A 9-Year Follow-Up of Attention-Deficit/Hyperactivity Disorder in a Population Sample

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ABSTRACT

Background: Prior follow-up studies of attention-deficit/hyperactivity disorder (ADHD) have mostly been from North America. They have provided a good deal of information about ADHD, but whether these results generalize to population samples and to other countries is not certain. Most prior studies have also not assessed predictors of possible new onsets of ADHD in non-ADHD youth or the validity of subthreshold forms of the disorder.

Methods: 1,012 families were recruited at baseline, when a telephone interview assessed a child in the 6–12 years age range. The interview covered symptoms of ADHD, conduct disorder, and oppositional defiant disorder as well as family living situation, school performance, sleep disturbance, eating habits, use of supplemental iron, and history of ADHD treatment. Nine years later, the persistence of ADHD and its impairments and the emergence of new conditions were assessed. *DSM-5* diagnostic criteria were used to diagnose ADHD.

Results: 492 of the 1,012 participants seen at baseline were followed up 9 years later, at a mean age of 18 years. At follow-up, 16.7% of the children diagnosed with ADHD at baseline met full criteria for ADHD and 11.1% met criteria for subthreshold ADHD, yielding a persistence rate of 27.8%. Among children not diagnosed with ADHD at baseline, 1.1% met criteria for ADHD at follow-up. The persistence of ADHD and new onsets of ADHD were predicted by several baseline clinical features and by a family history of ADHD.

Conclusions: We replicated predictors of the persistence of ADHD found in prior studies and provide new data about predictors of new ADHD onsets in the population. Our findings about subthreshold ADHD support a dimensional conceptualization of the disorder, highlighting the potential clinical utility of a subthreshold diagnostic category. This study also contributes to the ongoing debate regarding adult-onset ADHD.

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Much research shows that childhood onset attention-deficit/hyperactivity disorder (ADHD) impairs a majority of patients through adolescence into adulthood.[1–10] Impairing symptoms of ADHD persist in two-thirds of cases,[11] and persistence increases the risk for substance use disorders,[12] psychiatric comorbidity,[5,13,14] poor work and social functioning,[15,16] emotional dysregulation,[17] executive dysfunction,[18,19] and criminality.[20]

Although most longitudinal studies of ADHD have focused on establishing the nature and severity of outcomes associated with the disorder, clinicians would benefit from knowing the degree to which clinical features in childhood predict later outcomes. Several studies have addressed this issue. The most consistent finding is that diagnoses of conduct disorder and measures of aggression predict a more persistent course.[21–27] Other features predictive of persistent ADHD are psychosocial adversity and psychiatric comorbidity,[28–33] functional impairments,[28,29] family and school functioning,[30] symptoms of hyperactivity,[31] and exposure to maternal psychopathology.[28] In a retrospective study of adults, childhood ADHD severity and childhood treatment predicted persistence of ADHD.[32] In contrast, neurocognitive impairment has not consistently predicted persistence of ADHD.[34]

A family history of ADHD has been associated with greater persistence. In a study of 140 ADHD and 120 non-ADHD families, the prevalence of ADHD was significantly higher among the siblings and parents of persistent ADHD cases compared to the siblings and parents of remitted cases.[35,36] In that sample, familial ADHD predicted persistence out to a 10-year follow-up.[28] A link between familial and persistent ADHD was also seen in a longitudinal study of velo-cardio-facial syndrome.[31] A systematic review concluded that severity of ADHD, treatment for ADHD, comorbid conduct disorder, and comorbid major depressive disorder were well-replicated predictors of the persistence of ADHD.[37]

Although prior follow-up studies have yielded much information about ADHD, nearly all ascertained ADHD cases from clinical samples in North America. Thus, the degree to which these results generalize to population samples and to other countries is uncertain. Prior studies have not assessed predictors of new onsets of ADHD in non-ADHD youth, and much controversy

Clinical Points

- Although prior work has documented the persistence of ADHD into late adolescence and young adulthood, relatively few longitudinal studies have evaluated the validity of subthreshold and late onset forms of the disorder.
- The results of this study provide some support for the validity of subthreshold and late onset diagnoses. These data cannot be used to make recommendations about diagnostic practices, but they do suggest a need for future work toward understanding these atypical forms of ADHD so that they might be included in future versions of the diagnostic manuals.

has been generated about the validity of ADHD when it onsets in adulthood.[38] The present study seeks to fill these gaps by reporting a 9-year follow-up of a French population cohort.

METHODS

Ascertainment

The baseline sample was selected in December 2008 by the survey company IDDEM (http://www.iddem.com/).[39,40] Starting with 18 million French telephone numbers, 7,912 were randomly selected. When each phone number was called, questions were asked to determine these demographic features: administrative region, size of the village, town or city, marital status of the parents, and occupation of the head of household. Our goal was for the final distribution of our sample to match the distribution of the French population on each of these variables. Informed consent was obtained. The study was approved by the Institutional Review Board (Comité de Protection des Personnes—Ile de France 06).

Of the 7,912 telephone numbers, 1,663 were no longer in service; 2,063 were not eligible because the household did not have a child between the ages of 6 and 12 years or belonged to a quota stratum that had already been filled. Among the 4,186 eligible families, 1,012 (24.2%) were successfully recruited. The remaining households were not recruited for the following reasons: telephone was not answered, telephone was busy, telephone would not accept anonymous calls, the person answering the phone refused to participate, or the participant stopped the interview before completion.

Nine years after the baseline assessment, we attempted to contact each family by telephone to request their participation in the follow-up study. If no one answered the telephone, we attempted to make contact via mail. If the phone number was not working, we searched for the parents' names in the telephone directory of other regions.

Assessment

Baseline. After a parent consented to participate, the interviewer administered a questionnaire covering family living situation; school performance of the child; symptoms of ADHD, conduct disorder, and oppositional defiant disorder (ODD); sleep disturbance; eating habits; use of iron as a supplement; and history of treatment for ADHD. The interviewers were trained by one of the authors (M.L.). Questions about disorders were adapted from the Kiddie Schedule for Affective Disorders and Schizophrenia[41] updated with items addressing all *DSM-5* criteria by authors M.L. and E.K. Following prior reports,[42–45] we defined "subthreshold ADHD" by modifying *DSM-IV* criteria by only requiring 3 or more inattentive symptoms or 3 or more hyperactive-impulsive symptoms.

Four-year and 9-year follow-ups. At each follow-up,[10] the questionnaire including family living situation, school performance of the child, and symptoms of ADHD, conduct disorder, and ODD was administered to the sample. Family history of ADHD was collected at follow-up by asking the parent if any other family members had been diagnosed with the disorder. At the 4- and 9-year follow-ups, we added questions about video gaming.

Statistical Analysis

Statistical analyses used SPSS Version 24. χ^2 tests assessed the relationship between categorical variables. Analyses of variance were used for continuous variables. When an omnibus test was statistically significant, post hoc tests, adjusted using the Bonferroni correction for multiple comparisons, were calculated. All tests were 2-tailed and used a significance level of $P < .05$ unless otherwise indicated.

RESULTS

Four hundred ninety-two of the 1,012 participants seen at baseline (48.6%) were followed up 9 years later. Families lost and not lost to follow-up did not differ on the following baseline variables: age, sex, parental education, parental work status, diagnosis of ADHD, number of inattention symptoms, and number of hyperactive-impulsive symptoms (all P values $> .05$). Table 1 compares demographics of the ADHD and non-ADHD groups at follow-up. We found no significant group differences in sex, parental marital status, or parental education. We found significant differences in parental employment; parents of youth with subthreshold ADHD were less likely to be employed than parents in the other groups.

Nine-Year Outcome of ADHD

At follow-up, 16.7% of the children diagnosed with ADHD at baseline also met full criteria for ADHD. An additional 11.1% met criteria for subthreshold ADHD, yielding a persistence rate of 27.8%. Among children not diagnosed with ADHD at baseline, 1.1% were diagnosed with ADHD at follow-up. An additional 1.5% met our criteria for subthreshold ADHD. Figure 1 shows the numbers of participants with baseline ADHD and ODD diagnoses who showed evidence of persistent ADHD.

Table 2 presents rates of disorders and school problems at follow-up by baseline ADHD diagnosis. Those with full threshold ADHD at baseline showed increased rates of

conduct, oppositional defiant, and pervasive developmental disorders at follow-up along with a greater rate of school difficulties. They were more likely to use tobacco but not more likely to use alcohol or cannabis. Those with subthreshold ADHD at baseline showed an increased risk for school difficulties at follow-up.

Predictors of the Persistence of ADHD

We compared participants who had been diagnosed with ADHD at baseline (n = 18) who did and did not persist with ADHD at follow-up. Total ADHD symptoms at baseline significantly predicted ADHD diagnosis at follow-up ($F_{2,15} = 5.06$, $P = .021$, $\eta^2 = 0.40$). Those with full ADHD at follow-up had significantly more ADHD symptoms at baseline (mean ± SD, 13.7 ± 1.1) than those with no ADHD at follow-up (8.6 ± 2.7, $P = .019$), but the number of ADHD symptoms did not discriminate those with subthreshold ADHD at follow-up (9.5 ± 2.1) from those with full or no ADHD. The mean number of baseline inattentive symptoms (full ADHD: 7.3 ± 1.5; subthreshold ADHD: 4.0 ± 2.2; not ADHD: 5.1 ± 2.8) and the mean number of baseline hyperactive-impulsive symptoms (full ADHD: 6.3 ± 2.5; subthreshold ADHD: 5.5 ± 2.1; not ADHD: 3.5 ± 2.9) did not discriminate the 3 groups (inattentive: $F_{2,15} = 1.04$, $P = .376$, $\eta^2 = 0.12$; hyperactive-impulsive: $F_{2,15} = 1.50$, $P = .254$, $\eta^2 = 0.17$).

The mean number of ODD symptoms at baseline also predicted ADHD diagnostic status at follow-up ($F_{2,15} = 3.76$, $P = .048$, $\eta^2 = 0.33$). Those with full ADHD at follow-up had significantly more baseline symptoms (4.3 ± 0.6) than those with no ADHD at follow-up (1.2 ± 1.8, $P = .046$), but the number of baseline symptoms did not discriminate those with subthreshold ADHD at follow-up (2.0 ± 2.8) from those with full ADHD or no ADHD at follow-up. The mean number of conduct disorder symptoms at baseline (full ADHD: 1.7 ± 2.8; subthreshold ADHD: 0 ± 0; not ADHD: 0.5 ± 1.5) did not predict ADHD diagnosis at follow-up ($F_{2,15} = 0.73$, $P = .496$, $\eta^2 = 0.09$). As Table 3 shows, a baseline diagnosis of ODD predicted persistence, but other diagnoses, problems in school, or family history of ADHD did not.

Table 1. Demographics at the 9-Year Follow-Up

Item	*DSM-5* ADHD (n = 8), n (%)	Subthreshold ADHD (n = 9), n (%)	Not ADHD (n = 475), n (%)	χ^2 (*df*)	*P* Value
Sex				5.60 (2)	.06
Female	1 (12.5)	3 (33.3)	241 (50.7)		
Male	7 (87.5)	6 (66.7)	234 (49.3)		
Parental marital status				0.86 (4)	.93
Married	7 (87.5)	8 (88.9)	393 (82.7)		
Single	0 (0)	0 (0)	22 (4.6)		
Widowed/divorced/ separated	1 (12.5)	1 (11.1)	60 (12.5)		
Education of parent				3.08 (6)	.80
Elementary	0 (0)	0 (0)	5 (1.1)		
Secondary	2 (25.0)	1 (11.1)	72 (15.2)		
College	2 (25.0)	4 (44.4)	109 (22.9)		
Graduate	4 (50.0)	4 (44.4)	289 (60.8)		
Parent employed?				9.12 (2)	.01
Yes	8 (100)	5 (55.6)	415 (87.4)		
No	0 (0)	4 (44.4)	60 (12.6)		

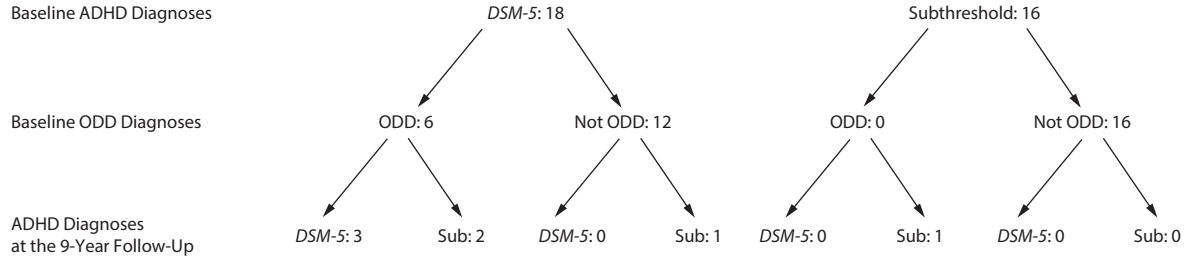
Abbreviation: ADHD = attention-deficit/hyperactivity disorder.

Predictors of New Onsets of ADHD

We next assessed which baseline features predicted new onsets of ADHD among those not diagnosed with ADHD at the baseline or year 4 follow-up assessments (Table 4). These analyses only included participants who had not been diagnosed with full threshold ADHD at either baseline or the year 4 follow-up. New onsets of *DSM-5* or subthreshold ADHD were predicted by past diagnoses of conduct or oppositional defiant disorder, having past school difficulties, repeating a grade in school, receiving treatment for ADHD, and having at least 1 sibling with ADHD. New onset cases were not more likely to have had subthreshold ADHD or a family history of ADHD. Baseline demographic variables did not predict new onsets of ADHD.

Those with new onset of ADHD at follow-up did not have more ADHD symptoms at baseline than those without new onset ADHD. We found no difference in the mean number of inattentive symptoms at baseline (full ADHD: 0.4 ± 0.5; subthreshold ADHD: 0.2 ± 0.4) compared with those who did not have ADHD at follow-up (0.3 ± 0.9; $F_{2,444} = 0.10$, $P = .903$, $\eta^2 < 0.01$). Those with new onsets also did not have more hyperactive-impulsive symptoms at baseline (full ADHD: 0.4 ± 0.9; subthreshold ADHD: 0 ± 0) compared with those who did not have ADHD at follow-up (0.2 ± 0.7; $F_{2,444} = 0.41$, $P = .662$, $\eta^2 = 0.02$) and did not differ in their number of total ADHD symptoms (full ADHD: 0.8 ± 1.3; subthreshold ADHD: 0.2 ± 0.4; not ADHD:

Figure 1. Numbers of Participants With Baseline ADHD and ODD Diagnoses With Evidence of Persistent ADHD



Abbreviations: ADHD = attention-deficit/hyperactivity disorder, ODD = oppositional defiant disorder.

Table 2. Psychiatric Disorders and School Problems at the 9-Year Follow-Up by Baseline ADHD Diagnosis

	Baseline ADHD Status				
	DSM-5 ADHD (n = 18), n (%)	Subthreshold ADHD (n = 16), n (%)	Not ADHD (n = 458), n (%)	χ^2 (*df* = 2)	*P* Value
Conduct disorder	1 (5.6)	0 (0)	1 (0.2)	12.25	.002
Oppositional defiant disorder	1 (5.6)	0 (0)	0 (0)	26.39	< .001
Alcohol use	1 (7.1)	2 (13.3)	31 (8.1)	0.55	.759
Tobacco use	2 (11.8)	0 (0)	5 (1.1)	12.79	.002
Cannabis use	0 (0)	1 (6.7)	5 (1.2)	3.71	.156
Pervasive developmental disorder	1 (5.6)	0 (0)	0 (0)	26.39	< .001
Depression	0 (0)	0 (0)	7 (1.5)	0.53	.768
Anxiety disorder or OCD	1 (5.6)	0 (0)	4 (0.9)	3.94	.139
Eating disorder	0 (0)	0 (0)	2 (0.4)	0.15	.928
Sleep disorder	1 (5.6)	0 (0)	30 (6.6)	1.14	.565
School difficulties	8 (44.4)	6 (37.5)	94 (20.5)	8.12	.017
Repeated a grade	7 (38.9)	4 (25.0)	83 (18.1)	5.20	.074

Abbreviations: ADHD = attention-deficit/hyperactivity disorder, OCD = obsessive-compulsive disorder.

Table 3. Association of Baseline Clinical Features and Family History With Persistence of ADHD[a]

	Time 3 ADHD Diagnosis				
Time 1 (Baseline) Feature	*DSM-5* ADHD (n = 3), n (%)	Subthreshold ADHD (n = 2), n (%)	Not ADHD (n = 13), n (%)	χ^2 (*df* = 2)	*P* Value
Conduct disorder	1 (33.3)	0 (0)	1 (7.7)	1.90	.386
Oppositional defiant disorder	3 (100)	1 (50.0)	2 (15.4)	8.14	.017
School difficulties	3 (100)	1 (50.0)	6 (42.6)	2.89	.236
Repeated a grade	2 (66.7)	1 (50.0)	1 (7.7)	5.91	.052
ADHD treatment	3 (100)	1 (50.0)	4 (30.8)	4.76	.093
Sibling ADHD diagnosis[b]	1 (33.3)	0 (0)	1 (7.7)	1.90	.386
Family history of adult ADHD[b]	1 (33.3)	0 (0)	1 (7.7)	1.90	.386

[a]Table includes only subjects who were diagnosed with ADHD at baseline.
[b]Assessed at Time 2 only.
Abbreviation: ADHD = attention-deficit/hyperactivity disorder.

Table 4. New Onsets of ADHD at the 9-Year Follow-Up by Baseline Clinical Features and Family History[a]

	New Onset ADHD at Follow-Up				
Time 1 (Baseline) Feature	*DSM-5* ADHD (n = 5), n (%)	Subthreshold ADHD (n = 6), n (%)	Not ADHD (n = 436), n (%)	χ^2 (*df* = 2)	*P* Value
Subthreshold ADHD[b]	1 (20.0)	0 (0)	34 (7.8)	1.54	.464
Conduct disorder[b]	3 (60.0)	2 (33.3)	49 (11.2)	13.65	.001
Oppositional defiant disorder[b]	3 (60.0)	3 (50.0)	87 (20.0)	7.96	.019
School difficulties[b]	2 (40.0)	3 (50.0)	57 (13.1)	9.65	.008
Repeated a grade[b]	2 (40.0)	2 (33.3)	50 (11.5)	6.38	.041
ADHD treatment[b]	2 (40.0)	0 (0)	20 (4.6)	13.56	.001
Sibling ADHD diagnosis[c]	0 (0)	2 (33.3)	25 (5.7)	8.27	.016
Family history of adult ADHD[c]	0 (0)	1 (16.7)	16 (3.7)	2.93	.231

[a]Table includes only subjects who had not been diagnosed with full ADHD at baseline or Time 2.
[b]Assessed at both baseline and Time 2.
[c]Assessed at Time 2 only.
Abbreviation: ADHD = attention-deficit/hyperactivity disorder.

0.5 ± 1.4; $F_{2,444} = 0.29$, $P = .749$, $\eta^2 = 0.01$). The number of ODD symptoms at baseline did not differentiate these groups (full ADHD: 0 ± 0; subthreshold ADHD: 0.7 ± 1.6; not ADHD: 0.3 ± 0.9; $F_{2,444} = 0.67$, $P = .510$, $\eta^2 = 0.03$), and neither did the number of conduct disorder symptoms at baseline (full ADHD: 0.2 ± 0.4; subthreshold ADHD: 0 ± 0; not ADHD: 0.2 ± 0.8; $F_{2,444} = 0.23$, $P = .792$, $\eta^2 = 0.01$).

Adolescents with new onset ADHD at follow-up did not differ in total ADHD symptoms at the year 4 follow-up from those without new onset ADHD (full ADHD: 2.0 ± 2.1; subthreshold ADHD: 1.3 ± 1.0; not ADHD: 0.9 ± 1.6; $F_{2,444} = 1.30$, $P = .272$, $\eta^2 = 0.06$). They also did not differ in the mean number of inattentive symptoms at year 4 (full ADHD: 1.6 ± 1.8; subthreshold ADHD: 0.3 ± 0.5, not ADHD: 0.6 ± 1.1; $F_{2,444} = 2.32$, $P = .100$, $\eta^2 = 0.01$) or in the mean number of hyperactive-impulsive symptoms at year 4 (full ADHD: 0.4 ± 0.5; subthreshold ADHD: 1.0 ± 1.2, not ADHD: 0.3 ± 0.9; $F_{2,444} = 1.77$, $P = .171$, $\eta^2 = 0.01$). However, they did differ in the mean number of ODD symptoms at year 4 ($F_{2,444} = 25.81$, $P < .001$, $\eta^2 = 0.10$).

Those with new onset full ADHD at year 9 had more ODD symptoms (9.2 ± 6.2) than those with subthreshold ADHD (5.3 ± 3.1, $P < .001$) and those with no ADHD (2.9 ± 2.0, $P = .007$). Those with subthreshold ADHD had more ODD symptoms than those with no ADHD ($P = .016$). They also differed in the mean number of conduct disorder symptoms at year 4 ($F_{2,444} = 23.46$, $P < .001$, $\eta^2 = 0.10$). Those with new onset full ADHD at year 9 had more conduct disorder symptoms (3.8 ± 2.9) than those with subthreshold ADHD (2.5 ± 0.8, $P < .001$) and no ADHD (2.1 ± 0.5, $P < .001$). Those with subthreshold ADHD did not have more conduct disorder symptoms than those with no ADHD ($P = .268$).

DISCUSSION

Our 9-year follow-up of a population sample to a mean age of 18 provides new data about the persistence of ADHD and the course of the disorder. We replicated prior predictors of ADHD's persistence and provide new data about predictors of new ADHD onsets in the population. We also addressed the validity of ADHD diagnoses that only meet subthreshold symptom criteria, which is an understudied area.

At a mean age of 18, 28% of the group with ADHD continued to show impairing symptoms of the disorder. Like prior follow-up studies, the

magnitude of ADHD's persistence depended upon how we defined remission.[29,30,35] The rate of persistence in our study is lower than the 67% rate computed in a meta-analysis of follow-up studies into young adulthood.[11] That work also showed that rates of persistence varied greatly with diagnostic methodology. Our telephone interview methodology may have had a low sensitivity, which is consistent with the relatively low prevalence we reported at the baseline assessment. The lower rate of persistence may also be due to our use of a population sample or to the site of ascertainment being France, a country that does not have a long history of recognizing and implementing the diagnosis of ADHD, particularly in adults. The lower severity of disorder in a non-referred population would lead to a lower level of persistence. This is especially true given data showing that increasing ADHD severity predicts greater persistence.[5,12–15,18,19,46,47]

At follow-up, the ADHD group had elevated rates of conduct disorder, ODD, school difficulties, and pervasive developmental disorders, which shows the continuing morbidity of the disorder. We did not, however, find significant differences in substance use disorders, which have been observed in other studies, such as that of Groenman et al.[12] Subsequent studies of this sample at older ages might see an emergence of these disorders. As reported by others, persistence of ADHD was predicted by baseline ADHD severity and ODD severity.[28,29,31,35–37,48,49] In contrast, we did not replicate prior reports that persistence could be predicted from conduct disorder, family history, or difficulties in school.[30,37]

If prior findings about familial ADHD predicting persistence were due to genetic effects, one would expect that twin studies of adults would yield higher heritabilities than twin studies of children. That is not the case,[50] which suggests that some familial environmental effect underlies the association between familiality and persistence. From our data, we cannot tell if this is a confounding effect, such as a parental bias in ratings, or if it is an environmental risk factor yet to be determined.

New onset cases of full ADHD were significantly more likely to have had subthreshold ADHD at baseline. New onsets of full ADHD or subthreshold ADHD were predicted by baseline measures of inattentive symptoms, hyperactive-impulsive symptoms, oppositional defiant symptoms, school difficulties, and ADHD treatment or having a family history of ADHD. As one might expect, participants with new onsets of ADHD were younger than other non-ADHD participants at baseline.

Our findings regarding subthreshold ADHD are consistent with prior cross-sectional and longitudinal work from population studies reporting that subthreshold ADHD predicted future onsets of the full threshold disorder.[51–54] Taken together, these findings suggest that subthreshold symptoms of the disorder foreshadow the future onset of the full ADHD diagnosis. The clinical relevance of these population findings was suggested in a study of 2,947 consecutive clinically referred youth which reported that subthreshold cases of ADHD had similar patterns of symptoms and impairment when compared with youth having the full threshold disorder.[45] This emerging research literature about subthreshold ADHD raises the question of whether it would be cost-effective to closely monitor children with subthreshold symptoms for future onset of the disorder, especially if they show associated features of ADHD such as ODD or school failure. Such monitoring would allow for prompt treatment with the emergence of the full threshold disorder but would also burden health care resources, patients, and families.

The results for subthreshold ADHD are consistent with the idea that ADHD is a dimensional syndrome[55] and that the clinical diagnosis corresponds to a cut-point on that dimensional continuum. This view of ADHD recently gained strong support from a large molecular genetic study of the disorder.[56] From the dimensional perspective, the category of subthreshold ADHD is analogous to borderline hypertension. Yet, clinicians should be careful in treating these cases, as that would greatly increase the prevalence. In our follow-up sample, the prevalence of *DSM-IV* ADHD was 3.0%, but when we add subthreshold cases, the prevalence climbs to 9.5%. A similar pattern of findings was seen in a family study of adult ADHD: subthreshold ADHD showed much evidence for validity, but its prevalence was too high to justify clinical utility.[43,57,58] More work is needed to better understand this subthreshold category. Moreover, we chose a definition of subthreshold ADHD we had used in prior work. There are no normative data on this definition nor is there a consensus about how subthreshold ADHD should be defined, especially in the context of comorbid disorders, most of which were not assessed in this study. Thus, the definition of subthreshold ADHD remains a fertile area for studies of diagnostic validity.

Our work should be viewed in the context of several methodological limitations. We conducted phone interviews with a parent rather than using an in-person interview because prior work suggests that telephone interviews are valid. Biederman et al[26] found that telephone interviews had high reliability ($\kappa = 0.93$, $P < .0001$), high sensitivity (95%), and high specificity (98%) when in-person interviews were used as gold standard diagnoses. Telephone interviews estimating the prevalence of ADHD[59] were replicated in work using in-person interviews,[60] and telephone interviews assessing functional impairments associated with ADHD[61] replicated our prior work using in-person interviews.[62] Other studies have documented the validity of telephone interviews for many psychiatric disorders.[63–70] Our assessment of family history most likely had low sensitivity because we did not inquire about ADHD diagnostic criteria, only whether the disorder had been diagnosed.

Another limitation is that we used only 1 informant (a parent) to collect information about the child. The estimated prevalence of ADHD is sensitive to the choice of informants and the number of informants.[71,72] In addition, many psychiatric disorders were not assessed, and only limited data on treatment were collected. Only 24% of eligible families agreed to participate at baseline, and 86.5% of these agreed

to participate at follow-up. Thus, any psychiatric phenomena due to nonparticipation could have skewed our results. Such biases, however, were not evident at follow-up based on comparisons of those lost and not lost to follow-up. Because we had small numbers in our 3 strata of persistence and in our late onset analyses, those results must be cautiously interpreted.

We must also be concerned about the generalizability of our results. Like all population studies, the number of affected members was relatively small, which raises the possibility that the small sample of ADHD youth at baseline was not representative of the population, although we did use a stratified sampling method that helped reduce that possibility. Also limiting generalization is that we conducted the work in France, a country that does not have a long history of recognizing and implementing the diagnosis of ADHD. Due to these factors, and the limitations noted above, our conclusions—particularly those that imply clinical relevance—require further work before they can be confidently accepted.

Despite these limitations, our work extends prior follow-up studies of ADHD from clinical samples to the general population and from North America to France. Our results regarding persistence are consistent with the prior literature, and we have also provided novel data about new onsets of ADHD in adolescence. If replicated, these data suggest that non-ADHD youth with associated features of the disorder should be monitored for the potential emergence of the disorder in adolescence. It is the task of future work to improve our ability to predict new onsets and confirm our finding that persistence of ADHD can be predicted with substantial accuracy.

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Potential conflicts of interest: In the past year, **Dr Lecendreux** has received consulting fees and been on advisory boards for Bioprojet, Jazz Pharma, UCB, and Shire. In previous years, he has received research support from Shire and received consulting fees from, been on advisory boards for, or been a speaker for Cephalon, UCB, Shire, and Eli Lilly. In the past year, **Dr Konofal** has received consulting fees and been on advisory boards for Shire. In previous years, he has received consulting fees from, been on advisory boards for, or been a speaker for H.A.C. Pharma, UCB, Janssen-Cilag, GlaxoSmithKline, and Vifor. He is CMO of NeuroLifeSciences, which is involved in experimental drug development. In the past year, **Dr Cortese** has received royalties from Aargon Healthcare Italy. In the past year, **Dr Faraone** received income, potential income, travel expenses, continuing education support, and/or research support from Vallon, Tris, Otsuka, Arbor, Ironshore, Shire, Akili Interactive Laboratories, VAYA, Sunovion, Supernus, and Genomind. With his institution, he has US patent US20130217707 A1 for the use of sodium-hydrogen exchange inhibitors in the treatment of ADHD. **Mr Silverstein** reports no potential conflict of interest.

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