Original Investigation

Attention-Deficit/Hyperactivity Disorder Trajectories From Childhood to Young Adulthood
Evidence From a Birth Cohort Supporting a Late-onset Syndrome

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IMPORTANCE The requirement of a childhood onset has always been a key criterion for the diagnosis of attention-deficit/hyperactivity disorder (ADHD) in adults, but recently this requirement has become surrounded by controversy.

OBJECTIVE To investigate whether impaired young adults with ADHD symptoms always have a childhood-onset disorder in a population-based longitudinal study.

DESIGN, SETTING, AND PARTICIPANTS Participants belonged to the 1993 Pelotas Birth Cohort Study, including 5249 individuals born in Pelotas, Brazil, in 1993. They were followed up to 18 to 19 years of age, with 81.3% retention. The data analysis was performed between August 8, 2015, and February 5, 2016.

MAIN OUTCOMES AND MEASURES The ADHD status was first ascertained at 11 years of age using a screening instrument (hyperactivity subscale of the Strength and Difficulties Questionnaire) calibrated for a *DSM-IV* ADHD diagnosis based on clinical interviews with parents using the Development and Well-Being Assessment. At 18 to 19 years of age, ADHD diagnosis was derived using *DSM-5* criteria, except age at onset. We estimated the overlap between these groups assessed at 11 and 18 to 19 years of age and the rates of markers of impairment in these 2 groups compared with those without ADHD.

RESULTS At 11 years of age, childhood ADHD (C-ADHD) was present in 393 individuals (8.9%). At 18 to 19 years of age, 492 individuals (12.2%) fulfilled all *DSM-5* criteria for young adult ADHD (YA-ADHD), except age at onset. After comorbidities were excluded, the prevalence of YA-ADHD without comorbidities decreased to 256 individuals (6.3%). Children with C-ADHD had a male preponderance not observed among children without ADHD (251 [63.9%] vs 1930 [47.9%] male, $P < .001$), whereas the YA-ADHD group had a female preponderance (192 [39.0%] vs 1786 [50.4%] male, $P < .001$). Both groups had increased levels of impairment in adulthood, as measured by traffic incidents, criminal behavior, incarceration, suicide attempts, and comorbidities. However, only 60 children (17.2%) with ADHD continued to have ADHD as young adults, and only 60 young adults (12.6%) with ADHD had the disorder in childhood.

CONCLUSIONS AND RELEVANCE The findings of this study do not support the assumption that adulthood ADHD is necessarily a continuation of childhood ADHD. Rather, they suggest the existence of 2 syndromes that have distinct developmental trajectories.

Editorial

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Attention-deficit/hyperactivity disorder (ADHD) has been traditionally conceptualized as a neurodevelopmental disorder. Most recently, *DSM-5* included ADHD in a specific section under this umbrella.[1] On the basis solely of clinical wisdom, *DSM-III* introduced ADHD criterion B, requiring symptoms to be present before the age of 7 years, and *DSM-IV-TR* added that impairment must also be present by this same age.[2,3] A systematic review of the literature[4] challenged the utility and validity of this criterion B. The *DSM-5* scientific committee decided to change the criterion to require symptoms before 12 years of age based on evidence that this threshold would capture almost every case presented in childhood, without increasing the prevalence rate.[1,5] Furthermore, *DSM-5* introduced the concept of adulthood ADHD as a disorder that begins in childhood and requires symptoms to be present before 12 years of age.

A recent report by Moffitt et al[6] presented data that diverge from this traditional perspective. In a representative birth cohort followed up to 38 years of age, prevalence rates of childhood ADHD (C-ADHD) and adulthood disorder were in accordance with estimates from the literature (6% and 3.1%, respectively). However, the 2 groups had only minimal overlap: 87% of those with adulthood ADHD did not have prior C-ADHD, and 85% of those with childhood ADHD did not continue to have ADHD in adulthood. Castellanos,[7] in an editorial about the report by Moffitt et al, emphasized the urgent need for replications to confirm or challenge these data.

In the present study, we report findings from a prospective, longitudinal study of a representative birth cohort in Brazil. We hypothesize that (1) prevalence rates of ADHD in childhood and young adulthood will be similar to that reported in the literature, (2) individuals with C-ADHD and adulthood ADHD will have higher levels of impairment markers than individuals without ADHD, and (3) groups will have little overlap. We extended the study by Moffitt et al[6] by examining the effects of comorbid disorders on ADHD continuity.

Methods

Design and Sample

Individuals enrolled in this study were participants in the 1993 Pelotas Birth Cohort. All children born in 1993 in the city of Pelotas, Brazil (5249 individuals), were assessed at multiple time points and followed up until 18 to 19 years of age, with a retention rate of 81.3%. Further information on the cohort design can be found elsewhere.[8,9] The institutional review board of the Federal University of Pelotas approved the study. Written informed consent was obtained from all participants, and all data were deidentified.

The assessment at 11 years of age included data on child mental health using the Brazilian Portuguese Version of the Strengths and Difficulties Questionnaire (SDQ), parent report and self-report. A subsample of 280 individuals was interviewed with the Development and Well-Being Assessment, and the optimal cutoff for ADHD was estimated to be 8 or more points on the SDQ hyperactivity scale as rated by parents.[10] The SDQ instrument accurately predicted ADHD diagnosis with an

Key Points

Question Do young adults with a *DSM-5* attention-deficit/hyperactivity disorder (ADHD) diagnosis always have a childhood-onset disorder?

Findings In this birth cohort study, 5249 individuals were assessed for ADHD at 11 and 18 to 19 years of age. Only 60 young adults with ADHD (12.6%) had the diagnosis in childhood.

Meaning These findings do not support the assumption that adulthood ADHD is necessarily a continuation of childhood ADHD.

area under the curve of 0.81 (95% CI, 0.74-0.88), and the cutoff of at least 8 points had 85.7% sensitivity and 67.4% specificity for the diagnosis.

In the last assessment, individuals at 18 to 19 years of age were interviewed by trained psychologists using specific modules for major depressive disorder (MDD), bipolar disorder (BD), generalized anxiety disorder (GAD), and social anxiety disorder (SAD) modified from the Mini-International Neuropsychiatric Interview.[11] The ADHD assessment was performed with a structured interview according to *DSM-5* criteria (eTable 1 in the Supplement).[12] For the present study, we did not require *DSM-5* criterion B (age at onset) for diagnosis of young adult ADHD (YA-ADHD). We defined C-ADHD as present when scores on the SDQ hyperactivity scale (parent report) were equal or higher to 8 points and associated with impairment, defined by at least 1 point in the impact supplement.

We also created a secondary category defined as YA-ADHD without comorbidities (YA-ADHD-WC), which excluded from the YA-ADHD group those individuals with comorbidities, including MDD, BD, GAD, SAD, and regular use of illicit drugs. This procedure was performed for secondary analyses to reduce confounding by these comorbidities that are likely to occur later in life and might contribute to inattentive and/or hyperactive-impulsive symptoms. We also excluded these comorbidities from comparison groups without ADHD. The comparison groups were defined as those without C-ADHD for childhood comparisons (C-ADHD vs those without C-ADHD) and those without YA-ADHD for adulthood comparisons (YA-ADHD vs those without YA-ADHD and YA-ADHD-WC vs those without YA-ADHD and comorbidities).

Correlates

Trained interviewers assessed correlates of tobacco use, illicit drug use, pregnancy, sexually transmitted diseases, and criminal behavior using confidential questionnaires. Suicide attempts were evaluated as part of the assessment of MDD. We estimated IQ with an abbreviated version of the Wechsler Adult Intelligence Scale–Third Edition.[13] Further details of assessments of correlates can be found in eTable 2 in the Supplement.

Statistical Analyses

We estimated differences between groups using χ^2 tests for the following variables: percentage male, ADHD subtype, traffic

Table. Characteristics and Outcomes of the C-ADHD, YA-ADHD, and YA-ADHD-WC Groups and Non-ADHD Comparisons in the 1993 Pelotas Birth Cohort Study[a]

Variable	Childhood Comparisons			Adulthood Comparisons					
	Without ADHD	C-ADHD	P Value	Without ADHD	YA-ADHD	P Value	Without YA-ADHD-WC	YA-ADHD-WC[b]	P Value
Total No. of individuals (prevalence rate)	4033 (91.1)	393 (8.9)	NA	3547 (87.8)	492 (12.2)	NA	2874 (91.8)	256 (8.2)	NA
Male sex	1930 (47.9)	215 (63.9)	<.001	1786 (50.4)	192 (39.0)	<.001	1479 (51.5)	115 (44.9)	.04
ADHD presentation in adulthood[c]									
Inattentive	225 (54.1)	31 (51.7)	.43	NA	262 (53.3)	NA	NA	151 (59.0)	NA
Hyperactive-impulsive	38 (9.1)	3 (5.0)		NA	44 (8.9)		NA	31 (12.1)	
Combined	153 (36.8)	26 (43.3)		NA	186 (37.8)		NA	74 (28.9)	
Outcomes									
Traffic incidents	619 (17.2)	77 (21.9)	.03	593 (16.7)	114 (23.2)	<.001	467 (16.2)	55 (21.5)	.03
Criminal behavior, violent	462 (14.5)	88 (28.7)	<.001	455 (14.4)	105 (24.4)	<.001	467 (11.8)	55 (18.5)	.003
Criminal behavior, any	527 (16.6)	95 (30.9)	<.001	519 (16.5)	117 (27.1)	<.001	352 (13.7)	47 (20.7)	.004
Incarceration	33 (0.9)	14 (4.0)	<.001	35 (1.0)	14 (2.8)	<.001	15 (0.5)	4 (1.6)	.04
Tobacco smoking	448 (13.3)	83 (25.9)	<.001	457 (13.7)	87 (19.0)	.003	270 (10.0)	28 (11.8)	.39
Illicit drug use	243 (6.8)	37 (10.8)	.006	246 (7.0)	44 (9.0)	.11	NA	NA	NA
Depression	133 (3.8)	24 (7.2)	.003	93 (2.7)	66 (13.6)	<.001	NA	NA	NA
Bipolar disorder	52 (1.5)	14 (4.2)	<.001	32 (0.9)	36 (7.4)	<.001	NA	NA	NA
Generalized anxiety disorder	255 (7.2)	36 (10.8)	.02	179 (5.1)	121 (24.9)	<.001	NA	NA	NA
Social anxiety disorder	228 (6.5)	34 (10.2)	.01	175 (5.0)	98 (20.2)	<.001	NA	NA	NA
Suicide attempt	213 (6.0)	35 (10.0)	.003	180 (5.1)	75 (15.2)	<.001	101 (3.5)	17 (6.6)	.01
Teenage pregnancy	250 (13.2)	29 (22.1)	.004	39 (13.0)	243 (13.9)	.64	185 (13.3)	14 (9.9)	.26
Sexually transmitted diseases	92 (2.6)	10 (2.9)	.72	79 (2.2)	26 (5.3)	<.001	56 (1.9)	12 (4.7)	.004
Personal income, mean (SD), $	510.5 (419)	543.5 (315.9)	.20	516.43 (422.9)	478.86 (263.6)	.11	517.5 (449.6)	489 (281.7)	.40
Length of schooling, mean (SD), y	8.63 (2.29)	8.45 (2.35)	.15	8.61 (2.29)	8.57 (2.26)	.70	8.62 (2.29)	8.56 (2.15)	.68
IQ, mean (SD)	97.2 (12.4)	89.7 (11.7)	<.001	96.7 (12.7)	95.3 (11.8)	.01	97.7 (12.2)	95.6 (11.9)	.008

Abbreviations: ADHD, attention-deficit/hyperactivity disorder; C-ADHD, childhood ADHD; NA, data not applicable; YA-ADHD, young adult ADHD; YA-ADHD-WC, young adult ADHD without comorbidities.

[a] Data are presented as number (percentage) of study participants unless otherwise indicated.

[b] The YA-ADHD-WC group represents 6.3% of the entire adult population and 8.2% of adults without comorbidities.

[c] Comparison with the χ^2 test in a 2 × 3 table considering YA-ADHD presentation in children with C-ADHD who continued to have YA-ADHD and those from the non-ADHD group in childhood who developed YA-ADHD.

incidents, illicit drug use, smoking, criminal behavior, correctional institution, comorbidities, and teenage pregnancy. We used one-way analysis of variance for continuous variables: personal income, years of schooling, and IQ. Effect sizes (ESs) for continuous variables were estimated with the Cohen d. All analyses were performed using SPSS statistical software, version 20.0 (SPSS Inc).

Results

Prevalence and Sex Distribution

At 11 years of age, C-ADHD was present in 393 individuals (8.9%). At 18 to 19 years of age, 492 individuals (12.2%) fulfilled all *DSM-5* criteria for YA-ADHD, except age at onset. After comorbidities were excluded, the prevalence of YA-ADHD-WC decreased to 256 individuals (6.3%). Children with C-ADHD had a male preponderance not observed among children without ADHD (251 [63.9%] vs 1930 [47.9%] male,

$P < .001$), whereas the YA-ADHD group had a female preponderance (192 [39.0%] vs 1786 [50.4%] male, $P < .001$). This difference persisted after excluding comorbidities (115 males [44.9%] in the group with YA-ADHD-WC and 1479 males [51.5%] in the group without YA-ADHD-WC, $P = .045$) (Table).

Persistence and Overlap

Among the 393 children in the C-ADHD group, 60 (15.3%) continued to have YA-ADHD (31 [7.9%] with at least 1 comorbidity and 29 [7.4%] with no comorbidity), 288 (73.3%) had no YA-ADHD in the assessment at 18 to 19 years old, and 45 (11.5%) were either unavailable for or lost to follow-up (Figure 1), resulting in a persistence rate of 17.2%. Furthermore, most individuals with C-ADHD presented with few symptoms in young adulthood (Figure 2), making it unlikely that a lower symptom cutoff would substantially change this result.

Among the 492 individuals in the YA-ADHD group, 60 (12.2%) had C-ADHD, 416 (84.6%) did not have C-ADHD, and 16 (3.3%) were not assessed with the SDQ at 11 years of age,

Figure 1. Follow-Forward and Follow-Back Analyses of Individuals With Attention-Deficit/Hyperactivity Disorder (ADHD)

C-ADHD indicates childhood attention-deficit/hyperactivity disorder; YA-ADHD, young adult attention-deficit/hyperactivity disorder; YA-ADHD-WC, young adult attention-deficit/hyperactivity disorder without comorbidities.
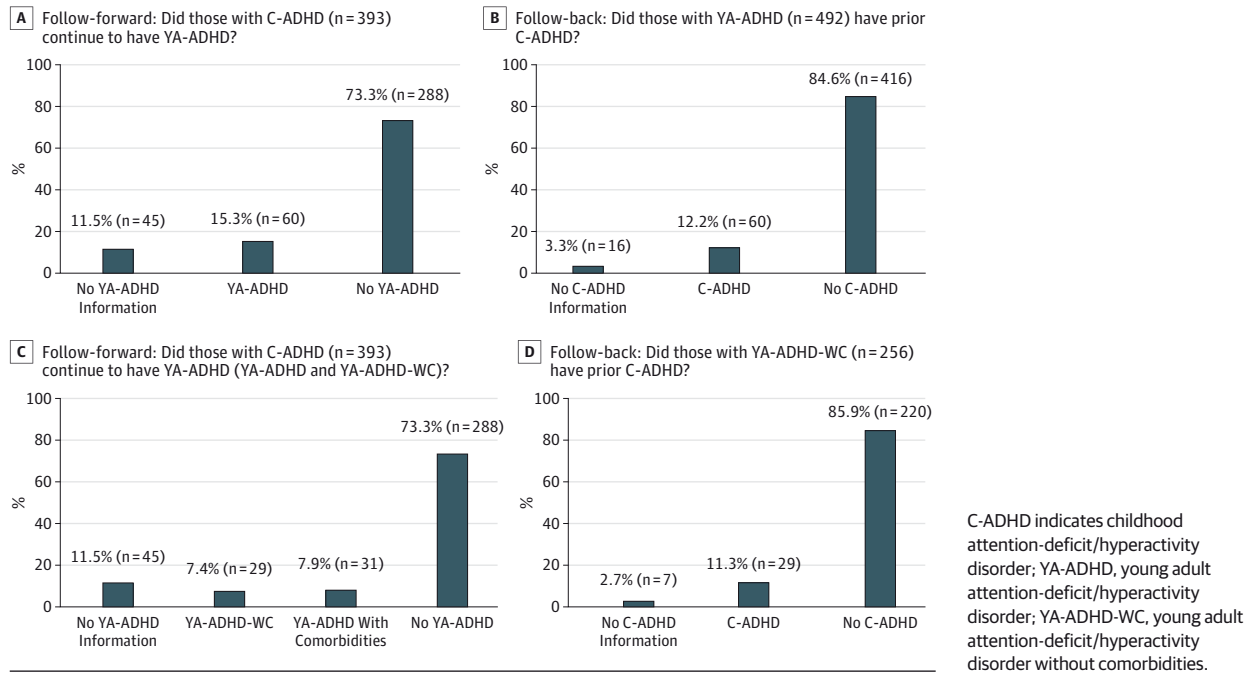
resulting in a prevalence of 12.6% of C-ADHD among the YA-ADHD group. Considering the 256 individuals in the YA-ADHD-WC group, 29 (11.3%) had C-ADHD, 220 (85.9%) did not have C-ADHD, and 7 (2.7%) had not been assessed with the SDQ at 11 years of age (Figure 1), resulting in a prevalence of 11.6% of C-ADHD among the YA-ADHD-WC group. See also the secondary analyses in eTables 3, 4, and 5 in the Supplement, checking the robustness of our findings.

YA-ADHD Presentation

Inattentive presentation prevailed in all groups in young adulthood: those with C-ADHD who continued to have YA-ADHD (31 [51.7%]), YA-ADHD (262 [53.3%]), and YA-ADHD-WC (151 [59.0%]) (see the Table for the other ADHD presentations).

Comorbidities and Suicide Attempts

Individuals with C-ADHD had significantly higher rates of comorbidities in young adulthood compared with those without C-ADHD. At 18 to 19 years of age, rates of MDD (24 [7.2%] vs 133 [3.8%], $P = .003$), BD (14 [4.2%] vs 52 [1.5%], $P < .001$), GAD (36 [10.8%] vs 255 [7.2%], $P = .02$), SAD (34 [10.2%] vs 228 [6.5%], $P = .01$), illicit drug use (37 [10.8%] vs 243 [6.8%], $P = .007$), and tobacco smoking (83 [25.9%] vs 448 [13.3%], $P < .001$) were all higher in the C-ADHD group. The YA-ADHD group had even higher levels of comorbidities, which were also significantly different from those of individuals without YA-ADHD for MDD (66 [13.6%] vs 93 [2.7%], $P < .001$), BD (36 [7.4%] vs 32 [0.9%], $P < .001$), GAD (121 [24.9%] vs 179 [5.1%], $P < .001$), SAD (98 [20.2%] vs 175 [5%], $P < .001$), and tobacco smoking (87 [19%] vs 457 [13.7%], $P = .003$). However, we did not find significant differences for illicit drug use (44 [9%] vs 246 [7%], $P = .11$). A self-reported suicide attempt in young

adulthood was more likely among children with ADHD than in children without ADHD (35 [10%] vs 213 [6%], $P = .003$) and among the YA-ADHD group than young adults without ADHD (75 [15.2%] vs 180 [5.1%], $P < .001$). The difference remained significant even after excluding comorbidities (17 [6.6%] in the group with YA-ADHD-WC vs 101 [3.5%] in the group without YA-ADHD-WC, $P = .01$) (Table).

Criminal Behavior and Incarceration

All 3 ADHD groups had higher levels of violent crimes compared with those without ADHD (C-ADHD: 88 [28.7%] vs 462 [14.5%], $P < .001$; YA-ADHD: 105 [24.4%] vs 455 [14.4%], $P < .001$; YA-ADHD-WC: 42 [18.5%] vs 303 [11.8%], $P = .003$). Accordingly, the 3 groups had significantly higher levels of incarceration compared with individuals without ADHD (C-ADHD: 14 [4.0%] vs 33 [0.9%], $P < .001$; YA-ADHD: 14 [2.8%] vs 35 [1.0%], $P < .001$; YA-ADHD-WC: 4 [1.6%] vs 15 [0.5%], $P = .04$) (Table).

Teenage Pregnancy and Sexually Transmitted Diseases

More girls with C-ADHD had teenage pregnancies compared with girls without ADHD (29 [22.1%] vs 250 [13.2%], $P = .004$). Differences in teenage pregnancy rates were not observed comparing YA-ADHD and YA-ADHD-WC groups and those without adulthood ADHD. A history of sexually transmitted diseases was more common in the YA-ADHD group than among young adults without ADHD (26 [5.3%] vs 79 [2.2%], $P < .001$), even controlling for comorbidities (12 [4.7%] in the group with YA-ADHD-WC vs 56 [1.9%] in the group without YA-ADHD-WC, $P = .004$). The C-ADHD group did not have a statistically significant different rate of sexually transmitted diseases compared with children without ADHD (10 [2.9%] vs 92 [2.6%], respectively; $P = .72$) (Table).

Figure 2. Number of Young Adult Attention-Deficit/Hyperactivity Disorder (YA-ADHD) Symptoms in the Childhood Attention-Deficit/Hyperactivity Disorder (C-ADHD), YA-ADHD, and Young Adult Attention-Deficit/Hyperactivity Disorder Without Comorbidities (YA-ADHD-WC) Groups
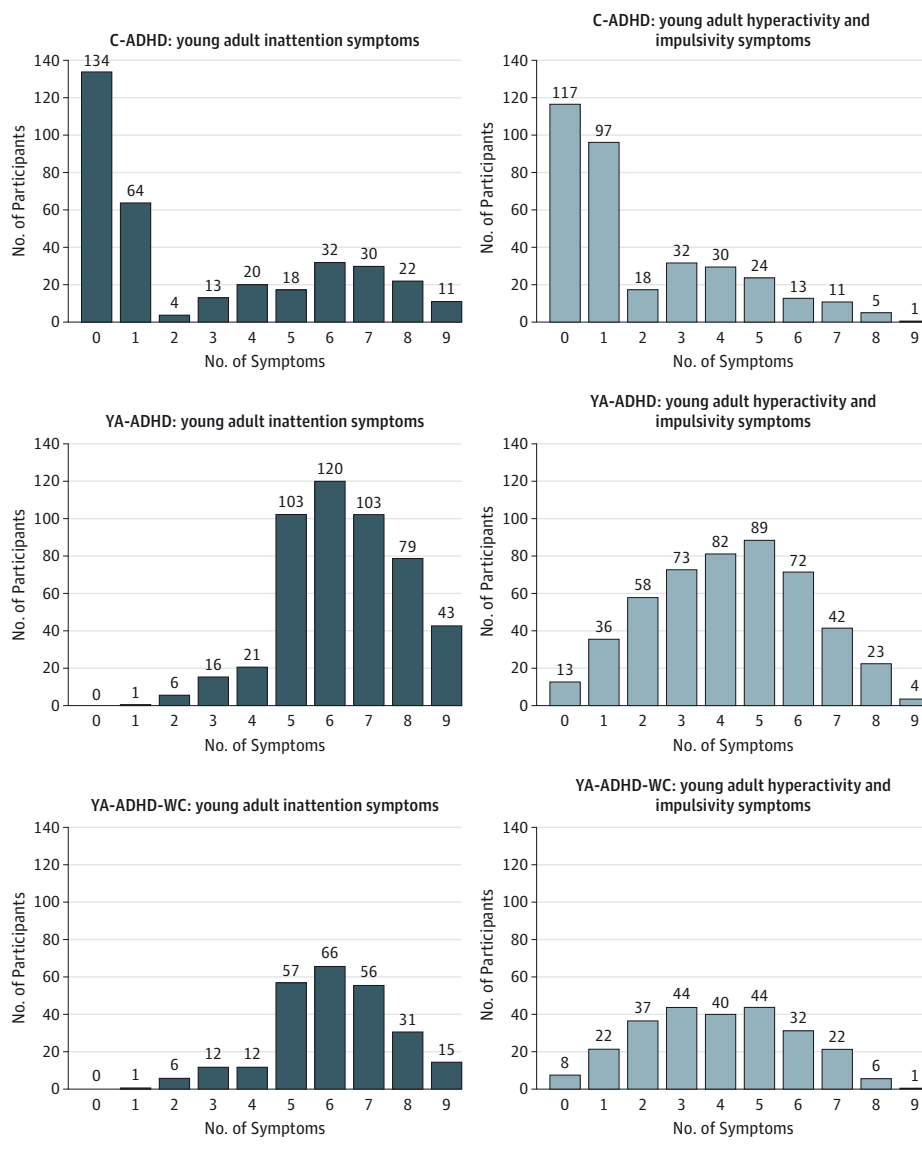
Traffic Incidents

Traffic incidents were significantly more likely among the ADHD groups than among those without ADHD (C-ADHD: 77 [21.9%] vs 275 [17.2%], *P* = .03; YA-ADHD: 114 [23.2%] vs 593 [16.7%], *P* < .001; YA-ADHD-WC: 55 [21.5%] vs 467 [16.2%], *P* = .03) (Table).

Personal Income and Years of Schooling

The C-ADHD, YA-ADHD, and YA-ADHD-WC groups did not differ from those without ADHD in terms of personal income and completed years of regular schooling.

IQ Levels

The C-ADHD and YA-ADHD groups had lower IQ levels than those without ADHD, and this difference was larger for the C-ADHD group (97.17 vs 89.74, ES = 0.61, *P* < .001) than

for the YA-ADHD (96.7 vs 95.28, ES = 0.12, *P* = .01) and YA-ADHD-WC (97.68 vs 95.59, ES = 0.17, *P* = .008) groups.

Discussion

The notion that adulthood ADHD is necessarily a continuation of C-ADHD is an established assumption in the field. Recently, a population-based birth cohort provided initial evidence suggesting the opposite (ie, 87% of adulthood ADHD cases without C-ADHD).[6] In the current study, we extended the findings of Moffitt et al[6] for young adults using a similar method. We identified that, among those with YA-ADHD that had been assessed in childhood, 416 (87.4%) did not have C-ADHD.

In the current study, young adults with self-reported ADHD symptoms had a consistent pattern of higher impairment than those without YA-ADHD, as determined by rates of traffic incidents, self-reported violent crimes, incarceration in correctional institutions, and comorbidities, which are analogous with previous findings in the literature.[14-17] Children and young adults with ADHD had higher rates of suicide attempts than their counterparts without the condition, which is also in accordance with the literature.[18] Because comorbidities might be responsible for the aforementioned differences, we ran the same analyses excluding those with co-occurring disorders from the ADHD group and similar results emerged, suggesting that comorbid disorders do not explain the adulthood ADHD impairments.

Although the expected prevalence rate of C-ADHD is approximately 5.3%,[19] we observed a notably inflated rate, approximately 8.9%. However, our estimate is similar to that reported by a prevalence study[20] that also used a screening instrument. Likewise, our adulthood ADHD prevalence rate was 12.2%, compared with a 2.5% to 5.0% prevalence rate suggested by meta-analyses.[21,22] It is likely that this difference occurred because of the lower symptom cutoff required by the *DSM-5* and because we did not require a childhood age at onset to make the diagnosis. Indeed, a previous report[12] in the same population found an ADHD prevalence rate of 3.5% using the age-at-onset criterion. Our estimate is considerably higher than that observed in the cohort of Moffitt et al,[6] in which they reported a 3.1% prevalence of ADHD in adults, even not requiring childhood age at onset. Such a difference might be explained by the fact that their sample was composed of individuals 20 years older, and there is a tendency for prevalence to decrease with increased age.[23] A lower male to female ratio is expected in adult samples compared with child ones, but we found a particularly low male to female ratio of 0.64:1, which may indicate that females are overrepresented in late-onset ADHD groups.

Our observed persistence rate of ADHD was 17.2%. This finding matched perfectly the persistence rate estimated by a previous meta-analysis.[24] Indeed, the cohort in the study by Moffitt et al[6] was the first population-based longitudinal sample to report an extremely low persistence rate of adulthood ADHD (5%). Again, the difference with our estimate was expected because the cohort in the study by Moffitt et al[6] is composed of older adults. It is important to bear in mind that our C-ADHD group continue to present significant impairments in adult life despite not continuing to qualify for an adulthood ADHD diagnosis. Three alternative hypotheses have been proposed to explain this finding: (1) impairments are a residual effect of the disorder, (2) impairments are owing to the effects of persistent comorbidities, and (3) there is an illusionary bias (eg, adults with ADHD do not perceive their ADHD symptoms). Future studies should try to elucidate these issues.

The main strengths of our study include a large representative sample not biased by clinical referral. Trained interviewers assessed our study participants at 11 and 18 to 19 years of age with substantial retention. Thus, we were able to report estimates of the overlap between ADHD in children and in adults, as well as their correlates, with rea-

sonable accuracy. On the other hand, some methodologic limitations should be taken into account in the interpretation of the results. First, diagnosis of C-ADHD was made using a screening instrument (SDQ hyperactivity scale). However, psychologists using the Development and Well-Being Assessment in a subsample of 280 individuals validated the SDQ hyperactivity scale cutoff scores against ADHD diagnosis in clinical interviews.[10] In addition, we checked a lower cutoff score that continued yielding significant lack of C-ADHD in those with YA-ADHD.

Second, we relied only on parent reports for C-ADHD and self-report for YA-ADHD. This might artificially increase diagnostic disagreement because of different information sources. However, when self-reported SDQ hyperactivity scores in childhood were used, both YA-ADHD groups (YA-ADHD and YA-ADHD-WC) were far below the threshold for C-ADHD diagnosis according to parent reports (score of 8), confirming that the individuals in the YA-ADHD group had few ADHD symptoms in childhood. In addition, these assessment procedures reflect more accurately what frequently occurs in clinical practice, where C-ADHD diagnosis relies much more on parent reports and adulthood ADHD diagnosis on self-report.[25] One could also question whether, if we had used parent reports of adulthood ADHD, a different group of ADHD cases in adulthood would have been identified. However, several previous investigations have found high agreement between self-reports and parent reports for ADHD diagnosis in adults.[26] Moreover, our ADHD cases defined by self-report have a clinical, comorbidity, and impairment profile similar to the one previously described for the disorder in adulthood. Finally, a recent report[27] suggests that even adult patients fulfilling *DSM-5* ADHD diagnosis by self-report, for whom other informants did not report ADHD symptoms in childhood, have the same clinical profile and response to treatment as those whose coinformants described ADHD symptoms in childhood.

Third, we did not have a formal diagnosis of some psychiatric disorders that could be the primary source of inattention and/or hyperactivity-impulsivity in adults, such as substance use disorders and personality disorders. Along the same line, diagnoses were ascertained with a structured interview rather than clinical judgment. Hence, the alternative explanation that another disorder might explain symptoms and impairment better than ADHD itself cannot be completely ruled out. However, 256 (6.3%) of the adult sample had ADHD without 4 important and frequent comorbidities or illicit drug use, and those individuals remained impaired compared with individuals without ADHD and comorbidities.

Fourth, our impairment measure was based only on the participant's perspective; a rater-derived score based on functional correlates was not used. However, physicians tend to see young adult patients without parents and to rely on self-perception about impairment more than on scales.

Fifth, our results in a community sample cannot be extrapolated to clinical samples in which most cases tend to be ADHD combined type with at least moderate severity. A final potential bias in our study, which indeed is inherent to most population-based studies in psychiatry, is the so-called

false-positive paradox that occurs when the rate of false-positive results based on the instrument used to assess the disorder is higher than the incidence of cases in the population.

Conclusions

In light of these findings, along with the study's strengths and limitations, we can draw some meaningful implications for practice and research. Above all, our findings do not support the premise that adulthood ADHD is always a continuation of C-ADHD. Rather, they suggest the existence of 2 syndromes that have distinct developmental trajectories, with a late on-set far more prevalent among adults than a childhood onset. This finding would not mean that ADHD could not be conceptualized as a neurodevelopmental disorder. Neurodevelopmental disorders may have a later onset, as is the case for schizophrenia.[28] In both clinical practice and research, it is important to differentiate early- and late-onset disorders, and future investigations should test whether they have different pathophysiologic mechanisms, treatment response, and prognosis. In addition, patients with late-onset adulthood ADHD have clear impairments, and their clinical profile cannot account for only the effect of comorbidities.

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