

Childhood Predictors of Adolescent Substance Use in a Longitudinal Study of Children With ADHD

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Children diagnosed with attention-deficit/hyperactivity disorder (ADHD; $n = 142$) were prospectively monitored into adolescence (13–18 years old) to evaluate their risk for elevated substance use relative to same-aged adolescents without ADHD ($n = 100$). Probands reported higher levels of alcohol, tobacco, and illicit drug use than did controls. Group differences were apparent for alcohol symptom scores but not for alcohol or marijuana disorder diagnoses. Within probands, severity of childhood inattention symptoms predicted multiple substance use outcomes; childhood oppositional defiant disorder/conduct disorder (ODD/CD) symptoms predicted illicit drug use and CD symptoms. Persistence of ADHD and adolescent CD were each associated with elevated substance use behaviors relative to controls. Further study of the mediating mechanisms that explain risk for early substance use and abuse in children with ADHD is warranted.

Attention-deficit/hyperactivity disorder (ADHD) is one of the most commonly diagnosed mental health disorders of childhood, occurring in 3% to 5% of school-age children (Barkley, 1998). In recent years, a focus on risk for substance use and substance use disorder (SUD) in this population has become a matter of public and scientific debate (National Institutes of Health, 2000). Well-known longitudinal studies of children with ADHD have examined rates of substance use and SUD by adolescence (e.g., Barkley, Fischer, Edelbrock, & Smallish, 1990; Biederman et al., 1997; Claude & Firestone, 1995; Gittelman, Mannuzza, Shenker, & Bonagura, 1985; Hartsough & Lambert, 1987; Loney, Kramer, & Milich, 1981; Mannuzza et al., 1991; Milberger, Biederman, Faraone, Chen, & Jones, 1997). These studies represent important initial efforts toward understanding risk for early substance use and SUD in this population, but more research is needed to explain

discrepant findings. For example, in the Milwaukee study of 123 adolescents with childhood hyperactivity, statistically significant group differences were found for cigarettes, alcohol, and marijuana, but Bonferroni corrections relegated two findings to statistical nonsignificance (Barkley et al., 1990). In the Berkeley sample of 54 adolescents with pervasive hyperactivity, Hartsough and Lambert (1987) reported group differences for cigarette use but not for alcohol or for other nonalcohol drugs. In the Boston study of 128 adolescents with childhood ADHD, SUDs were not significantly higher in the proband group (Biederman et al., 1997), but in the New York study of 101 adolescents with childhood hyperactivity, higher rates of drug (but not alcohol) dependence were found (Gittelman et al., 1985). On the surface, these discrepancies are puzzling. However, when the average age of the adolescents is considered, as well as the manner in which substance use/SUD was assessed, a need for further study becomes evident.

Most of the longitudinal research on childhood ADHD and later substance use/SUD has been conducted secondary to the initial goal of studying the long-term course of ADHD. Consequently, the detailed substance use assessment that is common in longitudinal studies of substance use has been missing in longitudinal studies of childhood ADHD. The result may be missed group differences in substance use behaviors prognostic of later abuse or dependence. Several methodological limitations have been noted in existing studies. First, diagnosing substance abuse or dependence in adolescence when rates of disorder have not yet reached their peak can miss emerging problems (e.g., Biederman et al., 1997; Gittelman et al., 1985; Mannuzza et al., 1991) that may be more appropriately measured as continuous variables. Second, for substances widely experimented with in adolescence (e.g., alcohol, cigarettes, marijuana), analysis of frequency or quantity of use, including heavy use, rather than any use over the lifetime or dependence, is more important and relevant toward development of later problematic use. Finally, age of first substance use is also a well-established predictor of later problematic substance use (Grant & Dawson, 1997; Robins & Przybeck, 1985) that has not

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been sufficiently included in ADHD studies. In the study here, we addressed these issues by assessing a range of substance use behaviors that are developmentally appropriate for the age of the probands.

Beyond establishing the magnitude of risk for early substance use in children with ADHD, we were interested in testing whether severity of childhood symptomatology prospectively predicted early substance use in the ADHD sample. We focused on severity of childhood ADHD symptomatology and comorbid oppositional defiant disorder/conduct disorder (ODD/CD) symptomatology because theoretical models of early substance abuse implicate a heterogeneous mix of childhood behavior problems as risk factors (Sher & Trull, 1994; Tarter et al., 1999; Wong, Zucker, Puttler, & Fitzgerald, 1999). This mix includes aspects of difficult childhood temperament that are very similar to the defining and associated features of ADHD (e.g., impulsivity/disinhibition). However, it remains unknown whether certain features of ADHD symptomatology, versus the comorbid behavior problems that develop in children with ADHD, are responsible for the risk within this population.

Early expressions of antisocial behavior, such as aggression, defiance, and other conduct problems in childhood, are common among children with ADHD (Hinshaw, 1987). Conduct problems are also well-established prospective predictors of early substance use and SUD in both clinical and nonclinical samples (e.g., Brook, Whiteman, Finch, & Cohen, 1995; Lynskey & Fergusson, 1995). Indeed, given the well-established link between substance use and abuse and other norm-violating behavioral problems in youth (Jessor & Jessor, 1977), it is not surprising that early expressions of conduct problems would be prognostic of substance abuse. Among ADHD children, however, little effort has been directed toward testing prospectively the contribution of this common comorbidity in childhood toward risk for early substance use and SUD in adolescence. Existing studies of clinic samples have shown that childhood aggression (Loney et al., 1981) or CD (Burke, Loeber, & Lahey, 2001) predicted alcohol and other substance use in adolescence, but that hyperactivity or inattention did not. However, childhood ADHD rather than CD predicted later tobacco use (Milberger et al., 1997), and adolescent inattention was correlated with tobacco use independent of adolescent CD (Burke et al., 2001).

Replication and expansion of these findings are needed for several reasons, including a broader assessment of substance use, as discussed previously. In addition, extant studies are quite variable in the adequacy with which they have assessed childhood ADHD and CD/aggression problems. There is now considerable empirical support for the distinctiveness of the inattention and impulsivity-hyperactivity dimensions of ADHD both from aggression/conduct problems and from each other (*Diagnostic and Statistical Manual of Mental Disorders, DSM-IV*, American Psychiatric Association, 1994; Milich, Balentine, & Lynam, 2001). For example, in the longest follow-up study (Loney et al., 1981), childhood "hyperactivity" was a composite measuring ADHD symptoms, including one or two each of inattention, hyperactivity, and impulsivity; thus, study of the two dimensions now known to be critical was not possible. Hence Loney et al.'s (1981) study may have underestimated the predictive power of ADHD symptom severity relative to co-occurring aggression and conduct problems in childhood.

Studies that have separated the ADHD symptoms into the two *DSM-IV* dimensions have shown differential relationships with later substance use. For example, Burke et al. (2001) found the inattention dimension to be significantly associated with concurrent tobacco use but not alcohol or other substance use. The empirically demonstrated salutary effects of tobacco on attentional functioning (Levin et al., 1998) and the possibility that tobacco use may reflect self-regulation of attention make further research on this ADHD dimension particularly important.

Other studies that have employed validated, contemporary dimensions of ADHD have suggested that the behavioral disinhibition component of ADHD might be particularly important for the development of substance use and substance use related impairment in adolescents (Molina, Smith, & Pelham, 1999). The idea that behavioral impulsivity, in interaction with environmental risk factors, is important for the development of serious chronic antisocial behavior is not new (Gorenstein & Newman, 1980; Moffitt, 1993) and is empirically supported (Taylor, Iacono, & McGue, 2000; White et al., 1994). Moreover, recent longitudinal data revealed that impulsivity-hyperactivity symptoms in childhood predicted later ODD symptoms in children, after controlling for prior inattention and ODD symptoms (Burns & Walsh, 2002), suggesting the prospective importance of early behavioral inhibition for the later development of problem behavior. Indeed, Barkley (1997) argued that disinhibition is the fundamental deficit in ADHD and accounts for most ADHD-related impairment.

In addition to the importance of using contemporary measures of childhood functioning to predict later substance use, previous research with clinic samples of children with ADHD has suggested that persistence of ADHD and the development of CD in adolescence are important for the initiation of substance use and for the development of substance dependence (Barkley et al., 1990; Gittelman et al., 1985). The development of CD has also been reported always to predate or coincide with the onset of late adolescent substance dependence in children with ADHD (Gittelman et al., 1985; Mannuzza et al., 1991). However, what has not yet been tested is whether adolescents with persistent ADHD report elevated levels of substance use compared to controls even when they do not develop CD in adolescence. Such a finding would support the hypothesis that persistence of ADHD, separate from deviant/antisocial behavior, is important when adolescents begin experimenting with drugs and alcohol. This hypothesis is consistent with Mason and Windle's (2002) recent finding that delinquent behavior and substance use in adolescence are strongly associated, yet empirically distinct, behaviors.

In summary, this study was designed to clarify the magnitude of risk for early substance use and SUD in clinic-referred children with ADHD compared to children without ADHD. As discussed previously, to extend the extant literature we employed contemporary measures in childhood of the two dimensions of ADHD, as well as a dimension of antisocial behavior. We tested whether severity of childhood symptomatology in the ADHD sample prospectively predicted their elevated substance use 5 years later. We included a full range of substance use variables that are developmentally appropriate for the adolescent age range, and potentially prognostic of later SUD, including age of onset, lifetime use, quantity and frequency of use, and SUD. Finally, we assessed the separate roles of the persistence of ADHD and the development of CD in adolescence on substance use.

Method

Participants

Participants were 142 adolescents with childhood ADHD (probands) and 100 demographically similar adolescents without childhood ADHD recruited from the Pittsburgh area (controls). Probands were recruited for this study from the attention deficit disorder (ADD) clinic records at Western Psychiatric Institute and Clinic, University of Pittsburgh Medical Center, for services received between 1987 and 1995. Probands all met diagnostic criteria for ADHD in childhood in accordance with the *Diagnostic and Statistical Manual of Mental Disorders*, 3rd rev. and 4th editions (*DSM-III-R* and *DSM-IV*; American Psychiatric Association, 1987, 1994). Participating probands were between the ages of 5 and 17 when they received services (88.7% were between 5 and 12). When they reached adolescence (13–18 years of age), eligible children and their parents were recontacted to participate in the study; 56.5% agreed to participate. An average of 5.26 years had elapsed between the childhood assessment and the interview in adolescence. Despite the modest rate of participation, comparisons between nonparticipants and 111 of the participants indicated no statistically significant differences in ADHD/ODD/CD symptoms or IQ/achievement scores (all $d_s < .20$).

The standard ADD Clinic assessment when the probands were children included the following packet of intake measures: the Disruptive Behavior Disorders Scale (DBD; Pelham, Gnagy, Greenslade, & Milich, 1992), IOWA/Abbreviated Conners Rating Scale (Goyette, Conners, & Ulrich,

1978; Loney & Milich, 1982), and Swanson, Nolan, and Pelham Rating Scale (SNAP; Atkins, Pelham, & Licht, 1985; Swanson, 1992). These scales are norm-referenced behavior-rating scales of *DSM-III-R* and *DSM-IV* ADHD symptoms and additional externalizing and social behaviors, and have acceptable psychometric properties comparable to those of other parent and teacher rating scales. These measures were completed by parents and teachers. A semistructured diagnostic interview with parents was also conducted by PhD clinicians to confirm ADHD symptomatology, assess comorbid problems, and rule out alternative diagnoses in the context of a broad clinical interview. Independent file reviews by a second clinician were conducted at referral on a subset of files to confirm diagnosis. Using the combined parent and teacher DBD data, all probands met *DSM-III-R* diagnostic criteria for ADHD, 74.3% of the probands met *DSM-III-R* diagnostic criteria for ODD, and 40.8% met *DSM-III-R* diagnostic criteria for CD, in childhood. Exclusionary criteria included an IQ less than 80, seizures or other neurological problems, or a history of pervasive developmental, psychotic, sexual, or organic mental disorders.

The 100 adolescents without childhood ADHD (controls) were recruited in adolescence from the greater Pittsburgh area using primarily newspaper and university advertisements (80%), with school fliers and word-of-mouth accounting for the remaining participants. A telephone screening was used to ascertain demographic characteristics and exclusionary criteria, and to administer a checklist of *DSM-III-R* ADHD symptoms. Control participants were matched to the ADHD group on age, gender, race, parental education, and single- versus two-parent household (see Table 1). Controls

Table 1
Demographic Characteristics of the Sample in Adolescence

Characteristic	Non-ADHD <i>n</i> = 100 % ^a	ADHD <i>n</i> = 142 % ^a
Demographic matching variables		
Age (<i>M, SD</i>)	15.18 (1.42)	15.18 (1.44)
Gender (female)	5.0	6.3
Racial minority	13.0	13.4
African American	8.0	10.6
Other	5.0	2.8
Highest parent education ^b (<i>M, SD</i>)	5.87 (0.96)	5.70 (1.03)
High school grad or GED	7.0	13.4
Part college or specialized training	32.0	31.7
College or university grad	28.0	26.1
Graduate professional training	33.0	28.9
% Single parent household	27.0	30.3
Other demographic variables		
Total parental income (<i>M, SD</i>) ^{c,d}	57,337 (38,246)	56,037 (42,986)
Median income	50,000	46,000
≤20K	11.22	14.96
21K–40K	26.53	27.56
41K–60K	28.57	25.20
61K–80K	15.31	18.11
81K–100K	11.22	6.30
≥101K	7.14	7.87
Grade in school or last grade completed		
Equivalent completed (<i>M, SD</i>)	9.73 (1.47)	9.35 (1.54)
Participated with a custodial biological parent	97.0	88.73*
Adopted	2.0	8.45**
Age at assessment in childhood (<i>M, SD</i>)	NA	9.93 (2.25)
Follow-up interval (<i>M, SD</i>)	NA	5.26 (2.22)

^a Except where indicated otherwise. ^b A 7-point scale was used to rate parent education, for example, 1 (<7th-grade education), 4 (high school graduate or GED), 5 (partial college or specialized training), 6 (standard college or university graduation), and 7 (graduate professional training). ^c Income data missing from 17 families. ^d In U.S. dollars.

* $p < .05$. ** $p < .01$.

were required to be free of a DSM-III-R ADHD diagnosis based on data collected at the telephone screening or interview (see the following), but presence of other mental health disorders was acceptable.

Procedure

ADHD and non-ADHD adolescents participated with their parents in a one-time office-based interview in which adolescents, mothers, and fathers were interviewed separately. Paper-and-pencil and interview questions were read aloud to adolescents who followed along on their own copy of the measures. Interviewers recorded the answers (substance use was an exception; see details later). Confidentiality of information was supported with a Certificate of Confidentiality from the Department of Health and Human Services with certain exceptions (e.g., suicidality, child abuse), and the protocol was approved by the University of Pittsburgh Institutional Review Board. At least three teachers of primary academic subjects were asked to complete ratings of behavior and academic performance.

Measures

Childhood inattention, impulsivity-hyperactivity, and ODD/CD symptoms. These data were available for probands only. Three continuous variables that indexed severity of inattention (5 items, $\alpha = .71$), impulsivity-hyperactivity (8 items, $\alpha = .85$), and ODD/CD symptoms (21 items, $\alpha = .85$) in childhood were calculated. *DSM-III-R* and *DSM-IV* symptoms of inattention, impulsivity-hyperactivity, and ODD/CD symptoms were taken from the parent and teacher DBD scales. All three scores were calculated as a mean of the symptom ratings, taking for each symptom the higher of the two ratings from the parent and teacher. Descriptive statistics for the sample were inattention ($M = 2.58$, $SD = 0.45$), impulsivity-hyperactivity ($M = 2.11$, $SD = 0.55$), and ODD/CD symptoms ($M = 1.04$, $SD = 0.40$). Intercorrelations among these variables were statistically significant but modest in magnitude: inattention with impulsivity-hyperactivity, $r = .27$, $p < .01$; inattention with ODD/CD

symptoms, $r = .17$, $p < .05$; and impulsivity-hyperactivity with ODD/CD symptoms, $r = .47$, $p < .001$.

ADHD and CD in adolescence. In adolescence, *DSM-III-R* diagnosis of ADHD was based on the DBD (parent and teacher report) and Diagnostic Interview Schedule for Children (DISC), Version 2.3, parent report (Shaffer et al., 1996). *DSM-III-R* diagnosis of CD was based on the DBD (parent and teacher report) and DISC 2.3 or DISC 3.0 (parent and adolescent report) for CD.

Substance use and SUD in adolescence. A structured paper-and-pencil substance use questionnaire was developed for this study as an adaptation and extension of existing measures (e.g., Health Behavior Questionnaire, Jessor, Donovan, & Costa, 1989; National Household Survey of Drug Abuse, NHSDA, 1992) with consultation from Drs. John Donovan and Oscar Bukstein, University of Pittsburgh School of Medicine. The questionnaire was divided into a screener section and a confidential section. For the screener section, adolescents provided answers directly to the interviewer regarding lifetime use of substances. Responses to the screener determined the need to administer the confidential section in which questions were read aloud by the interviewer but adolescents recorded answers privately and returned the completed questionnaire in a sealed envelope. This procedure was modeled after that used by the NHSDA. Use of licit and illicit substances was assessed, including any use in the lifetime ("Have you ever...?"), age of first use, and frequency/quantity of use in the past 6 months. Nonmarijuana illicit drugs (both inappropriate use of prescription drugs and use of street drugs, including inhalants, hallucinogens, cocaine, and heroin) were grouped together for analysis (no use vs. any use). Exploratory analyses with an emphasis on effect size were used to explore substance-specific trends in the data. Quantity of cigarettes was power-transformed using $1 - \text{cig}^{-1}$ to reduce skewness. Except for age of onset, substance variables in this study are listed in Table 2.

Alcohol and marijuana use disorders (abuse and dependence) were assessed using a highly structured interview version of the Structured Clinical Interview for *DSM-III-R* (SCID; Spitzer, Williams, & Gibbon,

Table 2
ADHD Versus Non-ADHD Group Differences in Substance Use

Variable	Non-ADHD <i>n</i> = 100 % ^a	ADHD <i>n</i> = 142 % ^a	Effect magnitude	
			Odds ratio	<i>d</i>
Any use of substance in lifetime				
Alcohol	46.0	52.1	1.28	—
Cigarettes	49.0	59.3	1.51	—
Marijuana	26.0	35.2	1.55	—
Illicit drugs (excluding marijuana)	7.0	20.4	3.41**	—
Level of substance use				
Frequency drunk past 6 mos ^b (<i>M</i> , <i>SD</i>)	1.47 (1.12)	1.91 (1.69)	—	.39*
Has been a daily smoker	12.1	30.4	3.16**	—
Quantity cigarettes past 6 mos ^{c,d} (<i>M</i> , <i>SD</i>)	1.20 (0.65)	1.55 (0.98)	—	.54***
Frequency marijuana past 6 mos ^e (<i>M</i> , <i>SD</i>)	1.58 (1.51)	2.30 (2.52)	—	.48**
Any illicit drugs past 6 mos.	6.0	14.8	2.72*	—
Substance misuse				
Alcohol use disorder	8.0	15.5	2.11	—
Marijuana use disorder	12.0	15.5	1.34	—
Alcohol problems (<i>M</i> , <i>SD</i>)	1.08 (2.51)	2.15 (4.12)	—	.43*
Marijuana problems (<i>M</i> , <i>SD</i>)	1.13 (2.94)	1.89 (4.99)	—	.26

Note. Significance levels are from associated chi-square or *t* tests comparing use in the ADHD and non-ADHD groups. Variances were significantly different for all continuous variables at $p < .01$ or $p < .001$; the degrees of freedom and associated *t* tests for unequal variances were used. Cohen's *ds* were calculated for continuous variables as the difference between group means divided by the standard deviation for the control group; odds ratios were calculated for dichotomous substance use variables.

^a Except where indicated otherwise. ^b 1 = never, 2 = once, 3 = 2–3 times. ^c 1 = less than 1 cigarette/day, 2 = 1–5 cigarettes/day, 3 = 10 cigarettes/day. ^d Means and standard deviations for cigarette quantity are presented in original (nontransformed) units. ^e 1 = never, 2 = once, 3 = 2–3 times.

* $p < .05$. ** $p < .01$. *** $p < .001$.

1987). We modeled our interview after the SCID substance use modules used by Martin and colleagues (Martin, Kaczynski, Maisto, Bukstein, & Moss, 1995; Martin, Pollock, Lynch, & Bukstein, 2000). These modules were adapted from the *DSM-III-R* SCID to include the *DSM-IV* criteria for the SUDs and symptoms appropriate for adolescents (e.g., drop in school grades due to drinking). Modules were administered as interviews by the research assistants (trained bachelor-level interviewers) and later translated into diagnoses by senior project staff blind to ADHD group membership. Interrater reliability with a psychiatrist board-certified in child psychiatry and addictions was acceptable for alcohol ($\kappa = .61$, 80.6% agreement) and for marijuana ($\kappa = .71$, 87.8% agreement). Diagnostic consensus conferences were subsequently used to reach agreement on 100% of the cases. These consensus diagnoses were used in this study as well as a sum of scores across each of the symptoms assessed. Each symptom score ranged from 0 (*never experienced the problem*) to 2 (*experienced the problem to a clinically significant degree*), and thus the summed symptom (problem) scores for alcohol and marijuana were used as developmentally sensitive indices of emerging alcohol and marijuana impairment in adolescence.

Results

Lifetime Use of Alcohol, Tobacco, Marijuana, and Illicit Drugs

As shown in Table 2, probands were not significantly more likely than controls to have tried alcohol, cigarettes, or marijuana. However, three times as many probands as controls reported lifetime use of at least one nonmarijuana illicit drug. Exploratory analyses conducted separately by illicit drug revealed that inhalants, hallucinogens, cocaine, and nonmedicinal or nonprescribed use of stimulants were responsible for the group differences. Polysubstance use was more frequent in probands, with 10.6% of probands versus 3.0% of controls reporting lifetime use of illicit drugs in two or more drug classes, $\chi^2(1, N = 242) = 5.44, p < .05$.

Level of Substance Use/Misuse

As shown in Table 2, medium-sized associations were found between ADHD group and current level of substance use. Probands reported more episodes of drunkenness in the past 6 months. In addition, 23.2% of probands versus 12.0% of controls were drunk more than once during this time. Significantly more probands than controls reported daily smoking at some time in their lives. On average, adolescents had been smoking cigarettes daily for 2 years ($M = 2.44, SD = 1.76$ for ADHD; $M = 2.08, SD = 1.16$ for non-ADHD) and of those who had ever smoked daily, most (92.7% of ADHD, 75.0% of non-ADHD) were smoking daily at the time of the interview. Thus, adolescents (especially probands) who had smoked daily were unlikely to have quit. Quantity of cigarettes smoked in the past 6 months was higher among the probands than among the controls. Although group differences were not statistically significant for alcohol or marijuana use disorders, probands had significantly higher alcohol problem scores indicating that they were more likely to experience some impairment due to drinking.

Age of First Substance Use

Kaplan–Meier survival analyses were used to examine childhood ADHD as a predictor of the age at which substances were first used. Probands smoked their first cigarette, they began daily

smoking, and they first used a nonmarijuana illicit drug at earlier ages than controls (see Figure 1). Survival curves were not significantly different between groups for age at first drink (Breslow statistic = 1.6 [1]), age when first drunk (Breslow statistic = 3.81, $p < .10$), and age when first used marijuana (Breslow statistic = 2.34).

Childhood Predictors of Substance Use in the Probands

Bivariate and multiple regression analyses (Cox, ordinary least squares, and logistic) were used to test prospective prediction of proband substance use from the childhood predictors of inattention, impulsivity–hyperactivity, and ODD/CD symptoms.¹ The findings are presented in Table 3.

In bivariate analyses of the childhood predictors, childhood inattention predicted substance use in seven of nine tests. Childhood impulsivity–hyperactivity and ODD/CD symptoms each predicted substance use in two of nine tests. In the multivariate predictions, the childhood inattention effect remained statistically significant after controlling for childhood impulsivity–hyperactivity and ODD/CD symptoms. One exception was for illicit drug use in the past 6 months: Childhood ODD/CD symptoms were significant predictors but childhood inattention was not. Childhood impulsivity–hyperactivity did not predict substance use after the effects of childhood inattention and ODD/CD symptoms were controlled. Most predictions from the childhood variables were modest in magnitude (R^2 range = .06–.10; Cohen, 1988). An exception was for illicit drug use (use in past 6 months and age of first illicit drug use), where large effects were found. For example, for a one standard deviation increase in ODD/CD symptoms, the odds of using illicit drugs at follow-up increased by 81%.

As a check on the specificity of these childhood predictors, we regressed the sum of 15 *DSM-IV* CD symptoms from the DISC, reported by either parent or adolescent at follow-up, onto these same childhood predictors. Childhood ODD/CD symptoms predicted adolescent CD symptoms ($B = .25, p < .01$) above and beyond childhood inattention ($B = .16, p = .06$), and impulsivity–hyperactivity ($B = .05, ns$), total $R^2 = .12, p = .001$. Thus, the results showed the expected prediction of later antisocial behavior from its precedent in childhood, and also showed, by way of comparison to Table 3, that antisocial behavior and substance use in adolescence were differentially predicted by childhood ODD/CD symptoms and inattention, respectively.

Adolescent ADHD and CD

To test whether diagnostic status (ADHD, CD) at follow-up was related to concurrent substance use, probands were divided into three groups: probands without ADHD or CD in adolescence ($n = 38$), probands with ADHD but not CD in adolescence ($n = 64$),

¹ Whether alcohol, cigarettes, or marijuana had ever been used in the lifetime (first three variables in Table 2) were excluded from the regressions on childhood predictors in the ADHD group because variability in usage was better captured with the current frequency/quantity variables. Similarly, alcohol and marijuana problems (continuous variables), but not alcohol or marijuana use disorder, were used in these regressions.

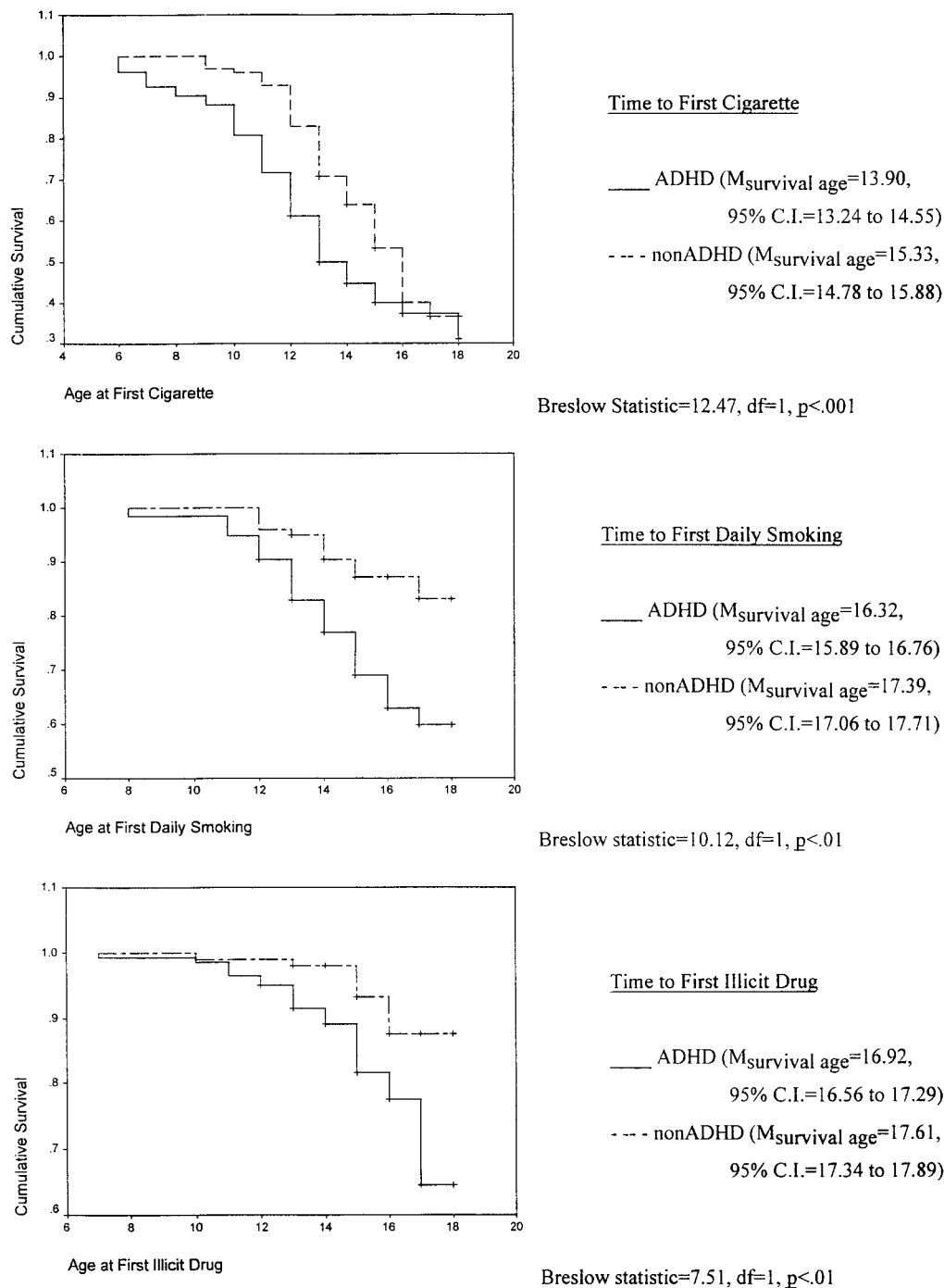


Figure 1. Survival curves showing initiation of substance use behaviors for ADHD and non-ADHD adolescents. ADHD = attention-deficit/hyperactivity disorder.

and probands with both ADHD and CD in adolescence ($n = 35$).² Substance use was dichotomized for use in logistic regressions in which age was treated as a covariate, and group membership was tested using dummy-coded variables. Results are shown in Table 4, where it can be seen that persistence of ADHD was associated with repetitive drunkenness, alcohol problems, and daily cigarette smoking, and persistence of ADHD plus development of CD was associated with alcohol problems in nearly half the probands, marijuana use, and illicit drug use.

Discussion

This study showed that when adolescent substance use is measured in a developmentally appropriate way, the presence of ADHD in childhood is associated with increased risk for elevated

² There were only 5 probands with CD and desistence of ADHD, reflecting the high degree of ADHD persistence in the probands who developed CD; these 5 probands were excluded from the current analysis.

Table 3
Prediction of Substance Use From Childhood Symptoms in the Probands

Childhood symptom	OR				B				
	Age 1st cigarette	Age 1st smoke daily	Age 1st illicit drug	Any illicit drugs past 6 mos.	Freq. drunk past 6 mos.	Alcohol Problems	Freq. Marj. past 6 mos.	Marj. Problems	Quantity Cigs. past 6 mos.
Bivariate associations									
Inattention	1.58	2.25	5.28*	4.98*	.32***	.25**	.27**	.23**	.22**
Impulsivity-hyperactivity	1.58*	1.66	2.13*	2.12	.08	.13	.11	.01	.13
ODD/CD	1.99*	2.12	2.58	5.03*	.05	.13	.15	.15	.13
Multivariate prediction									
Inattention	1.40	2.00	4.76*	4.74	.32***	.23**	.25**	.23**	.20*
Impulsivity-hyperactivity	1.28	1.22	1.44	1.07	-.01	.04	-.01	-.14	.04
ODD/CD	1.63	1.72	1.87	4.38*	.01	.08	.10	.18	.08
$\chi^2(3)$ or R^2	8.37*	7.01	12.43*	11.52**	.10**	.07*	.08**	.08*	.06*

Note. OR = odds ratio, denominated in units of the independent variable. B = standardized regression coefficient; standardized Bs are equivalent to Pearson correlations in bivariate analyses; highest variance inflation factor = 1.35. Freq. Marj. = frequency of marijuana use; ODD/CD = oppositional defiant disorder/conduct disorder. Ns range from 131 to 138 due to missing data. Thus, there is some shifting between analyses in standard errors of predictors and value of coefficient required to attain statistical significance at $p < .05$ or less (e.g., for childhood inattention predicting illicit drugs in past 6 months, OR = 4.74, $p = .05$).

* $p < .05$. ** $p < .01$. *** $p < .001$.

use and abuse of alcohol and heavier and earlier use of tobacco and other drugs by the teenage years. Furthermore, childhood ADHD symptoms, and particularly the inattention dimension of ADHD, predicted later substance use by the probands to a greater degree than childhood antisocial behaviors, suggesting clearly that severity of ADHD symptomatology is uniquely important for the early emergence of substance use behaviors. Finally, development of CD by adolescence in the ADHD probands was associated with the highest levels of substance use and associated problems, but persistence of ADHD diagnosis in probands was also important in characterizing probands with heavier levels of substance use relative to controls. Thus, the findings here represent a significant contribution to the literature that establishes childhood ADHD, variability in its underlying dimensions, and persistence of the disorder as risk factors for early substance use and emergence of SUD in adolescence.

In most cases, the magnitude of risk associated with childhood ADHD was clear, with medium effect sizes occurring when level of substance use—in particular, heavy use—was measured, rather than lifetime exposure or diagnosable disorder as in previous studies. These findings help to clarify previously discrepant reports regarding ADHD risk for substance use and abuse in adolescence, by demonstrating that developmentally appropriate measurement of substance use may capture group differences not apparent in earlier studies. Relative to other populations established as risk groups for early drinking and drug use, the risk associated with childhood ADHD is comparable. Among children of alcoholics in the community, for example, effect sizes measured as odds ratios range from 1.3 to 3.0 (Chassin, Pitts, Delucia, & Todd, 1999). Thus, ADHD diagnosed in childhood appears to be as strong a risk factor for substance use and abuse as having a positive family history of substance use disorder. Furthermore, the risk does not

Table 4
Substance Use by Diagnosis of ADHD and CD in Adolescence

Measure	% Using substances by diagnostic subgroup				Model χ^2 (4)	Odds ratios	
	Controls (n = 96)	ADHD desisters, No CD (n = 36)	ADHD persists, no CD (n = 59)	ADHD persists with CD (n = 34)		ADHD Persists, no CD vs. controls	ADHD Persists, with CD vs. controls
Drunk > once in past 6 months	11.3	15.8	21.9	28.6	27.85***	2.88*	3.60*
Alcohol problem score > 1 ^a	16.5	21.1	26.6	45.7	38.45***	2.40*	5.22***
Marijuana used in past 6 months	17.5	27.0	20.3	40.0	34.12***	1.48	3.68**
Smoked \geq 1 cigarette/day in past 6 months	9.4	13.5	28.8	44.1	28.35***	4.47**	8.11***
Used illicit drugs in past 6 months	6.2	10.5	10.9	20.0	8.74	2.06	3.90*

Note. Age was treated as a covariate in these analyses. Statistical significance of group comparisons are based on Wald's chi-square test. None of the ADHD desister versus control group comparisons were statistically significant.

^a An alcohol problem score of 2 or higher results from two symptoms coded as subthreshold (1) or one symptom coded as clinically present (2).

* $p < .05$. ** $p < .01$. *** $p < .001$.

appear to be specific to one substance, but cuts across alcohol, tobacco, marijuana, and other drugs. The mechanisms explaining the risk may vary with class of drug, however, and this point is discussed in the following.

In addition to its association with quantity–frequency measures and alcohol-related impairment, childhood ADHD was associated with earlier first use of cigarettes, earlier progression to daily smoking, and earlier use of illicit drugs. Early substance use is a well-established predictor of later SUD (Grant & Dawson, 1997; Robins & Pryzbeck, 1985). Thus, although substance use in this age group may not be sufficiently problematic to warrant diagnosis, significantly elevated risk for later nicotine dependence and other drug disorders in adulthood was evident at a young age. The extent to which these early substance use behaviors predict continued substance use and associated impairment in adulthood is the important question for future research. These findings also point to the importance of considering childhood precursors in research on age of substance use initiation. For example, recent research revealed that marijuana use before the age of 15 predicts cocaine use after age 26 (Gfoerer, Wu, & Penne, 2002). The young age at which our probands began experimenting with illicit drugs suggests that childhood ADHD may be an early detectable precursor to an early-appearing risk trajectory that includes illicit drug use 2 decades later.

Our finding that severity of childhood inattention symptoms prospectively predicted substance use outcomes, even after controlling for ODD/CD symptoms in childhood, suggests that this dimension of ADHD is uniquely prognostic of later drug and alcohol abuse within this population. Previous studies predicting substance use from variability in childhood characteristics may have missed this result because standardized ratings of ADHD symptoms were not routinely subdivided into inattention and impulsivity–hyperactivity dimensions until the advent of *DSM-IV* (cf. Loney et al., 1981). In addition, the use of continuous measures in childhood in this study probably increased power to detect effects relative to studies that employed diagnostic categories. Although the resulting longitudinal effects were small in size (e.g., correlations of .20 to .30), we emphasize that these effects are comparable in magnitude to effects found in other longitudinal studies predicting adolescent substance use from childhood variables (e.g., Block, Block, & Keyes, 1988; Burke et al., 2001; Fergusson, Lynskey, & Horwood, 1993, 1997). Arguably, these effects would be even larger if studied in a nonclinical population in which variability in the childhood predictors is greater.

What is unique about severity of inattention, as opposed to impulsivity–hyperactivity and ODD/CD symptoms, that is prognostic of later substance use? Executive functioning deficits associated with inattention, and not impulsivity–hyperactivity (Chhabildas, Bennington, & Willcutt, 2001), may be at the root of this mediational chain to substance abuse (Giancola & Moss, 1998; Pihl, Peterson, & Finn, 1990). We have previously hypothesized that inattention may influence substance use through mediational variables such as poor academic achievement and peer difficulties (Molina et al., 1999). With regard to academic achievement in this sample, we have previously shown that teacher ratings of inattention symptoms in adolescence were more strongly correlated with grade-point average than were ratings of impulsivity–hyperactivity and ODD (Molina, Smith, & Pelham, 2001), and that lower reading achievement scores in childhood, prospectively predicted alcohol use disorder in adolescence (Molina & Pelham,

2001). As suggested by drug abuse theories (e.g., Kaplan & Johnson, 1992), what may follow is gravitation away from conventional group values and behaviors that include academic success, and gravitation toward nonconformist peer groups where substance use is tolerated and modeled. Taken together with the substance use literature showing strong associations between academic failure and substance use (Hawkins, Catalano, & Miller, 1992), a pathway from inattention to substance use through academic impairment for some ADHD children seems plausible.

The absence of robust prediction from childhood levels of impulsivity–hyperactivity and ODD/CD symptoms in the probands was surprising. One reason may be that our measure of impulsivity was decidedly limited insofar as the *DSM-III-R* and *DSM-IV* do a poor job of assessing impulsivity (Pillow, Pelham, Hoza, Molina, & Stultz, 1998): the component of the impulsivity–hyperactivity dimension that has been most fruitful in previous drug abuse risk factor research. For example, only three of the *DSM-IV* items reflect impulsive behavior versus six reflecting excessive levels of activity and nine reflecting inattention. Improving measurement of the impulsivity dimension may change this result. For example, observations of ADHD children's impulsive behavior in a naturalistic setting may add significantly to the prediction of later drinking behavior, above and beyond parent and teacher ratings of behavior (Molina & Pelham, 1997). Alternatively, restriction of range in childhood symptomatology might have contributed to the results.

In contrast, our confidence in our measurement of childhood antisocial behavior, in the form of ODD/CD symptoms reported by teachers or parents, is good. The behaviors are not only easier to operationalize and to rate, but the predictive validity of this measure was good, with childhood ODD/CD symptoms predicting adolescent CD symptoms roughly 5 years later. Therefore, the failure of childhood ODD/CD to predict later substance use was not a methodological artifact; rather, it reflects the fact that ADHD symptomatology, rather than ODD/CD symptomatology within the ADHD sample, predicted adolescent substance use for alcohol, cigarettes, and marijuana. Our finding that childhood ODD/CD best predicted other illicit drug use suggests that early-occurring antisocial behaviors are uniquely helpful in the prospective prediction of especially deviant substance use behaviors in teenagers with ADHD. Not surprisingly, it was the ADHD probands who had retained or developed CD by adolescence who also reported illicit drug use at follow-up. Others have shown that it is this group that is at risk for drug dependence (Gittelman et al., 1985) or psychoactive substance use disorder (Biederman et al., 1997). Increased affiliation with substance-using peers among these probands may in part explain their vulnerability to deviant behavior that includes illicit drug use (Marshal, Molina, & Pelham, in press).

Previous research has suggested that persistence of ADHD is important for the development of drug dependence (Gittelman et al., 1985). Our study replicated that finding for adolescents, but extended the analysis to show that persistence of the disorder was relevant to regular or problematic substance use independent of CD diagnosis, even though emerging CD elevated risk even further. To the extent that persistence of ADHD and development of CD in some individuals is a natural outgrowth of ADHD pervasiveness and severity in childhood, which is suggested by previous research (Mannuzza, Klein, & Moulton, 2002), this finding fits with our predictions from childhood symptoms. That is, children

with the most severe ADHD should be the ones most at risk for continuing ADHD, development of CD, and onset of substance use and abuse by adolescence. Repetitive drunkenness and daily cigarette smoking were elevated in the ADHD persister group, but the highest rates were found in the subgroup that had developed CD. Thus, alcohol-related impairment and illicit drug use appear to develop side-by-side or follow the development of antisocial behavior. However, elevated use of alcohol and tobacco in adolescence may follow more uniquely from ADHD symptomatology. Ultimately, disentangling ADHD symptoms and antisocial spectrum behaviors is a difficult task in ADHD samples, because antisocial behavior and the severity and persistence of ADHD are so tightly intertwined. Following a larger sample of probands into adulthood should not only increase power to study ADHD persisters without CD, but also identify whether or not their elevated drinking and smoking persist beyond the teenage years, and enable testing of different mediational pathways (e.g., an academic failure pathway driven by inattention) that might explain differential vulnerability in subgroups of children with ADHD.

One third of our probands reported daily smoking, beginning at about age 16, and development of antisocial behavior in childhood or in adolescence was not a necessary precursor. Two previous studies have isolated cigarette smoking as uniquely associated with ADHD independent of CD (Burke et al., 2001; Milberger et al., 1997). The psychostimulant properties of this drug, combined with easy access and decreased legal sanctions compared to nonprescribed stimulant or cocaine use, suggest that children with ADHD might be particularly susceptible to cigarette smoking in adolescence. Specifically, speculations have arisen regarding the propensity for children with ADHD to abuse stimulant-type substances because of the attention-enhancing pharmacologic properties in these drugs (e.g., tobacco, amphetamines, cocaine) that are similar to those of methylphenidate and other central nervous system (CNS) stimulants (Khanzian, 1997; Milberger et al., 1997). Laboratory-based studies of attentional performance have found improvements in vigilance with nicotine challenges for both ADHD and non-ADHD adults (Conners et al., 1996; Levin et al., 1998). If improvements in cognitive functioning are more rewarding for individuals with ADHD, this would explain their rapid progression to nicotine addiction. A possible alternative pathway is behavioral sensitization to nicotine arising from previous exposure to therapeutic doses of CNS stimulants (Robinson & Berridge, 2000). Laboratory-based studies of nicotine responsivity in children with and without long-term CNS medication exposure are necessary to test this possibility.

A potential limitation regarding generalization of our findings to other ADHD children pertains to the clinic-referred nature of this sample. Insofar as diagnosed children referred for mental health services have higher rates of psychiatric comorbidity than nonreferred diagnosed children (Bird, Gould, & Staghezza, 1993), the risk for SUD may be a function of that referral bias (see Angold, Costello, & Erkanli, 1999; Caron & Rutter, 1991). Because comorbidity and impairment are positively correlated (Bird et al., 1993), externalizing comorbidity and greater impairment from both ADHD and comorbid psychopathology may be causing the increased risk for SUD in clinic-referred children with ADHD. Surprisingly, however, even though only 20% of children with ADHD appear to receive mental health services, those who do receive them do not have higher rates of psychiatric comorbidity (Bird, Gould, Staghezza-Jaramillo, 1994; Szatmari, Offord, &

Boyle, 1989). Thus, even though sensible arguments have been made about referral bias, it is not conclusive that referral bias is the cause of the SUD risk in this population.

Finally, two additional caveats are relevant. Few girls were included in our sample and testing of gender-specific associations was precluded. This is an important direction for future research because little is known about the long-term outcomes of ADHD in girls. Second, and perhaps most important toward future research endeavors, is the age range of our sample. Because the mean age was only 15, our findings are most relevant for understanding initiation and emergence of substance use rather than abuse. Our ongoing longitudinal research will monitor these children into late adolescence and adulthood to determine (a) whether their heavy use persists and leads to abuse of substances, and (b) the various mechanisms leading to abuse in this population.

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