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Proposed Terminology to Distinguish Adult-Onset from Late-Identified ADHD: A Preliminary Effort in a Large Survey of Adults Aged 18 to 80

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According to the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (*DSM-5*; American Psychological Association, 2013), attention-deficit/hyperactivity disorder (ADHD) can be diagnosed in a person age 17 or older if the following criteria are met: five or more symptoms of inattention (IA) and/or hyperactivity-impulsivity (HI) are evident; several of those symptoms emerged before age 12; symptoms are observed in two or more settings and are not better explained by a substance use or other mental health disorder; and symptoms are associated with significant impairment in functioning. In the last few years, there have been several publications exploring whether a late-onset version of ADHD exists, one that does not begin in childhood (Agnew-Blais et al., 2016; Asheron & Agnes-Blew, 2019; Caye et al., 2016; Chandra et al., 2016; Faraone & Biederman, 2016; Manfro et al., 2019; Moffitt et al., 2015; Klein et al., 2012; Sibley et al., 2018). There is a history of investigation along these lines during the *DSM-IV* (American Psychiatric Association, 1994) era, as well (Faraone et al., 2006a; Faraone et al., 2006b). This issue remains unresolved and has been complicated by variations, or a lack of specificity, in the definitions of late-onset ADHD as well as inconsistent assessment procedures (Sibley et al., 2016; Solanto, 2021).

Regarding the definition, *late-onset* ADHD could mean that symptoms did not begin in childhood, full criteria were not met until adulthood, and/or the individual was not diagnosed until adulthood. We suggest defining onset patterns by virtue of crossing *age of symptom onset* with *age of diagnosis*. First, we define *late symptom onset* as several symptoms presenting only after childhood (i.e., age 12 or later), in accordance with the *DSM-5* age of onset criterion and other prior work (Caye et al., 2017). The *DSM-5* does

Contents

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not define "several" in its criterion B for ADHD diagnosis, but an interpretation that others and we adopt (Caye et al., 2017) is three or more symptoms reported as present and affecting one's adjustment. Second, we define *late diagnosis* as diagnosis in adulthood (i.e., age 18 or later). We chose 18, rather than 12 or another age in adolescence, to correspond more with the typical conception of "adult" ADHD, and because if symptoms emerge in later childhood (e.g., 11 years of age), then a bona fide diagnosis just a few months or years later might not rightly be considered "late." Crossing these two factors results in four possible ADHD onset patterns.

We refer to the first onset pattern as *child-onset ADHD*, which occurs when several symptoms are observed before age 12 and diagnosis occurs prior to age 18—i.e., the traditional *DSM-5* presentation. We refer to the second pattern as *late-identified ADHD*, which occurs when several symptoms are observed before age 12 but diagnosis does not occur until age 18 or later. Individuals with late-identified ADHD also meet *DSM-5* criteria, and this pattern has been observed particularly among college students (DuPaul et al., 2009). The third pattern is *adolescent-onset ADHD*, which occurs when several symptoms are not observed until after age 12 but diagnosis still occurs prior to age 18. Individuals with adolescent-onset ADHD do not meet *DSM-5* criteria because they lack obvious, clinically meaningful symptoms by age 12. The final pattern is *adult-onset ADHD*, which occurs when symptoms are not observed in childhood or adolescence until after age 12 and diagnosis does not occur until age 18 or later. Individuals with adult-onset ADHD also do not meet *DSM-5* criteria because they too lack obvious, clinically meaningful symptoms by age 12.

Importantly, in their review, Asheron and Agnew-Blais (2019) concluded that common criticisms of potential late-onset ADHD (e.g., change in symptom reporters, presence of substance use or other comorbid disorders) cannot completely explain the presence of cases that appear to have significant ADHD in adulthood yet no clear signs or impairment in childhood. Further, Faraone and Biederman (2016) posited that adult-onset ADHD is possible and that many individuals so affected likely had unevaluated, subthreshold childhood symptoms.

It is also of note that, since the publication of the new diagnostic standards (including later age of onset criterion) in the *DSM-5*, several empirical studies using longitudinal (Agnew-Blais et al., 2016; Caye et al., 2016; Cooper et al., 2018; Moffit et al., 2015; Taylor et al., 2019; Sibley et al., 2017) and cross-sectional data (Chandra et al., 2016) have been published focusing on the question of late-onset ADHD. However, these vary with definitions of the late-onset construct and have limitations that we believe render further consideration necessary. As an example, Moffitt et al. (2015) examined ADHD in adults followed from childhood via the New Zealand birth cohort study (52% male). These authors defined late-onset ADHD solely by age of diagnosis and did not consider age of symptom onset. That is, they considered only those individuals who retrospectively met full *DSM* criteria but were diagnosed in adulthood (i.e., childhood-onset ADHD that has persisted into adulthood). Next, Caye et al. (2017) examined ADHD in adults who were included in a Brazilian birth cohort study (48% male). Similar to Moffitt et al. (2015), their conclusion is based on age of diagnosis and they did not consider age of symptom onset. Thus, of those adults who they identified as having late-onset ADHD, it is unclear how many would be categorized as late-identified, adolescent-onset, or adult-onset because they did not tap age of symptom onset. Agnew-Blais et al. (2016) studied ADHD in adults who were included in a birth cohort twin study in the United Kingdom (49% male). In this study, the authors compared those with persistent childhood-onset ADHD (i.e., met criteria in childhood and adulthood; 67% male) to those with late-identified ADHD (i.e., did not meet criteria in childhood but showed symptoms; 45% male). They did not argue for the existence of a late-onset version of ADHD that does not begin in childhood, although they referred to their late-identified group as having late-onset ADHD. Finally, in a

Swedish birth cohort twin study, Taylor et al. (2019) examined patterns of ADHD symptom onset in individuals who were first diagnosed in childhood, adolescence, or young adulthood. For those diagnosed in adulthood, they found that a large majority had subthreshold ADHD symptoms in childhood and were more likely to have been diagnosed with another mental health disorder in that period. Overall, they concluded that the evidence does not support the existence of adult-onset ADHD. Cooper et al. (2018) also drew similar conclusions, using the English Avon Longitudinal Study of Parents and Children (ALSPAC) birth cohort, although their consideration of late-onset data is limited to adolescence (i.e., to age 17).

In addition to these birth cohort studies, Chandra et al. (2016) conducted a cross-sectional study of ADHD in adults ages 18 to 55. Based on our four proposed onset patterns, participants in the Chandra et al. "full" ADHD group could have the child-onset pattern or the late-identified pattern, and those in the late-onset group could have the adolescent-onset or the adult-onset pattern. Next, Sibley et al. (2018) examined ADHD in adults who participated in the multimodal treatment of ADHD (MTA) study as children and were followed longitudinally. By design, none of them met full criteria for ADHD in childhood, but four appeared to have met full criteria in adolescence (child-onset ADHD, i.e., with subthreshold symptoms presenting first in childhood). When age of onset was considered, 12 had symptom onset during childhood (late-identified ADHD), 14 had symptom onset during adolescence (adolescent-onset ADHD), and 10 had symptom onset during adulthood (adult-onset ADHD). Sibley et al. (2018) concluded that out of 24 possible late-onset cases, only three remained viable as adolescent-onset or adult-onset ADHD cases. Of the 24 possible late-onset cases, over half only had impairing symptoms in the context of heavy substance use, five had impairing symptoms that were attributable to another mental health disorder, and two did not have interview data from childhood. These findings highlight the low likelihood of individuals having true late-onset ADHD and emphasize the importance of assessing for all *DSM* criteria when making a first-time diagnosis of ADHD in adulthood. For a more complete review of this existing literature, the reader is referred to the work of Asheron and Agnew-Blais (2019).

Still more recent is Solanto's (2021) review of methodological differences across this literature base. Attempting to reconcile inconsistent findings, she proposed that adult-onset ADHD is:

(a) etiologically distinct from child-onset ADHD
(b) a delayed manifestation of the same deficit(s) involved in child-onset ADHD
(c) a variant or subtype of ADHD as currently diagnosed

Solanto concluded, "regardless of methodology . . . there exists at least a small group of individuals who can be considered to be valid cases of adult-onset ADHD" (p. 7). Regarding (a), she argues for a distinct late-onset group by citing differences in genetic and neuroimaging data between persons diagnosed in childhood and those identified in adulthood. However, these differences could also be explained by child-onset ADHD being a more severe neurological condition than late-identified ADHD. With regards to (b), this possibility could also be consistent with late-identified ADHD in that these individuals with higher IQs and more support still show some symptoms in childhood, but only merit diagnosis later when the balance of support and demand changes (e.g., college; Agnew-Blais et al., 2019). Finally, in (c), Solanto proposes that adult-onset ADHD might be a subtype with a weaker genetic predisposition and a stronger environmental component. Again, this is consistent with the idea that adult-onset cases may be better understood as late-identified ADHD. Thus, these individuals may still have ADHD given they had childhood symptoms that do not reach clinical significance until later.

Finally, Taylor et al. (2021) also conducted a systematic review to examine whether the existing literature regarding adult ADHD was methodologically sufficient to empirically evaluate for adult-onset ADHD. The authors concluded that currently there is not clear and consistent evidence with which one can empirically evaluate whether adult-onset ADHD exists. Taylor et al. highlight the significant methodological heterogeneity among existent studies reviewed and suggest that part of this stems from the lack of a clear and consistent operationalization of adult-onset ADHD (for a nuanced review see Appendix C, Taylor et al., 2021). Among the many recommendations and findings laid out in the review, the authors also proposed that future research into adult-onset ADHD needs to separately and collectively examine both adolescent- and adult-onset ADHD. Quite recently, Riglin et al. (2022)—also using data from the ALSPAC study– in some ways follow suit, noting some similarities between individuals with research-criteria-defined child-onset persistent ADHD and those with "late-onset" ADHD. Unfortunately, the study's results are inconclusive, given discrepancies across different informants' data as well as a late onset group that combines late adolescent (age 17) and young adult (age 25) onset.

CURRENT STUDY

Regarding the definition, based on prior studies and rational consideration of current diagnostic criteria, late-onset ADHD could mean: (a) the symptoms did not begin in childhood, (b) full criteria were not met until adulthood, and/or (c) the individual was not diagnosed until adulthood. We define four onset patterns by crossing age of symptom onset with age of diagnosis, as noted above (see Figure 1). Given that three distinct types of late-onset ADHD have variously been included, but not explicitly defined as such, in the existent "late onset" studies (i.e., late-identified, adolescent-onset, adult-onset), an important consideration that is unexplored is to directly compare these nascent groups to child-onset cases and determine whether they are clinically distinct from non-ADHD cases. Further, prior research has consistently noted a smaller male-to-female ratio in diagnosed ADHD in adults, but it remains unclear how men and women may fall into these four putative onset categories

	Early diagnosis Diagnosis < age 18	Late diagnosis Diagnosis ≥ age 18
Early symptom onset Symptom onset < age 12	Early-Early *Childhood Onset*	Early-Late *Late Identified*
Late symptom onset Symptom onset ≥ age 12	Late-Early *Adolescent Onset*	Late-Late *Adult Onset*

Figure 1. Four possible patterns of ADHD onset

and whether that is associated with different symptom presentations, impairments, or other characteristics. Finally, most of the existing studies examining possible late-onset ADHD have included samples with a rather limited age range in adulthood (e.g., up to age 18 only or through young adulthood). In the current study, one research question was to explore whether symptoms and impairment related to each of the four possible onset patterns of ADHD differ, using a large, cross-sectional survey of non-referred adults representing a broad range of ages. A second question was whether there were notable sex differences across onset patterns. This latter goal is consistent with recent recommendations to examine sex differences more deliberately in psychopathology (Hartung & Lefler, 2019). In sum, we evaluated whether the four onset patterns differ from each other in terms of severity of symptoms and impairment, or by sex.

METHOD

Participants

Participants were 8,536 adults (62.1% females) who resided across all regions of the United States and ranged in age from 18 to 80 (M = 34.77, SD = 11.43). Report of a prior diagnosis of ADHD was used to differentiate those who would be grouped in the four ADHD-onset categories from non-diagnosed comparison participants; 1,076 (12.6%) reported a previous diagnosis of ADHD, and 7,460 (87.4%) did not. Participants in the sample who self-reported a previous diagnosis of ADHD ranged in age from 18 to 72 (M = 31.74, SD = 9.19; 56.4% females), and noted the timing of their initial diagnosis to be from 2 to 62 years of age (M = 18.59, SD = 10.25). Further information regarding these prior diagnoses was not collected.

All participants completed the online survey via Amazon's Mechanical Turk (MTurk; https://www.mturk.com) platform. The MTurk platform provides a number of advantages with regards to data collection. For instance, research demonstrates that MTurk samples provide high quality data (e.g., validity, reliability, accuracy in self-reporting, willingness to disclose) and increased diversity among participants that is more representative of the population compared to more traditional samples (e.g., in person studies involving college or community populations; Goodman & Paolacci, 2017). Further, prior research has established the value of the MTurk platform for examining psychopathology (Goodman & Paolacci, 2017; Shapiro et al., 2013). Specifically, with regard to the manifestation of ADHD, Wymbs and Dawson (2019) found that self-reported rates of ADHD in an MTurk sample were consistent with community samples. MTurk also has the potential advantage of facilitating large-sample data collection, which was deemed as critical by us to achieve sufficient power to detect what might be nuanced differences between onset groups and across sexes. However, a downside to data collection using this platform is that MTurk workers do not receive particularly large compensation for participation, which has the effect of streamlining the length of study procedures (e.g., abbreviated diagnostic measures vs. "gold standard" assessment).

Measures

Participants provided demographic information regarding age and biological sex, completed the Barkley Adult ADHD Rating Scale (BAARS-IV), and answered three questions about previous ADHD diagnosis:

1. Have you ever been diagnosed with ADHD?
2. At what age were you diagnosed?
3. How old were you when these symptoms began?

***BAARS-IV* (*Self-Report: Current Symptoms*)**. The BAARS-IV (Barkley, 2011) is a 30-item self-report scale measuring IA and HI symptoms of ADHD as well as related impairment in adults. The 18 ADHD items (9 IA, 9 HI) closely map onto the ADHD symptom stems in the *DSM-5* (American Psychiatric Association, 2013). Participants used a four-point Likert scale (1 = Never or rarely to 4 = Very often) to indicate the frequency of each item in the past six months. Participants were also asked to identify all settings in which their symptoms impair their functioning (i.e., school, home, work, social relationships).

Symptom counts for both IA and HI were determined by summing the number of items in each section rated Often or Very often. Thus, symptom counts for IA and HI ranged from 0 to 9. For impairment, the number of impaired settings was summed and the resulting range was from 0 to 4. The BAARS-IV has robust internal consistency, test-retest reliability, and inter-observer agreement across scales, as well as high construct and face validity (Barkley, 2011). Internal consistency in this sample ranged from α = .87 to .93.

Procedure

Institutional Review Board approval was obtained for the study procedures at the first author's university prior to beginning data collection. Participants on the MTurk website followed a link to the online survey created using the Qualtrics Research Suite (Qualtrics, 2015). First, participants provided informed consent. Next, participants completed a brief demographic questionnaire as well as the BAARS-IV. Participants were compensated with $0.15 for their time.

Table 1. Levels of self-reported, current symptoms and impairment by onset pattern and sex

	Child-onset (1.8M:1F)[a] *n* = 363 (4.3%)				Late-identified (1M:1.6F)[a] *n* = 251 (2.9%)				Adolescent-onset (2.2M:1F)[a] *n* = 223 (2.6%)				Adult-onset (1M:1.1F)[a] *n* = 239 (2.8%)				No diagnosis *n* = 7,460 (87.4%)			
	Males *n* = 187		Females *n* =176		Males *n* =70		Females *n* =181		Males *n* =129		Females *n* =94		Males *n* =83		Females *n* =156		Males *n* = 2,768		Females *n* = 4,692	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
IA	3.95	2.83	4.63	3.08	5.30	3.00	5.45	2.73	3.82	2.82	4.12	2.83	4.49	2.58	4.60	2.89	1.56	2.22	1.62	2.23
HI	3.45	2.91	4.10	2.71	4.20	2.50	4.15	2.69	3.53	2.78	3.36	2.64	3.53	2.20	3.84	2.75	1.51	1.98	1.46	1.99
IMP	2.94	1.11	3.28	0.91	3.33	1.03	3.51	0.74	2.60	1.13	2.72	1.18	2.82	1.11	2.92	1.03	1.75	1.43	1.81	1.45

Note. M = Males; F = Females; IA = inattention symptoms (range 0 to 9); HI = hyperactivity/impulsivity (range 0 to 9); IMP = areas of impairment (range 0 to 4).

[a]*Sex ratios were calculated based on the number of males and females reporting a diagnosis with a particular onset pattern when considering the number of males (37.9%)* and females (62.1%) in the total sample.

Analytic Approach

The original dataset (N = 8,696) was screened for missing data, failed attention checks, non US-based locations, and duplicate participation prior to analysis. Participants with missing data on the BAARS-IV or age of symptom onset or diagnosis (n = 143) were removed from the dataset. Additionally, data were removed for (n = 7) participants as geolocation based on IP address suggested their IP addresses originated from outside of the U.S. Finally, data of participants (n = 9) who did not endorse either male or female for biological sex were removed since there was not enough power to analyze this group separately. The remaining data (N = 8,536) approached normality across all variables of interest (e.g., skew ≤ |1.36|; kurtosis ≤ |1.97|), with the exception of one variable (i.e., prior ADHD diagnosis) which demonstrated larger skew and kurtosis (i.e., skew = 2.25; kurtosis = 3.08), but still approached normality given the sample size.

Analytic procedures. Sample characteristics were computed for three dependent variables (i.e., IA, HI, and impairment) across onset pattern and biological sex. First, we examined the distribution of participants with a past ADHD diagnosis across onset patterns and conducted a chi-square analysis to examine whether there were differences in sex distribution across the four onset patterns (i.e., child-onset vs. late-identified vs. adolescent-onset vs. adult-onset). Next, we conducted a one-way ANOVA to examine whether the three dependent variables differed by past ADHD diagnosis and/or sex, and another one-way ANOVA to examine whether the dependent variables differed by ADHD onset pattern and/or sex. Finally, we conducted analyses to examine whether there are differences in persistence of ADHD by onset pattern.

RESULTS

Distribution of participants with past diagnosis across onset patterns

Based on past diagnosis of ADHD, age of symptom onset, and age of diagnosis, participants were grouped into four ADHD-onset groups as defined above (*child-onset*, n = 363, 4.3%; *late-identified*, n = 251, 2.9%; *adolescent-onset*, n = 223, 2.6%; *adult-onset*, n = 239, 2.8%) and a non-diagnosed comparison group (n = 7,460, 87.4%). These groups were used in all further analyses reported below. Means for IA symptom count, HI symptom count, and number of settings of impairment are shown in Table 1 by sex and onset pattern. A chi-square analysis revealed significant differences in the distribution of sex across these groups, χ^2 (3, n = 1076) = 60.51, p < .001. The sex ratios for each onset pattern, after considering the number of men and women in the total sample, are also shown in Table 1. Additional chi-square analyses determined that the child-onset pattern was more common in men, χ^2 (1, N = 8,536) = 29.76, p < .001, the adolescent-onset patterns was more common in men, χ^2 (1, N = 8,536) = 38.62, p < .001, the late-identified pattern was more common in women, χ^2 (1, N = 8,536) = 11.06, p = .001, and the adult-onset pattern was equally common in men and women, χ^2 (1, N = 8,536) = 1.07, p = .302.

Comparison of symptom levels by ADHD diagnosis and sex

First, we conducted two (ADHD vs. non-ADHD) x two (female vs. male) ANOVAs to compare participants with and without a previous diagnosis of ADHD (i.e., lifetime) on ADHD symptoms and impairment. For all three ANOVAs, the main effects of group were statistically significant (ps < .001) with medium to large effect: IA, F(1, 8532) = 1444.97, η_p^2 = 0.15[1]; HI, F(1, 8532) = 1092.05, η_p^2 = 0.11; and impairment, F(1, 8532) = 734.37, η_p^2 = 0.08. With regard to the overall influence of sex on the self-described presence of ADHD symptomatology, for IA, F(1, 8532) = 17.09, and impairment, F(1, 8532) = 14.63, the main effects of sex were statistically significant (ps < .001), with women reporting higher levels than men; however, the effect size was negligible (η_p^2 < 0.01). In addition, two group x sex interactions were significant such that women with a past ADHD diagnosis reported higher IA, F(1, 8532) = 11.20, p = .001, and HI, F(1, 8532) = 7.89, p = .005, compared to men with a past diagnosis; however, again, the effect sizes were negligible (η_p^2 < 0.01).

Comparison of symptom levels by ADHD-onset patterns and sex

Next, we conducted four (child-onset vs. late-identified vs. adolescent-onset vs. adult-onset) x two (male vs. female) ANOVAs to compare participants across all four groups. For IA, the only significant effect was a main effect of group,

1 Partial-eta squared values of 0.01, 0.06, and 0.14 were considered small, medium, and large effect sizes, respectively (Cohen, 1988).

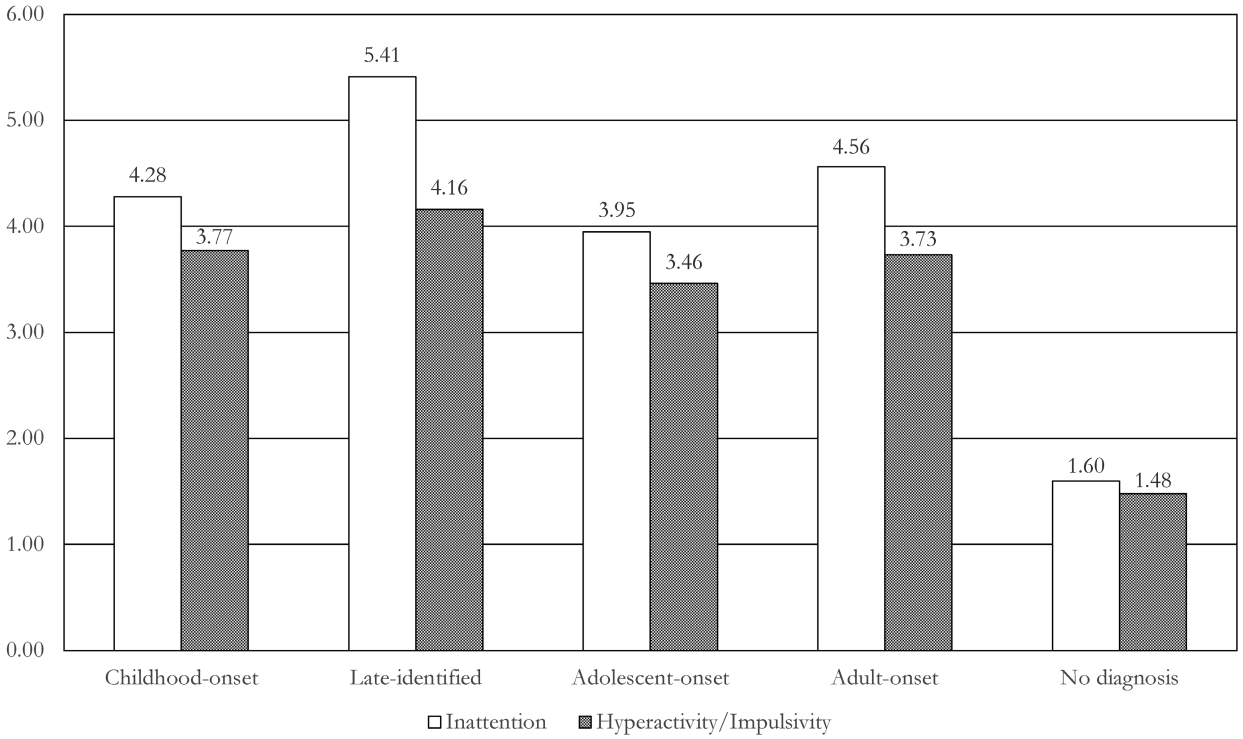

Figure 2. Current inattention and hyperactivity/impulsivity symtoms across onset patterns.

$F(3, 1068) = 9.52$, $p < .001$, $\eta_p^2 = 0.03$, with a small effect size. Post-hoc analyses demonstrated that the late-identified ($M = 5.41$, $SD = 2.80$) group reported significantly more IA symptoms than the child-onset ($M = 4.28$, $SD = 2.97$), adolescent-onset ($M = 3.95$, $SD = 2.82$), and adult-onset ($M = 4.54$, $SD = 2.90$) groups (see Figure 2). Similarly, for impairment, the main effect of group was significant, $F(3, 1068) = 21.97$, $p < .001$, $\eta_p^2 = 0.06$, with a medium effect size, such that the late-identified group reported the highest levels of current impairment and the adolescent-onset group reported the lowest levels. Post-hoc analyses demonstrated that the late-identified ($M = 3.46$, $SD = 0.84$) group reported significantly more settings of impairment than individuals in all other groups: child-onset ($M = 3.10$, $SD = 1.03$), adolescent-onset ($M = 2.65$, $SD = 1.15$), and adult-onset ($M = 2.88$, $SD = 1.06$). Further, individuals in the child-onset group reported significantly more settings of impairment than individuals in the adolescent-onset group. For impairment, the main effect of sex was also significant, $F(1, 1068) = 8.05$, $p = 0.005$, $\eta_p^2 = 0.01$; i.e., small effect), such that women reported impairment in more settings than men. For HI, there were no significant effects of group or sex. None of the group by sex interactions were significant for any of these variables.

Persistence of ADHD by onset pattern

We conducted some additional comparisons of these four groups. We calculated the percentage of participants who would currently meet criteria for ADHD based on the number of symptoms (i.e., 5+ symptoms of IA and/or HI) and impairment (i.e., impairment endorsed in 2+ settings) for each group. We did not consider age of symptom onset because this was already accounted for in how the groups were defined. We found that 52.6% of the child-onset group, 70.9% of the late-identified group, 43.9% of the adolescent-onset group, and 54.0% of the adult-onset group currently reported five or more symptoms of IA and/or HI and 2+ settings of impairment.

We then conducted a chi-square analysis to determine whether there were significant differences in the percentage who would meet current ADHD criteria across these groups, $\chi^2 (3, n = 1076) = 37.63$, $p < .001$. Post-hoc comparison of standardized residuals indicated that those in the late-identified group met this diagnostic standard for current ADHD significantly more frequently than other groups, and those in the adolescent-onset group fell below the diagnostic cutoff significantly more frequently (both $p < .001$).

DISCUSSION

Various definitions of late-onset ADHD have been adopted across prior studies (see Solanto, 2021 for a review), and we posit that more specific and consistent labeling of cases that do not fit the traditional child-onset pattern defined in the *DSM-5* may help us to better understand this phenomenon. We used data from a large sample of adults on Amazon's MTurk who resided in the US and who ranged in age from 18 to 80. Our first set of analyses focused on documenting that the differences between those without past ADHD and those with self-reported ADHD diagnosis of any timing (i.e., child-onset, late-identified, adolescent-onset, adult-onset) were

meaningful. As a whole, self-report of ADHD diagnosis was a robust predictor of risk for current, adult-persistent ADHD; across all ADHD-onset categories defined by these self-reports, 55.4% met the *DSM-5* standards of having five or more current symptoms of IA and/or HI and significant impairment in two or more settings. It is notable that this was the case even though some of these ADHD-onset categories (i.e., adolescent-onset, adult-onset) would not meet formal *DSM-5* criteria. In addition, women in this sample reported higher levels of IA and impairment than men.

Next, group by sex analyses clearly suggested that current IA and impairment is highest in those who self-reported symptoms in childhood that seem to have been unrecognized and were only identified through diagnosis in adulthood (i.e., late-identified group). This group also had, by far, the highest rate of current ADHD (70.9%, next highest being adult-onset at 54.0%), as assessed by self-report of symptoms and impairment. Adults diagnosed in childhood have been shown to under-report their symptoms as compared to their parents (Sibley et al., 2012); however, it should be noted that the Sibley et al. sample consisted mostly of men (i.e., 87%). In addition, part of the inclusion criteria for the late-identified group may inherently and naturally bias this group toward reporting *more* current, persisting symptoms: the very fact that these participants were judged by a professional to have above-threshold symptoms and impairment in adulthood.

Across all analyses, women with a lifetime ADHD diagnosis reported higher impairment and IA than men. This could be a result of women being more insightful about their symptoms and impairment (i.e., women more accurately reporting and men under-reporting; Breda et al., 2020) or men being more accurate about their symptoms and impairment (i.e., men more accurately reporting and women over-reporting; Jaconis et al., 2016). It also could be a true difference with women having more symptoms and impairment than men; however, this interpretation is inconsistent with literature suggesting that the male-preponderance in childhood ADHD is likely due to differences in genetic liability (Arnett et al., 2015). It is also the case that girls are more likely to have the predominantly inattentive presentation whereas boys are more likely to have the combined presentation (Biederman et al., 2002). As individuals develop, HI symptoms tend to wane more than the IA symptoms (Biederman et al., 2000; Holbrook et al., 2016). This could contribute to a change in the differential sex prevalence rate in adulthood with women and men being more equally impacted.

Our first important conclusion is that, as expected, groups that could be described as late-onset have distinct differences, and this in turn implies that researchers and clinicians would do well to more specifically define cases that do not fit the exact timing of the *DSM-5* criteria. As is seen in other disorders that can emerge in childhood (Hammen et al., 2008; Lahey et al., 1999), it appears from this data that early onset of ADHD symptoms, regardless of the timing of diagnosis per se, can be associated with a poor prognosis, in this case marked specifically by extent of impairment in adulthood. It is more a rule than an exception that individuals with early-presenting symptoms of psychological disorders tend to have more serious and persistent psychopathology (Hammen et al., 2008; Lahey et al., 1999). It is of note that this may be interpreted as providing some support for the current inclusion of an age-of-onset criterion in the diagnostic standards for ADHD; however, more research is needed on whether the age of 12 is the precise cut-off that should be used.

What is at least equally interesting is that those reporting early-appearing symptoms (i.e., before 12) who also report having been diagnosed with ADHD in adulthood had a current symptom profile that was marked by the highest IA among the ADHD onset groups. This suggests that adult-onset cases should be differentiated from those who are simply late-identified. It is worth remarking that the late-identified group has an unusual sex ratio, with women higher than men (i.e., 1.6 female:1 male). Further, the group reporting the second-highest current IA, the adult-onset group, was equally common across sexes (1.1 female:1 male). These statistics stand in contrast to the child- and adolescent-onset groups, which were predominantly male (1.8 male:1 female and 2.2 male:1 female, respectively). This fits with existing literature suggesting that girls with ADHD tend to present more often with predominant IA, which is less disruptive and may be associated with under-identification in childhood (Biederman et al., 2002). IA, however, has been shown to be the most developmentally persistent cluster of ADHD symptoms (Wilens et al., 2009), and it is possible that girls with elevated IA may have eluded clinical attention and grown to experience marked impairment in adulthood at which time they finally seek evaluation.

Overall, cases that are characterized as late-identified seem to have high relative severity, increased representation of women, and particularly elevated IA, and may be particularly worthy of clinical and research focus. There are several factors that may impact the age of ADHD diagnosis and thus lead to an individual being late-identified. First, given that the majority of existing childhood ADHD literature includes predominately-male samples, it is possible that women are more likely to be diagnosed in adulthood given the lack of understanding of symptom presentation in young girls. Another possibility is that individuals who were exhibiting symptoms of ADHD in childhood but were not diagnosed until adulthood (i.e., late-identified) did not receive clinical attention due to a lack of disruptive symptoms. Previous literature suggests that children with comorbid ADHD and oppositional defiant disorder (ODD) or conduct disorder (CD) are more likely to be diagnosed at a younger age compared to those without comorbid externalizing symptoms (Efron et al., 2016). It is also possible that individuals in the late-identified group did not have access to services during childhood due to lack of healthcare coverage, stigma surrounding mental health in their families or cultures, or coming from lower socioeconomic backgrounds. Thus, these late-identified individuals likely met criteria for ADHD during childhood but were not diagnosed until adulthood.

In addition to the aforementioned reasons for individuals being late-identified, it is possible that these individuals were late-identified due to not meeting full diagnostic criteria until adulthood. This group of individuals may have eluded a diagnosis of ADHD until adulthood due to having various protective factors. For example, they may have above average IQs that serve as a compensatory mechanism for their ADHD-related impairment. In this case, these individuals may not experience significant impairment until adulthood (e.g., when environmental demands increase). Thus, they may have had protective factors and compensatory strategies that mitigated their levels of impairment until adulthood. Although we have traditionally focused on the most disruptive cases of the disorder (i.e., child-onset ADHD with HI), we are now recognizing that individuals with less conspicuous cases can also be suffering from a version of this disorder that deserves clinical attention, such as those with late-identified ADHD. As awareness increases and our diagnostic tools become more sensitive, it is hoped that these individuals will be identified earlier. Ultimately, this could affect the sex distribution of ADHD if girls are being disproportionately underidentified in childhood because they are less likely to have comorbid HI or other disruptive behaviors (e.g., oppositionality and conduct problems).

It is important to keep in mind that the symptom profiles available in the current study are based on self-report. Prior research (e.g., Sibley et al., 2012) has suggested that the reports of adults with child-diagnosed ADHD, at least, tend to diverge from those of parents. Thus, all of the current results regarding symptomatic differences across onset categories should be interpreted as exploratory pending verification with collateral reports (e.g., significant others, parents). Although we were not able to conduct full evidence-based assessments with our MTurk sample, we were able to acquire a very large sample size, and there is precedent with other published studies on this topic relying on questionnaire data to distinguish subgroups of ADHD participants with varying onset (e.g., Riglin et al. 2022).

Relatedly, with these limited data it is impossible to know whether all reported ADHD symptoms can be attributed conclusively to that syndrome or whether they might be due more to comorbid or primary depression, anxiety, or other psychological or medical disorders (e.g., hyperthyroidism). Despite its limitations, as noted above, MTurk has been shown to provide a robust and valid tool for data collection and seems to be representative of the population as a whole, and specifically so with regards to the manifestation of ADHD (Wymbs & Dawson, 2019).

One additional limitation is that given the brief nature of the MTurk survey, we were not able to collect data such as ethnicity, socioeconomic status, current or past psychosocial or medication treatment, family support regarding ADHD, or type of diagnosing practitioner. As researchers engage in further work that aims to clarify distinctions among the groups of adults who present with ADHD, it will be important to tap these constructs and examine how they may predict or otherwise be associated with ADHD in this population.

While more research is needed in this vein, we believe the evidence from this exploratory study and other existent research suggests that researchers and clinicians should use more precise language when considering various patterns of late-onset ADHD, and, in fact, that this term is too vague. Specifically, while late-onset ADHD could be used to specify those whose symptoms first occur at or after after the *DSM-5* age-ceiling criterion of 12 years, these current data suggest that differentiation between adolescent-onset and adult-onset ADHD subgroups may be important. Adolescent-onset ADHD might be more similar to child-onset ADHD and in fact represent a kind of tail end of cases that are relatively mild and difficult to identify. Over time, comparison of these groups will provide more data that informs the appropriateness of the diagnostic criterion for age. Our limited data also suggest that the adult symptom levels and severity of those with adolescent-onset does not differ from those with clear child-onset, which could be interpreted as a strike against the 12-year-old cutoff. What is more, those in the late-identified group should be conceptually distinguished from others, both because they formally meet *DSM-5* criteria for diagnosis, but also because they may represent a particularly high-risk segment.

In clinical settings, diagnosis becomes especially thorny when adult clients present with clinically significant ADHD traits as well as symptoms of anxiety or depression. In keeping with the currently established standards, we would still counsel that without several symptoms of ADHD that emerge in childhood, diagnosis of ADHD should not be considered. However, a diagnosis of Other Specified ADHD (*DSM-5* code 314.01) remains possible if one notes that the age of onset criterion is not met. Caution should be exercised in this decision, using evidence-based assessment strategies and ruling out the possibility of malingering or another psychological condition that can explain the presence of a preponderance of the ADHD symptoms.

Another option that can apply here has been recommended by Youngstrom and colleagues (2005) in the assessment and diagnosis of bipolar disorder. They asserted that we should consider "extending the window of assessment" and that it is acceptable, in some circumstances, to conclude that we are not sure whether an individual meets criteria for a disorder (Youngstrom et al., 2005, p. 438). In these cases, we might provide treatment for comorbid concerns (e.g., anxiety, depression, sleep problems) as well as organization, time management, and planning skills training for IA symptoms and continue collecting information to inform our diagnostic impression during the course of treatment.

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