Which Aspects of ADHD Are Associated with Tobacco Use in Early Adolescence?

Jeffrey D. Burke

University of Pittsburgh Medical Center, U.S.A.

Rolf Loeber

University of Pittsburgh, U.S.A.

Benjamin B. Lahey

University of Chicago, U.S.A.

Several studies have found a relationship between attention-deficit hyperactivity disorder (ADHD) and substance use, primarily in the context of co-occurring conduct disorder (CD). However, very few have examined the associations between the individual dimensions of ADHD (hyperactivity-impulsivity and inattention) and substance use, even though these dimensions reflect distinct symptom groupings, both by clinical definition (DSM-IV, American Psychiatric Association, 1994) and through empirical demonstration (Lahey et al., 1988; McBurnett et al., 1999). This longitudinal study examines the relationship between dimensions of ADHD (as described by DSM) and substance use, accounting for other psychopathology and factors potentially related to substance use. Participants were 177 clinic-referred boys (initially between ages 7 and 12) followed up over nine annual phases until all participants had reached age 15. Annual assessment included structured clinical interviews with parent and child and self-report questionnaires of substance use, as well as questionnaires related to family factors and parenting behaviors. Seventy-eight per cent of participants reported use of tobacco, alcohol, marijuana, or other illicit drugs during adolescence, with 51% reporting any tobacco use. The inclusion of CD rendered all bivariate relationships with the full diagnosis of ADHD nonsignificant. However, adolescent inattention, considered independently, was associated with a 2.2 times greater risk for concurrent tobacco use, even after controlling for CD. Even when other factors, selected based on their associations with tobacco use in adolescence, were included in a regression model (concurrent adolescent CD odds ratio [OR] = 6.08), duration of tobacco use by age 12 (OR = 5.11), poor parental communication in childhood (OR = 2.9), African-American ethnicity (inversely predictive; OR = 0.15), inattention (OR = 2.3) remained significantly associated with tobacco use in early adolescence. These findings highlight the importance of considering the risks for comorbid substance use separately by individual dimensions of ADHD.

Keywords: ADD/ADHD, attention, hyperactivity, impulsivity, substance use.

Abbreviations: ADHD: attention deficit hyperactivity disorder; APD: antisocial personality disorder; CD: conduct disorder; DISC: Diagnostic Interview Schedule for Children; ODD: oppositional defiant disorder; OR: odds ratio; SADS: Schedule for Affective Disorders and Schizophrenia.

Introduction

Attention deficit hyperactivity disorder (ADHD) is recognized to be a chronic disorder that significantly affects children's functioning and places them at risk for associated difficulties, such as substance use (Wilens, Biederman, & Spencer, 1996), depression (Angold & Costello, 1993; Kovacs, Paulauskas, Gatsonis, &

Richards, 1988), and academic underachievement (Frick et al., 1991). ADHD has also been characterized as a disorder of notable heterogeneity in its composition, with symptom dimensions of inattention, hyperactivity, and impulsivity. These dimensions have been presented in three different frameworks in the last three editions of the DSM (American Psychiatric Association, 1980, 1987, 1994), with the current version distinguishing two dimensions: inattention and hyperactivity-impulsivity (American Psychiatric Association, 1994). DSM-IV includes nine symptoms of inattention, such as "often fails to give close attention to details … " and "often does not seem to listen when spoken to directly." Nine symptoms of hyperactivity-impulsivity include behaviors such as

Requests for reprints to: Jeffrey Burke, PhD, Life History Studies, Western Psychiatric Institute and Clinic, 3811 O'Hara Street, Pittsburgh, PA 15213, U.S.A.
(E-mail: burkejd@msx.upmc.edu).

Table 1
Symptom Assignment within Dimensions of ADHD

Hyperactivity-Impulsivity
(1) often fidgets with hands or feet or squirms in seat (in adolescents, may be limited to subjective feelings of restlessness)
(2) has difficulty remaining seated when required to do so
(4) has difficulty awaiting turn in games or group situations
(5) often blurts out answers to questions before they have been completed
(9) has difficulty playing quietly
(10) often talks excessively
(11) often interrupts or intrudes on others, e.g., butts into other children's games
(14) often engages in physically dangerous activities without considering possible consequences (not for the purpose of thrill-seeking), e.g., runs into street without looking
Inattention
(3) is easily distracted by extraneous stimuli
(6) has difficulty following through on instructions from others (not due to oppositional behavior or failure of comprehension), e.g., fails to finish chores
(7) has difficulty sustaining attention in tasks or play activities
(8) often shifts from one uncompleted activity to another
(12) often does not seem to listen to what is being said to him or her
(13) often loses things necessary for tasks or activities at school or at home (e.g., toys, pencils, books, assignments)

"often fidgets with hands or feet or squirms in seat" and "often has difficulty awaiting turn." See Table 1 for a more complete list of diagnostic criteria for inattention and hyperactivity-impulsivity as defined in this study.

There has been a greater degree of empirical support for the distinction between hyperactivity-impulsivity and inattention within ADHD than existed for the various symptom arrangements in previous editions of the DSM (Lahey et al., 1988; McBurnett et al., 1999). Yet, there is a striking paucity of literature investigating the individual dimensions of ADHD in terms of their relationship to other conditions.

Of particular importance among these other conditions is substance use, given both the pernicious effects of drug use and their deleterious impact in the presence of pre-existing psychopathology. With the varied effects of drugs of abuse, and the heterogeneity of ADHD symptoms, it is likely that the risk for substance use may vary with the dimensions of ADHD present. Additionally, it is possible that the role of ADHD in the development of substance use has been understated because of the focus on the unitary diagnosis, rather than the investigation of multiple symptom domains.

The potential exists for those with ADHD to engage in substance use to alleviate the deficits present as a result of the disorder, which has been described by some as "self-medication" (Khanzian, 1997). While several investigators have considered this issue (Coger, Moe, & Serafetinides, 1996; Horner & Scheibe, 1997), they have generally examined the use of substances for the purposes of alleviating symptoms of mood disorders comorbid with ADHD. Given the qualitative differences among the dimensions of ADHD (Lahey et al., 1988) it is important to investigate substance use in the context of dimensions of ADHD to fully identify specific deficits that might be alleviated by the use of substances.

Several studies investigating substance use as a composite (rather than by specific substances) show an association with ADHD (Milberger, Biederman, Faraone, Wilens, & Chu, 1997b; Thompson, Riggs, Mikulich, & Crowley, 1996; Whitmore et al., 1997). However, the extent of the association of ADHD with substance use appears to be tempered by the influence of other comorbid conditions, particularly conduct disorder (CD). Boyle and colleagues (1991), in cross-sectional analyses, and Biederman and colleagues (1997), in a longitudinal study, found no significant relationship between ADHD and substance use when CD is taken into account. In a review of literature on longitudinal and family-genetic studies relating to this issue, Wilens and colleagues (1996) concluded that there is an elevated risk of substance use in those with ADHD, which is likely to be mediated by co-occurring CD.

Regarding specific substances, studies have found an association between tobacco use and ADHD (Barkley, Fischer, Edelbrock, & Smallish, 1990; Hartsough & Lambert, 1987; Lambert & Hartsough, 1998). Milberger, Biederman, Faraone, Chen, and Jones (1997a) reported that ADHD in childhood predicted increased rates of smoking beyond that attributable to CD, other psychiatric disorders, and demographic factors. Lambert and Hartsough (1998) found higher proportions of smokers among adults with ADHD than a comparison group of adults with general behavioral problems. The finding of a relationship between ADHD and later tobacco use above that explained by comorbid CD differs from the literature on composite substance use. These studies, however, did not examine which dimensions of ADHD were particularly associated with tobacco use.

Molina, Smith, and Pelham (1999) did consider individual dimensions of ADHD and substance use, including tobacco. They cross-sectionally examined self-reported substance use in middle school students. Although they did not control for CD, the authors did include delinquency (as measured by the mean of a 24-item, 5-point self-report rating scale of conduct problems) in a regression model with inattention and hyperactivity-impulsivity (as measured by the means on a 4-point rating scale with 9 items and 8 items, respectively), and found that the dimension of hyperactivity-impulsivity was more strongly associated with cigarette and smokeless tobacco use than was the dimension of inattention.

Despite the findings of Molina and colleagues, the literature regarding the pharmacological effects of nicotine on cognition and behavior suggests that a link between ADHD and tobacco use would be more likely to involve inattention than hyperactivity-impulsivity. Studies have shown that the ingestion of nicotine is associated with improvements in general attention and simple task cognitive performance in humans and animals (Conners et al, 1996; Levin, 1992; Levin et al, 1998; Rusted & Warburton, 1992; Wesnes & Warburton, 1983),

and in the amelioration of attentional deficits secondary to pathologies such as ADHD and Alzheimer's disease in humans (Conners et al., 1996; Jones, Sahakian, Levy, Warburton, & Gray, 1992). In a review of the literature, Levin (1992) reported not only that nicotine improves learning and memory, but also that nicotine antagonists impair memory performance.

Transdermal patch administration has been shown to be effective not only in improving the attentional abilities of a nonsmoking, non-attentional-impaired group of participants compared to placebo treatment (Levin et al., 1998), but also in the improved clinical presentation and performance on a continuous performance task for participants with ADHD, for both smokers and non-smokers (Conners et al., 1996). Pritchard and colleagues (1995) reported improved task performance and increased cortical activation as measured by EEG.

Finally, the most direct laboratory comparison of inattention and hyperactivity relative to tobacco use was that of Aytaclar, Tarter, Kirisci, and Lu (1999). They investigated the impact of impairments in executive cognitive functioning (including impaired ability to inhibit responses) versus high behavioral activity level on substance use in early adolescence. Their findings suggested that, after controlling for CD, impairments in executive cognitive functioning more strongly predicted tobacco use than did behavioral activity level.

Animal studies also suggest that nicotine ameliorates attentional impairment. Prendergast and colleagues (1998) reported that administration of nicotine reduced the distractibility experienced by monkeys in a laboratory test, while the administration of specific nicotinic receptor agonists prevented this distractibility. Witte, Davidson, and Marrocco (1997) reported findings from a laboratory study in which monkeys fixated on a visual stimulus and were cued to respond to a visual target in one of two visual field locations. The administration of nicotine significantly reduced the reaction time of monkeys in the invalid cue trials, in which they had received a cue to the location opposite the actual target. They reported strikingly similar effects in human chronic tobacco smokers in performing the same task following cigarette smoking.

There are few studies supporting a link between tobacco use or nicotine administration and hyperactivity-impulsivity. Mitchell (1999) reported finding higher scores on personality and behavioral measures of impulsivity in a group of smokers compared to nonsmokers. Waldeck and Miller (1997) found higher self-reported use of nicotine among women with self-reported high impulsivity, compared to those with low impulsivity, but did not find the same to be true for men. Finally, animal studies also show a link between nicotine administration and hyperactivity in rats, including increased hyperactivity and changes in cortical receptor density in offspring following prenatal nicotine administration (Richardson & Tizabi, 1994; Tizabi, Popke, Rahman, Nespor, & Grunberg, 1997).

In summary, the available evidence regarding the pharmacological effects of nicotine consumption tends to be more suggestive of a relationship between tobacco use and changes in cognitive performance, including improvement in attentional ability, than between tobacco use and hyperactivity or impulsivity. However, very few studies have included measures of hyperactivity-impulsivity, inattention, conduct problems, and tobacco use. Questions remain regarding the strength and limits of the differential relationships between measures of psychopathology and tobacco use.

The differences between individual dimensions of ADHD may provide an hypothetical explanation for the mediation by CD of the relationship between ADHD and substance use, and the contrary findings when the focus is on tobacco use and ADHD. Other studies have demonstrated that hyperactivity-impulsivity symptoms are more associated with conduct problems than are inattention symptoms (Babinski, Hartsough, & Lambert, 1999) or that a measure of behavioral impulsivity (a lack of behavioral control) was more associated with delinquency than one of cognitive impulsivity (a lack of planful cognitive performance) (White et al., 1994). It is plausible that the presence of hyperactive-impulsive symptoms of ADHD are associated with general deviant or delinquent behavior, and therefore the risk for substance abuse from hyperactivity-impulsivity would not be specific, but more generally due to an overlap in deviant behavior. As such, the association between ADHD and substance use would be better accounted for by the diagnosis of CD, or conduct problems in general. However, problems of inattention may contribute to a specific risk for tobacco use because of the amelioration of attentional deficits experienced by the tobacco user.

The current investigation was conducted to contribute to the initial investigation of individual components of the diagnosis of ADHD and their relationship with use of tobacco, as well as alcohol, marijuana, and other illicit substances. Several questions served to guide the current study. First, is there a positive association between the diagnosis of ADHD and substance use? Second, do the individual components of ADHD, namely hyperactivity-impulsivity and inattention, differ in their associations with substance use? Finally, do the relationships identified in the preceding questions hold when other factors known to be related to ADHD and to substance use, including the diagnosis of CD, are taken into account?

Method

Participants

Data were gathered as part of a longitudinal study of 177 boys referred to one of three university outpatient clinics in Pennsylvania and Georgia (see site descriptions below). The methodology employed has been reviewed extensively in other publications (Loeber, Green, Lahey, Frick, & McBurnett, 2000). Participants were recruited to form a sample composed primarily of children who qualified for diagnoses of disruptive behavior disorders. Participants were 7 to 12 years of age at the time of the first assessment, in 1987, and were required to be living with at least one biological parent. Boys who exhibited mental retardation or psychosis, or who were taking psychotropic medication that could not be discontinued for 2 days prior to their scheduled assessment, were excluded from the study. In addition, families who indicated that they planned to move out of the area in the near future were also excluded. The sample was composed of Caucasian (70%) and African-American boys (30%), with families ranging across all five levels of the four-factor index of socioeconomic status (Hollingshead, 1975).

Pennsylvania site. Ninety-six participants were initially recruited by examining medical records of the child outpatient service of a large urban university psychiatric hospital. Referral sources for the service included school personnel, parents, physicians, and the courts. Of potential participants, 189 names were initially taken from the medical records and were contacted by phone: of those, 15 boys did not meet the criteria for inclusion, 28 refused before we could assess their eligibility, and

29 had disconnected phones with no new listing or did not answer after repeated attempts to contact. In addition, two participants were disqualified after the assessment because they did not meet criteria. Of the 115 qualifying participants, 19 repeatedly canceled appointments, leaving 96 participants.

Georgia site. Of the new male outpatients who scheduled appointments at a semi-rural university children's centre for psychological assessment, 70 were considered for inclusion in the study; 19 outpatient boys who met criteria were seen at a satellite of this center, a nearby urban university outpatient clinic. Referrals to the rural clinic came from schools, physicians, the courts, and parents. Referrals to the urban satellite center originated from the same sources but came through the university's department of psychiatry. Four participants were excluded after the assessment due to mental retardation, and five participants were excluded due to failure to keep appointments. The final sample included 81 boys (66 from the rural clinic and 15 from the satellite center).

Procedure

Participants at all sites were assessed using identical procedures. All initial assessments were conducted at the university clinics with both the child participant and a parent, concurrently but separately. The parent informant was the mother of the participant in 173 of the 177 cases. A structured diagnostic interview with a teacher, and teacher rating scales regarding the participant, were collected by telephone and mail.

Each child was reassessed annually on approximately the anniversary of the first assessment. Participants were not interviewed in the fifth year of the study (other than a shortened telephone interview with the parent) because of funding cuts. However, participants were recontacted in the sixth year, and interviewed annually until they reached the age of 18. Interviews were conducted by project staff members trained by the project coordinator and interviewer supervisors. Interviewers were blind to the hypotheses of the study, and unaware of the results of assessments conducted during the previous year(s). Follow-up interviews subsequent to the first year adhered to similar procedures as those described above, excepting that intelligence and personality tests were not repeated, and some children were interviewed in juvenile detention facilities, or at home if they could not come to the clinics. In addition, diagnostic interviews with teachers were discontinued after Year 4.

Development of age-based composites. Data from each phase were reordered by participant age because each project phase included a 5-year age range among participants (e.g. 7 to 12 in Year 1, 8 to 13 in Year 2, and so on). At the time of these analyses, complete data were available only for participants up to the age of 15. Constructs were developed for two age periods for each participant: 7 to 12 (hereafter referred to as "childhood") and 13 to 15 (hereafter "adolescence"). These constructs are described further in the Measures section. Substance use constructs were developed only in adolescence. Constructs related to parental and demographic factors were created using only Year 1 data. Therefore, no development of age-based composites was necessary for those constructs.

The number of time points for which each participant had available data for child composites varied, because at Year 1 the sample ranged in age from 7 to 12. Thus, for composites of childhood data, boys who were 7 in Year 1 would have a potential for six data points within the composite, while boys who were 12 in Year 1 would have only one. All participants provided data equally for adolescent composites.

Cases were coded as present for a disorder if there was at least one occurrence of that disorder by age 12. Cases were coded as absent for a disorder if there were no ages at which a positive report occurred, and if the participant had missed no more than one assessment from their age at the beginning of the study until the age of 12. Of the main diagnostic constructs of interest in this study (ADHD, hyperactivity-impulsivity, inattention, and CD) there were no missing cases for childhood composites.

Adolescent composites were coded as present if there was any instance of a disorder between ages 13 to 15. They were coded as absent for those participants with no instance of disorder, and no more than one missing assessment during that time period. For adolescent composites, five cases were missing on ADHD and on each dimension, and seven cases were missing on the CD composite. The dependent variables (drinking, tobacco use, marijuana use, and other drug use as described above) include only use during adolescence (ages 13 to 15). There were 13 cases that were missing on each of the four substance use composites, leaving 164 valid cases for each dependent variable.

Measures

Psychiatric diagnoses of the child. All constructs of psychiatric diagnoses of the child and parent in this study are dichotomous representations of the presence or absence of a disorder during a specified time period: either childhood or adolescence. The child, his parent, and his teacher were interviewed separately using parallel versions of the *NIMH Diagnostic Interview Schedule for Children* (DISC; Costello, Edelbrock, Dulcan, Kalas, & Klaric, 1987), modified to include all symptoms related to several disorders including the DSM-III-R diagnoses of ADHD, CD, Oppositional Defiant Disorder (ODD), Overanxious Disorder, and Separation Anxiety Disorder, Dysthymia, Major Depression, Enuresis, and Encopresis. The custodial biological parent who accompanied the child to the interview was administered a version (DISC-P) of the interview, and the child was administered a version of the interview (DISC-C) that was parallel to the DISC-P. Additionally, during the first four waves of the study, the child's teacher was also contacted by telephone and was administered an alternate version of the interview (DISC-T). Informants were asked in the DISC interview about symptoms that may have occurred during the most recent 6-month period, consistent with DSM-III-R criteria for symptom manifestation.

Each child was assigned all DSM-III-R diagnoses for which he met diagnostic criteria, based on the algorithmic scoring of the responses to the interview. Scoring for all diagnoses used a method of resolving among all informant reports by scoring a symptom positive if it was reported on any clinical interview with any informant. However, the portion of the DISC used to assess ADHD was dropped from the child assessment interview following Year 2. Therefore, ADHD diagnoses used in this study were generated using only parent and teacher report.

In order to assess the reliability of the structured diagnostic interviews of the child, parent, and teacher, 25% of the cases were selected to have a second interviewer observe and simultaneously score the DISC-C and DISC-P interviews through a one-way mirror or via videotape, and listen to the teacher interviews (during Years 1 through 4) over the phone. Agreement among diagnoses derived through the primary administration and the reliability review was consistently good during the entire project. Over Years 1 through 4, agreement for DSM-III-R diagnoses, as measured by Cohen's kappa (Cohen, 1960), for CD was .92, for ODD was .90, for ADHD was .95, and for Dysthymia was .97. Cohen's kappas for inter-rater reliability over Years 6 through 11 were .91 for CD, .95 for ODD, .94 for ADHD, and .80 for Dysthymia.

ADHD dimension constructs. Symptoms of ADHD used in DSM-III-R were assigned to the components of hyperactivity-impulsivity and inattention based on the domains detailed in DSM-III and DSM-IV. Symptoms, with numerical designations as given in DSM-III-R, were assigned to domains as depicted in Table 1. Resolved reports included parental report in all phases, teacher report during the first four phases, and child report during the first two phases. Collection of child report of ADHD symptoms was discontinued after the second phase of data collection. The influence of the use of child report data in the current analyses was assessed by creating constructs both using and excluding these reports. This resulted in only minimal differences in the final results.

For these analyses, a participant had to have at least six of the eight symptoms of hyperactivity-impulsivity, and at least four out of the six symptoms of inattention. These ratios were selected to conservatively approximate the ratio of symptoms

necessary to meet criteria for the two dimensions given in the current DSM-IV framework.

Child substance use. Self-report items regarding child substance use in four categories (tobacco, alcohol, marijuana, and other drugs) were included in the assessment battery beginning in the third year of the project, when the participants were 9 through 14 years of age. The category of other drug use included tranquilizers, barbiturates, codeine, amphetamines, LSD, cocaine, crack, heroin, and PCP. Participants were asked to estimate the number of days out of the past year they used a substance, and to provide an average weekly usage estimate. Due to the significantly skewed distributions of these variables, dichotomous constructs were created to reflect any reported days of use within each of the four categories during adolescence.

An additional substance use variable, the duration of tobacco use, was created by determining the age at which participants first prospectively reported substance use. This was used as a covariate to assess the influence of the duration of tobacco use history in analyses with adolescent tobacco use as the dependent variable.

Other child constructs. Intelligence scores were measured during Year 1 by administering the Wechsler Intelligence Scale for Children-Revised (WISC-R; Wechsler, 1974). These values were dichotomized such that scores below 100 on each of the Full, Verbal, and Performance Scales were coded as positive for low IQ. Perinatal problems were dichotomous constructs, determined from maternal report, and included complications at delivery and low birthweight. Birthweights less than 5.6 pounds were coded as low.

The Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988), a five-item index of physical maturation, was administered annually to both the mother and the child to measure development into puberty. Maternal and child scores were summed and averaged, and the mean of this score at ages 12 and 13 was calculated. Those in the highest quartile of this distribution were coded as positive for advanced pubertal development.

The child's use of prescribed medication for psychiatric disorder was obtained in Year 1 of the study and updated each subsequent year. The use of stimulants included any prescribed Ritalin, Dexedrine, or any other stimulant specifically prescribed for treatment of ADHD.

Parental psychopathology. To assess parental psychopathology at Year 1, a structured diagnostic interview was used that included DSM-III-R diagnoses of Antisocial Personality Disorder (APD), alcohol abuse, and drug abuse from the Schedule for Affective Disorders and Schizophrenia (SADS; Spitzer & Endicott, 1978). To assess the boy's biological father, the mother was asked all the relevant questions for the same diagnoses of the father. The interviewer administering the SADS was blind to the responses by the mother and child to the DISC interview. As described above for the DISC interviews, a second rater was used in 25% of the cases, and inter-rater reliabilities for the parental diagnoses were determined to be adequate (ranging from .57 for paternal unipolar depression, not used in this analysis, to .78 for the diagnosis of paternal APD). Maternal and paternal APD, both maternal and paternal substance use (coded as present if use of alcohol or drugs was reported), and prenatal maternal smoking and use of illicit substances were dichotomous parental psychopathology constructs used in this study.

Parenting. Several questions related to parenting (communication, consistency in discipline, and supervision of the child's behavior), were included in parent and child interviews (Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1998). All parental measures were created by dichotomizing scores on Year 1 measures, not because of skewed distributions in the data, but for conceptual purposes, so that the quartile of the distribution reporting the poorest communication, disciplinary consistency, or supervision represented a positive report for that construct. Parental communication was assessed from the perspective of the parent (denoted in this study as poor

Table 2
Independent Variables by Domain

Domain	Construct
Child factors	ADHD
	Inattention
	Hyperactivity-Impulsivity
	Anxiety
	CD
	Depression
	Duration of tobacco use by 12
	IQ
	Full Scale
	Verbal
	Performance
	Oppositional defiant disorder
	Perinatal problems
	Delivery complications
	Low birthweight
	Stimulant medication use
Adolescent factors	Advanced pubertal development
	ADHD
	Inattention
	Hyperactivity-Impulsivity
	Anxiety
	CD
	Depression
	Oppositional defiant disorder
	Stimulant medication use
Parental factors	Harsh discipline
	Inconsistent discipline
	Poor child communication
	Poor parent communication
	Poor supervision
	Psychopathology
	Maternal APD
	Paternal APD
	Substance use
	Maternal substance use
	Paternal substance use
	Prenatal maternal substance use
	Illicit substance use
	Smoking
Demographics	African-American ethnicity
	Broken family
	High number of siblings
	Low maternal age (21 or below)
	Low socioeconomic status
	Urban residence

parent communication) and from the perspective of the child (poor child communication). The designations assigned to the two communication constructs reflect the source of the perception of poor communication, but do not suggest who is responsible.

Demographics. Participant race, age, residential area, socioeconomic status, family size, and the age of the mother at the birth of her first child were ascertained at Year 1. Demographic constructs used in this study were African-American ethnicity, urban residence (population density of greater than 1000 per square mile, determined by the 1990 U.S. Census), low socioeconomic status (the lowest two of the five Hollingshead, 1975, categories), broken family (present if father was absent at Year 1), low maternal age (21 or younger), and continuous number of siblings.

Analyses

Unless otherwise specified, all constructs used in the study were dichotomous. Bivariate relationships between each category of substance use (alcohol, tobacco, marijuana, and other drug use) and each construct identified in Table 2 were assessed

using chi-square tests. Variables were selected for entry into logistic regression models based on the strength of their bivariate relationships. All logistic regressions used in the current analyses used the method of backwards variable selection unless otherwise specified. For each regression, assumptions of collinearity were assessed through the examination of correlation matrices generated through the SPSS logistic regression procedure, and through the evaluation of tolerance statistics, as described by Menard (1995). Assumptions of collinearity were met for each regression.

The number of constructs remaining as strong correlates of the substance use constructs would have been too great for inclusion in a single model. Therefore, several regressions were conducted, using each of the four dichotomized adolescent substance use variables as the dependent variable. These were structured by examining predictors within the domains indicated in Table 2. Those variables identified within the initial regression models as the strongest were retained and tested together to identify a final model as described in Results.

In the initial bivariate and regression analyses, an alpha level of .1 was adopted to identify those variables to be retained for subsequent analyses. An alpha level of .05 was adopted for statistical tests of the final model.

Finally, it should be noted that, because of the discontinuation of teacher interviews after Year 4, those participants who were age 7 or 8 at the beginning of the study would differ in the number of informants used to generate diagnostic constructs. Essentially, teacher ratings were "missing" for the childhood constructs of these participants at ages 11 and 12. To address this, and to partially address the fact that greater numbers of years of data were available for younger participants, we reanalyzed the final model developed below with the entire data set using only data from participants aged 9 through 12 at the beginning of the project. The results did differ; this will be discussed at the end of the Results section.

Results

During the period from 13 to 15 years of age, 51% of the boys used tobacco, 68% consumed alcohol, 26% used marijuana, and 12% used other drugs. Of those with any use, the mean number of days of annual use during that period was 121 for tobacco, 137 for alcohol, 36 for marijuana, and 15 for other drugs.

The initial question guiding our analyses was the strength and direction of any relationships between ADHD (and its symptom dimensions) and the use of tobacco, alcohol, marijuana, and illicit drugs in adolescence. In bivariate analyses, childhood ADHD was predictive of tobacco use in adolescence, $\chi^2(1) = 3.79$, $p = .05$; OR = 2.2. It was not predictive of any other category of substance use. During adolescence, ADHD was concurrently associated with tobacco use, $\chi^2(1) = 7.00$, $p = .008$; OR = 2.3, alcohol use, $\chi^2(1) = 5.45$, $p = .020$; OR = 2.2, and marginally related to drug use, $\chi^2(1) = 3.01$, $p = .083$; OR = 2.4. Adolescent ADHD was not concurrently associated with marijuana use.

Table 3 depicts the overlap between ADHD and CD in childhood and adolescence. As is evident, there was a high degree of overlap between the two disorders in childhood, with nearly half demonstrating both ADHD and CD during the period from age 7 to 12. In adolescence, the two disorders are still highly related, but the percentage of participants who demonstrate both disorders drops to approximately one-third of the total.

It was necessary to investigate the possible influence of CD on the above relationships between ADHD and tobacco, alcohol, and drug use, given findings in the literature (see earlier) regarding the mediating role of CD in the relationship between ADHD and substance use.

Table 3
Overlap between CD and ADHD in Childhood

CD	ADHD	
	Absent	Present
Absent	22	66
	12.4%	37.3%
Present	10	79
	5.6%	44.6%

Childhood CD and ADHD were significantly associated with one another; $\chi^2(1) = 5.66$, $p = .017$; OR = 2.6.
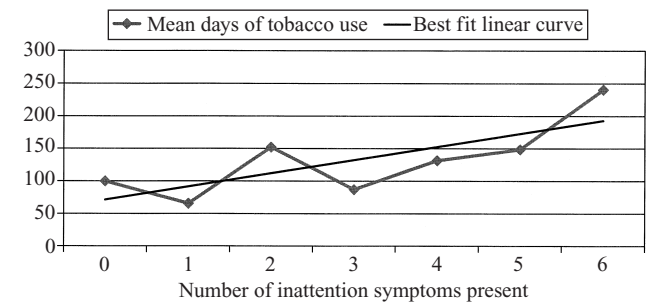

Figure 1. Mean number of days of tobacco use between the ages of 13 to 15 years as a function of the number of individual symptoms of inattention present during that same period.

Controlling for childhood CD reduced the relationship between childhood ADHD and adolescent tobacco use to nonsignificance, Wald (1) = 2.61, $p = .11$; OR = 1.98. Likewise, controlling for adolescent CD in a logistic regression reduced the relationship between ADHD and tobacco use to marginal significance, Wald (1) = 2.99, $p = .08$; OR = 1.79, and rendered the relationships between adolescent ADHD and both alcohol and drug use nonsignificant.

ADHD dimensions. Inattention and hyperactivity-impulsivity were highly correlated in both childhood and adolescence. In childhood, 68% of the participants showed both dimensions of ADHD, and the dimensions had a phi correlation of .47. In adolescence, the percentage with both inattention and hyperactivity-impulsivity dropped to 34%, but the correlation between them was still moderate (phi = .52) (see Table 4).

To examine the relationships among the individual ADHD dimensions and substance use constructs, chi-square tests were conducted for all combinations. None of the relationships between any childhood dimensions of ADHD and any substance use category approached significance.

In adolescence, hyperactivity-impulsivity was significantly associated with drinking, $\chi^2(1) = 4.69$, $p = .03$; OR = 2.2. Inattention was significantly associated with tobacco use, $\chi^2(1) = 7.82$, $p = .005$; OR = 2.5, and marginally associated with drug use, $\chi^2(1) = 2.91$, Fisher's exact $p = .098$; OR = 2.7. Figure 1 depicts the linear dose-response relationship that exists between the number of symptoms of inattention present and the mean days of tobacco use during adolescence. The chi-square test described above, between inattention and tobacco use, refers to the dichotomous constructs used throughout these analyses, while the graphic depiction in Fig. 1 utilizes continuous frequencies of use and sum of symptoms (so that the dose-response relationship may be clearly seen). A nonparametric test, the Jonckheere-

Table 4
Overlap between CD and ADHD in Adolescence

CD	ADHD	
	Absent	Present
Absent	54	28
	31.8%	20.6%
Present	28	53
	16.5%	31.2%

Adolescent CD and ADHD were significantly associated with one another; $\chi^2(1) = 11.58$, $p = .001$; OR $= 2.9$.

Terpstra Trend test, of the relationship between the sum of inattention symptoms and continuous tobacco use frequencies indicated that the relationship depicted was significant ($p = .003$).

To further examine these associations, logistic regressions controlling for adolescent CD were conducted. Inattention remained significantly associated with tobacco use, Wald (1) $= 5.24$, $p = .02$; OR $= 2.2$. However, neither of the other previously significant or marginal associations between dimensions of ADHD and substance use remained significant after controlling for CD. To directly compare the relative strengths of inattention and hyperactivity-impulsivity in their association with tobacco use, both were included in a regression, controlling for CD. Again, hyperactivity-impulsivity was not significantly associated with tobacco use, whereas the relationship between inattention and tobacco use was stronger in this model than it had been by itself, Wald (1) $= 6.86$, $p = .009$; OR $= 2.8$.

In addition, to investigate the specificity of the relationship between inattention and tobacco use, and the possibility that it was due to a nonspecific association between substance use and inattention, we regressed inattention on tobacco use after controlling for alcohol, marijuana use, and other drug use. Inattention and tobacco use remained significantly associated, Wald (1) $= 4.26$, $p = .039$; OR $= 2.1$.

Role of other risk factors in the model. To clarify the nature of the relationship between inattention, CD, and tobacco use, we sought to identify other potential risk factors that might influence tobacco use in adolescence. The results of initial chi-square tests are presented in Table 5.

Initial Regressions

The risk factors that were identified as significant in bivariate chi-square tests were entered into logistic regressions upon tobacco use by each domain, as described in the Analyses section.

Childhood psychopathology. Based on bivariate analyses, the duration of tobacco use by age 12, ADHD, and CD were the only significant predictors of adolescent tobacco use from the domain of childhood psychopathology. These were entered into a backwards logistic regression, with tobacco use as the dependent variable. Duration of tobacco use by age 12, Wald (1) $= 6.74$, $p = .009$; OR $= 3.21$, and ADHD, Wald (1) $= 3.14$, $p = .076$; OR $= 2.13$, were retained in the model, while CD was removed.

Adolescent factors. Stimulant medication use, inattention, ADHD, and CD were significant in bivariate analyses with tobacco use. Because inattention is a conceptual and diagnostic subset of ADHD, and because analyses conducted to assess the first set of questions for this study demonstrated that ADHD was not significantly associated with tobacco use beyond the effects of CD, ADHD was excluded from this regression. CD remained in the model, Wald (1) $= 14.11$, $p < .001$; OR $= 3.53$, as did inattention, Wald (1) $= 4.73$, $p = .03$; OR $= 2.09$, while stimulant medication use was removed.

Parenting and demographic factors. Variables from the parental (prenatal smoking and poor child communication) and demographic (African-American ethnicity) domains were included together in a regression. SES and urban residence were included in the model for theoretical purposes, although they were not individually associated with tobacco use. African-American ethnicity, Wald (1) $= 11.84$, $p = .001$; OR $= .27$, remained in the model (inversely associated with tobacco use), along with poor child communication, Wald (1) $= 9.07$, $p = .003$; OR $= 3.48$; prenatal smoking, SES, and urban residence were removed. Interactions were assessed among ethnicity and all other variables in the model, and no significant interactions were found.

Final Model

Variables that were retained in the analyses based on the initial regressions from all domains were: duration of

Table 5
Risk Factors Associated with Tobacco Use During Adolescence

Risk factor[a]	No tobacco use (%)	Tobacco use (%)	χ^2	p	OR
Inattention	39.8	60.2	6.96	.008	2.32
ADHD	38.8	61.2	7.00	.008	2.31
Conduct disorder	32.1	67.9	16.66	.000	3.76
Stimulant medication use	33.3	66.7	3.51	.061	2.19
Duration of tobacco use by age 12	15.0	85.0	10.40	.001	6.50
Childhood ADHD	45.1	54.9	3.79	.052	2.21
Childhood CD	41.0	59.0	4.11	.043	1.89
African-American ethnicity	68.6	31.4	11.67	.001	0.30
High maternal prenatal smoking	36.6	63.4	3.25	.071	2.98
Poor communication (son)	29.3	70.7	8.33	.004	2.98
	Mean	SD	χ^2	p	OR
Duration of tobacco use by age 12	0.22	0.67	11.18	.025	n/a

[a] Age of measurement is 13–15 years unless otherwise indicated.
None of the other risk factors described in the Methods section were significantly associated with tobacco use at alpha $< .10$.

Table 6
Regression Analyses of Risk Factors Significantly Associated with Adolescent Tobacco Use

Variable	B	SE	Wald	df	p	r	OR
African-American ethnicity	−1.92	0.49	15.34	1	.0001	−.24	0.15
Inattention	0.82	0.39	4.46	1	.035	.10	2.26
Conduct disorder	1.80	0.45	16.09	1	.0001	.25	6.08
Poor communication (son)	1.07	0.46	5.53	1	.019	.13	2.93
Duration of tobacco use by age 12	1.63	0.71	5.32	1	.021	.12	5.11

Childhood ADHD was removed from the backwards regression model. SES and urban residence were not significant in any model, neither independently nor in any interaction.

tobacco use by 12, adolescent inattention, adolescent CD, childhood ADHD, poor child communication, and African-American ethnicity. These variables were included together in a regression to identify a final model of risk factors associated with tobacco use. The results are presented in Table 6.

To assess for interactions among the variables in the final model, we compared each pair in forced entry logistic regressions with each variable and the interaction term containing the two entered in separate steps. There were no significant interactions identified for any pair of variables in the final model.

Reanalysis using restricted data set. As described in the Analysis portion of the Methods section, the final model was analyzed a second time, using data only from participants between the ages of 9 and 12 at the beginning of the study. In this model, the duration of tobacco use and poor child communication are statistically removed from the model. Adolescent inattention is associated with greater risk in this model than in the model using the entire data set, Wald (1) = 6.20, $p = .013$; OR = 3.49, despite the fact that childhood ADHD is also retained in the model, Wald (1) = 5.54, $p = .019$; OR = 3.9. CD, Wald (1) = 10.78, $p < .001$; OR = 6.3, and African-American ethnicity, Wald (1) = 14.6, $p < .001$; OR = .09, are also retained in the model.

The results of this latter model, in terms of the primary questions of interest of this study, are essentially similar to the first summary model. Because of this, the discussion will focus on the findings derived from the entire data set.

Discussion

The findings of the study should be seen in light of its limitations. Within the data in this study, no indicators of post-natal tobacco use exposure were available. Thus, we were not able to test the association between adolescent tobacco use and dimensions of ADHD after accounting for the effects of having been exposed to the use of tobacco by others, particularly within the household, while growing up. Additionally, the sample in the present study was derived from clinic referrals. This sample will therefore differ from community-based and other samples, presumably in the direction of overall greater presence of pathology. This implies that extension of the findings to other populations should be based on replication of these findings on samples representative of those populations.

Although we found a concurrent relationship between ADHD and the use of tobacco and marijuana in adolescence, this relationship was mediated by the presence of CD. This is consistent with previous findings (see review by Wilens and colleagues, 1996) and is suggestive of the hypothesis that findings of an association between

ADHD, CD, and substance use may be a function of the behavioral problems associated with the conditions.

The dimensions of ADHD demonstrated two bivariate associations with the four categories of substance use. Neither dimension in childhood was predictive of later substance use in early adolescence. In adolescence, however, inattention was concurrently associated with tobacco use, whereas hyperactivity-impulsivity was associated with alcohol use.

Such findings are of interest for their implications regarding the composition of ADHD. First, differential associations among dimensions suggest that they are likely measures of differing processes. Second, they indicate that the underlying dimensions of ADHD may have unique associations with other disorders and conditions, meaning that the degree to which these dimensions are present within the overall presentation of symptoms may suggest differing risk for comorbid conditions. Finally, it may be the case that the unitary diagnosis of ADHD masks underlying comorbid risk that may be present differentially among dimensions.

However, the context of the associations between individual dimensions and other conditions is crucial to their meaning. In this study, of the significant relationships between substance use variables and ADHD dimensions, only that between inattention and tobacco use exceeded the explanation provided by CD, suggesting that the presence of symptoms of inattention places boys at increased risk for tobacco use. It is plausible that a specific neurological condition is reflected in the expression of problems of inattention, and the effects of nicotine may serve to reinforce the use of tobacco by providing relief, if temporarily, from impairments in attention (Conners et al., 1996; Jones et al., 1992; Levin, 1992; Levin et al., 1998; Rusted & Warburton, 1992; Wesnes & Warburton, 1983). This possible specific effect of adolescent inattention on tobacco use is additionally supported by the finding that the association remains significant after controlling for the effects of alcohol, marijuana, and other drug use, which suggests that it is not a more general process of inattention tending to associate with substance use.

Regarding other factors associated with substance use, three of the variables in the final model in this study have been consistently associated with tobacco use: CD, duration of early tobacco use, and ethnicity. The strong relationship found here between CD and tobacco use is unlikely to be the result of the same psychophysiological processes that we propose are in effect between tobacco use and inattention. Rather, it is likely that there is a more general mechanism than the alleviation of symptoms involved in the relationship between tobacco use and CD, such as the identification with deviant peer groups or the display of norm-violating behaviors.

It is not surprising that the duration of tobacco use by age 12 is predictive of tobacco use between ages 13 to 15. This suggests that once begun, tobacco use is likely to persist, and that greater duration of early use may indicate more risk of chronic and intense later use, consistent with findings from other studies (e.g., Lewinsohn, Rohde, & Brown, 1999). African-American ethnicity has been associated with lowered risk for tobacco use in adolescence in other studies as well (e.g., Epstein, Botvin, & Diaz, 1998).

An association between problems of inattention and tobacco use suggests that efforts in the prevention of tobacco use may be particularly warranted for those demonstrating attentional deficits in late childhood or early adolescence. Not only is this of concern given the deleterious health effects of tobacco use, but also given the associations demonstrated in other studies between tobacco use or smoking and the onset of abuse of other substances. It may be necessary to provide more specific evaluation of tobacco and substance use for adolescents in treatment for ADHD of the subtypes "Primarily Inattentive" or "Combined." Additionally, since children with inattention symptoms may be less likely to be referred for treatment than those demonstrating more obvious symptoms of hyperactivity-impulsivity, they may be at greater risk for tobacco use. Further research will be needed to understand the relationship between stimulant medication, ADHD, and substance use. However, if adolescents do consume nicotine to alleviate attention problems, the use of stimulant medication would reduce the need to turn to an alternative such as cigarettes.

It should be noted that our findings indicate a concurrent relationship between inattention and tobacco use, but not a predictive relationship. The concurrent nature of the relationship should not be taken lightly, as these processes are very likely reciprocal, on a proximal basis. That is, the use of nicotine to alleviate deficits of inattention is likely to increase the need for nicotine. Nicotine rapidly asserts its influence on cognitive performance (see review by Heishman, Taylor, & Henningfield, 1994), establishing the potential for a short-term reciprocity, but problems of inattention tend to persist throughout adolescence and into adulthood (Hart, Lahey, Loeber, Applegate, & Frick, 1995). Individuals with problems of inattention, particularly in the context of ADHD, may be at risk for addiction to tobacco and other perils associated with the chronic use of tobacco.

Further research should be undertaken to replicate these findings, and to investigate the prospect that symptom reduction may increase the risk of nicotine use in those with attentional deficits. Given changes between the DSM-III-R and the DSM-IV, these findings should be replicated using current diagnostic criteria. Of additional importance is the need to explore the possibility that increased risk of tobacco use among adolescents with problems of inattention further increases the risk of developing additional substance abuse problems.

Note added in proof

We recently discovered that the ADHD data in these analyses were conservatively scored, requiring that each symptom have an onset before age 7. We rescored the data to remove this requirement. Similar results to those reporetd above were obtained when inattention was defined as having five, rather than four or more symptoms. The overall changes were subtle, and the relative strengths of the variables in the final model were the same. When defined by four or more symptoms, childhood inattention was predictive of adolescent tobacco use, even after controlling for the variables in the final model, while adolescent inattention was dropped. This provides even more compelling support for the role of inattention in the development of tobacco use. Details of these analyses are available from the senior author.

References

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: U.S. Government Printing Office.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: U.S. Government Printing Office.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: U.S. Government Printing Office.

Angold, A., & Costello, E. J. (1993). Depressive comorbidity in children and adolescents: Empirical, theoretical, and methodological issues. *American Journal of Psychiatry*, *150*, 1779–1791.

Aytaclar, S., Tarter, R. E., Kirisci, L., & Lu., S. (1999). Association between hyperactivity and executive functioning in childhood and substance use in adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry*, *38*, 172–178.

Babinski, L. M., Hartsough, C. S., & Lambert, N. M. (1999). Childhood conduct problems, hyperactivity-impulsivity, and inattention as predictors of adult criminal activity. *Journal of Child Psychology and Psychiatry*, *40*, 347–355.

Barkley, R. A., Fischer, M., Edelbrock, C. S., & Smallish, L. (1990). The adolescent outcome of hyperactive children diagnosed by research criteria: I. An 8-year prospective follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *29*, 546–557.

Biederman, J., Wilens, T., Mick, E., Faraone, S. V., Weber, W., Curtis, S., Thornell, A., Pfister, K., Jetton, J. G., & Soriano, J. (1997). Is ADHD a risk factor for psychoactive substance use disorders? Findings from a four-year prospective follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 21–29.

Boyle, M. H., & Offord, D. R. (1991). Psychiatric disorder and substance use in adolescence. *Canadian Journal of Psychiatry*, *36*, 699–705.

Coger, R. W., Moe, K. L., & Serafetinides, E. A. (1996). Attention deficit disorder in adults and nicotine dependence: Psychobiological factors in resistance to recovery? *Journal of Psychoactive Drugs*, *28*, 229–240.

Cohen, J. (1960). A coefficient of agreement for nominal scales. *Education and Psychological Measurement*, *20*, 37–46.

Conners, C. K., Levin, E. D., Sparrow, E., Hinton, S. C., Erhardt, D., Meck, W. H., Rose, J. E., & March, J. (1996). Nicotine and attention in adult Attention Deficit Hyperactivity Disorder (ADHD). *Psychopharmacology Bulletin*, *32*, 67–73.

Costello, A. J., Edelbrock, C., Dulcan, M. K., Kalas, R., & Klaric, S. (1987). *Diagnostic Interview Schedule for Children (DISC)*. Pittsburgh, PA: Western Psychiatric Institute and Clinic, School of Medicine, University of Pittsburgh.

Epstein, J. A., Botvin, G., & Diaz, T. (1998). Ethnic and gender differences in smoking prevalence among a longitudinal sample of inner-city adolescents. *Journal of Adolescent Health*, *23*, 160–166.

Frick, P. J., Kamphaus, R. W., Lahey, B. B., Loeber, R.,

Christ, M. A. G., Hart, E. L., & Tannenbaum, L. E. (1991). Academic underachievement and the disruptive behavior disorders. *Journal of Consulting and Clinical Psychology*, *59*, 289–294.

Hart, E. L., Lahey, B. B., Loeber, R., Applegate, B., & Frick, P. J. (1995). Developmental change in attention-deficit hyperactivity disorder in boys: A four-year longitudinal study. *Journal of Abnormal Child Psychology*, *23*, 729–749.

Hartsough, C. S., & Lambert, N. M. (1987). Pattern and progression of drug use among hyperactives and controls: A prospective short-term longitudinal study. *Journal of Child Psychology and Psychiatry*, *28*, 543–553.

Heishman, S. J., Taylor, R. C., & Henningfield, J. E. (1994). Nicotine and smoking: A review of effects on human performance. *Experimental and Clinical Psychopharmacology*, *2*, 345–395.

Hollingshead, A. B. (1975). *Four Factor Index of Social Status*. New Haven, CT: Yale University.

Horner, B. R., & Scheibe, M. A. (1997). Prevalence and implications of attention-deficit hyperactivity disorder among adolescents in treatment for substance abuse. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 30–36.

Jones, G. M. M., Sahakian, B. J., Levy, R., Warburton, D. M., & Gray, J. A. (1992). Effects of acute subcutaneous nicotine on attention, information processing and short-term memory in Alzheimer's disease. *Psychopharmacology*, *108*, 485–494.

Khanzian, E. J. (1997). The self-medication hypothesis of substance use disorders: A reconsideration and recent applications. *Harvard Review of Psychiatry*, *4*, 231–244.

Kovacs, M., Paulauskas, S., Gatsonis, C., & Richards, C. (1988). Depressive disorders in childhood: III. A longitudinal study of comorbidity with and risk for conduct disorders. *Journal of Affective Disorders*, *15*, 205–217.

Lahey, B. B., Pelham, W. E., Schaughency, E. A., Atkins, M. S., Murphy, H. A., Hynd, G., Russo, M., Hartdagen, S., & Lorys-Vernon, A. (1988). Dimensions and types of Attention Deficit Disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *27*, 330–335.

Lambert, N. M., & Hartsough, C. S. (1998). Prospective study of tobacco smoking and substance dependencies among samples of ADHD and non-ADHD participants. *Journal of Learning Disabilities*, *31*, 533–544.

Levin, E. D. (1992). Nicotinic systems and cognitive function. *Psychopharmacology*, *108*, 417–431.

Levin, E. D., Conners, C. K., Silva, D., Hinton, S. C., Meck, W. H., March, J., & Rose, J. E. (1998). Transdermal nicotine effects on attention. *Psychopharmacology*, *140*, 135–141.

Lewinsohn, P. M., Rohde, P., & Brown, R. A. (1999). Level of current and past adolescent cigarette smoking as predictors of future substance use disorders in young adulthood. *Addiction*, *94*, 913–921.

Loeber, R., Farrington, D. P., Stouthamer-Loeber, M., & Van Kammen, W. B. (1998). *Antisocial behaviors and mental health problems: Explanatory factors in childhood and adolescence*. Mahwah, NJ: Lawrence Erlbaum.

Loeber, R., Green, S. M., Lahey, B. B., Frick, P. J., & McBurnett, K. (2000). Findings on disruptive behavior disorders from the first decade of the Developmental Trends Study. *Clinical Child and Family Psychology Review*, *3*, 37–60.

McBurnett, K., Pfiffner, L. J., Willcutt, E., Tamm, L., Lerner, M., Ottolini, Y. L., & Furman, M. B. (1999). Experimental cross-validation of DSM-IV types of attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *38*, 17–24.

Menard, S. (1995). Applied logistic regression analysis. *Sage University Paper series on Quantitative Applications in the Social Sciences, series no. 07–106*. Thousand Oaks, CA: Sage.

Milberger, S., Biederman, J., Faraone, S. V., Chen, L., & Jones, J. (1997a). ADHD is associated with early initiation of cigarette smoking in children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 37–44.

Milberger, S., Biederman, J., Faraone, S. V., Wilens, T., & Chu, M. P. (1997b). Associations between ADHD and psychoactive substance use disorders: Findings from a longitudinal study of high-risk siblings of ADHD children. *The American Journal on Addictions*, *6*, 318–329.

Mitchell, S. H. (1999). Measures of impulsivity in cigarette smokers and nonsmokers. *Psychopharmacology*, *146*, 455–464.

Molina, B. S. G., Smith, B. H., & Pelham, W. E. (1999). Interactive effects of attention deficit hyperactivity disorder and conduct disorder on early adolescent substance use. *Psychology of Addictive Behaviors*, *13*, 348–358.

Petersen, A. C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity and initial norms. *Journal of Youth and Adolescence*, *17*, 117–133.

Prendergast, M. A., Jackson, W. J., Terry, A. V., Decker, M. W., Arneric, S. P., & Buccasfusco, J. J. (1998). Central nicotinic receptor agonists ABT-418, ABT-089, and nicotine reduce distractibility in adult monkeys. *Psychopharmacology*, *136*, 50–58.

Pritchard, W. S., Robinson, J. H., DeBethizy, J. D., Davis, R. A., & Stiles, M. F. (1995). Caffeine and smoking: Subjective, performance, and psychophysiological effects. *Psychophysiology*, *32*, 19–27.

Richardson, S. A., & Tizabi, Y. (1994). Hyperactivity in the offspring of nicotine-treated rats: Role of the mesolimbic and nigrostriatal dopaminergic pathways. *Pharmacology, Biochemistry and Behavior*, *47*, 331–337.

Rusted, J. M., & Warburton, D. M. (1992). Facilitation of memory by post-trial administration of nicotine: Evidence for an attentional explanation. *Psychopharmacology*, *108*, 452–455.

Spitzer, R. L., & Endicott, J. (1978). *Schedule for Affective Disorders and Schizophrenia*. New York: New York State Psychiatric Institute.

Thompson, L. L., Riggs, P. D., Mikulich, S. K., & Crowley, T. J. (1996). Contribution of ADHD symptoms to substance problems and delinquency in conduct-disordered adolescents. *Journal of Abnormal Child Psychology*, *24*, 325–347.

Tizabi, Y., Popke, E. J., Rahman, M. A., Nespor, S. M., & Grunberg, N. E. (1997). Hyperactivity induced by prenatal nicotine exposure is associated with an increase in cortical nicotinic receptors. *Pharmacology, Biochemistry and Behavior*, *58*, 141–146.

Waldeck, T. L., & Miller, L. S. (1997). Gender and impulsivity differences in licit substance use. *Journal of Substance Abuse*, *9*, 269–275.

Wechsler, D. (1974). *Manual for the Wechsler Intelligence Scale for Children-Revised*. New York: Psychological Corporation.

Wesnes, K., & Warburton, D. M. (1983). Smoking, nicotine and human performance. *Pharmacology and Therapeutics*, *21*, 189–208.

White, J. L., Moffitt, T. E., Caspi, A., Bartusch, D. J., Needles, D. J., & Stouthamer-Loeber, M. (1994). Measuring impulsivity and examining its relationship to delinquency. *Journal of Abnormal Psychology*, *103*, 192–205.

Whitmore, E. A., Mikulich, S. K., Thompson, L. L., Riggs, P. D., Aarons, G. A., & Crowley, T. J. (1997). Influences on adolescent substance dependence: Conduct disorder, depression, attention deficit hyperactivity disorder, and gender. *Drug and Alcohol Dependence*, *47*, 87–97.

Wilens, T. E., Biederman, J., & Spencer, T. J. (1996). Attention-deficit hyperactivity disorder and the psychoactive substance use disorders. *Adolescent Substance Abuse and Dual Disorders*, *5*, 73–91.

Witte, E. A., Davidson, M. C., & Marrocco, R. T. (1997). Effects of altering brain cholinergic activity on covert orienting of attention: Comparison of monkey and human performance. *Psychopharmacology*, *132*, 324–334.