Persistence, Remission and Emergence of ADHD in Young Adulthood:

Results from a Longitudinal, Prospective Population-Based Cohort

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ABSTRACT

Importance: ADHD is now recognized to occur in adulthood and is associated with a range of negative outcomes. However, less is known about the prospective course of ADHD into adulthood, the risk factors for its persistence, and the possibility of its emergence in young adulthood in non-clinical populations.

Objective: To investigate childhood risk factors and young adult functioning of individuals with persistent, remitted and late-onset young adult ADHD.

Design, Setting and Participants: The study sample is the Environmental Risk (E-Risk) Longitudinal Twin Study, a nationally-representative birth cohort of 2,232 twins born in England and Wales in 1994–1995.

Main Outcome Measures: We ascertained ADHD diagnoses in childhood at ages 5, 7, 10, and 12 and in young adulthood at age 18. Childhood predictors of ADHD included pre/perinatal factors, child clinical characteristics and aspects of the family environment. Age-18 outcomes included DSM-5 ADHD symptoms and associated impairment, overall functioning and other mental health disorders.

Results: Among individuals with childhood ADHD (n=247), 21.9% met diagnostic criteria for the disorder at age 18. Persistence was associated with more symptoms and lower IQ in childhood. Persistent individuals had more functional impairment and higher rates of other mental health disorders at age 18 compared to those who remitted. Among individuals with adult ADHD (n=166), 67.5% did not meet criteria for ADHD at any assessment in childhood. Results from logistic regressions indicated that individuals with late-onset ADHD showed fewer behavior problems ($p<.001$) and higher IQ ($p=.001$) in childhood compared to the persistent group; at age 18, however, they showed comparable ADHD symptoms and impairment, and similarly elevated rates of mental health disorders.

Conclusion and Relevance: We identified heterogeneity in the DSM-5 young-adult ADHD population such that this group consisted of a large late-onset ADHD group with no childhood diagnosis, and a smaller group with persistent ADHD. The extent to which childhood-onset and late-onset adult ADHD may reflect different etiologies has implications for genetic studies and treatment of ADHD.

Introduction

To date, adult attention deficit hyperactivity disorder (ADHD) has been conceptualized as a continuation of childhood ADHD. However, recent findings have suggested that for some ADHD may not arise until adolescence or adulthood and may be associated with different risk factors and outcomes than childhood ADHD.[1] In the current study, we take a prospective, developmental approach to clarifying the origins and correlates of young adult ADHD in a general population cohort.

While ADHD was originally described as childhood-limited,[2,3] prospective follow-up studies of clinic-referred children with ADHD indicate that approximately 15% will continue to meet full diagnostic criteria, and an additional 50% will continue to have impairing ADHD symptoms as young adults.[4] These studies have identified childhood risk factors associated with a more persistent course, including higher levels of symptoms, comorbid oppositional-defiant disorder (ODD), lower IQ, and family socioeconomic disadvantage.[5-9] However, the vast majority of follow-up studies of children with ADHD have been conducted with clinical samples, which may not represent the overall ADHD population.[10] Additionally, individuals who do not meet diagnostic criteria in childhood are generally not included in studies following children with ADHD, resulting in a limited understanding of the potential emergence of the disorder in later life.

Our investigation aims to characterize young adult ADHD by examining the persistence of the disorder from childhood to age 18, and its possible emergence in young adulthood. First, we examined childhood predictors of persistence, including pre/perinatal, clinical, and family environmental factors. Second, we assessed whether some individuals who did not have an ADHD diagnosis in childhood developed the disorder by age 18, and described childhood risk factors among these individuals. Third, we investigated the functioning of persistent, remitted, and late-

onset ADHD groups at age 18 to understand how these groups differ or resemble one another in young adulthood.

Methods

Study cohort

Participants were members of the Environmental Risk (E-Risk) Longitudinal Twin Study, a birth cohort of 2,232 British children. The sample was drawn from a larger birth register of twins born in England and Wales in 1994-95.[11] Full details about the sample are reported elsewhere.[12] The E-Risk sample was constructed in 1999-2000, when 1,116 families (93% of those eligible) with same-sex 5-year-old twins participated in home-visit assessments. This sample comprised 55% monozygotic and 45% dizygotic twin pairs; sex was evenly distributed within zygosity (49% male). Families were recruited to represent the UK population with newborns in the 1990s, on the basis of residential location throughout England and Wales and mother's age. Teenaged mothers with twins were over-selected to replace high-risk families who were selectively lost to the register through non-response. Older mothers having twins via assisted reproduction were under-selected to avoid an excess of well-educated older mothers. At follow up, the study sample represented the full range of socioeconomic conditions in the UK.[13,14]

Follow-up home visits were conducted when the children were aged 7 (98% participation), 10 (96%), 12 (96%), and 18 years (93%). Home visits at ages 5, 7, 10, and 12 years included assessments with participants and their mother; we conducted interviews only with participants at age 18 (n=2,066). There were no differences between those who did and did not take part at age 18 in socioeconomic status when the cohort was initially defined (X^2=0.86, p=0.65), age-5 IQ (t=0.98, p=0.33), internalizing or externalizing problems (t=0.40, p=0.69 and t=0.41, p=0.68), or rates of childhood ADHD at ages 5, 7, 10 or 12 (X^2=2.08, p=0.72). With parents' permission, questionnaires

were mailed to the children's teachers, who returned questionnaires for 94% of children at age 5, 93% of those followed up at age 7, 90% at age 10, and 83% at age 12. At age 18, participants were asked to identify individuals who know them well to act as co-informants; 99.3% of participants at age 18 had co-informant data. Study interviewers completed post-assessment questionnaires about their own impressions of the participants' mental health and personality including 6 characteristics related to ADHD. The Joint South London and Maudsley and the Institute of Psychiatry Research Ethics Committee approved each phase of the study. Parents gave informed consent and twins gave assent between 5-12 years and then informed consent at age 18.

Childhood ADHD diagnosis

We ascertained ADHD diagnosis on the basis of mother and teacher reports of 18 symptoms of inattention and hyperactivity-impulsivity derived from DSM-IV diagnostic criteria and the Rutter Child Scales.[15-17] Participants had to have six or more symptoms reported by mothers or teachers in the past 6 months, with the other informant endorsing at least two symptoms. We considered participants to have a diagnosis of childhood ADHD if they met criteria at age 5, 7, 10 or 12. In total, 247 participants (12.1%) met criteria for ADHD across childhood: 6.8% at age 5 (n=131), 5.4% age 7 (n=102), 3.4% age 10 (n=65), and 3.4% age 12 (n=64). Additional information is provided in Supplemental Table 1.

Young adult ADHD diagnosis

We ascertained ADHD at age 18 based on private structured interviews with participants regarding 18 symptoms of inattention and hyperactivity-impulsivity according to DSM-5 criteria.[1] Participants had to endorse five or more inattentive and/or five or more hyperactivity–impulsivity symptoms to be diagnosed; we also required that symptoms interfered with individual's "life at home, or with family and friends" and "life at school or work" as rated 3 or higher on a scale from

"1=mild interference" to "5=severe", thereby meeting criteria for impairment and pervasiveness. The DSM-5 requirement of symptom onset prior to age 12 was met if parents/teachers reported more than 2 ADHD symptoms at ages 5, 7, 10 or 12. Analyses were restricted to 2,040 individuals with ADHD information in childhood and adulthood. A total of 166 (8.1%) participants met criteria for age-18 ADHD. We fitted an ACE model and identified heritability estimate of ADHD symptoms of 35% (95% CI:25-41%). Co-informants rated participants on 8 ADHD symptoms at age 18. Heritability estimates were virtually identical using co-informant reports, indicating that these estimates were not simply an artifact of twins' self-report. Additional information is provided in Supplemental Table 1.

Persistent, remitted, and late-onset ADHD groups

Among individuals who met diagnostic criteria for ADHD in childhood or adulthood, we identified three mutually exclusive groups (Figure 1): individuals with persistent ADHD who met full diagnostic criteria both in childhood and at age 18 (n=54, 2.6% of the total sample); individuals with remitted ADHD who met diagnostic criteria in childhood but did not meet full diagnostic criteria at age 18 (n=193, 9.5%); and individuals with late-onset ADHD who did not meet criteria in childhood but had elevated symptoms and impairment at age 18 (n=112, 5.5%). A total of 1,681 (82.4%) participants did not meet criteria for ADHD in childhood or adulthood. Supplemental Figures 1 shows the distribution of inattentive and hyperactive/impulsive symptoms in childhood and at age 18 among different ADHD groups.

Statistical analyses

We compared individuals with persistent, remitted and late-onset ADHD to non-ADHD controls on *a priori* selected factors using logistic regressions. We contrasted individuals who persisted to those who remitted to identify risk factors for persistence. We compared late-onset individuals to those

who persisted to characterize childhood features of the adult ADHD groups which differ on their childhood ADHD status. We examined functional outcomes at age 18 by comparing each ADHD group to controls. We compared persistent to remitted individuals to examine the impact of ADHD remission on functioning, and persistent to late-onset groups to capture the extent to which these groups differed on age-18 characteristics. We used linear regression to assess whether characteristics associated with persistence were similar when age-18 ADHD symptoms were assessed with co-informant report. Analyses were corrected for the non-independence of twin observations with tests using the sandwich variance estimator in Stata version 11.[18]

Results

Participants who met diagnostic criteria for ADHD in childhood or adulthood both differed from controls on pre/perinatal factors, clinical features and family environment (Table 1).

Childhood characteristics of persistent versus remitted ADHD

Among individuals who met diagnostic criteria for ADHD in childhood, 21.9% still met full criteria at age 18. Few childhood characteristics distinguished individuals with persistent and remitted ADHD (Table 1): persistent individuals had more symptoms across childhood and lower IQ compared to those who remitted. Overall, characteristics of the family environment did not distinguish individuals who persisted from those who remitted, except that families of persistent individuals had comparatively higher maternal warmth and less maternal depression.

Childhood characteristics of late-onset versus persistent ADHD

Among individuals with adult ADHD (n=166), 67.5% had late-onset ADHD. Late-onset individuals were more likely to be female and, controlling for gender, had fewer childhood behavioral problems

and higher IQ compared with persistent individuals (Table 1). Pre/perinatal factors and characteristics of the family environment did not differ between these groups.

Young adult functioning of persistent versus remitted ADHD

At age 18, co-informants (i.e. parents and co-twins), rated individuals with persistent ADHD as having more symptoms compared to remitted individuals, and interviewers rated them as less conscientious, diligent and persevering (Table 2). Individuals with persistent ADHD had higher rates of generalized anxiety disorder, conduct disorder and marijuana dependence compared those who remitted. However the remitted group still showed impairment: compared to controls, individuals with remitted ADHD had more self-rated and co-informant-rated ADHD symptoms, lower life satisfaction and job preparedness, and higher rates of major depression and conduct disorder.

Young adult functioning of late-onset versus persistent ADHD

Individuals with late-onset ADHD differed from the persistent group on few age-18 variables (Table 2). Late-onset individuals had higher age-18 IQ than the persistent group, but the two groups did not differ on life satisfaction, job preparedness, and rates of being in formal education. Late-onset and persistent ADHD individuals did not differ on age-18 psychiatric comorbidity: both had elevated rates of generalized anxiety disorder, conduct disorder, and marijuana dependence. Late-onset individuals had significantly higher rates of alcohol dependence compared to those with persistent ADHD. We examined whether having a co-twin with childhood ADHD conferred increased risk for late-onset ADHD and found no difference in the proportion of individuals who developed late-onset ADHD among those who had a co-twin with childhood ADHD (7.9%) or those who did not (6.0%) ($p=0.39$).

Childhood characteristics of co-informant-rated ADHD symptoms at age 18

As when predicting age-18 ADHD using self-report, the number of childhood ADHD symptoms was the most significant predictor of co-informant-reported symptoms of ADHD at age 18. Maternal stress during pregnancy, male gender, and comorbid CD and higher externalizing score in childhood were also associated with more co-informant-rated symptoms.

Discussion

Our study was particularly well-suited to investigate the persistence and emergence of DSM-5 adult ADHD, given its prospective follow-up of a general population sample of children with and without ADHD from early childhood to young adulthood. We found that ADHD persistence was associated with more ADHD symptoms and lower IQ in childhood. Additionally, we identified heterogeneity in the young adult ADHD population, such that this group consisted of a minority of individuals for whom ADHD persisted from childhood, and a larger proportion who did not meet criteria for the disorder until young adulthood. Our results suggest that adult ADHD is more complex than a straightforward continuation of the childhood disorder.

Persistence and remission of ADHD from childhood to age 18

While we examined a wide range of risk factors, we found persistence to be most strongly associated with severity of childhood ADHD symptoms, consistent with several,[5,19] but not all,[6,20] prospective studies in clinical samples. We also found that lower IQ was associated with persistence. While most pre-/perinatal and family environment factors were associated with the incidence of ADHD in childhood, overall they were not associated with its persistence into adulthood. It may be that remission of ADHD at age 18 is associated with the increased opportunities young adults have to select environments more suited to their ADHD symptoms; in

this way, concurrent lifestyle factors, versus childhood environment, may more important for determining remission of ADHD at age 18.

The majority of individuals with ADHD in childhood no longer met full criteria at age 18. However, this remitted group reported interference with functioning due to their ADHD symptoms. In addition to showing more ADHD symptoms, the remitted group continues to have lower IQ and higher rates of depression and conduct disorder, which could also negatively impact functioning at age 18. While this group no longer meets full diagnostic criteria for ADHD, residual ADHD symptoms, comorbidity, and functional impairment suggest that they may require clinical attention.

ADHD among women and girls

While boys are more commonly diagnosed with ADHD than girls in childhood, epidemiologic surveys of adult ADHD identify a gender ratio closer to 1:1.[21] The larger proportion of women in the E-Risk adult ADHD group is due to a higher number of women with late-onset ADHD joining the population in adulthood, rather than childhood symptoms being especially persistent in women. ADHD symptoms in girls may be less likely to come to the attention of parents and teachers due to lower rates of externalizing-type behaviors,[22] resulting in fewer girls diagnosed with ADHD in childhood.

What is late-onset ADHD?

A few studies point to the possibility of ADHD emergence after childhood; they offer suggestive evidence that for some, ADHD symptoms may increase into adolescence and adulthood.[23,24] Findings from the Dunedin Study found that 90% of the individuals with adult ADHD at age 38 did not meet criteria for the disorder in childhood.[1] We found that already by age 18, late-onset individuals constitute a large proportion of the adult ADHD population. However, many questions

remain as to the nature of late-onset ADHD. We considered three possibilities. First, late-onset individuals may have the same underlying liability for ADHD as those with childhood ADHD, but the disorder may be masked in childhood due to protective childhood factors, such as particularly supportive family environments or highly developed cognitive skills. In such cases, symptoms may not become impairing until increasing challenges of later, more demanding schooling.[25]

Second, late-onset individuals may not have ADHD at age 18, but rather another disorder with similar symptoms. We found that late-onset individuals exhibit elevated rates of anxiety, depression, and marijuana and alcohol dependence. To investigate whether the late-onset group is entirely accounted for by ADHD-like symptoms from other disorders, we excluded individuals with diagnoses of anxiety, depression, and marijuana and alcohol dependence. We found about a third of the late-onset group remained after excluding individuals with these comorbidities, and presented similar levels of ADHD impairment and co-informant-rated ADHD symptoms. However, late-onset ADHD individuals may have other (e.g., OCD, social anxiety) or sub-threshold comorbidity that account for ADHD symptoms.

Third, late-onset adult ADHD could be a distinct disorder. The late-onset ADHD group shows several characteristics that differ from childhood-onset ADHD, including a dissimilar gender composition (the late-onset group includes more women), and lower heritability. Indeed, these differences are consistent with the extant research on the characteristics of childhood and adult ADHD populations. We found that the risk of developing late-onset ADHD was similar whether or not the participant's co-twin had childhood ADHD. The extent to which etiology differs between childhood-onset and late-onset ADHD has broad implications for our understanding of the adult ADHD population. Studies of adult ADHD that examine the genetic origins of the disorder, or the

effectiveness of different treatments, may benefit from taking into account this heterogeneity of the adult ADHD population.

Limitations

Our findings should be considered in light of potential limitations. First, we have diagnostic information on ADHD at age 18 based on self-report only. However, a strength of our study is the availability of reports from co-informant at age 18. Co-informants rated the persistent group as having more symptoms than the remitted group, corroborating self-reports, and childhood risk factors for persistence were similar using age-18 co-informants reports (Table 3).

Second, we defined young adult ADHD using an age of onset criterion; therefore the late-onset ADHD group has young adult onset of the full ADHD *syndrome* rather than ADHD *symptoms.* This could be considered a limitation, as we do not focus on individuals with have ADHD at age 18 but had no apparent ADHD symptoms in childhood. However, individuals with no reported childhood ADHD symptoms are a somewhat distinctive group, as it was normative to display some ADHD behavior in childhood (over 85% of our sample has some mother or teacher reported ADHD symptoms in childhood). Including the age of onset criterion is also a strength, as our adult ADHD group meets DSM-5 criteria, which stipulate symptom onset before age 12, and are comparable with the extant literature on adult ADHD which include this criterion. Third, our study - as an epidemiological cohort - does not have clinician interviews and may include false positives. However, we provide evidence of construct validity as our ADHD groups were associated with known correlates and were rated as having more ADHD symptoms by co-informants. Taken together, this network of information lends validity to our diagnostic procedures. Fourth, results regarding childhood characteristics and age-18 functioning do not differ if we remove the age of onset criterion for the young adult ADHD diagnosis. Third, the sample comprised twins, so results

may not generalize to singletons. However, our prevalence of childhood ADHD at each age is well within the range of 3.4-11% estimated previously.[26,27] Additionally, our rate of ADHD persistence is similar to that found in a meta-analysis.[4]

Conclusions and implications

Due to the prospective, longitudinal design of the E-Risk study, we were able to identify heterogeneity in the adult ADHD population. Our findings highlight the importance of taking a developmental approach to understanding ADHD. While many questions remain as to the nature of late-onset ADHD, we found this group showed significant levels of ADHD symptoms and impairment, as well as poor functioning and high rates of psychiatric comorbidity. Therefore, the absence of a childhood diagnosis of ADHD should not preclude adults with ADHD from receiving clinical attention. Whether individuals with late-onset versus childhood-onset ADHD respond differently to treatment is an open question, and further research is required to better understand the etiology, course and optimal treatment of late-onset ADHD.

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Conflict of Interest Disclosures

All authors report no conflict of interest

Role of Funder/Sponsor Statement

No funders/sponsors had any role in design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

Access to Data and Data Analysis

Drs Agnew-Blais and Arseneault had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

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Figure 1. Groups of individuals with childhood ADHD, adult ADHD, and subgroups of remitted, persistent and late-onset ADHD
Legend: ADHD = Attention Deficit Hyperactivity Disorder; n = number of participants; % = percentage

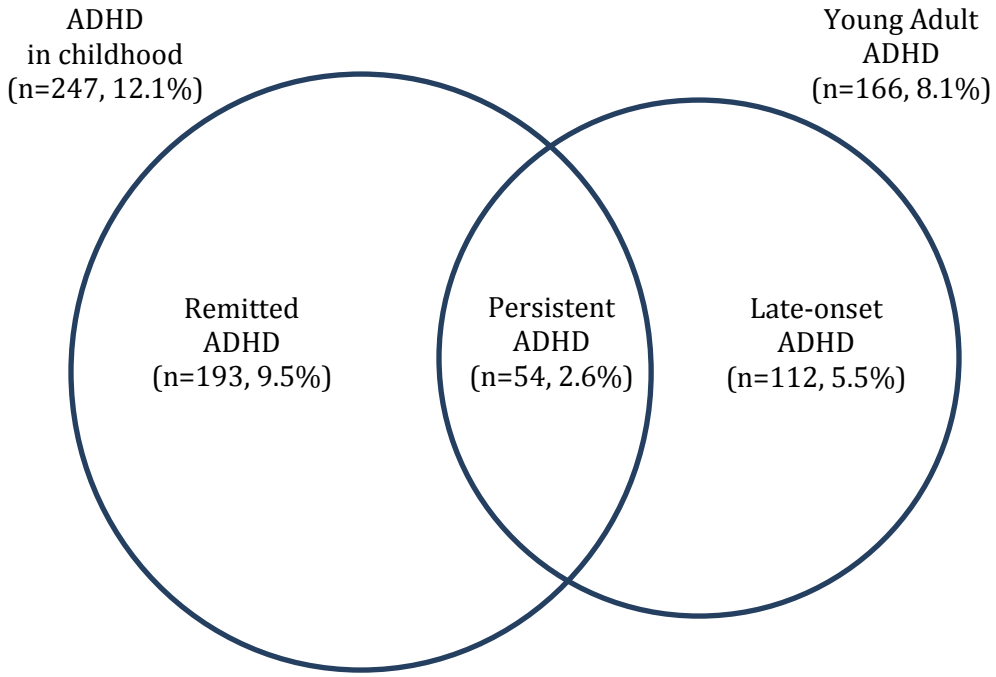

Table 1. Pre- and perinatal, clinical, and family environment factors in childhood among individuals with and without ADHD

			Young adult ADHD			
		Childhood ADHD				
	No ADHD dx φ *N (%)* *1681 (82.4)*	Remitted ADHD dx α *N (%)* *193 (9.5)*	Persistent ADHD dx α *N (%)* *54 (2.6)*	Late-onset ADHD dx *N (%)* *112 (5.5)*	Persistent vs remitted	Persistent vs late-onset α
Pre- and perinatal factors						
Male gender, n (%)	743 (44.2)	140 (72.5)***	36 (66.7)**	49 (44.6)		*
Birth weight (gr), mean (SD)	2449.0 (539.7)	2372.2 (503.6)*	2364.4 (538.1)	2467.8 (529.5)		
Stress during pregnancy, n (%)	329 (20.6)	45 (26.0)	17 (32.1)~	29 (26.4)		
Smoking during pregnancy, n (%)	372 (23.3)	65 (39.4)***	23 (43.4)**	34 (31.5)~		~
Child clinical characteristics						
Age 5-12 childhood ADHD symptoms β						
Total inattentive symptoms, mean (SD)	0.83 (1.2)	4.96 (2.6)***	6.32 (3.2)***	1.72 (1.3)***	**	***
Total hyp/impul symptoms, mean (SD)	1.33 (1.5)	5.53 (2.5)***	6.63 (3.0)***	2.41 (1.6)***	*	***
Total symptoms, mean (SD)	2.15 (2.4)	10.49 (4.2)***	12.93 (5.5)***	4.14 (2.5)***	**	***
Age 5-12 comorbid problems						
ODD, n (%)	167 (9.9)	83 (43.0)***	25 (46.3)***	26 (23.2)***		*
CD, n (%)	168 (10.0)	90 (46.6)***	28 (51.9)***	33 (29.5)***		*
Internalizing score, mean (SD)	10.72 (6.2)	15.81 (8.4)***	17.45 (9.2)***	12.71 (6.7)**		**
Externalizing score, mean (SD)	14.20 (9.8)	31.87 (14.9)***	36.85 (17.9)***	22.24 (11.2)***	~	***
Age-5 IQ and executive functioning						
IQ, mean (SD)	101.38 (14.6)	93.04 (14.6)***	87.96 (14.7)***	96.91 (15.7)**	*	**
Performance IQ, mean (SD)	9.98 (2. 8)	8.53 (2.7)***	7.43 (2.9)***	9.23 (2.9)**	*	**
Verbal IQ, mean (SD)	9.12 (3.0)	7.81 (3.1)***	7.35 (2.8)***	8.44 (3.2)*		*
Executive functioning, mean (SD)	11.84 (3.0)	10.54 (3.3)***	10.64 (3.4)*	10.97 (2.8)**		
Family environment						
Parental antisocial behavior, n (%)	422 (25.2)	82 (42.7)***	23 (42.6)*	37 (33.0)~		
Parental substance use problems, n (%)	381 (22.8)	66 (34.4)**	25 (46.3)**	42 (37.5)**		
Maternal depression, n (%)	438 (26.2)	90 (46.9)***	15 (28.3)	34 (30.4)	*	
Low social class, n (%)	513 (30.5)	94 (48.7)***	24 (44.4)*	47 (42.0)*		
Maternal warmth, mean (SD)	3.33 (1.0)	2.83 (1.1)***	3.17 (1.0)	3.17 (0.9)~	~	
Maternal negativity, mean (SD)	1.45 (0.9)	2.01 (1.1)***	1.96 (1.1)**	1.71 (1.0)**		
Domestic violence exposure, n (%)	670 (40.2)	102 (53.1)**	26 (48.2)	59 (52.7)*		
Child maltreatment, n (%)	198 (11.8)	44 (22.8)***	9 (16.7)	24 (21.4)**		

* p<0.05, ** p<0.01, *** p<0.001, ~ p<0.10

φ Comparisons are between remitted, persistent and late-onset ADHD groups with no ADHD diagnosis controls.

α Statistical comparisons adjusted for gender using logistic regression.

β Childhood ADHD symptoms from ages 5 to 12 are computed as mean of the sum of mother reported and teacher reported ADHD symptoms across ages 5, 7, 10, and 12. The range of total inattentive symptoms in childhood was 0 to 18 (range of 0 to 9 for mother and teacher report separately); the range of total hyperactive/impulsive symptoms in childhood was 0 to 18; the range of total ADHD symptoms in childhood 0 to 36.

Table 2. Age-18 functioning among individuals with no ADHD, and persistent, remitted, and late-onset ADHD

			Young adult ADHD			
		Childhood ADHD				
	No ADHD dx φ *N (%) 1681 (82.4)*	Remitted ADHD dx α *N (%) 193 (9.5)*	Persistent ADHD dx α *N (%) 54 (2.6)*	Late-onset ADHD dx *N (%) 112 (5.5)*	Persistent vs remitted	Persistent vs late-onset α
Age-18 ADHD symptoms and impairment						
Self-report						
# inattentive symptoms, self-report, mean (SD)	2.63 (2.2)	3.51 (2.4)***	5.72 (2.2)***	6.13 (1.8)***	***	
# hyp/impulsive symptoms, self-report, mean (SD)	2.44 (2.2)	3.33 (2.4)***	5.63 (2.2)***	5.27 (2.4)***	***	
# total symptoms, self-report. mean (SD)	5.08 (3.9)	6.89 (4.3)***	11.37 (3.5)***	11.42 (3.2)***	***	
ADHD interference at school or work, mean (SD)	1.93 (1.0)	2.03 (1.1)	3.73 (1.1)***	3.91 (0.8)***	***	
ADHD interference at home or with friends, mean (SD)	1.55 (0.8)	1.65 (0.8)~	3.59 (1.0)***	3.45 (0.7)***	***	
Co-informant report						
# any informant ADHD sx, mean (SD)	0.42 (1.1)	1.25 (1.9)***	2.31 (2.6)***	1.37 (2.2)***	**	*
Interviewer personality impressions						
Not conscientious, n (%)	151 (9.0)	38 (19.9)***	21 (38.9)***	21 (18.9)**	**	*
Not diligent, n (%)	240 (14.4)	56 (29.2)***	28 (54.9)***	30 (27.3)***	**	**
Not planful, n (%)	290 (17.3)	66 (34.6)***	16 (30.2)*	37 (33.0)***		
Disorderly, n (%)	49 (2.9)	15 (7.8)**	7 (13.2)***	13 (11.6)***		
Not focused, n (%)	219 (13.1)	49 (25.7)***	20 (37.0)***	25 (22.3)**		~
Not persevering, n (%)	133 (8.1)	35 (18.5)***	20 (37.0)***	19 (17.0)**	**	**
Functioning						
IQ, mean (SD)	102.49 (14.5)	93.33 (15.9)***	89.78 (14.8)***	96.12 (14.3)***		**
Life satisfaction, mean (SD)	3.92 (0.7)	3.75 (0.7)**	3.48 (0.9)***	3.44 (0.8)***	~	
Job preparedness, mean (SD)	17.23 (2.4)	16.45 (2.9)***	14.64 (4.2)***	15.42 (3.0)***	**	~
Currently studying, n (%)	1,231 (73.2)	113 (58.6)***	33 (61.1)~	71 (63.4)*		
Comorbid diagnoses						
Generalized anxiety disorder, n (%)	108 (6.4)	11 (5.8)	13 (24.1)***	18 (16.1)***	**	
Major depressive episode, n (%)	300 (17.9)	41 (21.4)~	19 (35.2)**	48 (42.9)***	*	
Conduct disorder, n (%)	200 (11.9)	45 (23.6)**	20 (38.5)***	39 (35.1)***	*	
Marijuana dependence, n (%)	54 (3.2)	11 (5.7)	8 (14.8)**	13 (11.6)***	*	
Alcohol dependence, n (%)	182 (10.8)	32 (16.6)~	7 (13.0)	35 (31.5)***		*

* p<0.05, ** p<0.01, *** p<0.001, ~ p<0.10

φ Comparisons are between remitted, persistent and late-onset ADHD groups with no ADHD diagnosis controls.

α Statistical comparisons adjusted for gender using logistic regression.

Table 3. Associations between childhood pre- and perinatal, clinical and social environment characteristics and age-18 informant report of ADHD symptoms among individuals with childhood ADHD

	Total ADHD symptoms at age 18 among individuals with childhood ADHD (N=239)
Pre- and perinatal factors	Standardized β
Male gender, n (%)	0.14~
Birth weight (gr), mean (SD)	0.13
Stress during pregnancy, n (%)	0.19*
Smoking during pregnancy, n (%)	0.15~
Child ADHD characteristics	
Age 5-12 childhood ADHD symptoms	
Total inattention symptoms, mean (SD)	0.37***
Total hyp/impul symptoms, mean (SD)	0.37***
Total symptoms, mean (SD)	0.43***
Age 5-12 comorbidity	
ODD, n (%)	0.15*
CD, n (%)	0.19**
Internalizing score, mean (SD)	0.13~
Externalizing, mean (SD)	0.30***
Age-5 IQ and executive functioning	
IQ, mean (SD)	-0.12
Performance IQ, mean (SD)	-0.13
Verbal IQ, mean (SD)	-0.06
Executive functioning, mean (SD)	-0.07
Family environment	
Parental antisocial behavior, n (%)	-0.08
Parental substance use problems, n (%)	-0.04
Maternal depression, n (%)	0.00
Low social class, n (%)	0.02
Maternal warmth, mean (SD)	0.02
Maternal negativity, mean (SD)	0.08
Domestic violence exposure, n (%)	-0.11
Child maltreatment, n (%)	0.02

* p<0.05, ** p<0.01, *** p<0.001, ~ p<0.10