Published in final edited form as:
*Alcohol Clin Exp Res*. 2007 April ; 31(4): 643–654. doi:10.1111/j.1530-0277.2007.00349.x.

# Attention-Deficit/Hyperactivity Disorder Risk for Heavy Drinking and Alcohol Use Disorder Is Age Specific

**Brooke S. G. Molina**[1], **William E. Pelham**[2], **Elizabeth M. Gnagy**[2], **Amanda L. Thompson**[3], and **Michael P. Marshal**[4]

1 *Departments of Psychiatry & Psychology, University of Pittsburgh*

2 *Departments of Psychology & Pediatrics, The State University of New York at Buffalo, New York*

3 *Department of Psychology, University of Pittsburgh, Pittsburgh, Pennsylvania*

4 *Department of Psychiatry, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania*

## Abstract

**Background—**This study was designed to assess age specificity in the risk for heavy drinking and alcohol use disorder (AUD) among adolescents and young adults with Attention-Deficit/Hyperactivity Disorder (ADHD) diagnosed in childhood.

**Method—**Children diagnosed with ADHD (*n* = 364 probands) were interviewed an average of 8 years later in the Pittsburgh ADHD Longitudinal Study, either as adolescents (11–17 years old) or as young adults (18–28 years of age). Demographically similar age-matched participants without ADHD were recruited as adolescents (*n* = 120) or as adults (*n* = 120) for comparison with the probands. Alcohol involvement was assessed comprehensively to include measures of heavy drinking that are standard in alcoholism research and prognostic of later alcohol-related problems.

**Results—**Results revealed age specificity in the association such that episodic heavy drinking (measured as 5+ drinks per occasion), drunkenness, DSM-IV AUD symptoms, and DSM-IV AUD were elevated among 15- to 17-year-old probands, but not among younger adolescents. Among young adults, drinking quantity and AUD were elevated among probands with antisocial personality disorder. Childhood predictors indexing antisocial behavior were also examined.

**Conclusions—**The age- specificity of these findings helps to explain prior inconsistencies across previous studies regarding risk for alcohol-related outcomes among children with ADHD.

### Keywords

ADHD; Attention Deficit Disorder; Binge Drinking; Alcoholism; Adolescent Alcohol

Children with Attention-Deficit/Hyperactivity Disorder (ADHD) are theoretically at risk for alcoholism because of their behavioral profiles in early childhood (Pelham and Lang, 1993; Smith et al., 2002). Impulsivity, distractibility, hyperactivity, and, in general, cognitive and behavioral under-regulation describe the symptoms of ADHD as well as the larger construct of behavioral under-control implicated in alcoholism theory (Sher, 1991; Tarter et al., 1990; Zucker et al., 1995). Many longitudinal studies have found a prospective connection between these early-appearing behavioral traits and later alcohol-related outcomes (Caspi et al., 1996; Masse and Tremblay, 1997; Niemela et al., 2006; Tarter et al., 2004). However, empirical tests

Reprint requests: Brooke Molina, Ph.D., Western Psychiatric Institute & Clinic, 3811 O'Hara Street, Pittsburgh, PA 15213; Fax: 412-246-5650; E-mail: E-mail: molinab@upmc.edu.

of the hypothesis that childhood ADHD diagnosis elevates risk for alcohol-related outcomes have produced inconsistent findings. A failure to consider age specificity in the association may have contributed to the discrepancies in the literature.

## Alcoholism Vulnerability in Adolescence

Several studies have investigated the association between childhood ADHD and later alcohol use in adolescence; the findings suggest no substantially increased risk for diagnosable alcohol use disorder (AUD) for ADHD children as a group. Biederman et al. (1997) conducted a 4-year follow-up study of 140 children diagnosed with ADHD and found similar rates of AUD in proband and control adolescents. We followed 142 children with ADHD and did not find significantly more AUD compared with same-aged youth without an ADHD history (Molina and Pelham, 2003). In both of these studies, some participants were quite young (9 years old in the Biederman study, 13 years old in our study). In addition, the average age at follow-up was about 15, which is younger than the age range associated with peak risk for AUD (Grant et al., 1994). The inclusion of children or young adolescents in these comparisons may explain failure to detect group differences in certain alcohol-related outcomes because AUDs have not yet developed, and indices of alcohol consumption other than diagnosis of alcohol abuse or dependence may be more appropriate (Martin and Winters, 1998). Other studies have grouped together participants ranging widely in age, which may have obscured important associations (Gittelman et al., 1985; Mannuzza et al., 1991).

Other findings from these studies suggest that important effects of childhood ADHD may be masked by the over-focus on diagnosable AUD in adolescence. In the Biederman study, children with ADHD had an earlier age of symptom onset than controls for most substance use disorders (that included AUD). Probands also had a significantly shorter span of time between onset of substance abuse and onset of substance dependence, suggesting rapid progression of alcohol-related problems after experimental drinking. Similarly, in the Molina and Pelham study, probands reported more frequent drunkenness, a younger age when first drunk, and more alcohol-related problems that included subclinical symptomatology. Symptoms of AUD in adolescence (Robins and Regier, 1991) and alcohol consumption before age 14 (Grant and Dawson, 1997) are prognostic of later AUD. Thus, probands with ADHD appear to differ from comparison youth without ADHD on more developmentally informed indicators of alcoholism vulnerability in adolescence.

One particularly important measure of problematic drinking among young people is episodic heavy drinking, often defined as 5 or more drinks consumed per drinking occasion (sometimes referred to as “binge drinking”). Although experimentation with alcohol during the teen-age years is commonplace, such early heavy drinking is significantly less benign (Chassin et al., 2002) and predicts AUD and other adverse adult functioning outcomes (Hill et al., 2000). For example, Chassin et al. (2002) found that adolescent heavy drinking predicted alcohol and drug abuse, antisocial personality disorder (ASP) symptoms, and less college attendance by the early 20s. Our earlier findings suggest that drinking alcohol to the point of intoxication (drunkenness) is more prevalent among adolescents with childhood ADHD than among adolescents without childhood ADHD (Molina and Pelham, 2003). Given the long-term risks associated with early heavy drinking, these findings need to be replicated. Research on ADHD and adolescent alcohol use also needs to expand drinking behavior assessments to include the standard set of drinking variables commonly seen in studies of adolescent alcohol use (5 or more drinks per occasion).

## Alcoholism Vulnerability in Early Adulthood in ADHD

In the most recent prospective examination of childhood ADHD and adult drinking, childhood ADHD failed to predict AUDs or other drug use disorders by 20–21 years of age (Fischer et

al., 2002). Other well-known longitudinal studies of children with hyperactivity or ADHD also failed to find ADHD–nonADHD group differences in young adult AUD (Claude and Firestone, 1995; Gittelman et al., 1985; Lambert and Hartsough, 1998; Mannuzza et al., 1991, 1993). These findings are puzzling because elevated rates of drug use disorders in the probands have been reported for all of these samples (for an exception, see Fischer et al., 2002), and because comorbidity between drug and AUDs is common (Robins and Regier, 1991). Moreover, there is growing evidence of sustained educational and occupational impairment in adulthood following childhood ADHD (Mannuzza et al., 1993), and these are correlates of adult problem drinking. Finally, given the contribution of disinhibitory traits to the development of alcoholism (e.g., Sher et al., 2005), childhood ADHD should, theoretically, predict adult AUD. As with adolescents, age of assessment may be a factor. Because heavy drinking is prevalent among young adults (SAMHSA, 2003), group differences between adults with and without childhood ADHD may not be obvious until later when adult roles such as marriage and stable employment are typically assumed. For example, in the Rutgers Health and Human Development Project, Bates and Labouvie (1997) reported a deceleration in the growth of drinking around the early 20s, and reductions in drinking as individuals aged to their late 20s. Thus, there is evidence that the underlying trajectory of drinking behavior in young adulthood is shaped quite differently from that in adolescence, with heavy drinking reaching its peak during this age range, and subsequently declining as adults mature. Longitudinal studies of children with ADHD may need to consider this pattern by examining drinking behavior separately for young adults and for adolescents, and by allowing for the possibility of age-specific ADHD group differences within young adulthood.

## Conduct Problems and Alcoholism Vulnerability Among Children With ADHD

Conduct problems are well-established prospective predictors of early substance use and substance use disorder in longitudinal studies of community samples (Brook et al., 1995; Lynskey and Fergusson, 1995; White et al., 2001). Thus, it is often hypothesized that among children with ADHD, for whom antisocial behaviors are common (Hinshaw, 1987), that symptoms of comorbid behavior disorders (i.e., oppositional defiant disorder, ODD, or conduct disorder, CD) should identify probands at risk for AUD or other substance use/disorders. Although some studies have found that the presence of later externalizing disorders is important for substance use risk in children with ADHD (August et al., 2006; Barkley et al., 1990; Gittelman et al., 1985), a surprisingly small number of studies have specifically examined prediction from childhood symptoms of ODD or CD explicitly. Among clinic samples of children with ADHD followed into adolescence, childhood aggression, conduct problems, or CD significantly predicted alcohol use in 2 studies (Burke et al., 2001; Loney et al., 1981) and failed to predict it in 3 others (Fischer et al., 1993; Mannuzza et al., 2004; Molina and Pelham, 2003). Among children with ADHD followed into early adulthood, childhood conduct problems did not predict weekly alcohol use or drunkenness (Barkley et al., 2003). Thus, although there is considerable evidence that conduct problems co-occur with heavy drinking in adolescence, it is unclear whether conduct problems *in childhood* are robust markers of alcoholism risk among children with ADHD. An answer to this question would help to establish whether an early and widely assessed comorbidity in children being assessed for ADHD is a marker for alcoholism vulnerability.

## The Current Study

The Pittsburgh ADHD Longitudinal Study (PALS) was designed to address the questions raised above. The PALS is a large longitudinal study of children diagnosed with ADHD and a demographically similar group of youth without ADHD recruited for comparison purposes (Faden et al., 2004).[1] The sample includes both adolescents and young adults, allowing examination of questions regarding the age specificity of associations with ADHD. In the

current study, we focused on the alcohol consumption and AUD symptoms/diagnoses of our participants within the developmentally distinct periods of adolescence and young adulthood, when trajectories of drinking behavior assume different shapes. In addition to measures of AUD, we tested the hypothesis that childhood ADHD increased risk for heavy drinking and early drinking, variables that are prognostic of later drinking difficulties (e.g., frequency of episodic heavy drinking, age when first drunk), and yet that are typically absent in longitudinal studies of children with ADHD. Finally, we tested the hypothesis that childhood conduct problems predict alcohol- related outcomes among children with ADHD, and that heavy alcohol use and AUD are comorbid with concurrent conduct problems (CD for adolescents; ASP for young adults).

## MATERIALS AND METHODS

### Participants

**Probands—**Probands were 364 individuals recruited from a pool of 516 children (70.5% participation rate) previously diagnosed with DSM-III-R or DSM-IV ADHD at the ADD Clinic at the Western Psychiatric Institute and Clinic (WPIC) in Pittsburgh, PA during the years from 1987 to 1996. Age at initial evaluation ranged from 5.0 to 16.92 years ($M$ = 9.40, SD = 2.27), with 90% in their elementary school-aged years (ages 5–12). All probands participated in the Summer Treatment Program (STP) for children with ADHD, an 8-week intervention that included behavioral modification, parent training, and psychoactive medication trials where indicated (Pelham and Hoza, 1996). Of the 516 potential participants, 23 could not be located at follow-up and 129 refused or failed to participate. Participating probands were compared with nonparticipating probands on demographic (e.g., age at first treatment, race, parental education level, and marital status) and diagnostic (e.g., parent and teacher ratings of ADHD and related symptomatology) variables. Only one of 14 comparisons was significant at the $p < 0.05$ significance level.[2] Probands were between the ages of 11 and 28 at the time of their first follow-up interview in the PALS, with the majority (99%) falling between 11 and 25 years of age and an average of 8.35 (SD = 2.79) years having elapsed since the probands' initial assessment in the ADD program.

Diagnostic information for the probands was collected in childhood using several sources, including the parent and teacher disruptive behavior disorder (DBD) Rating Scale, which assesses the DSM-III-R and DSM-IV symptoms of the disruptive behavior disorders (Pelham et al., 1992). Parents completed a semi-structured diagnostic interview with PhD-level clinicians consisting of the DSM-III-R or DSM-IV descriptors for ADHD, ODD, and CD, with supplemental probe questions regarding situational and severity factors. The interview also included queries about other comorbidities to determine whether additional assessment was needed (instrument available at http://wings.buffalo.edu/adhd, or through the second author). Following DSM guidelines, diagnoses were made if a sufficient number of symptoms were endorsed (considering information from both parents and teachers) to result in diagnosis. Two Ph.D.-level clinicians independently reviewed all ratings and interviews to confirm the DSM diagnoses. When the 2 clinicians disagreed, a third clinician reviewed the file and the majority decision was used. Exclusionary criteria for participation in the follow-up study were also assessed in childhood: a full-scale IQ < 80, a history of seizures or other neurological problems,

[1]Some of the PALS *young adults* participated as adolescents in the Molina and Pelham (2003) study: 24% of the PALS probands (86/364) and 20% of the PALS controls (47/240). There is no overlap in participants for the adolescents in the two studies.

[2]Participants had a slightly lower average CD symptom rating as indicated by a composite of parent and teacher ratings on the Disruptive Behavior Disorders (DBD) Rating Scale (Pelham et al., 1992) (participants $M$ = 0.43, nonparticipants $M$ = 0.53, $d$ = .30). Other effect sizes ranged from 0.06 to 0.14. Odds ratios were computed for categorical variables (e.g., race); all odds ratios were < 1.4 and associated chi-squared statistics were nonsignificant.

and/or a history of pervasive developmental disorder, schizophrenia, or other psychotic or organic mental disorders.

**Non-ADHD participants—**Two-hundred forty participants without ADHD (controls) were recruited from the Pittsburgh area between 1999 and 2001 for their demographic similarity to the probands at follow-up (e.g., age range between 11 and 25). Most of the minors were recruited through several large pediatric practices in Allegheny County serving patients from diverse socioeconomic backgrounds (40.8% of the control sample). The remaining controls were recruited via advertisements in local newspapers and the university hospital staff newsletter (27.5%), local universities and colleges (20.8%), and other methods (Pittsburgh Public Schools, word of mouth, etc.). A telephone screening interview administered to parents of the controls gathered basic demographic characteristics, history of diagnosis and treatment for ADHD and other behavior problems, presence of exclusionary criteria as previously listed for probands, and a checklist of ADHD symptoms. Young adults (18+) also provided self-report. Attention-deficit/hyperactivity disorder symptoms were counted as present if reported by either the parent or young adult. Individuals who met DSM-III-R criteria for ADHD (presence of 8 or more symptoms)—either currently or historically—were excluded. Control participants with subthreshold ADHD symptomatology, or other psychiatric disorders, were retained.

The control participants were selected to ensure that the 2 groups were equivalent in proportion on several demographic characteristics. As a result, the probands and controls did not differ in age (for ADHD, $M = 17.74$, SD = 3.38; for nonADHD, $M = 17.17$, SD = 3.16), sex (for ADHD, 89.6% male; for nonADHD, 88.7% male), ethnicity/racial minority (for ADHD, 18.4% were minority, 11.0% were African American; for nonADHD, 15.4% were minority, 9.2% were African American), and highest parent education (for ADHD, $M = 7.14$, SD = 1.62; for nonADHD, $M = 7.41$, SD = 1.65, on a scale of 1 (less than seventh-grade education) to 9 (graduate professional training), with 7 = Associate's or a 2-year degree. A higher percentage of probands than controls were from single parent households (33.2% of probands vs 23.6% of controls, $p < 0.05$), probands' parents had lower incomes than controls' parents (for ADHD, $M = \$62{,}959$, SD = 47,971, *Mdn* = $52,000; for nonADHD, $M = \$76{,}091$, SD = 58,140, *Mdn* = $67,318, $p < 0.01$), and more probands than controls had been adopted (8.0% of probands vs 0.4% of controls, $p < 0.01$). The last grade in school completed by the probands and controls was not significantly different between the groups (for ADHD, $M = 11.34$, SD = 2.79; for nonADHD, $M = 11.78$, SD = 3.36).

Because of the wide age range in the PALS sample and our interest in age-specific associations between ADHD and alcohol use, we compared the adolescent (11–17 years old at follow-up) and adult (18+ at follow-up) probands on the same 14 demographic and diagnostic childhood measures examined above. Adolescent probands had significantly higher ($p < 0.01$) reading and math achievement scores, and yet more deviant scores on parent and teacher peer-relations ratings and teacher Abbreviated Conners ratings ($p < 0.05$); no other diagnostic or demographic variables were different between the proband subgroups. Thus, the proband subgroup differences do not suggest a consistent pattern of greater symptom severity and impairment in one age subgroup than in the other.

## Procedure

Follow-up interviews in adolescence and young adulthood were conducted in the ADD Program offices by postbaccalaureate research staff. Interviewers were not blind to recruitment source (i.e., presence or absence of ADHD in childhood), but they were trained to avoid bias in data collection. Moreover, many of the PALS questionnaires are completed privately by participants (e.g., alcohol consumption measures), which helps to minimize interviewer

contamination. Informed consent was obtained and all participants were assured confidentiality of all disclosed material, except in cases of impending danger or harm to self or others (reinforced with a DHHS Certificate of Confidentiality). In cases where distance prevented participant travel to WPIC, information was collected through a combination of mailed and telephone correspondence; home visits were offered as need dictated. Self-report questionnaires were completed either with paper and pencil or web-based versions on a closed-circuit internet page.

## Measures

**Alcohol consumption—**Alcohol use at follow-up was evaluated with a structured paper-and-pencil substance use questionnaire (SUQ) that is an adaptation of existing measures, including the Health Behavior Questionnaire (Jessor et al., 1989) and National Household Survey of Drug Abuse interview (NHSDA, 1992). The SUQ includes both lifetime exposure questions (e.g., have you ever had a drink, have you ever been drunk, age of first drink) and quantity/frequency questions. Four variables indicating alcohol use in the previous 12 months were used in the current study: (1) Frequency of drinking ("In the past 12 months, how often did you drink beer, wine, wine coolers, or liquor?"); (2) Typical quantity of drinking ("How much did you usually drink each time?"); (3) Frequency of episodic heavy drinking ("In the past 12 months, how often did you drink 5 or more drinks when you were drinking?"); and (4) Frequency of drunkenness ("In the past 12 months, how often have you gotten drunk or "very, very high" on alcohol?"). Except for typical quantity, responses ranged from 0 "not at all," to 11 "several times a day". For a typical quantity, responses ranged from 0 "I didn't drink in the past 12 months" and 1 "less than one can or glass", to 13 "more than 25 drinks".

**AUD—**The presence of AUD (lifetime and current) at follow-up was assessed with the Structured Clinical Interview for DSM-IV, Non-Patient Edition (First et al., 1996) for adults (participants aged 18 or older), or a structured paper-and-pencil clinical interview of DSM-IV alcohol abuse and dependence symptoms for minors (age 17 or younger). The Structured Clinical Interview for DSM-IV Axis 1 Disorders—Nonpatient edition (SCID-NP) has well-established psychometric properties. The interview for minors was a fully structured and expanded adaptation of the DSM-IV SCID AUD module by Martin (1995, 2000). Thirty questions addressed lifetime occurrence of alcohol-related problems (e.g., getting into serious trouble at school or having significant arguments with family members because of drinking; drinking to the exclusion of spending time at hobbies or with family/friends), the severity and persistence of the problem, and ages of onset and offset. As with the SCID-NP, a diagnosis of abuse required the presence of at least 1 abuse symptom at the clinical level (score of 2 on a 0–2 scale); a diagnosis of dependence required the presence of at least 3 dependence symptoms at the clinical level. As per DSM-IV, the diagnosis of alcohol dependence precluded a diagnosis of alcohol abuse. Inter-rater reliability for scoring of alcohol abuse and dependence based on these questions, with a psychiatrist board-certified in child and addiction psychiatry, was 0.90 for current AUD and 0.92 for lifetime AUD. A dimensional measure of impairment due to alcohol use was calculated by summing the 11 DSM-IV abuse and dependence symptom scores for all participants (range 0–22).

**Conduct problems in the probands—**Parent and teacher DBD ratings of childhood ODD and CD symptoms were averaged, using as customary (Lahey et al., 1994) the higher rating between the 2 reporters for each symptom. Ratings of symptoms ranged from 0 "not at all" to 3 "very much." Items were those in common between the DSM-III-R and DSM-IV (8 ODD and 13 CD symptom items, Cronbach's alpha = 0.93, $M = 1.01$, SD = 0.42). These ratings were collected when the probands were children, at the time of entry into the STP (Molina and Pelham, 2003). Based on these ratings, 47% of the sample had DSM-IV ODD and 36% of the sample had DSM-IV CD in childhood. The average rating for each child of ODD and CD

symptoms was tested as a predictor of proband alcohol use; the diagnosis of childhood CD was also used for comparison with previous studies and for application to clinical settings.

**Concurrent CD and ASP—**At follow-up, a current diagnosis of CD for adolescents was made using a combination of parent, teacher, and self-report on the DBD adapted for adolescents, and parent and self-report on the DISC (Shaffer et al., 2000). If each symptom of CD was endorsed by any rater, it was counted and adolescents who exceeded DSM-IV symptom cutoffs were diagnosed as having CD. For young adults, parents and young adults completed the Anti-social Personality Disorder portion of the SCID II interview. Because evidence of childhood CD is required for a diagnosis of ASP, parent and teacher ratings collected during childhood (at time of STP enrollment) on the DBD were examined; any young adult who had met CD diagnostic criteria in childhood and exceeded the clinical cutoff of 3 or more items endorsed on the ASP interview was diagnosed as having ASP. (Retrospective DBD self-reports and parent reports describing childhood functioning were used for young adult controls.)

# RESULTS

## Substance Use in the Sample

For descriptive purposes, Table 1 shows the extent of alcohol use, AUD symptoms, and AUD for the PALS sample as a whole and for the proband and control sub-groups. Original values for the continuous drinking variables are recoded for ease of interpretation. Statistical tests of group differences, including tests of interactions with age, are presented below (not in Table 1). Table 1 data are presented separately for adolescents (11–17 years of age) and for young adults (18–25 years of age). The 3 oldest probands (26–28 years old) were deleted from this table and from all subsequent analyses because there were no age-matched controls. Similar to national survey data (SAMHSA, 2003), about 40% of adolescents and over 80% of young adults in our sample had used alcohol at least once in their lives. As predicted, ADHD group differences were not suggested for lifetime use. As a group, the adolescents on average reported drinking less than once a month and drinking heavily about once every other month. Heavy drinking (frequency of 5 or more drinks; frequency of drunkenness), in contrast to the AUD variables (AUD symptoms; diagnosed AUD), suggested the possibility of group differences, with much higher means and standard deviations among the proband than control adolescents. These group differences were not apparent among the young adults.

## Analyses Testing Age Specificity

Ordinary least squares regressions were used to test the hypothesis that risk for alcohol involvement is age specific and measure specific. Given the large differences in alcohol behavior between the adolescent and young adult subsamples, the analyses were conducted separately for these 2 age subgroups. The 4 alcohol use consumption variables and the AUD symptom score were each regressed on age at follow-up, childhood ADHD (no/yes), age squared (to test for a possible nonlinear association), and childhood ADHD×age. There were no statistically significant interactions of childhood ADHD by age squared; results are presented without this term. Age was centered to reduce multicollinearity; influential outliers were examined using DFFITS and DFBETAS (Fox, 1991). Statistically significant interactions were interpreted using calculation of simple slopes at each year of age (Aiken and West, 1991). The results did not change after controlling for the 3 demographic variables that were different between probands and controls (parent income, marital status, and adoption); results are presented from analyses excluding these covariates. Finally, additional analyses were conducted to determine whether the findings were robust to analytic method (i.e., using logistic regression with dichotomized outcomes); the results did not change.

### Adolescent Subsample

Regression results are given in Table 2. There were significant linear and nonlinear associations between age and all of the drinking variables, and significant ADHD by age interactions for 3 variables. The linear and nonlinear age effects reflected the expected higher levels of drinking among older adolescents. The ADHD by age interactions revealed significant prediction of episodic heavy drinking, drunkenness, and AUD symptoms from childhood ADHD beginning at age 15. Specifically, childhood ADHD was not associated with episodic heavy drinking, drunkenness, or AUD symptoms for youth younger than 15, *p*'s = 0.09 to 0.90. However, associations were significant for these alcohol variables at ages 15, *p*' = 0.000 to 0.011; 16, *p*'s = 0.000 to 0.004; and 17, *p*'s = 0.000 to 0.004. For example, for AUD symptoms, $B = 0.20$, $p = 0.000$ at 15; $B = 0.31$, $p = 0.000$ at 16; and $B = 0.42$, $p = 0.000$ at 17. These findings make sense, given the low rates of alcohol use among the adolescents below 15 years of age (e.g., only 16.1% of 11–14-year-old controls and 14.5% of 11–14-year-old probands had consumed alcohol; only 3.6 and 4.3% of these groups had ever been drunk).

### Conduct Problems in Childhood as Predictors of Adolescent Alcohol Involvement

The alcohol variables in Table 2 were regressed on childhood ODD/CD symptoms, child age at follow-up, and childhood ODD/CD symptoms×age, for the adolescent proband subsample (recall that ODD/CD symptoms were not collected in childhood for the controls). Neither the main effect of ODD/CD symptoms, nor the interaction with age, were statistically significant. Zero-order correlations ranged from −0.05 to 0.02.

As a check on the specificity of the childhood ODD/CD symptom variable, we regressed adolescent CD diagnosis on this variable using logistic regression. The prediction was statistically significant, Wald $\chi^2 = 4.43$, df = 1, $p = 0.04$, OR = 2.45.

Conduct disorder diagnosis in childhood predicted AUD symptoms, $B = 0.16$, $p = 0.03$, but not the 4 alcohol use variables (*p*-values higher than 0.16).

### Concurrent CD in Adolescence

The alcohol variables in Table 2 were regressed on age at follow-up and 2 dummy-coded variables indicating the effect of ADHD+concurrent CD versus controls, and ADHD/no concurrent CD versus controls. Eleven to 14 year olds were excluded because of their low levels of alcohol use; 15- to 17-year-old controls with CD ($n = 2$) were also excluded. Across all 5 alcohol variables, probands with concurrent CD had significantly higher alcohol use and AUD symptom scores than controls: for frequency of drinking, $B = 0.43$, $p = 0.00$; for typical quantity, $B = 0.32$, $p = 0.00$; for episodic heavy drinking, $B = 0.53$, $p = 0.00$; for frequency of drunkenness, $B = 0.47$, $p = 0.00$; and AUD symptoms, $B = 0.54$, $p = 0.00$. Probands without concurrent CD did not have higher alcohol use and AUD symptom scores than controls: *p*'s = 0.23 to 0.96.

Table 3 shows the extent of alcohol use, AUD symptom scores, and percents with alcohol abuse and dependence, among the 15 to 17 year olds separately for controls, for all probands, for probands without concurrent CD, and for probands with concurrent CD. Consistent with the regression results, probands with concurrent CD had significantly elevated levels of alcohol use across all 4 of the alcohol consumption variables and the AUD symptom score when compared with controls. Also as expected, compared with controls, probands with CD were more likely to have an AUD (32.1 vs 0% for controls), $\chi^2(1) = 23.14$, $p = 0.000$, and probands without CD fell in between controls and probands with CD, but were still more likely than controls to have an AUD (6.3 vs 0% for controls), $\chi^2(1) = 5.55$, $p = 0.019$.

**Age of onset—**Survival analyses were used to compare age of initial alcohol consumption and drunkenness between the proband and control adolescents (allowing information from all 11–17 year olds to contribute to the analysis). Attention-deficit/hyperactivity disorder group differences were not found for age of first drink, Breslow statistic = 0.34, df = 1, $p = 0.56$, with both groups reporting their first drink of alcohol (more than a sip) at about age fifteen ($M$ = 15.16 for controls, $M$ = 15.00 for probands). Probands reported on average an earlier age of first drunkenness but the group difference was not statistically significant at $p < 0.05$, Breslow statistic = 2.96, df = 1, $p = 0.09$, ($M$ = 16.37 for controls, $M$ = 16.00 for probands). Conduct disorder in adolescence was associated with an earlier age at first drink, Wald $\chi^2(1) = 9.14$, $p = 0.002$, and first drunk, Wald $\chi^2(1) = 15.56$, $p = 0.000$, compared with controls without CD; absence of CD was associated with a later age of first drink for probands versus controls, $\chi^2(1) = 3.96$, $p = 0.047$, but not first drunkenness, $\chi^2(1) = 0.41$, $p = 0.522$.

## Young Adult Subsample

The results of the multiple regressions for the young adults (lower half of Table 2) revealed only linear and nonlinear associations between age and the alcohol involvement variables. The age effects reflected a strong positive association between age and drinking/AUD symptoms among the younger adults (e.g., $B = 0.57$, $p = 0.001$, at age = 18.06, for episodic heavy drinking), and a plateau in this association among the older adults (e.g., $B = -0.07$, $p = 0.59$, at age 21.85, for episodic heavy drinking). There were no statistically significant predictions from childhood ADHD, either alone or in interaction with age at follow-up.

## Conduct Problems in Childhood as Predictors of Adult Alcohol Involvement

There were 2 marginally significant findings ($p < 0.11$) for the young adult probands ($B = 0.81$, $p = 0.10$ for frequency of drinking, $B = 0.12$, $p = 0.08$ for AUD symptoms). The remaining 3 predictions were not significant ($p$-values higher than 0.10). Zero-order correlations ranged from 0.08 to 0.12 for the young adult subsample.

Conduct disorder diagnosis in childhood predicted typical quantity of drinking, $B = 0.16$, $p = 0.03$ and AUD symptoms, $B = 0.15$, $p = 0.03$. The remaining 3 predictions were not significant ($p$-values higher than 0.19).

## Concurrent ASP in Young Adulthood

Antisocial personality disorder comorbidity was examined using dummy coding and after excluding controls with ASP ($n = 4$). Compared with controls, probands with ASP had significantly higher scores for drinking quantity, $B = 0.15$, $p = 0.02$, and AUD symptoms, $B = 0.18$, $p = 0.00$, while probands without ASP did not, $B = -0.07$, $p = 0.25$, and $B = -1.17$, $p = 0.24$, respectively. None of the other comparisons were statistically significant at $p < 0.05$.

The effect of sub grouping the young adult probands into those with, and without, ASP can be seen in Table 4. In general, there was a consistent pattern of higher drinking scores among those with concurrent ASP. As expected from the regression results, probands with ASP were more likely than controls to have an AUD (42.6 vs 22.4% for controls), $\chi^2(1) = 4.95$, $p = 0.03$, but probands without ASP were not (19.7 vs 22.4% for controls), $\chi^2(1) = 0.38$, $p = 0.54$.

**Age of onset—**Attention-deficit/hyperactivity disorder group differences were not found for age of first drink, Breslow statistic = 0.04, df = 1, $p = 0.85$, nor for age of first drunkenness, Breslow statistic = 0.33, df = 1, $p = 0.57$. Probands and controls recalled similar ages for first drink ($M$ = 15.43 for probands, $M$ = 15.68 for controls) and for first time drunk ($M$ = 17.57 for probands, $M$ = 17.76 for controls). We found older ages for these first experiences compared with the adolescents because more adults than adolescents had consumed alcohol by their first follow-up interview. Antisocial personality disorder in adulthood was associated with an earlier

age at first drink, Wald $\chi^2(1) = 5.71$, $p = 0.017$, and first drunk, Wald $\chi^2(1) = 6.59$, $p = 0.010$, compared with controls without ASP; probands without ASP did not report an earlier age at first drink, $\chi^2(1) = 1.84$, $p = 0.175$, nor an earlier age at first drunkenness, $\chi^2(1) = 1.39$, $p = 0.239$.

## DISCUSSION

We examined adolescent and young adult alcohol involvement in the PALS. We hypothesized that the association between childhood ADHD and later alcohol use/AUD would be age specific. This prediction was partially confirmed. Childhood ADHD predicted heavy drinking, drunkenness, AUD symptoms, and AUD for 15 to 17 year olds but not for 11 to 14 year olds. For example, 15- to 17-year-old probands, on average, reported consuming 5 or more drinks of alcohol 17 times in the past year versus an average of only 2 times for same-aged controls. Given the prognostic implications of such early heavy drinking (Chassin et al., 2002), this level of consumption is of concern. Childhood ADHD did not predict young adult drinking/AUD (age 18–25 years), when frequent heavy drinking was typical regardless of ADHD history. These findings underscore the importance of considering both age and measurement issues when assessing risk for alcohol involvement in this population. We found the expected comorbidity at follow-up between antisocial behavior (CD or ASP) and problem drinking for both adolescents and young adults. For young adults with ASP (28% of the young adult probands), drinking quantity and AUD were significantly elevated compared with controls. Predictions to probands' later drinking/AUD symptoms from childhood conduct problems were either non-significant (ODD/CD symptoms) or modest (CD diagnosis).

### Alcohol Risk for the Adolescents

The findings for the adolescents help to explain the discrepant results across earlier studies of childhood ADHD and alcohol use. Prior studies have been limited by their measurement of any alcohol use (Barkley et al., 1990; Hartsough and Lambert, 1987) or diagnosable AUD (Biederman et al., 1997) for wide age ranges spanning early to late adolescence. Failure to find group differences in alcohol use that includes experimental drinking is not surprising, given the typicality of such behavior in the teenage years (Baumrind, 1987; SAMHSA, 2003). Previous failures to detect ADHD group differences in adolescent AUD (Biederman et al., 1997; Molina and Pelham, 2003) may be explained by smaller numbers of youth in the age of highest risk (roughly high school age) in those studies. In the current study, testing the association in a larger sample of mid-to-late adolescent drinkers allowed a more focused (and better powered) examination of problem drinking. We also replicated our previous finding that children with ADHD report more frequent drunkenness as adolescents (Molina & Pelham, 2003), and we extended these findings to include binge drinking (consumption of 5 or more drinks at a time).

Similar to other studies (August et al., 2006; Barkley et al., 1990; Gittelman et al., 1985), alcohol use/AUD risk was specific to adolescent probands who also had CD at the time of follow-up. Moreover, when we added this comorbidity to the analysis, frequency and quantity of drinking were elevated and ages of first drink and drunkenness were younger for the ADHD/CD comorbid probands relative to the controls. We reported similar findings in Molina and Pelham (2003), and also strong associations between delinquent behavior and emerging substance use among the 11- to 13-year-old children in the Multimodal Treatment of ADHD study (Molina et al., 2007). A tendency toward deviance in general among drug and alcohol-using adolescents has been reported for some time (Donovan and Jessor, 1985; Jessor and Jessor, 1977). Thus, it is not surprising that children with ADHD who reported the most alcohol use as adolescents are the same ones who also have CD or otherwise antisocial behavior. In fact, the literature is converging to indicate that a common underlying vulnerability toward

disinhibited behavior is heritable and leads not only to early drinking but also to antisocial behavior and abuse of other substances (Kendler and Prescott, 2006; McGue et al., 2001). This predisposition, seen at young ages in community samples as disinhibited traits or personality (Caspi et al., 1996; Masse and Tremblay, 1997), eventually blossoms for a subset of youth into serious behavior problems, early alcohol consumption, and eventually AUD (Moffitt et al., 2002). Importantly, we do not interpret these findings to mean that childhood ADHD is unimportant as a risk factor for alcohol abuse, but rather that ADHD is associated with higher rates of both antisocial behavior and problem drinking that tend to co-occur in adolescents.

In contrast, prospective prediction of the probands' drinking outcomes from the childhood conduct problem variables was only modest. Oppositional defiant disorder and CD symptom ratings did not predict alcohol outcomes at all; childhood CD diagnosis predicted alcohol abuse and dependence symptoms but not alcohol consumption. Mannuzza et al. (2004) also reported a failure to predict adolescent substance use disorder from childhood symptoms of ODD and CD in their longitudinal study of children with ADHD. Oppositional defiant disorder and CD symptoms in the current sample were not entirely devoid of utility. They predicted CD in adolescence, they predicted illicit drug use in our earlier adolescent follow-up (Molina & Pelham, 2003), and even low levels of CD symptoms predicted later CD in the Mannuzza et al. (2004) study. Thus, there appears to be specificity of association among variables indexing especially deviant behavior that includes negative social consequences. The failure of these variables to be more prognostic may be due to other influences that shape individual children's trajectories toward later antisociality and alcohol use (e.g., parental supervision and control affecting persistence or desistance of antisocial behavior (Maughan et al., 2000; Patterson et al., 1998).

The juxtaposition of the alcoholism (e.g., Sher, 1991; Sher et al., 2005) and ADHD literatures (Barkley, 2006) provides clues to the AUD risk pathways for our probands beyond a simplistic discussion of antisocial behavior comorbidity. For example, ADHD vulnerabilities toward negative peer influences (Marshal et al., 2003), presumably following from the well-established social difficulties experienced by children (Pelham and Bender, 1982) and adolescents with ADHD (Bagwell et al., 2001), suggest the potential importance of social influence processes in ADHD risk for AUD. These appear to be especially problematic for the same youth who have severe ADHD symptoms as children, and who have also developed other antisocial behaviors by adolescence (Marshal and Molina, 2006). Academic difficulties which typify children with ADHD (Barkley, 2006) and that predict alcohol use in adolescence (Hawkins et al., 1992) are probably important in maintaining an ADHD child's progression along a vulnerability pathway. Expectations about the effects of alcohol may operate differently for youth with and without ADHD (McCrady et al., 2006). Finally, elevated rates of alcoholism and other forms of distress among the parents of children with ADHD (Biederman et al., 1992) may indicate both inherited and socialization influences. These and other simultaneously operating risk processes may explain ADHD vulnerability toward problem alcohol use and are the subject of other manuscripts in preparation (McCrady et al., 2006).

It will be important to follow these adolescents into adulthood to test whether their elevated drinking is truly prognostic of later alcohol-related problems—as has been suggested in other studies of nonADHD samples (Chassin et al., 2002). We are currently following all of the PALS participants longitudinally and will eventually be able to test this possibility. We will also study whether heavy drinking among the teenage probands leads to delayed attainment of social and educational milestones, as has been found in other longitudinal studies of drinking adolescents (Chassin et al., 2002; Hill et al., 2000). Research has shown that heavy-drinking adolescents have significantly less positive psychosocial outcomes by age 21, such as less high school completion, involvement in prosocial activities, and family bonding (Hill et al., 2000). Given the growing evidence that children with ADHD already have difficulty meeting their

educational and occupational goals in adulthood (Fischer et al., 2002; Mannuzza et al., 1993), our findings suggest the possibility that early drinking may partly contribute to these adverse outcomes for at least some children with ADHD.

## Alcohol Risk for Young Adults

Our failure in general to find proband–control differences in AUD by young adulthood replicates earlier reports (Claude and Firestone, 1995; Fischer et al., 2002). Given others' findings of group differences in *drug* use disorder for this age range (Mannuzza et al., 1991, 1993), these results may be due to the typicality of heavy drinking among young adults in the general population. AUD (abuse or dependence) reaches its peak at 17.7%of 18 to 25 year olds in the general population, after which it declines substantially, to 6.2% of 26+ year olds (SAMHSA, 2003). Heavy drinking is also common for this age (Griffin et al., 2000; SAMHSA, 2003). Thus, our failure and that of others to detect overall group differences in early adulthood AUD and alcohol consumption may be due to this temporary elevation in heavy drinking for many adults experimenting with newfound independence and celebratory drinking. If there are overall ADHD group differences in AUD to be found in adulthood, they may not emerge until the PALS participants as a group are older, and longitudinal data can distinguish individuals with chronic versus decreasing drinking trajectories, as have been found in other longitudinal studies of adults (Schulenberg et al., 1996).

When the adult probands with ASP were separated out from the remaining probands, however, more heavy drinking, AUD symptoms, and AUD diagnoses were found compared with controls (43 vs 22%). Our rates of AUD were consistent with those recently reported by August and colleagues (46% for 18-year-old probands with childhood ADHD and ODD or CD in the interim; 22% for probands without ODD/CD; 28% for controls; August et al., 2006). Thus, although heavy drinking is commonplace among young adults, it is especially prevalent among children with ADHD who have persisting antisociality. Perhaps as expected, diagnosis of CD in childhood also predicted quantity of alcohol consumption and AUD symptoms. Quite a few studies, including our data, show elevated risk for ASP among children with ADHD (e.g., Mannuzza et al., 1993, 2004; Satterfield and Schell, 1997; but see Lahey et al., 2005 for an exception). Previous research has shown such youth to be at a particularly elevated risk for substance dependencies by adulthood (Moffitt et al., 2002) and this includes AUD well beyond the 18- to 25-year-old age range (Kessler et al., 1997). Consequently, we anticipate the possibility that this subgroup of probands will continue to experience AUD into middle adulthood. Presently, we do not know whether another subgroup of drinkers likely to persist with heavy alcohol involvement exists in the remaining adults. Notably, only a subset of adult problem drinkers are antisocial (Kessler et al., 1997; Robins and Regier, 1991). As the PALS adults mature beyond the period of normative heavy drinking, we will be able to test whether certain characteristics (antisociality, family history of alcoholism, expectations of benefits from alcohol use, impulsivity, and deficient coping skills) distinguish heavy drinkers who persist in their habits from those who desist.

## Study Limitations

We have implied the possibility of different drinking behavior trajectories over time in probands versus controls, or in subgroups of probands versus controls. Obviously, the current data, which are from the first wave of follow-up interviews in the PALS, do not provide direct evidence of such trajectories. Prospective multi-wave data are needed as evidence of this and these data are being collected now. Additional limitations may include the generalizability of our findings to a larger population of children with ADHD. The PALS is a follow-up of clinic-referred children diagnosed with and treated for ADHD in childhood. It is not known whether our findings would apply to the larger population identified in epidemiological studies of children who exhibit symptoms of ADHD and impairment consistent with the disorder. The

probands recruited into the PALS had an average CD symptom rating ($M$ = 0.43) lower than the average rating of the probands not recruited for PALS follow-up ($M$ = 0.53). Although antisociality may be underestimated in the PALS, this difference in a variable scaled from 0 to 3 does not suggest substantial sampling bias. Finally, our sample is primarily male, and our power to test sex differences is decidedly limited.

## Acknowledgements

This research was supported by Grant AA11873 from the National Institute of Alcohol Abuse and Alcoholism. Research was also supported in part by AA00202, AA08746, AA12342, AA0626, and grants from the National Institute on Drug Abuse (DA12414, DA05605, F31 DA017546), the National Institute on Mental Health (MH12010, MH4815, MH47390, MH45576, MH50467, MH53554), and the National Institute of Environmental Health Sciences (ES0515-08).

## References

Aiken, LS.; West, SG. Multiple Regression: Testing and Interpreting Interactions. Sage Publications; Newbury Park, CA: 1991.

August GJ, Winters KC, Realmuto GM, Fahnhorst T, Botzet A, Lee S. Prospective study of adolescent drug use among community samples of ADHD and Non-ADHD participants. J Am Acad Child Adolescent Psychiatry 2006;45:824–832.

Bagwell CL, Molina BSG, Pelham WE, Hoza B. Attention-deficit hyperactivity disorder and problems in peer relations: predictions from childhood to adolescence. J Am Acad Child Adolescent Psychiatry 2001;40:1285–1292.

Barkley, RA. Attention-Deficit Hyperactivity Disorder. Vol. 3. The Guilford Press; New York: 2006.

Barkley RA, Fischer M, Edelbrock CS, Smallish L. The adolescent outcome of hyperactive children diagnosed by research criteria: I. An 8-year prospective follow-up study. J Am Acad Child Adolescent Psychiatry 1990;29:546–557.

Barkley RA, Fischer M, Smallish L, Fletcher KE. Does the treatment of Attention-Deficit/Hyperactivity Disorder with stimulants contribute to drug use/abuse? A 13-year prospective study. Pediatrics 2003;111:97–109. [PubMed: 12509561]

Bates ME, Labouvie EW. Adolescent risk factors and the prediction of persistent alcohol and drug use into adulthood. Alcohol Clin Exp Res 1997;21:944–950. [PubMed: 9267549]

Baumrind, D. A developmental perspective on adolescent risk taking in contemporary America. In: Irwin, CE., editor. Adolescent Social Behavior and Health. Vol. 37. Jossey-Bass; San Francisco: 1987. p. 93-125.

Biederman J, Faraone SV, Keenan K, Benjamin J, Krifcher B, Moore C, Sprich-Buckminster S, Ugaglia K, Jellinek MS, Steingard R, Spencer T, Norman D, Kolodny R, Kraus I, Perrin J, Keller MB, Tsuang MT. Further evidence for family-genetic risk factors in Attention Deficit Hyperactivity Disorder. Arch Gen Psychiatry 1992;49:728–738. [PubMed: 1514878]

Biederman J, Wilens T, Mick E, Faraone SV, Weber W, Curtis S, Thornell A, Pfister K, Jetton JG, Soriano J. Is ADHD a risk factor for psychoactive substance use disorders? Findings from a four-year prospective follow-up study. J Am Acad Child Adolescent Psychiatry 1997;36:21–29.

Brook JS, Whiteman M, Cohen P, Shapiro J, Balka E. Longitudinally predicting late adolescent and young adult drug use: childhood and adolescent precursors. J Am Acad Child Adolescent Psychiatry 1995;34:1230–1238.

Burke JD, Loeber R, Lahey BB. Which aspects of ADHD are associated with tobacco use in early adolescence? J Child Psychol Psychiatry Allied Disciplines 2001;42:493–502.

Caspi A, Moffitt TE, Newman DL, Silva PA. Behavioral observations at age 3 years predict adult psychiatric disorders. Arch Gen Psychiatry 1996;53:1033–1039. [PubMed: 8911226]

Chassin L, Pitts SC, Prost J. Binge drinking trajectories from adolescence to emerging adulthood in a high-risk sample: predictors and substance use outcomes. J Consulting Clin Psychol 2002;70:67–78.

Claude D, Firestone P. The development of ADHD boys: a 12-year follow-up. Can J Behav Sci 1995;27:226–249.

Donovan JE, Jessor R. Structure of problem behavior in adolescence and young adulthood. J Consulting Clin Psychol 1985;53:890–904.

Faden VB, Day NL, Windle M, Windle RC, Grube JW, Molina BSG, Pelham WEJ, Gnagy EM, Wilson TK, Jackson KM, Sher KJ. Collecting longitudinal data through childhood, adolescence, and young adulthood: methodological challenges. Alcohol Clin Exp Res 2004;28:330–340. [PubMed: 15112941]

First, MB.; Spitzer, RL.; Gibbon, M.; Williams, JBW. Structured Clinical Interview for DSM-IV Axis 1 Disorders—Non-patient edition (SCID-1/NP, Version 2.0). Biometrics Research Department, New York State Psychiatric Institute; New York, NY: 1996.

Fischer M, Barkley RA, Gletcher KE, Smallish L. The adolescent outcome of hyperactive children: predictors of psychiatric, academic, social, and emotional adjustment. J Am Acad Child Adolescent Psychiatry 1993;32:324–332.

Fischer M, Barkley RA, Smallish L, Fletcher KE. Young adult follow-up of hyperactive children: self-reported psychiatric disorders, comorbidity, and the role of childhood conduct problems and teen CD. J Abnormal Child Psychol 2002;30:463–475.

Fox, J. Regression Diagnostics. Vol. 79. Sage Publications; Newbury Park, CA: 1991.

Gittelman R, Mannuzza S, Shenker R, Bonagura N. Hyperactive boys almost grown up: I. Psychiatric status Arch Gen Psychiatry 1985;42:937–947.

Grant BF, Dawson DA. Age at onset of alcohol use and its association with DSM-IV alcohol abuse and dependence: results from the National Longitudinal Epidemiologic Survey. J Substance Abuse 1997;9:103–110.

Grant BF, Harford TC, Dawson DA, Chou P, Dufour M, Pickering K. Prevalence of DSM-IV alcohol abuse and dependence. Alcohol Health Res World 1994;18:243–248.

Griffin KW, Botvin GJ, Epstein JA, Doyle MM, Diaz T. Psycho-social and behavioral factors in early adolescence as predictors of heavy drinking among high school seniors. J Studies on Alcohol 2000;61:603–606.

Hartsough CS, Lambert NM. Pattern and progression of drug use among hyperactives and controls: a prospective short-term longitudinal study. J Child Psychol Psychiatry 1987;28:543–553. [PubMed: 3654806]

Hawkins JD, Catalano RF, Miller JY. Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: implications for substance abuse prevention. Psychol Bull 1992;112:64–105. [PubMed: 1529040]

Hill KG, White HR, Chung IJ, Hawkins JD, Catalano RF. Early adult outcomes of adolescent binge drinking: person- and variable-centered analyses of binge drinking trajectories. Alcohol Clin Exp Res 2000;24:892–901. [PubMed: 10888080]

Hinshaw SP. On the distinction between attentional deficits/hyper-activity and conduct problems/aggression in child psychopathology. Psychol Bull 1987;101:443–463. [PubMed: 3602250]

Jessor, R.; Donovan, JE.; Costa, FM. Health Behavior Questionnaire. Institute of Behavioral Science, University of Colorado; Boulder, CO: 1989.

Jessor, R.; Jessor, SL. Problem Behavior and Psychosocial Development: A Longitudinal Study of Youth. Academic Press; New York: 1977.

Kendler, KS.; Prescott, CA. Understanding the Causes of Psychiatric and Substance Use Disorders. The Guilford Press; New York, NY: 2006. Genes, Environment, and Psychopathology.

Kessler RC, Crum RM, Warner LA, Nelson CB, Schulenberg J, Anthony JC. Lifetime Co-occurence of DSM-III-R Alcohol Abuse and Dependence With Other Psychiatric Disorders in the National Comorbidity Survey. Arch Gen Psychiatry 1997;54:313–321. [PubMed: 9107147]

Lahey BB, Applegate B, Barkley RA, Garfinkel B, McBurnett K, Kerdyk L, Greenhill L, Hynd GW, Frick PJ, Newcorn J, Biederman J, Ollendick T, Hart EL, Perez D, Waldman I, Shaffer D. DSM-IV field trials for oppositional defiant disorder and conduct disorder in children and adolescents. Am J Psychiatry 1994;151:1163–1171. [PubMed: 8037251]

Lahey BB, Loeber R, Burke JD, Applegate B. Predicting future antisocial personality disorder in males from a clinical assessment in childhood. J Consulting Clin Psychol 2005;73:389–399.

Lambert NL, Hartsough CS. Prospective study of tobacco smoking and substance dependencies among samples of ADHD and non-ADHD participants. J Learning Disabilities 1998;31:533–544.

Loney, J.; Kramer, J.; Milich, RS. The hyperactive child grows up: predictors of symptoms, delinquency and achievement at follow-up. In: Gadow, KD.; Loney, J., editors. Psychosocial Aspects of Drug Treatment for Hyperactivity. Westview Press Inc; Boulder, CO: 1981. p. 381-415.

Lynskey MT, Fergusson DM. Childhood conduct problems, attention deficit behaviors, and adolescent alcohol, tobacco, and illicit drug use. J Abnormal Child Psychol 1995;23:281–302.

Mannuzza S, Klein RG, Abikoff H, Moulton JL III. Significance of childhood conduct problems to later development of conduct disorder among children with ADHD: a prospective follow-up study. J Abnormal Child Psychol 2004;32:565–573.

Mannuzza S, Klein RG, Bessier A, Malloy P, LaPadula M. Adult outcome of hyperactive boys. Educational achievement, occupational rank, and psychiatric status. Arch Gen Psychiatry 1993;50:565–576. [PubMed: 8317950]

Mannuzza S, Klein R, Bonagura N, Malloy P, Giampino TL, Addalli KA. Hyperactive boys almost grown up. V. Replication of psychiatric status. Archf Gen Psychiatry 1991;48:77–83.

Marshal MP, Molina BSG. Antisocial behaviors moderate the deviant peer pathway to substance use in children with ADHD. J Clin Child Adolescent Psychol 2006;35:216–226.

Marshal MP, Molina BSG, Pelham WE. Childhood ADHD and adolescent substance use: an examination of deviant peer group affiliation as a risk factor. Psychol Addict Behav 2003;17:293–302. [PubMed: 14640825]

Martin CS, Kaczynski NA, Maisto SA, Bukstein O, Moss HB. Patterns of DSM-IV alcohol abuse and dependence symptoms in adolescent drinkers. J Studies on Alcohol 1995:672–680.

Martin CS, Pollock NK, Lynch KG, Bukstein OG. Inter-rater reliability of the SCID substance use disorder section in adolescents. Drug Alcohol Depend 2000;59:173–176. [PubMed: 10891630]

Martin CS, Winters KC. Diagnosis and assessment of alcohol use disorders among adolescents. Alcohol Health Res World 1998;22:95–105. [PubMed: 15706783]

Masse LC, Tremblay RE. Behavior of boys in kindergarten and the onset of substance use during adolescence. Arch Gen Psychiatry 1997;54:62–68. [PubMed: 9006402]

Maughan B, Pickles A, Rowe R, Costello EJ, Angold A. Developmental trajectories of aggressive and nonaggressive conduct problems. J Quantitative Criminol 2000;16:199–221.

McCrady BS, Zucker RA, Molina BSG, Ammon L, Ames GM, Longabaugh R. Social environmental influences on the development and resolution of alcohol problems. Alcohol Clin Exp Res 2006;30:688–699. [PubMed: 16573588]

McGue M, Iacono WG, Legrand LN, Elkins I. The origins and consequences of age at first drink. I. Associations with substance-use disorders, disinhibitory behavior and psychopathology, and P3 amplitude. Alcohol Clin Exp Res 2001;25:1156–1165. [PubMed: 11505047]

Moffitt T, Caspi A, Harrington H, Milne BJ. Males on the life-course-persistent and adolescence-limited antisocial pathways: follow-up at age 26 years. Dev Psychopathol 2002;14:179–207. [PubMed: 11893092]

Molina BSG, Flory K, Hinshaw SP, Greiner AR, Arnold LE, Swanson JM, Hechtman L, Jensen PS, Vitiello B, Hoza B, Pelham WE, Elliott GR, Wells K, Abikoff HB, Gibbons RD, Marcus S, Epstein JA, Greenhill LL, Newcorn J, Severe J, Wigal T. Delinquent behavior and emerging substance use in the MTA at 36-months: prevalence, course, and treatment effects. J Am Acad Child Adolescent Psychiatry. 2007in press

Molina BSG, Pelham WE. Childhood predictors of adolescent substance use in a longitudinal study of children with ADHD. J Abnormal Psychol 2003;112:497–507.

NHSDA. US Department of Health and Human Services, Public Health Service. Alcohol, Drug Abuse and Mental Health Administration. National Institute on Drug Abuse; Rockville, MD: 1992. National Household Survey on Drug Abuse.

Niemela S, Sourander A, Poikolainen K, Helenius H, Sillanmaki L, Parkkola K, Piha J, Kumpulainen K, Almqvist F, Moilanen I. Childhood predictors of drunkenness in late adolescence among males: a 10-year population-based follow-up study. Addiction 2006;101:512–521. [PubMed: 16548931]

Patterson GR, Forgatch MS, Yoerger KL, Stoolmiller M. Variables that initiate and maintain an early-onset trajectory for juvenile offending. Dev Psychopathol 1998;10:531–547. [PubMed: 9741681]

Pelham, WE.; Bender, ME. Peer relationships in hyperactive children: description and treatment. In: Bailer, KGI., editor. Advances in Learning and Behavioral Disabilities. Vol. 1. JAI Press; Greenwich, CT: 1982. p. 366-436.

Pelham WE, Gnagy EM, Greenslade KE, Milich R. Teacher ratings of DSM-III-R symptoms for the disruptive behavior disorders. J Am Acad Child Adolescent Psychiatry 1992;31:210–218.

Pelham, WE.; Hoza, B. Intensive treatment: a summer treatment program for children with ADHD. In: Hibbs, ED.; Jensen, PS., editors. Psychosocial Treatments for Child and Adolescent Disorders: Empirically Based Strategies for Clinical Practice. American Psychological Association Press; New York: 1996. p. 311-340.

Pelham WE, Lang AR. Parental alcohol consumption and deviant child behavior: laboratory studies of reciprocal effects. Clin Psychol Rev 1993;13:1–22.

Robins, LN.; Regier, DA. Psychiatric Disorders in America: The Epidemiologic Catchment Area Study. The Free Press; New York: 1991.

SAMHSA. 2002 National Survey on Drug Use and Health: Detailed Tables. Vol. 2004. Substance Abuse and Mental Health Services Administration; Rockville, MD: 2003.

Satterfield JH, Schell A. A prospective study of hyperactive boys with conduct problems and normal boys: adolescent and adult criminality. J Am Acad Child Adolescent Psychiatry 1997;36:1726–1735.

Shaffer D, Fisher P, Lucas CP, Dulcan MK, Schwab-Stone ME. NIMH Diagnostic Interview Schedule for Children Version IV (NIMH DISC-IV): Description, differences from previous versions, and reliability of some common diagnoses. J Am Acad Child Adolescent Psychiatry 2000;39:28–38.

Schulenberg J, O'Malley PM, Bachman JG, Wadsworth KM, Johnston LD. Getting drunk and growing up: trajectories of frequent binge drinking during the transition to young adulthood. J Studies Alcohol 1996;57:289–304.

Sher, KJ. A Critical Appraisal of Theory and Research. The University of Chicago Press; Chicago: 1991. Children of Alcoholics.

Sher KJ, Grekin ER, Williams NA. The development of alcohol use disorders. Ann Rev Clin Psychol 2005;1:493–523. [PubMed: 17716097]

Smith BH, Molina BSG, Pelham WE. The clinically meaningful link between alcohol and Attention-Deficit/Hyperactivity Disorder. Alcohol Health Res World 2002;26:122–129.

Tarter RE, Kabene M, Escallier EA, Laird SB, Jacob T. Temperament deviation and risk for alcoholism. Alcohol Clin Exp Res 1990;14:380–382. [PubMed: 2378421]

Tarter RE, Kirisci L, Habeych M, Reynolds M, Vanyukov M. Neurobehavioral disinhibition in childhood predisposes boys to substance use disorder by young adulthood: direct and mediated etiologic pathways. Drug Alcohol Depend 2004;73:121–132. [PubMed: 14725951]

White HR, Xie M, Thompson W, Loeber R, Stouthamer-Loeber M. Psychopathology as a predictor of adolescent drug use trajectories. Psychol Addict Behav 2001;15:210–218. [PubMed: 11563798]

Zucker, RA.; Fitzgerald, HE.; Moses, HD. Emergence of alcohol problems and the several alcoholisms: a developmental perspective on etiologic theory and life course trajectory. In: Cicchetti, D.; Cohen, DJ., editors. Developmental Psychopathology, Risk, Disorder, and Adaptation. Vol. 2. Wiley; New York: 1995. p. 677-711.

## Table 1

Alcohol Involvement in the PALS Sample.

| | PALS Sample | PALS Subgroups | |
|---|---|---|---|
| | **Aggregated** | **Control** | **ADHD** |
| *Adolescents (11–17 years old)* | *n* = 283 | *n* = 120 | *n* = 163 |
| Ever used alcohol (%) | 38.9 | 40.8 | 37.4 |
| Ever been drunk (%) | 19.5 | 17.5 | 21.0 |
| Times drank alcohol in the past year, *M* (SD) | 8.1 (29.6) | 2.9 (7.4) | 12.0 (38.1) |
| Usual number of drinks in the past year, *M* (SD) | 1.3 (2.5) | 1.2 (2.0) | 1.5 (2.8) |
| Times drank 5+ drinks in the past year, *M* (SD) | 6.5 (28.6) | 1.6 (6.7) | 10.1 (36.9) |
| Times drunk in the past year, *M* (SD) | 5.8 (26.8) | 1.5 (6.2) | 8.9 (34.6) |
| AUD symptom score total, *M* (SD) | 0.7 (2.0) | 0.3 (0.9) | 1.0 (2.4) |
| Lifetime AUD | | | |
| Lifetime Alcohol Abuse (%) | 3.9 | 0 | 6.8 |
| Lifetime Alcohol Dependence (%) | 1.4 | 0 | 2.5 |
| *Young Adults (18–25)* | *n* = 313 | *n* = 118 | *n* = 195 |
| Ever used alcohol (%) | 85.3 | 88.1 | 83.6 |
| Ever been drunk (%) | 71.2 | 74.6 | 69.2 |
| Times drank alcohol in the past year, *M* (SD) | 55.4 (74.3) | 53.9 (67.5) | 57.0 (78.2) |
| Usual number of drinks in the past year, *M* (SD) | 4.2 (3.75) | 4.0 (3.05) | 4.3 (4.1) |
| Times drank 5+ drinks in the past year, *M* (SD) | 33.9 (65.15) | 29.7 (56.4) | 36.4 (69.9) |
| Times drunk in the past year, *M* (SD) | 24.9 (48.5) | 24.8 (48.5) | 25.2 (49.5) |
| AUD symptom score total, *M* (SD) | 2.7 (4.5) | 2.6 (4.2) | 2.7 (4.7) |
| Lifetime AUD | | | |
| Lifetime Alcohol Abuse (%) | 16.0 | 18.0 | 14.6 |
| Lifetime Alcohol Dependence (%) | 9.7 | 8.3 | 11.1 |

For drinking frequency variables, original values were recoded into number of times per year (range = 0–365) for ease of interpretation; original values were used in statistical comparisons. AUD symptom scores are sums calculated from 11 symptoms (4 abuse, 7 dependence), each with possible scores of 0 (symptom rated as absent), 1 (symptom rated as subthreshold), or 2 (symptom rated as present).

ASP, antisocial personality disorder; PALS, Pittsburgh ADHD Longitudinal Study.

**Table 2**

Regression Results for Prediction of Alcohol Involvement By Childhood ADHD

| | Frequency of Drinking | | Typical Quantity of Drinking | | Frequency of Episodic Heavy Drinking | | Frequency Drunk | | AUD Symptoms | |
|---|---|---|---|---|---|---|---|---|---|---|
| **Predictors** | ***β*** | ***B*** | ***β*** | ***B*** | ***β*** | ***B*** | ***β*** | ***B*** | ***β*** | ***B*** |
| *Adolescents* | | | | | | | | | | |
| Age[a] | 0.38*** | 0.36 | 0.73*** | 0.49 | 0.24** | 0.25 | 0.23** | 0.25 | 0.14 | 0.13 |
| Childhood ADHD (no/yes) | 0.29 | 0.08 | 0.06 | 0.01 | 0.44* | 0.13 | 0.40* | 0.12 | 0.66** | 0.17 |
| Age squared | 0.07* | 0.12 | 0.12** | 0.15 | 0.08** | 0.16 | 0.07* | 0.15 | 0.09* | 0.14 |
| ADHD by age | 0.15 | 0.11 | 0.05 | 0.02 | 0.23* | 0.19 | 0.20† | 0.16 | 0.42** | 0.29 |
| $R^2$ | 0.19*** | | 0.23*** | | 0.18*** | | 0.16*** | | 0.17*** | |
| *Young adults* | | | | | | | | | | |
| Age[a] | 0.47** | 0.32 | 0.23 | 0.14 | 0.37* | 0.25 | 0.21 | 0.16 | 0.75** | 0.31 |
| Childhood ADHD (no/yes) | −0.30 | −0.05 | 0.00 | 0.00 | 0.08 | 0.01 | −0.19 | −0.04 | 0.00 | 0.00 |
| Age squared | −0.10* | −0.17 | −0.09† | −0.13 | −0.12** | −0.21 | −0.09* | −0.16 | −0.14* | −0.15 |
| ADHD by age | 0.05 | 0.03 | 0.11 | 0.06 | 0.01 | 0.01 | 0.05 | 0.03 | 0.10 | 0.04 |
| $R^2$ | 0.07*** | | 0.02 | | 0.04** | | 0.02 | | 0.07*** | |

[a]Age at follow-up. Sample sizes varied across models from 281 to 283 for adolescents, and from 312 to 318 for young adults. *β* = unstandardized regression coefficient. B = standardized regression coefficient. Highest Variance Inflation Factor = 2.43 for adolescents; 3.60 for young adults. Frequency of episodic heavy drinking = frequency of 5 or more drinks consumed at a time.

†$p$ = .053 for ADHD×Age with Frequency Drunk, adolescents; $p$ = .068 for Age Squared with Typical Quantity, young adults.

*$p < .05$;

**$p < .01$;

***$p < .001$.

ASP, antisocial personality disorder; ADHD, attention-deficit/hyperactivity disorder.

**Table 3**

Alcohol Involvement Among 15 to 17-Year-Old Probands and Controls

| | | | ADHD Subgroups | |
|---|---|---|---|---|
| | **Control** | **ADHD** | **ADHD No Concurrent CD** | **ADHD With Concurrent CD** |
| *Adolescents (15–17 years old)* | *n* = 61 | *n* = 94 | *n* = 63 | *n* = 29 |
| Ever used alcohol (%) | 60.7 | 54.3 | 39.7 | 82.8 |
| Ever been drunk (%) | 26.2 | 33.3 | 21.0 | 58.6 |
| Times drank alcohol in the past year, *M* (SD) | 4.26 (8.60) | 19.69 (48.54) | 6.09 (19.13) | 49.71 (74.73) |
| Usual number of drinks in the past year, *M* (SD) | 1.73 (2.16) | 2.28 (3.21) | 1.44 (2.79) | 4.19 (3.34) |
| Times drank 5+ drinks in the past year, *M* (SD) | 2.13 (7.70) | 16.94 (47.38) | 4.07 (18.01) | 45.53 (73.74) |
| Times drunk in the past year, *M* (SD) | 1.80 (6.91) | 14.76 (44.54) | 4.37 (18.29) | 37.90 (70.58) |
| AUD symptom score total | 0.31 (0.92) | 1.57 (3.01) | 0.60 (1.55) | 3.76 (4.21) |
| Lifetime AUD | | | | |
| Lifetime alcohol abuse (%) | 0 | 9.7 | 4.8 | 20.7 |
| Lifetime alcohol dependence (%) | 0 | 4.3 | 1.6 | 10.3 |

For drinking frequency variables, original values were recoded into number of times per year (range = 0–365) for ease of interpretation; original values were used in statistical comparisons (regressions). Controls who met diagnostic criteria for CD (*N* = 2) were excluded.

ASP, antisocial personality disorder; ADHD, attention-deficit/hyperactivity disorder; CD, conduct disorder.

**Table 4**

Alcohol Involvement Among Young Adult Controls and Probands With and Without ASP Comorbidity Control

| | | ADHD Subgroups | |
|---|---|---|---|
| | **Control** | **ADHD No ASP** | **ADHD With ASP** |
| *Young Adults (18–25 years old)* | *n* = 116 | *n* = 143 | *n* = 54 |
| Ever used alcohol (%) | 87.7 | 79.4 | 96.2 |
| Ever been drunk (%) | 73.7 | 63.8 | 84.9 |
| Times drank alcohol in the past year, *M* (SD) | 52.43 (65.42) | 49.37 (72.68) | 77.47 (89.02) |
| Usual number of drinks in the past year, *M* (SD) | 3.98 (3.09) | 3.72 (3.81) | 5.90 (4.52) |
| Times drank 5+ drinks in the past year, *M* (SD) | 30.02 (57.07) | 29.11 (61.18) | 55.91 (86.90) |
| Times drunk in the past year, *M* (SD) | 22.48 (42.15) | 18.46 (38.16) | 43.08 (68.88) |
| AUD symptom score total | 2.44 (4.06) | 1.86 (3.80) | 4.96 (6.15) |
| Lifetime AUD | | | |
| Lifetime alcohol abuse (%) | 16.2 | 13.3 | 18.5 |
| Lifetime alcohol dependence (%) | 6.8 | 6.3 | 24.1 |

For drinking frequency variables, original values were recoded into number of times per year (range = 0–365) for ease of interpretation; original values were used in statistical comparisons (regressions). Controls who met diagnostic criteria for ASP (*N* = 4) were excluded.

ASP, antisocial personality disorder; ADHD, Attention-deficit/hyperactivity disorder.