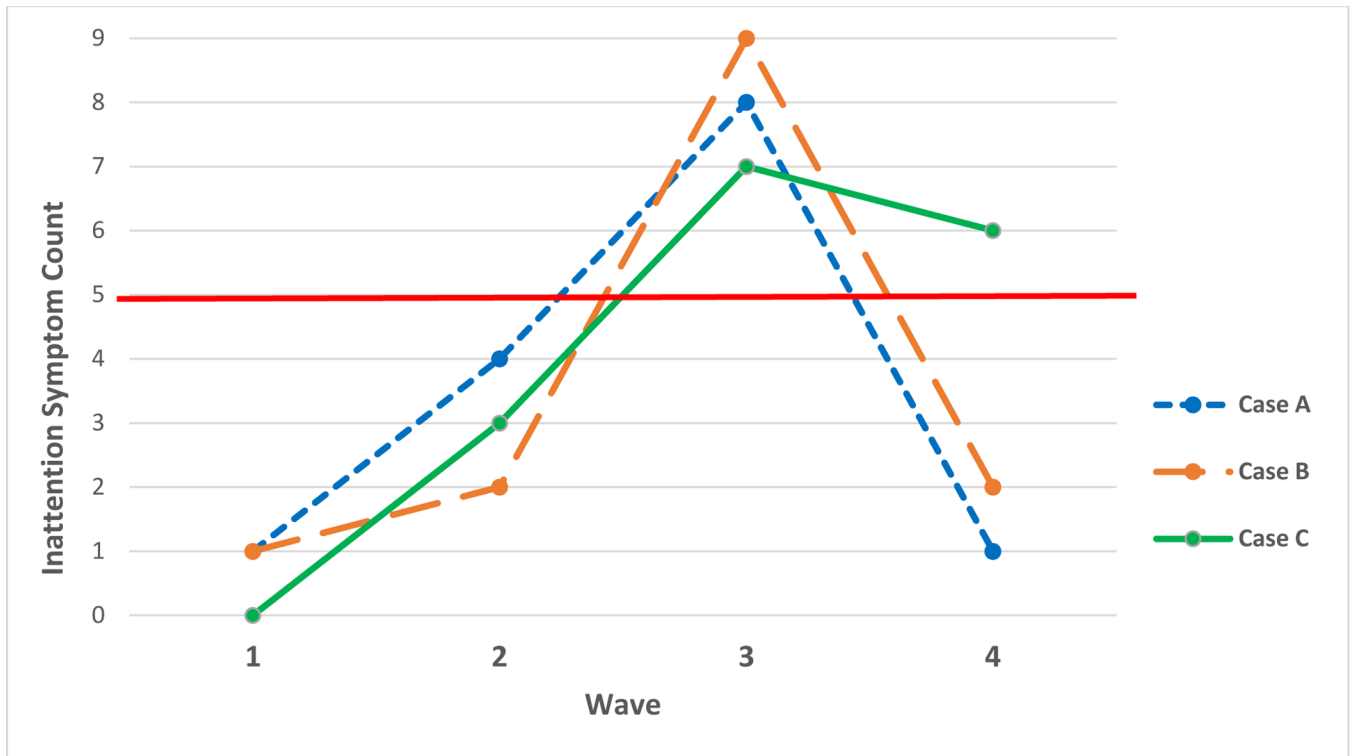
This study highlights the importance of performing a thorough clinical interview, obtaining a developmental history, and utilizing multiple sources when diagnosing ADHD in adulthood. It also emphasizes the importance of screening for comorbid psychiatric diagnoses. In addition, findings caution against making a first-time diagnosis of ADHD in adulthood without evidence of symptom onset in childhood or adolescence.

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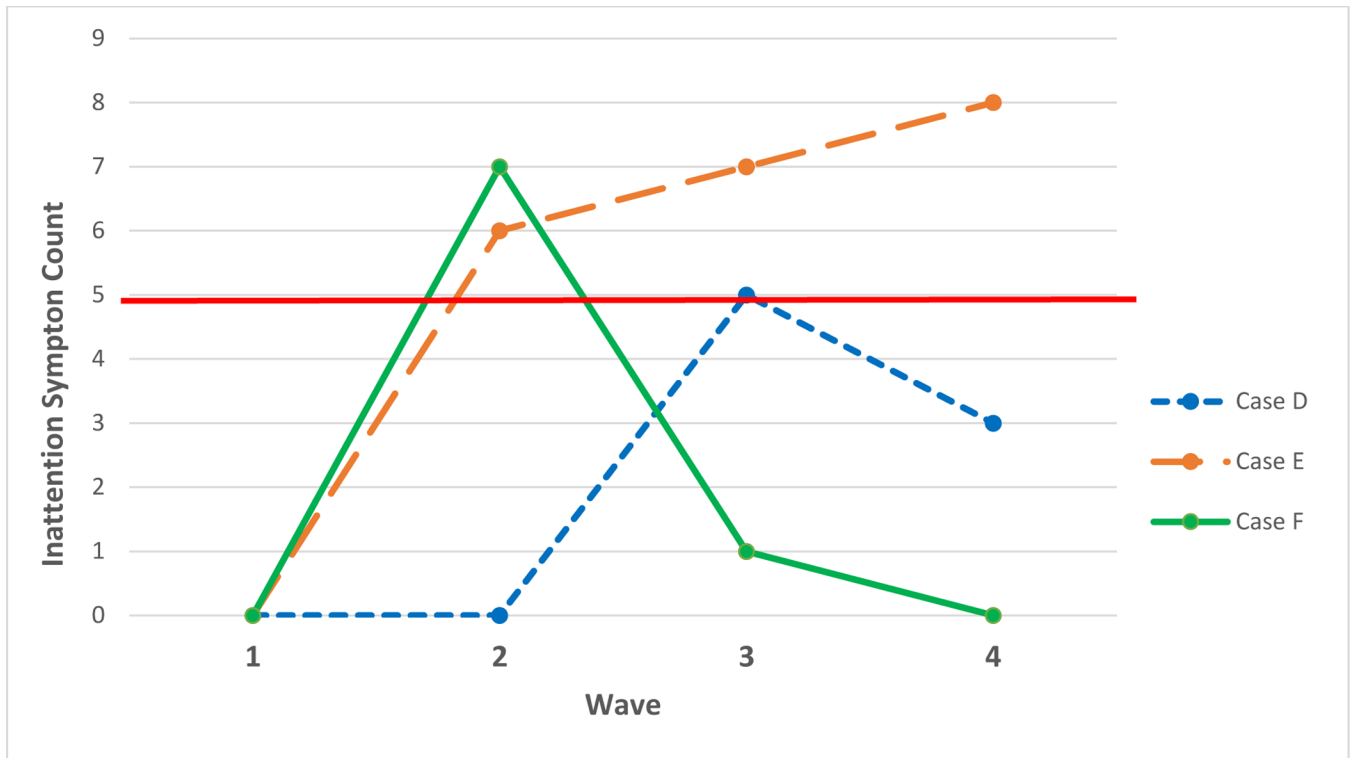
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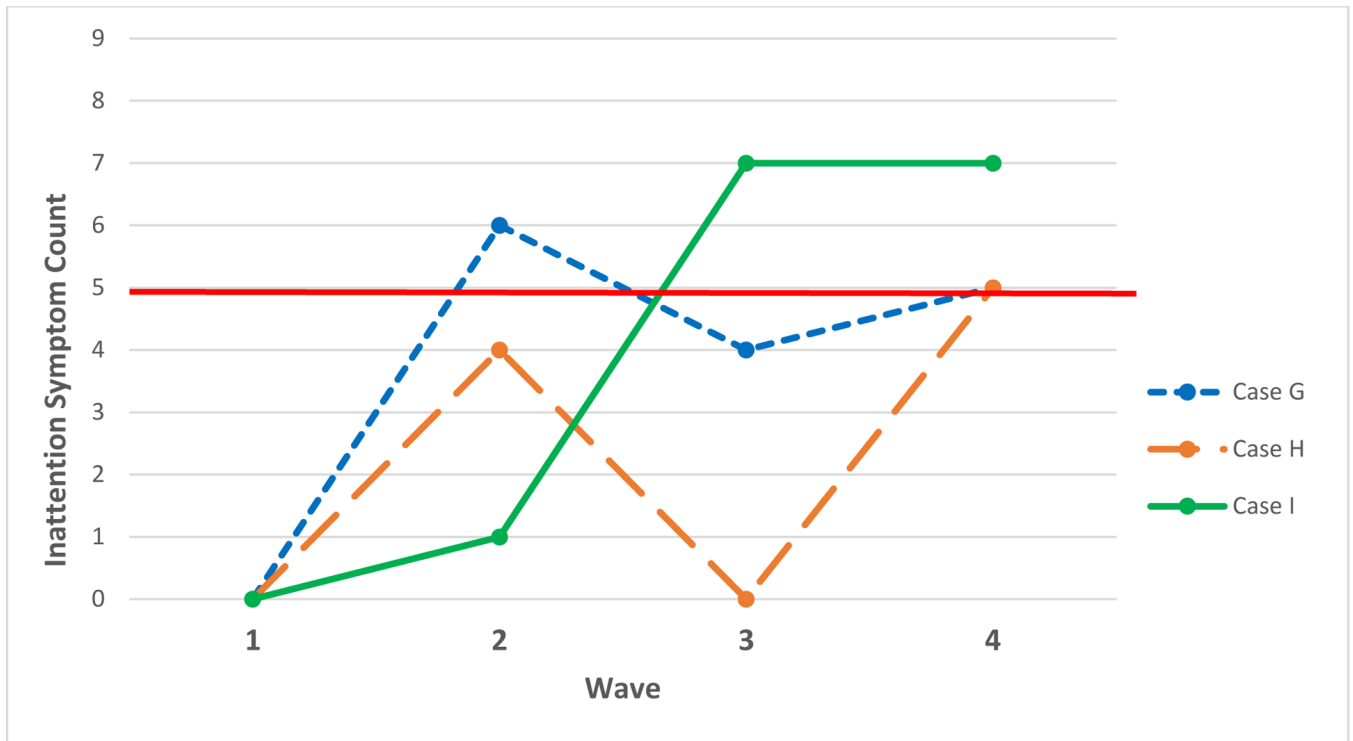
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**Figure 1a.** Inattention symptom trajectory by Wave. This figure illustrates the total inattentive symptom count across both informants by wave for the first three cases. Note that Case C met diagnostic criteria for ADHD-I at both W3 and W4.



**Figure 1b.** Inattention symptom trajectory by Wave. This figure illustrates the total inattentive symptom count across both informants by wave for the second three cases. Note that Case E met diagnostic criteria for ADHD-C (combined type) at W3 only, and did not meet criteria at W2/W4 due to lack of impairment.



**Figure 1c.** Inattention symptom trajectory by Wave. This figure illustrates the total inattentive symptom count across both informants by wave for the final three cases. Note that Case G met diagnostic criteria for ADHD-I at both W2 and W4, and Case I met diagnostic criteria for ADHD-I at both W3 and W4.

**Table 1**

## ADHD and Comorbid Diagnoses by Wave

Age (in years) and Diagnosis									
Case	Wave 1		Wave 2		Wave 3		Wave 4		
	Age	Diagnosis	Age	Diagnosis	Age	Diagnosis	Age	Diagnosis	
A	9.0		13.2		18.6	ADHD-I, SU	25.5		
B	11.4		15.6		20.9	ADHD-I	27.6	SU	
C	9.0	Ach-Int	14.0		19.0	ADHD-I, PTSD, Anxiety, MDD, SU	25.2	ADHD-I, MDD, PDD, SU	
D	7.2		11.7		17.0	ADHD-I, ODD	21.5	SU	
E	9.9	Anxiety, ODD	14.5	Anxiety, SU	19.7	ADHD-C, MDD	24.9	MDD, PDD, BPD	
F	9.9		14.7	ADHD-I, Ach-Int, ODD, SU	19.7	PTSD, SU	25.2	SU	
G	8.6	ODD	12.9	ADHD-I, ODD	18.0	ASPD, SU	23.1	ADHD-I, MDD, SU	
H	8.5		12.7	Anxiety	17.7		23.3	ADHD-I, MDD, PDD	
I	8.9	Ach-Int	13.2		18.3	ADHD-I, Anxiety, ASPD, MDD	23.9	ADHD-I, MDD, PDD	

Note: Ach-Int = Achenbach Internalizing scale (T score  $\geq 70$ ), ADHD-I = ADHD, inattentive type/presentation, ADHD-C = ADHD, combined type/presentation, SU = substance use, PTSD = post-traumatic stress disorder, Anxiety = anxiety-related disorder, MDD = major depressive disorder, PDD = persistent depressive disorder/dysthymia, ODD = oppositional defiant disorder, BPD = borderline personality disorder, ASPD = antisocial personality disorder