Attention Problems in Childhood and Adult Substance Use

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Objective To assess the link between childhood attention problems (AP) and substance use 18 years later.

Study design This cohort study was conducted in a community sample of 1103 French youths followed from 1991 to 2009. Exposures and covariates were childhood behavioral problems (based on parental report at baseline), early substance use, school difficulties, and family adversity. Outcome measures were regular tobacco smoking, alcohol problems, problematic cannabis use, and lifetime cocaine use (based on youth reports at follow-up).

Results Individuals with high levels of childhood AP had higher rates of substance use (regular tobacco smoking, alcohol problems, problematic cannabis use, and lifetime cocaine use). However, when taking into account other childhood behavioral problems, early substance use, school difficulties, and family adversity, childhood AP were related only to regular tobacco smoking and lifetime cocaine use. Early cannabis exposure was the strongest risk factor for all substance use problems.

Conclusion This longitudinal community-based study shows that, except for tobacco and cocaine, the association between childhood AP and substance use is confounded by a range of early risk factors. Early cannabis exposure plays a central role in later substance use. *(J Pediatr 2013;163:1677-83)*.

A considerable body of research has focused on the link between childhood attention deficit/hyperactivity disorder (ADHD) and later substance use disorders (SUDs).[1,2] Studies based on clinic-referred samples have emphasized this deleterious association. Adolescent and adult patients with ADHD have shown increased rates of tobacco, alcohol, cannabis, and other drug use disorders in both cross-sectional and longitudinal settings.[3-5] Conversely, patients treated for SUDs display an overrepresentation (more than 25% of patients with an SUD) of both concurrent and retrospective ADHD[6,7]; however, because clinical samples tend to select the most severely affected individuals, these patients are not representative of the entire ADHD population. In addition, there are at least 2 problems with using the available clinical data. First, behavioral comorbidities (ie, externalizing and internalizing problems) often have been insufficiently controlled for, and when considered, they frequently acted as confounders or moderators of the association between ADHD and SUD.[8] Second, males have been investigated more often than females, precluding any inference of the findings in both sexes.

Longitudinal population-based studies are needed in this area. These studies can yield different results than clinical studies and can improve the generalizability of findings and possible inferences on causality. Early prospective studies found discrepant results. Some did not find any contribution of ADHD to later substance use, abuse, and dependence after controlling for conduct problems and confounders,[9,10] whereas others noted significant independent links only between tobacco smoking and ADHD and/or its specific dimensions (ie, inattention or hyperactivity-impulsivity).[11,12] Finally, some prospective community studies have reported a link between ADHD and/or its specific dimensions and the use of substances other than tobacco.[13,14]

Beyond the methodological variations in the way in which ADHD and substance use were measured, there have been some common limitations with this type of research. First, substance use outcomes were generally assessed when participants were age <18 years (ie, before they entered young adulthood, when dependence emerges), except for 2 studies that extended to 21 years[12] and 25 years.[10] Second, early first tobacco and cannabis use, which are considered to serve as gateway substance toward heavier forms of consumption, were most often not taken into account. Third, ADHD was often considered as a category rather than a dimensional measure,[9,11,14] even though growing evidence suggests

ADHD	Attention deficit/hyperactivity disorder
AP	Attention problems
CBCL	Child Behavior Checklist
DSM	*Diagnostic and Statistical Manual of Mental Disorders*
GAZEL	Electricity of France -Gas of France
SUD	Substance use disorder
TEMPO	Trajectoires épidémiologiques en population (epidemiological population trajectories)

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Table I. Characteristics of the TEMPO sample

Characteristic	Value
Sex, % (n/N)	
Male	41.2 (454/1103)
Female	58.8 (649/1103)
Age at baseline, y, mean (SD)	11.0 (3.7)
Age at follow-up, y, mean (SD)	28.9 (3.7)
Parental divorce, % (n/N)	14.8 (154/1043)
Parental depression, % (n/N)	29.5 (324/1098)
Parental alcohol problems, % (n/N)	23.0 (253/1099)
Parental tobacco use, % (n/N)	22.1 (244/1103)
Low family income at baseline, % (n/N)	34.8 (373/1071)
Participant position at follow-up, % (n/N)	
Student	9.3 (102/1097)
Worker	82.0 (900/1097)
Job-seeker	7.4 (81/1097)
Inactive	1.3 (14/1097)
Substance use at follow-up, % (n/N)	
Regular tobacco smoking	35.8 (385/1075)
Alcohol problems	14.3 (155/1086)
Problematic cannabis use	6.3 (68/1078)
Lifetime cocaine use	7.8 (83/1069)
Early first tobacco smoking	20.6 (227/1103)
Early first cannabis smoking	21.1 (233/1103)

that ADHD symptoms are continuously distributed in the population.[15] Thus, the dimensional approach is necessary to limit possible underestimation of the associations between ADHD symptoms and SUD.[2,15]

Overall, research examining the connection between ADHD and SUD remains controversial with respect to the independence of childhood ADHD in predicting adult SUD. In this study, we tested the hypothesis that attention problems (AP) in childhood and adolescence (age 4-16 years) are associated with substance use (tobacco, cannabis, alcohol, cocaine) in adulthood (age 22-35 years) independent of other factors (ie, childhood conduct problems and anxiety-depression, school difficulties, early tobacco/cannabis smoking, and family risk characteristics) in a community sample followed for up to 18 years.

Methods

Data for this study came from 2 French sources, young adults participating in the Trajectoires épidémiologiques en population (epidemiological population trajectories) (TEMPO) study (http://www.tempo.inserm.fr/) and their parents who are part of the Electricity of France–Gas of France (GAZEL) cohort study (http://www.gazel.inserm.fr/). The latter was set up in 1989 and included 20 624 men and women aged 35-50 years, employed in a variety of occupations ranging from manual worker to manager and living in France. Since study inception, participants have been followed yearly via self-report questionnaires. The TEMPO study was initiated in 2009 among young adults (age 22-35 years) who had previously taken part in a study of children's psychological problems and access to mental health care in 1991. The original sample in 1991 was selected among 4- to 16-year-olds whose parents were participants in the GAZEL study. The original sample (n = 2582) was selected to match the socioeconomic and family characteristics of French families in the 1991 census.[16,17]

In 2009, we asked parents of youths who had taken part in the 1991 survey to forward the TEMPO study questionnaire to their sons and daughters. Of the 2498 youths whose parents were alive and could be contacted, 16 had died since 1991 and 4 were too ill or disabled to respond. The overall response rate to the 2009 TEMPO questionnaire was 44.5% (n = 1103), which is comparable to response rates reported in other mental health surveys in France. Leading reasons for nonparticipation were nontransmission of the questionnaire by the parent (34.8%) and the youth's lack of interest (28.5%). Regarding baseline features, compared with participants, nonparticipants were more likely to be male (59.8% vs 41.2%), to come from a nonintact family (8.5% vs 5.5%), to come from a lower socioeconomic background (42.3% vs 34.8%), and to have parents who smoked tobacco (21.9 vs 18.7%) and abstained from alcohol (3.5% vs 1.5%). Participants and nonparticipants did not vary in terms of Child Behavior Checklist (CBCL) total score or parental depression. The TEMPO study was approved by the Commission Nationale Informatique et Liberté, the French national committee for data protection.

Youths' Mental Health at Baseline

Youths' psychopathology was assessed in 1991 when parents completed the CBCL.[18,19] The French version of the CBCL has been validated in previous clinical and epidemiologic studies.[20] This widely used tool includes 118 items on youths' behavioral problems in the preceding 6 months. The CBCL makes it possible to construct empirically based scales (based on factor analyses that identify syndromes of co-occurring problem items) and *Diagnostic and Statistical Manual of Mental Disorders* (DSM)-oriented scales (constructed from problem items that resemble DSM criteria for categorical diagnosis). DSM-oriented scales were proposed by Achenbach et al[19] as proxies for DSM diagnostic categories. They are built with items that do not include all DSM criteria, but nonetheless are considered satisfactorily consistent with

Table II. Substance use problems at follow-up by levels of AP

	Regular tobacco smoking	Alcohol problems	Problematic cannabis use	Lifetime cocaine use
AP ≥90th percentile, % (n/N)	52.8 (56/106)	16.7 (18/108)	9.4 (10/106)	14.4 (15/104)
AP 50th-90th percentile, % (n/N)	41.2 (169/410)	16.5 (68/413)	7.3 (30/411)	9.8 (40/407)
AP ≤50th percentile, % (n/N)	28.5 (156/548)	12.1 (67/554)	4.9 (27/550)	4.6 (25/547)
P value	<.0001	.0579	.0389	.0002

Mantel-Haenszel χ^2 test of linearity.

Table III. Correlation and phi coefficients among variables of interest

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13
1. AP	1												
2. Conduct disorder	0.44*	1											
3. Anxious-depressed	0.36*	0.22*	1										
4. Regular tobacco smoking	0.17*	0.15*	−0.01	1									
5. Alcohol problems	0.04	0.04	−0.08*	0.23*	1								
6. Problematic cannabis use	0.06*	0.12*	−0.03	0.31*	0.27*	1							
7. Lifetime cocaine use	0.14*	0.14*	−0.08*	0.29*	0.19*	0.30*	1						
8. School difficulties	0.37*	0.15*	0.17*	0.09*	0.03	0.03	0.11*	1					
9. Family risk index	0.06*	0.05	0.10*	0.10*	0.02	0.03	0.02	0.01	1				
10. Early tobacco initiation	0.06	0.10*	−0.04	0.24*	0.12*	0.11*	0.14*	0.04	0.09*	1			
11. Early cannabis initiation	0.07*	0.13*	−0.03	0.29*	0.21*	0.17*	0.24*	0.03	0.04	0.42*	1		
12. Sex	−0.14*	−0.13*	0.08*	−0.07*	−0.17*	−0.11*	−0.12*	−0.15*	−0.01	0.05	−0.01	1	
13. Age	0.09*	−0.14*	0.06*	−0.04	−0.12*	−0.06	0.01	0.14*	0.03	−0.07	−0.16*	−0.02	1

The Pearson correlation coefficient was used for continuous-continuous measure association; point-biserial correlation was used for dichotomous-continuous associations, and the Phi coefficient was used for dichotomous-dichotomous associations.
*$P < .05$.

DSM categories. The empirically based AP scale (Cronbach's alpha = 0.72) comprises the following items: "cannot concentrate," "daydreams," "impulsive," "cannot sit still," "acts young," "confused," "stares blankly," and "poor school work." The AP scale has been shown to be a good predictor of ADHD diagnosis.[21] We kept a single combined variable because factor analysis of the CBCL did not yield separate factors for inattention and hyperactivity-impulsivity.[19]

To take into account potential confounders, we accounted for baseline psychiatric comorbidity using symptoms of conduct disorder, using the DSM-oriented scale (16 items; Cronbach's alpha = 0.69), and symptoms of anxiety/depression (13 items; Cronbach's alpha = 0.75), using the corresponding CBCL empirically based scale. We chose conduct disorder DSM-oriented scales rather than the aggressive/delinquency empirically based scales. Indeed, aggressive/delinquency empirically based scales reflect a distinction between aggressive and nonaggressive conduct problems. In contrast, the DSM combines aggressive and nonaggressive conduct problems into the single category of Conduct Disorder.[19] Because we wanted to assess the moderating role of conduct disorder symptoms on the relationship between hyperactivity-inattention symptoms and academic outcomes, we deemed it appropriate to use the conduct disorder DSM-oriented scales. Data missing on each CBCL scale were imputed when less than one-third were missing. We generated an ordinal variable (symptom level: high, ≥90th percentile; intermediate, 50th-90th percentile; low, ≤50th percentile) for each CBCL problem scale.

Family Characteristics

Family data were obtained primarily from parents' yearly reports in the GAZEL study between 1989 and 2009. Low income at baseline was defined as <23 800 euros/year (lowest tertile) vs ≥23 800 euros/year. Parental separation or divorce (yes vs no) was reported in the yearly GAZEL questionnaire. Parental depression (yes vs no) was defined as at least 2 parental self-reports of depression in the yearly GAZEL study questionnaire or by TEMPO participants' report of parental lifetime depression ascertained using a questionnaire adapted from the National Institute for Mental Health's Family Interview for Genetic Studies.[22] Parental alcohol problem (high alcohol use present vs absent) was defined as at least 2 parental self-reports of high alcohol use in the yearly GAZEL study questionnaire (≥21 glasses of alcohol/per week in women, ≥28 glasses of alcohol/per week in men), and TEMPO participants' reports of parental alcohol dependence were ascertained using a questionnaire adapted from the Family Interview for Genetic Studies.[22]

Parental tobacco smoking was assessed by yearly self-report from 1989 to 2009 in the GAZEL study. Each year, reference parents reported whether they smoked tobacco and, if so, the daily number of cigarettes, pipes or cigars. This information was used to construct longitudinal trajectories of parental tobacco use for no smoking (77.8% of parents), declining smoking (13.0%), or persistent smoking (9.2%). These trajectories were identified empirically using a semiparametric mixture model. For each tobacco smoking group, the model defined the shape of the trajectory and the proportion of parents in each group. The validity of this classification was confirmed using the Bayes information criterion. A binary variable (declining or persistent smoking vs no smoking) was then constructed. The overall family risk index was created by summing the risk for parental low income, parental separation or divorce, parental depression, parental alcohol problem, and parental tobacco smoking. The cumulative risk was then dichotomized (≥2 vs <2).

Youths' Substance Use at Follow-Up

In 2009, study participants were asked to report their past 12-month use of tobacco, alcohol, and cannabis. Past 12-month regular tobacco smoking was defined as ≥1 cigarette per day/less than 1 cigarette per day. Alcohol-related problems were assessed using the French version of the Alcohol Use Disorders Identification Test, a 10-item screening test developed by the World Health Organization and validated against clinical diagnosis,[23,24] which captures alcohol abuse and dependence (referred to as alcohol abuse hereinafter).[25] According to published guidelines, men who scored ≥8 and women who scored ≥7 were considered to have signs of

Table IV. Multivariate modeling of substance use problems at follow-up in function of childhood AP and other covariates (ordinal approach)

	Regular tobacco smoking		Alcohol problems		Problematic cannabis use		Lifetime cocaine use	
	OR 1 (95% CI)	OR 2 (95% CI)	OR 1 (95% CI)	OR 2 (95% CI)	OR 1 (95% CI)	OR 2 (95% CI)	OR 1 (95% CI)	OR 2 (95% CI)
CBCL problems								
Attention								
≥90th percentile	2.74 (1.79-4.21)*	2.19 (1.31-3.68)*	1.45 (0.83-2.56)	1.43 (0.71-2.86)	2.23 (1.04-4.80)*	1.35 (0.53-3.43)	3.10 (1.56-6.17)*	2.73 (1.16-6.45)*
50th-90th percentile	1.73 (1.32-2.27)*	1.47 (1.08-2.01)*	1.43 (1.00-2.06)†	1.31 (0.86-1.99)	1.57 (0.92-2.69)	1.06 (0.58-1.96)	2.10 (1.25-3.54)*	1.80 (0.99-3.28)†
≤50th percentile	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
Conduct								
≥90th percentile	2.43 (1.57-3.76)*	1.61 (0.97-2.66)†	1.68 (0.98-2.90)†	0.97 (0.51-1.84)	2.58 (1.25-5.31)*	2.27 (0.98-5.25)†	2.40 (1.18-4.88)*	1.57 (0.68-3.64)
50th-90th percentile	1.71 (1.31-2.24)*	1.47 (1.08-2.01)*	1.16 (0.80-1.69)	1.02 (0.68-1.52)	1.54 (0.87-2.69)	1.42 (0.77-2.61)	1.90 (1.14-3.18)*	1.45 (0.81-2.58)
≤50th percentile	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
Anxious-depressed								
≥90th percentile	1.14 (0.75-1.73)	0.74 (0.45-1.21)	0.56 (0.28-1.12)	0.48 (0.23-1.03)	0.56 (0.20-1.62)	0.39 (0.12-1.24)	0.38 (0.13-1.06)	0.16 (0.05-0.50)*
50th-90th percentile	0.92 (0.71-1.21)	0.78 (0.58-1.06)	0.89 (0.62-1.29)	0.81 (0.54-1.21)	1.02 (0.61-1.72)	0.90 (0.51-1.59)	0.71 (0.43-1.15)	0.46 (0.26-0.82)*
≤50th percentile	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
School difficulties (yes vs no)	1.47 (1.12-1.93)*	1.18 (0.86-1.62)	1.10 (0.76-1.61)	1.05 (0.69-1.59)	1.15 (0.67-1.96)	1.07 (0.59-1.93)	1.99 (1.25-3.17)*	2.09 (1.22-3.58)*
Family risk index (≥2 vs <2)	1.54 (1.18-2.00)*	1.44 (1.08-1.91)*	1.17 (0.81-1.68)	1.08 (0.74-1.59)	1.36 (0.82-2.25)	1.36 (0.80-2.31)	1.18 (0.74-1.88)	1.23 (0.74-2.05)
Early initiation								
Tobacco (yes vs no)	3.31 (2.43-4.50)*	1.85 (1.30-2.63)*	2.24 (1.52-3.29)*	1.36 (0.87-2.12)	2.68 (1.59-4.51)*	1.56 (0.85-2.88)	2.99 (1.86-4.80)*	1.33 (0.75-2.38)
Cannabis (yes vs no)	4.30 (3.15-5.87)*	3.05 (2.15-4.31)*	3.22 (2.21-4.67)*	2.74 (1.80-4.18)*	3.69 (2.21-6.14)*	2.71 (1.50-4.88)*	5.63 (3.51-9.06)*	4.89 (2.80-8.53)*

OR 1, age- and sex-aOR; *OR 2*, fully aOR.
*$P < .05$.
†$P < .10$.

alcohol abuse or dependence.[25] Problematic cannabis use was assessed by 7 questions adapted from the Cannabis Abuse Screening Test.[26] The total score ranged from 0 to 6, with a score of ≥2 considered indicative of problematic use according to the test authors' recommendations. Participants were also asked about age at first tobacco and cannabis use. Lifetime cocaine use was defined as ≥1 occasion. Early tobacco initiation (age ≤14 years) and early cannabis initiation (age ≤16 years) were defined consistent with measures used in previous studies.[27,28]

Youths' School Difficulties

School difficulties were assessed by combining youths' poor academic performance in 1991 (in French, mathematics, sciences, foreign language; prevalence of failing at least one subject, 4.9%), learning difficulties in 1991 (prevalence = 13.7%), and grade retention reported in 2009. Because grade retention is common in France, only participants who were retained at least twice were considered to have had school difficulties (prevalence of ≥2 grade retentions, 19.1%).

Statistical Analyses

We first described sample characteristics and position at follow-up, then described substance use at follow-up by AP level at study baseline. We then sought to estimate the strength of the association between childhood AP (main exposure) and substance use 18 years later (outcome), controlling for potential confounders (CBCL problems and early tobacco and cannabis initiation) and covariates (age, sex, school difficulties, and family risk index). Analyses were performed using logistic regression models adjusted for CBCL problems, sex (male vs female), age (continuous), school difficulties (yes vs no), family risk index (≥2 vs <2), early tobacco initiation (age ≤14 years vs >14 years), and early cannabis initiation (age ≤16 years vs >16 years). We established 2 sets of multivariate models to study behavioral problems measured using the CBCL as ordinal variables and as *z*-scores. We tested statistical interactions among AP, sex, and all significant variables in the models. Multicollinearity was tested using the Belsley criteria. Statistical significance was determined at a level of $P < .05$. All calculations were performed using SAS version 9.1 (SAS Institute, Cary, North Carolina). To test the robustness of our findings, we conducted complementary sensitivity analyses using multiple-imputation models to account for missing data, removing variables highly correlated to AP (main exposure) to rule out possible overadjustment.

Results

Table I presents the main sociodemographic features of the sample. Table II characterizes participants' substance use at follow-up by AP level at study baseline. Participants with high AP levels were more likely to exhibit higher rates of substance use problems. Table III presents correlation and phi coefficients for the variables of interest. Table IV provides the results of regression analyses for substance use

problems at follow-up, with CBCL problems coded as ordinal variables. Table V (available at www.jpeds.com) presents the results of regression analyses for substance use problems at follow-up, with CBCL problems coded as continuous *z*-standardized variables. All multivariate models were significant (Ns range was 1057-1074; $P < .0001$ for all Wald χ^2). The categorical and dimensional approaches provided similar results. AP was significantly related to regular tobacco smoking 18 years later and to lifetime cocaine use. Early cannabis initiation was significantly associated with all types of substance use problems. There was no significant statistical interaction. The percentage of missing data ranged from 2.7% to 4.2% in the different models using the sample with data available at both baseline and follow-up. Sensitivity analyses demonstrating the same pattern of results (not shown) using multiple-imputation models and when removing conduct problems, anxious-depressed, and school difficulties from the multivariate modeling.

Discussion

In this longitudinal French population-based study, individuals with higher levels of childhood and adolescent AP demonstrated higher rates of substance use problems in adulthood. However, after accounting for a range of potential confounders (ie, childhood conduct problems or anxiety/depression, school difficulties, family risk, and early tobacco/cannabis initiation), AP were independently associated only with regular tobacco smoking and lifetime cocaine use. Interestingly, early cannabis initiation appeared to be the strongest risk factor of later substance use, regardless of the type of substance considered.

Our study confirms the results reported by Pingault et al[12] showing that AP are independently associated with subsequent nicotine problems, but not with cannabis or alcohol problems. We also extend these previous results to a later age group and account for early cannabis initiation. However, Pingault et al found no association between AP and cocaine use. Of note, cocaine use was measured differently in the 2 studies (cocaine abuse/dependence vs lifetime cocaine use), which limits comparisons. In addition, our findings partially diverge from 2 other recently reported longitudinal and community-based studies. The Christchurch Health and Development Study found no independent association between AP and tobacco use/dependence and only a weak association with cannabis abuse/dependence.[10] However, this earlier study involved a younger age group (ie, 18-25 years), and the analyses were not adjusted on early cannabis use. The Minnesota Twin Family Study reported a significant link between hyperactivity-impulsivity and most substance abuse/dependence outcomes at age 18 years.[13] Insufficient covariate control in this study with no adjustment for anxious-depressed symptoms, family risk, or early first substance use, may explain the discrepancies.

The link between AP and tobacco smoking is consistent in both the present study and in the literature, making a direct causal pathway or genetic/environmental commonalities plausible. Tobacco could be used to self-medicate by relieving the impairment owing to the core characteristics of ADHD, such as poor concentration and executive dysfunctions. This idea is supported by studies showing improved attention after nicotine administration[29] and a possible role of nicotinic $\alpha 7$ receptors in learning, memory, and attention.[30] In addition, ADHD physiology could involve nicotinic-acetylcholinergic circuits and the dopamine reward-processing system.[31] The link between AP and cocaine may reflect a propensity to experiment with illicit substance use, especially stimulating substances. We could not assess the strength or the specificity of the relationship in more depth, however, because we did not consider diagnoses of substance abuse or dependence. Our findings partially support the dual pathway model,[10] because ADHD was not related to alcohol problems or cannabis problematic use once comorbidity (notably conduct problems) and early substance initiation were accounted for.

Of importance, we were able to consider the timing of tobacco and cannabis exposure (first use), an issue rarely addressed in previous ADHD-addiction research. We found that early cannabis initiation was the strongest predictor of all substance use problems and often confounded the effect of other risk factors. This is consistent with previously reported evidence suggesting that early substance use, particularly early cannabis initiation, can play an important role in increased later risk of adult substance dependence.[32,33] Cannabis may play a central role through a "gateway" effect and by mediating the influence of childhood and adolescent risk factors on later substance use problems. This is a crucial finding that has been insufficiently considered in previous research. Some recent results suggest that this association may be partially mediated by increasing exposure to and opportunity to use other illicit drugs.[34] Early cannabis use also may reflect an underlying common vulnerability implicating striatal dopamine, reward sensitivity, and risk-taking.[35]

Within the addiction and ADHD research field, the strengths of this study are its use of a longitudinal community sample, follow-up to a mean age of 29 years, a dimensional approach to ascertain AP, adjustment for behavioral comorbidities, and early tobacco/cannabis initiation. Some caveats inherent to this type of survey should be taken into account when interpreting the results, however. First, the use of CBCL to measure psychiatric problems precluded consideration of functional impairment, symptom duration, and ADHD subtypes. Nevertheless, CBCL scales have shown high levels of validity compared with DSM clinical diagnoses in independent samples.[19] Second, attrition was notable in this longitudinal dataset. Reassuringly, comparisons between participants and nonparticipants in 2009 revealed no significant differences regarding youth and parental psychiatric problems, suggesting that nonresponse did not induce any meaningful bias in connection with these features. Third, attrition was selective. Individuals with lower socioeconomic status at baseline were underrepresented, because participants came from families in which 1 parent had good job security, and

the likelihood of participating at follow-up is greater in families with more favorable socioeconomic status. This might have biased our results toward minor cases and thus may have generated more conservative findings. Fourth, we did not consider other potential confounders, such as ADHD symptoms at follow-up, IQ, learning disability, executive dysfunction, bipolar disorder, child maltreatment, biological factors, parental ADHD, or ADHD treatment status. However, in view of the study period and the French setting, this sample is unlikely to have been exposed to stimulant medications.

Childhood ADHD appears to be an independent early risk factor only for regular adult tobacco smoking and lifetime cocaine use. Owing to its prevalence and chronicity, considering early ADHD and its contribution to smoking could help mitigate later dependence. Similarly, seeking and treating undiagnosed ADHD in adults with nicotine dependence may be useful in tobacco addiction treatment. In addition, early cannabis initiation and, to a lesser extent, childhood conduct problems also contributed to the emergence of substance use problems. This is crucial, because many youths experiment with cannabis early on. This experimentation plays an important long-term role not only with regard to substance use problems, but also in other negative outcomes, such as risky behaviors, low educational attainment, and psychopathological difficulties. Prevention efforts should address substance use initiation through broad universal interventions as well as selective programs targeting early experimenters and youths who exhibit conduct problems. ■

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Table V. Multivariate modeling of substance use problems at follow-up in function of childhood AP and other covariates (continuous approach)

	Regular tobacco smoking		Alcohol problems		Problematic cannabis use		Lifetime cocaine use	
	OR 1 (95% CI)	OR 2 (95% CI)	OR 1 (95% CI)	OR 2 (95% CI)	OR 1 (95% CI)	OR 2 (95% CI)	OR 1 (95% CI)	OR 2 (95% CI)
CBCL problems (continuous)								
Attention	1.42 (1.24-1.61)*	1.34 (1.13-1.58)*	1.13 (0.96-1.33)	1.18 (0.94-1.48)	1.26 (1.01-1.58)*	1.07 (0.78-1.47)	1.49 (1.21-1.82)*	1.47 (1.10-1.97)*
Conduct disorder	1.35 (1.19-1.54)*	1.16 (1.00-1.35)†	1.12 (0.96-1.30)	0.94 (0.77-1.15)	1.32 (1.10-1.59)*	1.28 (1.02-1.60)*	1.39 (1.17-1.65)*	1.23 (0.97-1.55)†
Anxious-depressed	1.00 (0.88-1.13)	0.85 (0.73-0.99)*	0.80 (0.65-0.97)*	0.74 (0.59-0.94)*	0.88 (0.67-1.17)	0.78 (0.57-1.09)	0.71 (0.53-0.94)*	0.48 (0.33-0.68)*
School difficulties (yes vs no)	1.47 (1.12-1.93)*	1.15 (0.84-1.58)	1.10 (0.76-1.61)	1.05 (0.69-1.61)	1.15 (0.67-1.96)	1.06 (0.58-1.93)	1.99 (1.25-3.17)*	1.87 (1.07-3.26)*
Family risk index (≥2 vs <2)	1.54 (1.18-2.00)*	1.44 (1.09-1.92)*	1.17 (0.81-1.68)	1.11 (0.76-1.62)	1.36 (0.82-2.25)	1.32 (0.78-2.25)	1.18 (0.74-1.88)	1.23 (0.73-2.05)
Early initiation								
Tobacco (yes vs no)	3.31 (2.43-4.50)*	1.83 (1.29-2.60)*	2.24 (1.52-3.29)*	1.36 (0.87-2.12)	2.68 (1.59-4.51)*	1.50 (0.82-2.76)	2.99 (1.86-4.80)*	1.28 (0.72-2.28)
Cannabis (yes vs no)	4.30 (3.15-5.87)*	3.07 (2.17-4.34)*	3.22 (2.21-4.67)*	2.77 (1.82-4.23)*	3.69 (2.21-6.14)*	2.75 (1.53-4.94)*	5.63 (3.51-9.06)*	5.02 (2.87-8.77)*

*$P < .05$.
†$P < .10$.