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ORIGINAL CONTRIBUTION

# Metacognitive and motivation deficits, exposure to trauma, and high parental demands characterize adolescents with late-onset ADHD

**Margaret H. Sibley[1,2] · Mercedes Ortiz[1] · Paulo Graziano[1] · Anthony Dick[1] · Elena Estrada[1]**

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## Abstract

The objective of this study is to evaluate support for three hypotheses about the etiology of adolescent-onset ADHD symptoms: (1) a "cool" cognitive load hypothesis, (2) a "hot" rewards processing hypothesis, and (3) a trauma exposure hypothesis. Participants ($N=50$) were drawn from two public high schools in a culturally diverse metropolitan area. A detailed procedure for identifying and confirming late-onset ADHD cases is described. Adolescents with late-onset ADHD ($n=15$) were identified and compared to childhood-onset ($n=17$) and non-ADHD classmates ($n=18$). Adolescents and parents completed measures of neurocognition, rewards' processing, clinical profile, and environmental demands. Late-onset cases were clinically and neurocognitively indistinguishable from childhood-onset cases; however, they experienced higher demands from parents ($d=1.09$). Compared to the non-ADHD group, late-onset cases showed significant deficits in metacognition ($d=1.25$) and academic motivation ($d=0.80$), as well as a pronounced history of multiple trauma exposure (OR 11.82). At 1-year follow-up, ADHD persisted in 67.7% of late-onset cases. Late-onset cases (26.7%) were more likely than childhood-onset cases (0.0%) to transfer to alternative schools by 1-year follow-up. Multiple factors may contribute to adolescent-onset ADHD. Adolescents with metacognition and motivation deficits may be at greatest risk for the late-onset ADHD phenotype, particularly in highly demanding environments. Exposure to traumatic stress may play a key role in the exacerbation of existing deficits or onset of new symptoms. Late-onset ADHD was persistent in most cases and associated with higher risk for school disengagement than childhood-onset ADHD. Further work is needed to better understand the etiologies of late-onset ADHD symptoms.

**Keywords** Adolescence · ADHD · Late-onset · Cognition

## Introduction

Since its initial appearance in the Diagnostic and Statistical Manual of Mental Disorders (DSM), Attention-Deficit/Hyperactivity Disorder (ADHD) has been conceptualized as a chronic neurodevelopmental disorder with biological origins in childhood [1]. The current DSM-5 ADHD nosology reflects this view in its B criterion, which requires evidence of several inattentive or hyperactive/impulsive symptoms prior to age 12 [2]. Yet, the notion that ADHD is strictly a childhood-onset disorder has undergone increasing scrutiny in recent years. Beginning in 2015, a series of birth-cohort studies (from the United Kingdom, Brazil, and New Zealand) reported that 2.5–10.7% of their samples experienced clinically significant ADHD symptoms and related impairments in adulthood, in the absence of childhood ADHD [3-5]. Skeptics criticized the cursory nature of birth-cohort diagnostic assessments, suggesting that ADHD symptoms may have been missed in childhood or conflated with other etiologies in adulthood [6-8]. To address this possibility, the Multimodal Treatment of ADHD (MTA) group scrutinized comprehensive psychiatric data collected over 15 years of long-term follow-up (ages 10–25) and confirmed that a handful of non-ADHD individuals in its comparison group (2.1%) showed onset of ADHD after age 12 [9]. Thus, late-onset ADHD appears to be a valid phenomenon.

✉ Margaret H. Sibley
margaret.sibley@seattlechildrens.org

[1] Florida International University, Miami, USA

[2] Department of Psychiatry and Behavioral Sciences, University of Washington School of Medicine, Center for Child Health, Behavior, and Development, Seattle Children's Hospital, 2001 8th Ave., Suite 400, Seattle, WA 98121, USA

Despite documentation of its occurrence, the roots of this phenomenon remain poorly understood. It is not clear if the late-onset phenotype shares a diathesis with childhood-onset ADHD or represents a completely separate entity. In the wake of the birth-cohort studies, continuing research on late-onset ADHD revealed additional clues. The MTA group ruled out most adult-onset cases due to alternative symptom explanations. Its remaining late-onset cases typically first exhibited symptoms between ages 12 and 15, with most cases experiencing remission by age 19. They also found that a majority of late-onset symptoms were reported in the school context [9]. Secondary analyses of birth-cohort data revealed that individuals with childhood- and late-onset ADHD show indistinguishable clinical profiles at age 18 follow-up [10]. In contrast, childhood polygenetic and cognitive testing revealed that late-onset cases do not share the signature risks associated with childhood-onset ADHD [3, 11, 12].

In concert, these findings suggest that individuals with late-onset ADHD appear typically developing in childhood, but are indistinguishable from childhood-onset cases by the cusp of young adulthood. Thus, endogenous and exogenous factors in adolescence may be key to understanding the etiology of late-onset ADHD. Adolescence is the chief onset period for a range of psychiatric disorders that reflect dysfunction in emotion regulation, inhibitory control, and/or information processing [13, 14]. Etiological models for adolescent-onset psychopathology suggest that new-onset mental health symptoms can emerge due to adolescent-specific developmental and environmental factors [15]. Thus, late-onset symptoms may emerge if brain regions associated with ADHD are strained by adolescent contextual or developmental factors.

## Neurocognitive model of ADHD

Prevailing models of ADHD delineate two neural pathways that contribute to cognitive and behavioral symptoms. The first is a "cool" executive functioning (EF) deficit associated with mesocortical dopamine circuits and impairments in cognitive control. These functions include lower order EFs such as working memory and inhibitory control, as well as higher order EFs such as cognitive flexibility, metacognition, and planning [16, 17]. The second is a "hot" rewards processing deficit associated with cortical–striatal dopamine loops and difficulties with delay discounting, delay aversion, risky decision-making, and motivation [16, 17]. The "cool" circuits typically correlate with inattentive symptoms, whereas the "hot" circuits often correlate with hyperactive/impulsive symptoms. As such, individuals with ADHD may experience dysfunction in one or both circuits with individual differences in how deficits manifest [16, 17]. Below, we outline three ways that the adolescent context may disrupt functioning in these circuits, potentially producing adolescent-onset ADHD symptoms.

## Hypothesis 1: increased cognitive load exacerbates "Cool" EF vulnerabilities

Adolescence is characterized by increased academic demands, including heavier workloads, less guidance from teachers, and a new expectation for self-regulated learning [18, 19]. If this increased cognitive load surpasses one's capabilities, an adolescent may experience new difficulties with concentration, memory, or self-regulation. Individuals with certain cognitive vulnerabilities may be at highest risk for these environmentally triggered ADHD-like symptoms. Vulnerabilities might include slow cortical maturation relative to adolescent norms, subclinical EF deficits, or below average intellectual functioning [20]. Under extremely demanding conditions, even individuals with neurotypical cognitive abilities may experience impairments [21]. In support of this hypothesis, the transition to secondary school is associated with a spike in ADHD symptoms among individuals with and without ADHD [22]. Furthermore, adolescent-onset symptoms in the MTA study were most commonly reported by teachers and often desisted at the conclusion of high school [9].

## Hypothesis 2: adolescent rewards response mimics "Hot" rewards processing deficits

Adolescent rewards processing is qualitatively distinct from that of children and adults and implicated in adolescent-limited impulsivity and sensation seeking [23]. Evidence suggests that some adolescents experience heightened difficulties suppressing disadvantageous responses to immediately rewarding stimuli. This inability to delay gratification is associated with impulsive behaviors in adolescence [24]. Some adolescents also experience a peak in sensation seeking characterized by preference for low probability high payouts over gradual low payouts that maximize long-term gain [25]. This urge is posited to underlie adolescent-limited risk behaviors [26]. Despite these developmental trends, adolescent development is heterotypical, and not all teenagers may experience dysfunctional rewards processing [24]. It may be the case that adolescents with particularly heightened rewards responses show motivational and behavioral difficulties that mimic ADHD symptoms or exacerbate existing cognitive vulnerabilities. In support of this hypothesis, one study demonstrated that sensation seeking in childhood predicted adolescent-onset ADHD symptoms [27]. Furthermore, a majority of adolescent-onset cases detected in the MTA study experienced symptom remission by age 19, when adolescent-limited risk behaviors also typically subside [9, 28]. Adolescent-onset rewards processing deficits

also could emerge due to adverse experiences, rather than pubertal changes [29, 30].

### Hypothesis 3: exposure to traumatic stress produces ADHD-like self-regulatory deficits

Ongoing traumatic stress exposure disrupts a number of self-regulatory processes related to cognition, emotion, and behavior [31]. However, traumatic stress experienced during adolescence may be amplified by the brain's increased plasticity, which creates heightened environmental sensitivity and vulnerability to psychopathology [32]. Traumatic stress exposure in adolescence may also interfere with the process of pruning [33]. If disruptions to neural reorganization impact "cool" EF or "hot" rewards processing regions, symptoms that resemble ADHD may emerge. In support of this hypothesis, negative experiences in adolescence have a greater influence on ADHD symptom trajectories than childhood factors [34] and expression of trauma-related cognitive and behavioral symptoms is often indistinguishable from ADHD [35].

### Present investigation

The present study investigates the hypotheses above by examining clinical characteristics, neurocognitive profiles, and environmental experiences of late-onset ADHD cases compared to non-ADHD and childhood ADHD peers. Participants ($N = 50$) were drawn from two public high schools in a culturally diverse metropolitan area. To evaluate support for hypothesis 1, we investigated group differences in cognitive vulnerabilities (i.e., working memory, cognitive flexibility, metacognition, inhibitory control, processing speed, and full-scale IQ). We hypothesized that late-onset ADHD would be associated with increased cognitive vulnerabilities compared to the non-ADHD group, but fewer cognitive vulnerabilities than the childhood-onset group. We also examined group differences in environmental demands and hypothesized that individuals in the late-onset ADHD group would experience higher environmental demands (i.e., parental academic expectations and extracurricular demands) than the childhood-onset group (but equal demands to the non-ADHD group). For hypothesis 2, we investigated differences in rewards processing functions (i.e., risky decision-making, delay discounting, and motivation). We hypothesized that the late-onset group would experience significantly greater deficits in rewards processing than the non-ADHD group, but equal deficits to the childhood-onset group (in the absence of childhood trait impulsivity). For hypothesis 3, we assessed group differences in trauma exposure. We hypothesized that the late-onset ADHD group would experience significantly higher rates of trauma exposure than both the childhood-onset and non-ADHD groups. One-year follow-up was also conducted to assess persistence of late-onset ADHD symptoms and group differences in school disengagement.

## Method

### Participants

Participants were regular education ninth grade students ($N = 50$) at two public high schools in a culturally diverse metropolitan region in the eastern United States. Students with ADHD ($n = 32$) were recruited from a larger trial on academic interventions for high school students with ADHD symptoms. At baseline, parents of participants at two schools in the trial were approached with an opportunity to participate in the current study, which included an extended cognitive, behavioral, and neuroimaging battery designed to study ADHD symptom expression in adolescence. Out of 48 eligible students, 36 consented to the extended battery, and four were excluded due to not meeting criteria for ADHD (which was not a requirement of the larger trial). Participating and non-participating students with ADHD showed no differences in free/reduced lunch status, gender, ethnicity, medication status, GPA, or IQ (all $p > 0.20$). Non-ADHD participants ($n = 18$) were recruited from the same classrooms as ADHD participants and were matched to ADHD participants by school and demographic profile. There were no significant group differences in age, free/reduced lunch status, gender, or ethnicity (see Table 1). However, non-ADHD participants were more likely than both ADHD subgroups (described below) to have a parent with at least a 2-year college degree. As a result, parent education level served as a covariate in all analyses that included the non-ADHD group.

### Procedures

#### ADHD group recruitment

Regular education ninth grade teachers at two high schools were asked to nominate students who displayed symptoms of ADHD in their classrooms. Teachers obtained written parental permission to nominate and completed a DSM-5 ADHD checklist and measures of academic impairment [36-38]. Students were eligible for participation in the larger trial if they displayed at least four symptoms of either inattention or hyperactivity/impulsivity and significant academic impairment, defined as meeting two of the following four criteria: (1) at least one D or F in a core academic class; (2) at least 20% of assignments missing in one class; (3) at least a "3" on the academic impairment item of the 0–6 teacher Impairment Rating Scale [36]; or (4) elevated academic

**Table 1** Demographic characteristics of the sample

| | Childhood-onset ($n=17$) | Late-onset ($n=15$) | Non-ADHD ($n=18$) | $p$ |
|---|---|---|---|---|
| Male (%) | 76.5 | 60.0 | 66.7 | 0.602 |
| Race/ethnicity (%) | | | | 0.357 |
| African-American | 29.4 | 20.0 | 5.6 | |
| Hispanic (any race) | 58.8 | 60.0 | 77.8 | |
| White, non-Hispanic | 5.9 | 0.0 | 0.0 | |
| Mixed race | 5.9 | 20.0 | 16.7 | |
| Free/reduce lunch (%) | 94.1 | 100.0 | 83.3 | 0.197 |
| Age *M* (SD) | 14.59 (0.79) | 14.87 (0.74) | 14.50 (0.79) | 0.387 |
| Parent Ed: AA or higher (%)* | 41.1 | 33.4 | 76.5 | 0.005 |

*$p<0.05$

problems on the teacher Adolescent Academic Problems Checklist (AAPC; four items endorsed as "pretty much" or "very much") [35]. Participants were also required to demonstrate an IQ ≥ 70 on the Wechsler Abbreviated Scale of Intelligence, 2nd edition (WASI-II) [39]. Parents of 48 enrolled participants in the larger trial were phoned by project staff to present the current study opportunity.

In addition to the criteria above, participants in the current study were required to meet DSM-5 A (symptom count) and C–E criteria (impairment, pervasiveness, ruling out other disorders) for ADHD according to combined report on a parent and adolescent semi-structured diagnostic interview (Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version-DSM-5; KSADS-PL) and teacher symptom and impairment ratings [2, 40]. An item-level "or" rule was used to determine symptom presence [41]. Independent diagnoses were made by two licensed clinical psychologists and all cases of disagreement were resolved through discussion.

### Age of onset

The B criterion (age of onset) determined categorization as a childhood- or late-onset case. To carefully assess age of onset, we utilized supplemental probes on the K-SADS-PL building on similar retrospective reporting procedures described by Chandra et al. [42]. For each ADHD symptom on the K-SADS-PL, parents and teens independently reported the age at which the symptom first appeared and the age at which the symptom was most severe. This information was used to build a comprehensive timeline of each participant's symptom-onset and escalation, incorporating both informants' recollections. We utilized a strict definition for late-onset ADHD, requiring participants to demonstrate two or fewer symptoms of both inattention and hyperactivity/impulsivity prior to age 12 according to both parent and teen report (using an item level "or" rule). This definition is consistent with the wording of the DSM-5 B criterion, symptom count norms for children without ADHD, guidelines for assessing ADHD in adolescence, and conceptualization of late-onset ADHD as a low prevalence disorder [2, 5, 9, 10, 41].

### Non-ADHD group recruitment

A research assistant visited all regular education classrooms with at least one participant in the ADHD groups and provided a brief presentation on the current study, which was described as an investigation of the teenage brain at the transition to high school. The research assistant distributed an informational flyer and a parent permission to contact form. Teacher reports of ADHD symptoms were also obtained. Students were eligible for the non-ADHD group if they: (1) possessed an IQ ≥ 70, (2) possessed three or fewer current symptoms of inattention and three or fewer symptoms of hyperactivity/impulsivity according to combined reports (using an "or" rule) on the K-SADS-PL and teacher ratings, (3) had no evidence of ADHD in childhood (using the procedures described for the ADHD group), and (4) increased the non-ADHD group's similarity to the ADHD group (based on sex, ethnicity, and school). Comparison participants were permitted to display academic impairment and mental disorders other than ADHD.

To promote demographic equivalence, comparison recruitment intentionally lagged behind ADHD group enrollment. As they were screened, senior research staff members met to review each potential comparison participant to see if the participant was demographically appropriate for the study. Potential comparison participants were examined on three demographic characteristics: (1) school (2) gender, and (3) ethnicity. A comparison participant was deemed study-eligible if his or her enrollment increased the comparison group's demographic similarity to the ADHD group. At the end of the recruitment process, the ADHD and comparison groups were equivalent on the three demographic variables noted above. This procedure previously has been used in

ADHD longitudinal studies (i.e., Pittsburgh ADHD Longitudinal Study, PALS) to recruit demographically equivalent non-ADHD peers.

### Data collection

Participants and parents completed two study visits of approximately 3 h each. Visit 1 was held at the university or adolescent's home (according to parent preference). Visit 2 was held at the university neuroimaging center. Parents and participants each received $100 for completing visit 1. For visit 2, parents received $50 to offset transportation costs and students received $100 and a photographic image of their brain for participation. Written informed parental consent and youth assent were obtained for all participants. During visit 1, students completed cognitive tasks, a semi-structured diagnostic interview (K-SADS-PL) and self-ratings, while parents completed parent ratings and the K-SADS-PL [38]. During visit 2, students completed a neuroimaging protocol. Neuroimaging data were not utilized in the present investigation. All participants who received psychoactive medication underwent a 24-h washout period prior to both visits. A follow-up visit was conducted with late-onset cases 1 year after the initial assessment. Parents, teachers, and adolescents provided symptom and impairment reports electronically. Each informant received $20 for completing follow-up ratings. Participation in the 1-year follow-up was 86.7%. For both ADHD groups, official records were obtained from the school district to assess school placement in 10th grade. These records were obtained for 100% of participants.

## Measures

### Clinical profile

Trained interviewers administered the full K-SADS-PL to parents and teens [40]. This measure was used to assess DSM-5 diagnoses (including ADHD). Interviews were conducted in Spanish or English, according to the informant's preference. Interviewers were extensively trained through several weeks of didactic instruction, role plays, shadowing at least three assessments with previously trained interviewers, and meeting an inter-rater reliability criterion for symptom endorsement (at least 90% agreement with a trained rater) prior to conducting independent interviews. During the interview period, weekly supervision was provided to prevent rater drift. All K-SADS-PL interviews were audio recorded for the purpose of supervision and to inform final diagnoses, which were made by two licensed clinical psychologists. Clinicians administering the K-SADS-PL considered both parent and youth report when determining symptom presence and were trained to query discrepancies prior to final determination. Assessed DSM-5 diagnoses included 12 mood disorders, five psychotic disorders, 12 anxiety disorders, four eating disorders, 13 developmental disorders, and two substance use disorders. The number of comorbidities was calculated as the total number of mental disorders other than ADHD for which full diagnostic criteria were met (after applying differential diagnoses). Medication status, previous ADHD diagnosis, and ADHD subtype were also assessed using the K-SADS-PL.

Teacher ratings of ADHD symptom count were measured using a standard DSM-5 ADHD symptom checklist [38]. Teachers rated symptoms of ADHD as 0 (*not at all*)–3 (*very much*). A symptom was rated as present if endorsed as 2 (*pretty much*) or 3 (*very much*). Psychometric properties of the measure are very good, with empirical support for internally consistent IN and HI subscales [38]. Subscale alpha in the current study ranged from 0.95 to 0.96.

Our clinical profile measures also included two indices of impairment. Academic impairment was measured by the participant's current grade point average. Electronic gradebook data were obtained directly from schools. Grade point average was calculated by converting all academic grades (i.e., English, Math, Science, and History) to a 5-point scale (i.e., 4.0 = A, 3.0 = B, 2.0 = C, 1.0 = D, 0.0 = F). Grades were not weighted for class level (e.g., Honors vs. Regular). Family impairment was measured using the parent-report Conflict Behavior Questionnaire-20 (CBQ-20) [41]. Parents rated statements about the parent-teen relationship on a five-point scale from 1-*strongly agree* to 5-*strongly disagree*. The CBQ-20 is a 20-item scale adapted from the 73-item CBQ. The CBQ-20 items best discriminated distressed from non-distressed families. It yields a single score that correlates 0.96 with the CBQ [43]. In this study, alpha was 0.92.

Finally, our clinical battery included two indices of substance use. On the Substance Use Questionnaire (SUQ), adolescents reported their use frequency for alcohol and other drugs during the past 3 years [44]. In this study, lifetime alcohol use was defined as consuming an entire drink of alcohol (not just a sip). Lifetime marijuana use was defined as any use.

### Cognitive vulnerability

Six aspects of executive functioning were measured. Working memory was measured using the National Institute of Health (NIH) Toolbox List Sorting Working Memory Test [45]. In this task, a series of stimuli is presented visually and orally. Participants are instructed to recall the stimuli in the order of size, from smallest to largest. The List Sorting task takes approximately 7 min to administer and test scores consist of total items correct across all trials. This task shows excellent test–retest reliability and convergent and discriminant validity [46]. Cognitive flexibility was measured using the NIH Toolbox Dimensional Change Card Sort Test [45].

In this task, a target visual stimulus must be matched to 1 of 2 choice stimuli according to shape or color. The relevant sorting criterion word, "color" or "shape," appears on the screen. An algorithm weights accuracy and reaction time. A total of 40 trials require 4 min. The task shows excellent developmental sensitivity and convergent validity [47]. Response inhibition was measured using the NIH Toolbox Flanker Task [45]. On this task, participants indicate the left–right orientation of a stimulus presented in the center of the screen while inhibiting their attention to incongruent stimuli on either side. Psychometrics for the flanker task are excellent [47]. The NIH Toolbox Pattern Comparison task was used as a measure of processing speed [45]. This timed task requires participants to compare two pictures and determine if they are the same or different, completing as many items as possible during a 90-s period. This task shows good convergent and discriminant validity [48].

The 32-item metacognition index of the parent-report Behavior Rating Index of Executive Function (BRIEF) measures an adolescent's ability to initiate, plan, organize, self-monitor, and sustain working memory [49]. The BRIEF is a well-validated measure of executive function for youth ages 5–18 [49]. Parents rate 86 items describing youth executive functions on a three-point scale across nine subscales. In the current study, alpha was 0.97 for the metacognition index.

Full-scale IQ was measured using a composite score from the Matrix Reasoning and Vocabulary subtests of the Wechsler Abbreviated Scale of Intelligence-2nd Edition (WASI-II) [39]. The WASI-II is a well-established test that has been validated for use with children, adolescents, and adults.

#### Environmental demands

Parental academic demands were measured using an item commonly utilized in the educational literature: "What grade do you want your child to get on a test?" Response options ranged from "at least an A" (4.0) to "at least a D" (1.0) [50]. Extracurricular demands were quantified as parent report of the average number of hours per day that the student participates in extracurricular activities such as athletics, band, church groups, volunteer work, and school organizations. School demands were held constant across groups, as participants were drawn from the same general education classrooms.

#### Rewards processing

A computerized Iowa gambling task (Hungry Donkey Task) was administered as a measure of risky decision-making [51]. Participants were told to assist the hungry donkey to collect as many apples as possible by pressing one of four keys corresponding to four separate doors. The future yield of each door varied, with higher wins at the high paying doors (A and B), and lower wins at low paying doors (C and D). Selecting door A or B resulted in a gain of four apples, whereas door C or D resulted in a gain of two apples. The number of low-risk doors selected minus number of high-risk doors selected was computed as an index of risky decision-making [51]. The task shows good convergent validity in adolescents [52]. Delay discounting was measured using a computerized Choice-Delay Task in which participants were instructed to make repeated choices between a small variable reward (0, 2, 4, 6, 8, or 10 cents) that would be delivered immediately (i.e., after 0 s) and a large constant (10 cents) reward that would be delivered after a variable delay of 0, 5, 10, 20, or 30 s [53]. After completion of the task, participants received the total earnings from the examiner. The total amount of money earned served as an index of delay discounting. This task shows developmental sensitivity and correlates with symptoms of ADHD [53, 54]. The Expectancy-Value Theory of Motivation Measure-Student Version (EVTMM) is a gold-standard self-report measure of student motivation with excellent psychometric properties that consists of 11 items measured on a 5-point scale [55]. This measure previously was validated in a sample of adolescents with ADHD [56]. The two importance items (i.e., "for me being good in school is important…" "compared to most of your other activities, how important is it for you to be good in school…") were averaged to provide an index of academic motivation. In the current study, alpha for this subscale was 0.82.

#### Trauma exposure

Lifetime exposure to 13 categories of trauma (e.g., abuse, violence, car accidents, and loss of a close family member) were queried as a part of the K-SADS-PL Post-Traumatic Stress Disorder screener [40]. Exposure to multiple traumas was defined as endorsement of more than one trauma item. Multiple trauma exposure was selected as an index of trauma after examining sample moments and determining that a binomial distribution best represented the data (i.e., the sample mode was one trauma). For comparison to ADHD age of onset, age of trauma exposure was queried for each reported trauma.

#### Follow-up measures

At 1-year follow-up, parents, teachers, and adolescents completed the DSM-5 ADHD checklist [38] and the Impairment Rating Scale (IRS) [36]. A score of at least "3" on the overall impairment item of the 0–6 Impairment Rating Scale was considered clinically significant according to established norms for the measure [36]. To combine symptom and

impairment ratings across informants, procedures were followed as described for the initial assessment. Official district records provided information about school placement.

### Analytic plan

First, the late-onset and childhood-onset ADHD groups were compared on clinical profile. For continuous indices, group differences were examined using a General Linear Model (GLM) with group (two levels: late-onset ADHD and childhood-onset ADHD) as the independent variable. Cohen's *d* standardized effect sizes were computed using a baseline pooled standard deviation and group means. For binary indices, 2 × 2 Chi-square analyses were conducted.

In testing study hypotheses, orthogonal comparisons were conducted to evaluate differences between (1) the late-onset ADHD group and the childhood-onset ADHD group and (2) the late-onset ADHD group and the non-ADHD group. Parent 2-year degree or higher (dummy coded: no degree = 0, degree = 1) was covaried to account for higher parent education level in the non-ADHD group. For continuous outcomes, we conducted linear regressions; for binary and ordinal outcomes, we employed logistic regression. For continuous outcomes, Cohen's *d* standardized effect sizes were computed using a baseline pooled standard deviation and estimated marginal means after inclusion of the covariate. A false-discovery rate correction was applied within domain and contrast (late-onset vs. childhood-onset; late-onset vs. non-ADHD) to correct for multiple comparisons [57]. For follow-up analyses, we conducted 2 × 2 Chi-square analyses for group (0 = childhood-onset, 1 = adolescent-onset) by 10th grade educational setting (0 = regular high school, 1 = alternative school). One-year persistence of late-onset ADHD was calculated descriptively according to a range of persistence definitions [41].

## Results

### Clinical profile

There were no differences between the late- and childhood-onset groups on any aspect of clinical profile (see Table 2).

### Hypothesis 1: increased cognitive load exacerbates "Cool" EF vulnerabilities

Full results are presented in Table 3. Adolescents with late-onset ADHD did not significantly differ from the childhood-onset group on any indices of cognitive vulnerability. However, the late-onset group had significantly higher parental academic demands than the childhood-onset group ($p = 0.003$, $d = 1.09$). Compared to the non-ADHD group, the late-onset group showed significant deficits in metacognition ($p = 0.001$, $d = 1.25$). The late-onset group also possessed lower full-scale IQs than the non-ADHD group, although this effect did not survive correction for multiple comparisons ($p = 0.049$, $d = 0.73$). The late-onset group did not differ from the non-ADHD group on environmental demands.

**Table 2** Clinical characteristics of childhood and adolescent-onset ADHD groups

| | Childhood-onset | Late-onset | *p* | *d* |
|---|---|---|---|---|
| ADHD subtype | | | | |
| ADHD-PI (%) | 70.6 | 66.7 | 0.811 | – |
| ADHD-C (%) | 29.4 | 33.3 | – | – |
| Lifetime comorbidities *M* (SD) | 1.06 (0.90) | 0.93 (1.33) | 0.714 | −0.11 |
| Lifetime alcohol use % (*n*) | 0.0 (0) | 13.3 (2) | 0.212 | – |
| Lifetime marijuana use % (*n*) | 5.9 (1) | 26.7 (4) | 0.106 | – |
| KSADS symptom count *M* (SD) | | | | |
| Current inattention | 5.00 (2.98) | 3.53 (2.88) | 0.091 | −0.50 |
| Current *H/I* | 1.82 (2.51) | 1.13 (1.60) | 0.266 | −0.33 |
| Teacher symptom count *M* (SD) | | | | |
| Current inattention | 6.82 (1.94) | 7.40 (1.80) | 0.309 | 0.31 |
| Current *H/I* | 2.35 (3.35) | 3.53 (3.29) | 0.217 | 0.36 |
| Academic impairment *M* (SD) | 1.44 (1.10) | 1.16 (0.85) | 0.387 | 0.28 |
| Family impairment *M* (SD) | 2.47 (0.83) | 2.28 (0.83) | 0.442 | −0.23 |
| ADHD medication % (*n*) | 11.8 (2) | 0.0 (0) | 0.274 | – |
| Previous ADHD diagnosis % (*n*) | 29.4 (5) | 13.3 (2) | 0.272 | – |

*d* Cohen's *d* effect size calculated using pooled standard deviation, *ADHD-PI* predominantly inattentive, *ADHD-C* combined subtype

**Table 3** Group differences in cognitive vulnerabilities and environmental demands

| | Childhood-onset | Late-onset | Non-ADHD | Late-onset vs. childhood-onset | | | Late-onset vs. non-ADHD | | |
|---|---|---|---|---|---|---|---|---|---|
| | *M* (SD) | *M* (SD) | *M* (SD) | *b* (SE) | *p* | *d* | *b* (SE) | *p* | *d* |
| Cognitive vulnerability | | | | | | | | | |
| IQ-WISC | 98.62 (11.33) | 91.02 (11.94) | 101.07 (15.52) | 7.60 (4.67) | 0.111 | 0.65 | 10.05 (4.97) | 0.049 | 0.73 |
| Cognitive flexibility-NIH Dimensional change Card Sort Test | 87.36 (10.39) | 90.95 (9.74) | 100.69 (18.18) | − 3.59 (4.87) | 0.465 | 0.36 | 9.74 (5.19) | 0.067 | 0.67 |
| Working Memory-NIH List Sorting Test | 97.68 (15.86) | 98.26 (15.10) | 95.39 (15.01) | − 0.58 (5.48) | 0.916 | 0.04 | − 2.87 (5.84) | 0.625 | − 0.19 |
| Response Inhibition-NIH Flanker Task | 78.29 (10.44) | 81.19 (11.07) | 84.66 (12.96) | − 2.90 (4.12) | 0.485 | 0.27 | 3.47 (4.39) | 0.434 | 0.29 |
| Processing Speed-NIH Pattern Comparison Test | 87.70 (21.54) | 96.12 (25.75) | 106.07 (23.21) | − 8.42 (8.40) | 0.322 | 0.35 | 9.95 (8.95) | 0.272 | 0.41 |
| Metacognition problems-BRIEF parent report | 62.36 (10.34) | 56.32 (14.46) | 42.06 (7.11) | 6.04 (3.67) | 0.107 | 0.48 | − 14.26 (3.91) | 0.001 | 1.25 |
| Environmental demands | | | | | | | | | |
| Parental expectations | 3.11 (0.78) | 3.79 (0.41) | 3.55 (0.51) | − 0.68 (0.21) | 0.004 | 1.09 | − 0.24 (0.23) | 0.219 | 0.52 |
| Extracurricular activities | 0.90 (1.09) | 1.61 (2.37) | 1.30 (1.28) | − 4.99 (4.13) | 0.233 | 0.38 | − 2.14 (4.40) | 0.629 | 0.16 |

*M* estimated marginal mean after controlling for parent education level, *b* unstandardized beta coefficient, *SE* standard error, *d* Cohen's *d* effect size

**Table 4** Group differences in rewards processing

| | Childhood-onset | Late-onset | Non-ADHD | Late-onset vs. childhood-onset | | | Late-onset vs. Non-ADHD | | |
|---|---|---|---|---|---|---|---|---|---|
| | *M* (SD) | *M* (SD) | *M* (SD) | *b* (SE) | *p* | *d* | *b* (SE) | *p* | *d* |
| Delay Discounting-Choice-Delay Task | 322.76 (64.45) | 278.07 (51.01) | 323.53 (66.90) | 44.69(21.88) | 0.047 | 0.77 | 45.46 (23.32) | 0.058 | 0.76 |
| Risky Decision-Making-IOWA Gambling Task | − 3.92 (13.81) | − 0.2.51 (8.04) | 1.15 (10.93) | − 1.44 (3.61) | 0.692 | 0.12 | 3.66 (3.85) | 0.347 | 0.38 |
| Academic Motivation | 3.87 (0.58) | 3.94 (1.07) | 4.61 (0.54) | − 0.05 (0.26) | 0.850 | 0.06 | 0.68 (0.27) | 0.016 | 0.80 |

*M* estimated marginal mean after controlling for parent education level, *b* unstandardized beta coefficient, *SE* standard error, *d* Cohen's *d* effect size

## Hypothesis 2: adolescent reward response mimics "Hot" reward processing deficits

Full results are presented in Table 4. The late-onset group showed lower performance on the delay discounting task than the childhood-onset group, but this effect did not survive correction for multiple comparisons ($p = 0.047$, $d = 0.77$). The late-onset group showed lower levels of academic motivation than the non-ADHD group ($p = 0.016$, $d = 0.80$).

## Hypothesis 3: exposure to traumatic stress leads to ADHD-like self-regulatory deficits

The late-onset and childhood-onset groups did not significantly differ on multiple trauma exposure (86.7% versus 64.7%; $b = 1.24$, SE 0.93, $p = 0.182$, OR 3.44). Compared to the non-ADHD group (33.3% multiple trauma exposure), the late-onset group experienced a significantly higher rate of multiple trauma exposure ($b = 2.47$, SE 0.95, $p = 0.010$, OR 11.82). In the late-onset group, all but one participant with multiple trauma exposure experienced the reported traumas prior to the onset of ADHD symptoms.

## One-year follow-up

Using DSM-5 symptom criteria, late-onset ADHD persistence was 50.0% at 1-year follow-up. Using impairment-based criteria, 67.7% of late-onset cases experienced clinically significant impairment paired with elevated ADHD symptoms (i.e., at least five symptoms of either inattention or hyperactivity/impulsivity) at follow-up. At 1-year

follow-up, participants in the late-onset group (26.7%) were more likely than the childhood-onset group (0.0%) to be moved to an alternative high school for tenth grade [$X^2$ (1) = 5.18, $p$ = 0.023].

## Discussion

Very little is known about the etiology of late-onset ADHD symptoms. In this study, we identified and comprehensively assessed 15 individuals with late-onset ADHD, comparing them to 17 childhood-onset cases and 18 non-ADHD classmates. The resulting late-onset group was clinically and neurocognitively indistinguishable from their childhood-onset counterparts; however, they possessed parents with higher academic expectations. Compared to the non-ADHD group, late-onset cases showed significant deficits in metacognition and academic motivation, as well as a pronounced history of multiple trauma exposure. In approximately two-thirds of cases, there was evidence of persistent late-onset ADHD at 1-year follow-up. Compared to childhood-onset ADHD, late-onset ADHD was also associated with increased risk for academic disengagement (0.0% vs 26.7%) characterized by transfer to an alternative high school by 1-year follow-up.

Late-onset ADHD is a low base-rate phenomenon and adolescents with this phenotype appear less likely to present in clinical settings [9, 10, 58]. Thus, identification and recruitment of adolescents with late-onset ADHD can be challenging. Using an empirically-informed strategy, we successfully identified a meaningful subgroup of late-onset cases by: (1) deliberately recruiting from an age-bracket at the height of late-onset ADHD symptoms (ages 14–15) (2) limiting the sample to general education settings, and (3) using teacher, rather than parent ratings, to screen for late-onset ADHD. Thus, we purposefully sought a sample that contained a high incidence of late-onset cases and this sample cannot be used to estimate late-onset ADHD prevalence. To protect against false-positive cases, we used the MTA methodology to require evidence of clinically significant impairment, cross-situational symptoms, and rule out other disorders and substance use as the source of symptoms [9]. We also augmented an existing retrospective reporting methodology to create procedures for documenting a comprehensive history and timeline of ADHD symptom onset and escalation [42]. Replicating these methods may aid researchers in the recruitment and study of valid late-onset ADHD cases.

We proposed three potential etiologies for adolescent-onset ADHD symptoms: (1) a cognitive load hypothesis, (2) a rewards processing hypothesis, and (3) a trauma exposure hypothesis. Based on our results, all three warrant further study. With respect to cognitive load, late-onset cases demonstrated vulnerabilities in metacognition and potentially IQ (though this effect did not survive false-discovery rate correction). Despite these weaknesses, late-onset students faced regular high school coursework and possessed parents with very high academic expectations (A-; see Table 3). Thus, in line with cognitive load theory, there may be a discrepancy between student ability and environmental demands [21]. Though IQ is conceptualized as a stable construct, it is unclear if the "cool" EF deficits indicated by the metacognitive index were present since childhood [59]. On one hand, schools may not demand regular use of higher order EFs (like metacognition) until the secondary grades, veiling these deficits in elementary school [19]. On the other hand, it is possible that higher order "cool" EF deficits can first emerge in adolescence due to maturational or experiential factors [20]. It is also possible that high parental expectations are a protective factor in elementary school, mitigating "cool" EF-related impairments until the more demanding adolescent context.

With respect to rewards processing, late-onset cases displayed lower academic motivation than the non-ADHD comparison group. In addition, late-onset cases earned characteristically low scores on the delay discounting, but not risky decision-making task (see Table 4). These results are striking given that the relationship between delay of gratification and adolescent school grades is mediated by academic motivation [60]. These reward processing deficits may have been present but unimpairing in childhood, because elementary school students are infrequently required to self-regulate their motivational state [18]. They also may newly emerge due to adolescent brain maturation or adverse experiences [23, 29, 30]. Although most adolescents in our sample exhibited the ADHD-Predominantly Inattentive (PI) presentation, we do not believe that this reduces support for the rewards processing hypothesis; one criticism of the DSM-5 is that its hyperactivity/impulsivity symptoms are developmentally inappropriate for adolescents, misclassifying cases with an ADHD-Combined phenotype as ADHD-PI [38, 41].

Our trauma exposure hypothesis was also supported. Over 80% of the adolescents in the late-onset group experienced multiple trauma exposures prior to the onset of their ADHD symptoms—in the demographically similar non-ADHD group, trauma risk was nearly three times lower. These data support a prominent role of adverse life experiences in the development of late-onset ADHD. Notably, the childhood-onset group also experienced elevated rates of multiple trauma (64.7%); however, a majority of these traumas occurred *after* the onset of childhood ADHD symptoms. This finding is not surprising as childhood ADHD is conceptualized as a risk factor for Post-Traumatic Stress Disorder [61]. Previous work suggests that exposure to trauma and chronic stress may impact both higher order EFs and delay discounting [62, 63]. Therefore, the relationship between ADHD and trauma may be complex and bidirectional: the

pathway through which trauma begets risk for adolescent-onset ADHD may be distinct from the pathway through which childhood ADHD begets risk for later trauma. Furthermore, linkages between our three hypotheses are likely to be present. Future longitudinal work with larger samples should investigate the pathways through which exposure to adverse experiences may lead to the appearance of late-onset ADHD symptoms. This work should include study of three-way interactions between the constructs of trauma, "cool" EFs, and "hot" rewards processing circuits.

At 1-year follow-up, most of the sample (67.7%) demonstrated persistence of elevated ADHD symptoms and impairment, while 50.0% of the sample met formal DSM-5 ADHD symptom criteria. These data are consistent with the MTA findings, which demonstrated that a majority of adolescent-onset ADHD cases experienced impairing symptoms at multiple adolescent assessment points [9]. Our findings also suggest that late-onset ADHD may be associated with a school disengagement trajectory; 4 of 15 adolescent-onset cases were reassigned to an alternative high school in 10th grade due to persistent course failure (compared to 0 of 17 childhood-onset cases). This finding is consistent with other work suggesting that late-onset ADHD may be associated with poorer long-term outcomes compared to childhood-onset ADHD [3]. Further work is needed to understand the mechanisms through which late-onset symptoms are associated with high school disengagement.

The field will struggle to design prospective studies that identify predictive pathways to late-onset ADHD. For one, late-onset cases are impossible to detect prior to the onset of their symptoms. Researchers can rely on samples of convenience, such as existing longitudinal studies; however, these studies are unlikely to serendipitously include ideal measures to test late-onset ADHD hypotheses. Attempts to recruit an original sample prior to symptom onset will require very large sample sizes to capture a meaningful late-onset subgroup. For example, using comprehensive diagnostic methods, the British ALSPAC sample required 4,953 participants to detect 19 cases of late-onset ADHD [11]. Thus, the most efficient way to achieve large samples of late-onset cases is through retrospective follow-back methodology.

Thus, one inevitable limitation of our study was reliance on retrospective report of ADHD symptom-onset and psychiatric history to rule out other disorders as the source of late-onset symptoms. However, we believe that our diagnostic and onset classification methodologies were maximally rigorous for a retrospective design. In addition, we cannot be sure that high environmental demands begot increased cognitive load—in some cases, adolescents may be apathetic to the demands placed upon them. Although the late-onset ADHD group's rewards processing deficits did not manifest behaviorally in childhood, we cannot be certain that onset was related to adolescent development. Our participants primarily hailed from low income or working-class homes—while this may have increased our likelihood of detecting late-onset cases, it also may mean that some findings do not generalize to individuals in higher socioeconomic brackets [63]. Similarly, our culturally diverse sample consisted primarily of ethnic minority participants; thus, findings may not generalize to ethnic groups that were underrepresented in our sample (i.e., adolescents of non-Hispanic European, Asian, and Native American descent). For example, most participants with ADHD did not have a previous diagnosis (see Table 1), which likely reflects decreased mental health service access in ethnic minority youth [64, 65]. Although our non-ADHD participants were systematically selected to increase the comparison group's demographic similarity to ADHD participants, we did not employ a case–control-matching procedure. Finally, our sample size ($N = 50$) was modest and we were only powered to detect large effects. Therefore, some small-to-medium effects may have gone undetected in this study. However, the results of this study remain meaningful, because several large effects were present after Type I error corrections were imposed.

In conclusion, it is likely that multiple factors contribute to late-onset ADHD symptoms and that the late-onset phenotype possesses heterogeneous etiologies. Future work should disentangle how environmental (i.e., parental demands and exposure to trauma) and cognitive risks (i.e., deficits in metacognition and motivation) may be sensitized by the adolescent context, leading to an adolescent-limited form of ADHD [9]. We suggest that this work incorporate data from multiple levels of analysis (i.e., genetics, neuroimaging, tasks of cognition, and behavioral measures). Longitudinal work is also needed to understand trajectories of symptom persistence and desistence among late-onset cases. Clarifying the nature of late-onset ADHD will allow for refinement of the DSM nosology and may aid clinical providers in appropriate diagnosis of individuals who present with first-time ADHD symptoms in adolescence.

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## Compliance with ethical standards

**Conflict of interest** On behalf of all authors, the corresponding author states that there is no conflict of interest.

**Ethical standards** This study was approved by the local university ethics committee and have, therefore, was performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

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