Childhood Attention-Deficit/Hyperactivity Disorder (ADHD) and Growth in Adolescent Alcohol Use: The Roles of Functional Impairments, ADHD Symptom Persistence, and Parental Knowledge

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Research on the relation between childhood attention-deficit/hyperactivity disorder (ADHD) and adolescent alcohol use has found mixed results. Studies are needed that operationalize alcohol use in developmentally appropriate ways and that test theoretically plausible moderators and mediators in a longitudinal framework. The current study tested childhood ADHD as a predictor of alcohol use frequency at age 17 and age-related increases in alcohol use frequency, through adolescence for 163 adolescents with ADHD diagnosed in childhood and 120 adolescents without ADHD histories. Childhood ADHD did not predict either alcohol outcome. However, parental knowledge of the teen's friendships, activities, and whereabouts moderated the association such that childhood ADHD predicted alcohol use frequency at age 17 when parental knowledge was below median levels for the sample. Mediational pathways that explained this risk included social impairment, persistence of ADHD symptoms, grade point average, and delinquency. Social impairment was positively associated with alcohol use frequency through delinquency; it was negatively associated with alcohol use frequency as a direct effect independent of delinquency. These nuanced moderated-mediation findings help to explain previously inconsistent results for the ADHD-adolescent alcohol use association. The findings also imply that future research and intervention efforts should focus on ADHD-related social and academic impairments as well as symptom persistence and parenting efforts.

Keywords: ADHD, attention deficit, alcohol, adolescence, parenting

Attention-deficit/hyperactivity disorder (ADHD) is among the most common behavioral disorders of childhood, with a prevalence of approximately 7.8% in the United States (Centers for

Disease Control [CDC], 2005). Onset of the disorder is early in a child's life (Lahey et al., 2004) and heritability is well established (Wallis, Russell, & Muenke, 2008). ADHD symptoms persist into adolescence for a majority of diagnosed children (Barkley, Fischer, Edelbrock, & Smallish, 1990; Biederman, Faraone, Milberger, Curtis, et al., 1996; Sibley et al., 2012). Impaired academic performance and social functioning commonly persist and conduct problems often continue or develop (Barkley et al., 1990; Lee, Lahey, Owens, & Hinshaw, 2008; Molina et al., 2009). A specific area of concern beginning in adolescence is increased risk of alcoholism for children with ADHD (Charach, Yeung, Climans, & Lillie, 2011; Lee, Humphreys, Flory, Liu, & Glass, 2011).

The symptoms of ADHD (inattention, impulsivity, and hyperactivity) are prominently featured in alcoholism theory as part of the larger construct of behavioral disinhibition integral to alcoholism risk (Iacono, Malone, & McGue, 2008; Pelham & Lang, 1993; Sher, Grekin, & Williams, 2005; Tarter, Kirisci, Feske, & Vanyukov, 2007; Zucker, 2006). Traits such as "impulsive," "restless," "hyper," and "distractible" are thought to reflect a broad behavioral phenotype indicative of inherited family level risk for alcoholism. This risk may suggest not only biological tendencies to respond differently to alcohol, but also vulnerability to intervening

risk factors that elevate risk, such as deviance proneness and vulnerability to social influences (Derefinko & Pelham, in press; Molina & Pelham, 2003). Studies of these traits in community samples not selected for ADHD have supported these pathways (Caspi, Moffitt, Newman, & Silva, 1996; Martel et al., 2009; Másse & Tremblay, 1997; Niemelä et al., 2006; Tarter et al., 2007; Tarter, Kirisci, Habeych, Reynolds, & Vanyukov, 2004). These early signs of behavioral disinhibition are among the first visible signs of risk for the male-dominated alcoholism subtype that begins at a young age, is accompanied by antisocial behavior, and is severe and chronic in course (Zucker, 2006). Assuming the veracity of this pathway, children (and particularly boys) diagnosed with ADHD should have an increased risk of alcohol misuse beginning in adolescence.

Tests of this prediction, however, have produced variable findings. Although meta-analytic reviews indicate ADHD-associated risk for alcohol use disorder (AUD) by adulthood (Charach et al., 2011; Lee et al., 2011), indication of risk at earlier ages is inconsistent. For example, several well-known longitudinal studies of children diagnosed with ADHD did not find greater rates of adolescent AUD (either abuse or dependence) when compared to adolescents without ADHD histories (Biederman, Wilens, Mick, & Faraone, 1997; Gittelman, Mannuzza, Shenker, & Bonagura, 1985; Mannuzza et al., 1991). These null results may stem from a mismatch between the studies' operationalization of alcohol use and the mean ages of the participants at follow-up (Molina, 2011).

AUDs do not reach their peak prevalence until early adulthood, but alcohol consumption that occurs at greater frequency than is typical for teenage experimentation is associated with later substance dependence (Chassin, Pitts, & Prost, 2002). Alcohol use that begins at a young age, typically defined as before age 15, also predicts later alcohol dependence, abuse of other substances, and other negative health outcomes (e.g., Hingson, Heeren, & Winter, 2006; Odgers et al., 2008). Relatively few studies of ADHD have assessed alcohol use in a manner that would detect consumption atypical for age. Barkley and colleagues' initial follow-up of children with ADHD into adolescence found a small group difference for any alcohol use by the mean age of 15 (Barkley et al., 1990). The early adolescent follow-up of the children in the MTA (multimodal treatment of ADHD) found more alcohol use in the ADHD than in the non-ADHD comparison group (Molina, Flory, et al., 2007). In our first adolescent follow-up of children with ADHD in Pittsburgh, we found more frequent drunkenness but not more frequent AUDs for the adolescents with versus without childhood ADHD (Molina & Pelham, 2003). Another small study reported negative results for alcohol use (Ernst et al., 2006).

In the sample of interest for the current study, the Pittsburgh ADHD Longitudinal Study (PALS), we investigated several alcohol use variables in relation to childhood ADHD, separately for the younger (11–14 years old) and older (15–17 years old) teens, at the first follow-up assessment (Molina, Pelham, Gnagy, Thompson, & Marshal, 2007). We found that frequency of heavy drinking and AUD was associated with childhood ADHD for the older (15–17 years old) but not for the younger (11–14 years old) teens. We did not find ADHD group differences in drinking frequency. These results suggest more rapid growth in alcohol use for adolescents with childhood ADHD such that experimental drinking typical of teens leads more quickly to frequent and eventually problematic drinking. However, a prospective test of acceleration in consump-

tion through adolescence has yet to be conducted for this population. Since the initial PALS publication, the teens have been followed longitudinally. The current study uses these data to compare growth in drinking frequency between adolescents with, and without, childhood ADHD, where "growth" is acceleration in drinking frequency through adolescence and mean drinking frequency at age 17.

Another reason for the modest and inconsistently demonstrated association between childhood ADHD and drinking may rest with the need to consider a key contributor to child adjustment that is also known to affect children with ADHD: parenting. Theories of problem behavior (Donovan & Jessor, 1985; Jessor & Jessor, 1977) and more specifically of teen drinking (Barnes, 1990; Zucker, Donovan, Masten, Mattson, & Moss, 2008) have long emphasized the potential impact of efficacious parenting in promoting positive outcomes for youth. This includes clearly communicated and age-appropriate limit-setting with consistent follow-up, appropriate consequences, and a supportive parent–child relationship (Barnes & Farrell, 1992; Barnes, Reifman, Farrell, & Dintcheff, 2000; Jackson, Henriksen, Dickinson, & Levine, 1997; Wills, Resko, Ainette, & Mendoza, 2004). Parental supervision and monitoring, especially, have been widely studied for their relationships to teen drinking (Barnes et al., 2000; Dishion & Kavanaugh, 2003).

Effective parental monitoring may play an important role in moderating ADHD-related vulnerability to alcohol use. If so, it would help to clarify circumstances under which childhood ADHD leads to teen drinking. Parenting behaviors have been found to moderate genetic and environmental influences on adolescent substance use. Dick and colleagues reported a greater impact of genetic vulnerability to smoking for adolescents whose parental monitoring levels were low (Dick et al., 2007). Barnes and colleagues found that parental monitoring dampened the longitudinal associations between peer deviance and alcohol misuse (Barnes, Hoffman, Welte, Farrell, & Dintcheff, 2006). These results suggest the potential for important interactive influences between individual and contextual vulnerability factors, parental monitoring, and alcohol use.

Research in recent years has taken the additional step of distinguishing between parent reports of their efforts to *monitor* their children, which tend to be positively biased, from parents' actual *knowledge* of their teens' friends, activities, and whereabouts based on adolescent report (Stattin & Kerr, 2000). Adolescents' reports of these and other parenting behaviors are stronger predictors of teen drinking than parent report (Latendresse et al., 2009). However, no research has explicitly tested whether more monitoring or better parental knowledge of children with ADHD affects escalation in teen drinking. This is an important gap in the literature given the well-established evidence base for behavioral parent training on the functioning of children with ADHD (Pelham & Fabiano, 2008). In the current study we test whether successful parental monitoring, defined as *parental knowledge based on adolescent report*, moderates ADHD-related risk for alcohol use in adolescence.

Theoretical models of alcoholism vulnerability include adjustment problems in adolescence that directly overlap with the major domains of impairment common in ADHD, namely, academics and social functioning. Co-occurring conduct problems common to children with ADHD are also implicated in these models. Diffi-

culties in these three domains predict later alcohol and substance misuse in non-ADHD samples (Hawkins, Catalano, & Miller, 1992). Thus, in addition to inherited tendencies to experience alcohol differently (a subject of future studies), increased risk for alcohol misuse among children with ADHD may be partly driven by the academic, social, and behavioral problems they experience in adolescence. However, beyond considering conduct disorder comorbidity and cross-sectional relations with peer substance use (Marshal, Molina, & Pelham, 2003), explanatory models of ADHD risk for alcohol misuse have not directly tested these variables as mediators. Moreover, these potential pathways to alcohol have not been simultaneously tested to allow examination of each variable's unique associations with alcohol use above and beyond effects of the other.

One potential ADHD-related pathway to alcohol use is through grades earned in school. Academic problems in childhood and in adolescence predict substance use in longitudinal studies (Brook, Whiteman, Cohen, Shapiro, & Balke, 1995; Wills et al., 2004). Developmental theories of adolescent alcohol use suggest that failure to engage in activities that promote future goal attainment, such as academic success, creates vulnerability to alcohol misuse (Brook, Brook, Gordon, Whiteman, & Cohen, 1990; Jessor & Jessor, 1977). Children with ADHD are certainly at risk for this particular pathway. Many studies have shown that children with ADHD perform worse on standardized tests of academic achievement than children without ADHD (Barkley, 2006). Multiple negative academic outcomes in adolescence have been associated with childhood ADHD, including lower test scores, grade point averages (GPAs), grade retentions, suspensions, expulsions, and drop-out (Barkley et al., 1990; Biederman, Faraone, Millberger, Guite et al., 1996; Claude & Firestone, 1995; Kent et al., 2011; Molina et al., 2009). GPA may be particularly important as an indicator of academic goals because it is the most visible, recurring, and frequently discussed barometer of school success. As such, children with ADHD should theoretically have increased risk of alcohol misuse partly due to their lower GPAs than adolescents without ADHD histories. Moreover, chronic failure in school should feed the deviance process (Jessor & Jessor, 1977); lower GPAs for adolescents with ADHD histories should increase engagement in delinquent activities and ultimately set the stage for alcohol use.

Social influence and selection processes are well-established as important in the development of alcohol use and other drug use among adolescents (e.g., Curran, Stice, & Chassin, 1997). Affiliation with deviant peer networks is among the strongest predictors and correlates of problem alcohol consumption among youth (Hawkins et al., 1992). Such affiliation is usually believed to result from social difficulties resulting in exclusion from conventional peer groups. Support for this notion is provided by several community studies of children followed longitudinally (Hops, Davis, & Lewin, 1999; Kellam, Ensminger, & Simon, 1980; Másse & Tremblay, 1997). Children who mismanaged conflict with peers, who were aggressive, and who were less accepted by peers were more likely than socially skilled and accepted children to use drugs and alcohol as adolescents (Hops et al., 1999). First grade aggression and "shyness," which included having few friends, together predicted teenage antisocial behavior and drug use that included alcohol (Kellam et al., 1980). These profiles include two key areas of difficulty for children with ADHD, namely, social impairment

that may be caused or exacerbated by ADHD (e.g., difficulty with making and keeping friends), and antisocial behavior that may or may not include aggression. These types of social difficulties are hallmark characteristics of children with ADHD (McQuade & Hoza, 2008; Pelham & Bender, 1982). As such, children with ADHD should theoretically be at increased risk of alcohol use in adolescence because of their social impairments in addition to their well-documented risk of antisocial behavior. The extent to which social impairments operate independently of, or in tandem with, additional behavioral problems also remains to be answered. Finally, given the equifinality of delinquency outcome from preexisting social and academic difficulties, tests of the unique contributions of these variables as mediators in developmental pathways to alcohol use are needed.

The Current Study

The PALS is a multiple-wave follow-up study of children diagnosed with ADHD. A non-ADHD comparison group is included, and yearly assessments through adolescence allow longitudinal modeling of hypothesized associations. The adolescents in the PALS provided four annual waves of data on alcohol consumption, parental knowledge, GPA, social functioning, and delinquent behavior. Thus, the data provide the opportunity to test whether age-related increases in alcohol use between ages 14 and 17, including drinking frequency by age 17, are predicted by childhood ADHD, whether these associations vary as a function of parenting (parental knowledge), and whether academic and social impairment and delinquency mediate observed associations. We used latent growth curve modeling to estimate alcohol growth (escalation with age and level by age 17) and to test our hypotheses; use of these methods improves upon traditional regression techniques by allowing simultaneous estimation of multiple mediational pathways to alcohol growth, thereby demonstrating unique effects of individual pathways while controlling for others. Finally, because many studies show concurrent relations between behavioral disinhibition and substance use, because not all children with ADHD are symptomatic as teens, and to distinguish between areas of impairment and persistence of symptoms, we included ADHD symptoms in adolescence in our models. Together these analyses provided a new opportunity to consider previously untested multivariate pathways to alcohol use for adolescents with, versus without, childhood ADHD.

Method

Participants

ADHD probands. Participants with childhood ADHD were diagnosed with *DSM–III–R* or *DSM–IV* ADHD at the ADD Clinic, Western Psychiatric Institute and Clinic, in Pittsburgh, Pennsylvania, between 1987 and 1996. Average age at initial evaluation was 9.40 years old (*SD* = 2.27 years, range = 5.0–16.92). Ninety percent of children were diagnosed in their elementary school-aged years (ages 5–12). ADHD probands were selected for longitudinal follow-up with annual interviews due to their diagnosis of ADHD and participation in a summer treatment program for children with ADHD, an 8-week intervention that included behav-

ioral modification, parent training, and psychoactive medication trials where indicated (Pelham & Hoza, 1996).

Diagnostic information for ADHD probands was collected in childhood using standardized parent and teacher *DSM–III–R* and *DSM–IV* symptom rating scales (Disruptive Behavior Disorders [DBD]; Pelham, Gnagy, Greenslade, & Milich, 1992) and a standardized semistructured diagnostic interview administered to parents by a Ph.D. level clinician. The interview consisted of the *DSM–III–R* or *DSM–IV* descriptors for ADHD, oppositional defiant disorder (ODD), and conduct disorder (CD) with supplemental probe questions regarding situational and severity factors. It also included queries about other comorbidities to determine whether additional assessment was needed (instrument available through coauthor W.E.P.). Following DSM guidelines, diagnoses of ADHD, ODD, and CD were made if a sufficient number of symptoms were endorsed to result in diagnosis (counting each symptom as positive if endorsed by either parent or teacher in the structured interview or on either parent or teacher rating scale). Two Ph.D. level clinicians independently reviewed all ratings and interviews to confirm DSM diagnoses and when disagreement occurred, a third clinician reviewed the file and the majority decision was used. Exclusion criteria for follow-up was assessed in childhood and included a full-scale IQ <80, a history of seizures or other neurological problems, and/or a history of pervasive developmental disorder, schizophrenia, or other psychotic or organic mental disorders.

Of those eligible for follow-up in the PALS ($n = 516$), 70.5% ($n = 364$) participated ($M = 8.35$ years after childhood diagnosis, $SD = 2.79$). A minority could not be located ($n = 23$); 129 refused or failed to participate. Participating and nonparticipating ADHD probands were compared on 14 demographic, diagnostic, and related symptomatology variables collected in childhood, with only one of 14 comparisons statistically significant ($p < .05$). Participants had a slightly lower average CD symptom rating (participants $M = .43$, nonparticipant s $M = .53$, Cohen's $d = .30$). Average *DSM–III–R* ADHD symptom rating was 2.26, $SD = .45$, on a scale of 0 to 3; average number of *DSM–III–R* ADHD symptoms endorsed by parent or teacher was 12.56, $SD = 1.78$; percent with *DSM–III–R* ODD was 47%; percent with *DSM–III–R* CD was 36%. At the first PALS follow-up interview, which occurred on a rolling basis between 1999 and 2003, mean age was 17.75 years, $SD = 3.39$ years, range = 11 to 28 (three subjects were 26–28 years old), 89.6% were male, and 18.4% were a racial/ethnic minority.

Non-ADHD comparison group. Individuals without ADHD were recruited into the PALS at the same time as the ADHD probands' recruitment into the follow-up study. Non-ADHD comparison participants were recruited on a rolling basis to ensure demographic similarity to the probands as a group (age within 1 year, sex, race, highest parental education). They were recruited from the greater Pittsburgh area from several sources including pediatric practices serving patients from diverse socioeconomic backgrounds, advertisements in local newspapers and the university hospital staff newsletter, local universities and colleges, and other miscellaneous sources. A telephone screening with parents gathered demographic characteristics, history of diagnosis and treatment for ADHD and other behavior problems, presence of exclusionary criteria as previously listed for ADHD probands, and a checklist of ADHD symptoms. Young adults (18+) also pro-

vided self-report. Individuals who met *DSM–III–R* criteria for ADHD, were excluded. Non-ADHD comparison participants with subthreshold ADHD symptomatology or with other psychiatric disorders other than those listed above as exclusionary were retained. There were no statistically significant differences between the 364 ADHD probands and 240 non-ADHD comparison participants on age, sex, ethnicity/racial minority status, or highest parental education. As with the ADHD probands, the non-ADHD comparison participants were interviewed on an annual basis once recruited into the PALS.

Subsample for the current study. Data were selected from the first four annual interviews of the PALS for any participants who were 14, 15, 16, or 17 years old at any of these interviews. This sampling resulted in 283 participants (163 probands; 120 comparison participants). There were no statistically significant differences between these probands and comparison participants on sex, $\chi^2(1) = .001$, *ns*, or ethnicity/racial minority, $\chi^2(1) = .611$, *ns*, but highest parent education was lower in the proband than in the comparison group, $\chi^2(5) = 13.989$, $p < .05$. Younger teens were excluded due to their smaller numbers and low rates of drinking (Molina, Pelham, et al., 2007); 18 year olds were excluded because of the associated educational and residential transitions at that age that have implications for alcohol consumption. Driven by our hypotheses about rate of increase in alcohol consumption with age, we modeled alcohol use by age rather than by year of the annual interview as recommended when age varies considerably within the sample in a given year or "wave" (Bollen & Curran, 2006). For example, for those who were 15 years old at the first annual interview, their alcohol use at ages 15, 16, and 17 was included in the analyses. This resulted in the following numbers of participants providing data for alcohol use one ($n = 91$), two ($n = 84$), three ($n = 82$), or four ($n = 24$) times. The procedure also resulted in the following numbers of probands and comparison participants providing data for alcohol use at ages 14 ($n = 63$ and 49), 15 ($n = 85$ and 70), 16 ($n = 88$ and 64), and 17 ($n = 103$ and 79).

Procedure

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. Interviews with participants and parents were conducted in the ADD program offices by postbaccalaureate research staff. PALS questionnaires are completed privately by participants and their parents via paper and pencil, web-based versions, or secure Internet connection. Teachers and guidance counselors completed measures by mail. Where distance prevented office visits, mail and telephone were used, with home visits as needed. Privacy was reinforced with a Department of Health and Human Services Certificate of Confidentiality.

Measures

Alcohol consumption. Adolescent alcohol use was assessed annually with a structured paper-and-pencil substance use questionnaire (Molina & Pelham, 2003; Molina, Pelham, et al., 2007) that is an adaptation of existing measures (Jessor, Donovan, & Costa, 1989; NHSDA, 1992). The substance use questionnaire

includes both lifetime exposure questions (e.g., age of first drink) and quantity/frequency questions for alcohol and other substances. Pertinent to the current study is the item assessing frequency of alcohol use in the past 12 months ("In the past 12 months, how often did you drink beer, wine, wine coolers, or liquor?"). Responses ranged from 0, *not at all*, to 11, *several times a day*. The percentages of adolescents who reported any alcohol consumption in the past year increased, as expected, from 16.96% (19/112) at age 14 to 57.37% (109/190) at age 17.

Parental knowledge. Parent knowledge of the adolescent's activities was assessed annually in adolescence using adolescent report of the Behavioral Supervision and Strictness subscale of the Authoritative Parenting measure (Steinberg, Lamborn, Dornbusch, & Darling, 1992). The subscale assesses adolescent-perceived attempted parental monitoring and knowledge of the adolescent's friendships, location at night, how money was spent, how free time was used, and where the adolescent was after school. For the current analyses, the items assessing actual (adolescent-perceived) knowledge were used. For these five items, the question stem was, "During the past 12 months, how much did either of your parents *really* know . . .". Responses ranged from 1, *Didn't know*, to 5, *Knew all the time*. Item responses were averaged. Internal consistency of this subscale was high, average $\alpha = .86$.

Academic performance. Average school grades earned in adolescence was calculated annually from participants' report cards obtained directly from guidance counselors. Grades earned closest in time to the participants' interview were coded using a $90\% = A$, $80\% = B$, and so forth, coding scheme and were averaged across courses. This GPA ranged from 19.33 to 99.43. The variable was divided by 10 to render its scale similar to other variables for ease of model estimation.

Social functioning. Parents annually provided ratings of the adolescent's impairment in multiple domains of functioning using the Impairment Rating Scale (IRS; Fabiano et al., 2006). The IRS can be completed by multiple informants from natural settings and has acceptable reliability and validity for children and adolescents (Fabiano et al., 2006: Sibley et al., 2012). For the current analyses, we used the item assessing impairment in relationships with same-aged people. Parents rated "How your son's or daughter's problems affect his or her relationships with other people his or her age." Response options ranged from 0, *No problem, definitely does not need treatment, counseling, or extra help*, to 6 *Extreme problem, definitely needs treatment, counseling, or extra help*. Scores ranged from .22 to 5.13.

Delinquent behavior. Delinquency data were collected annually in adolescence with the Self-Reported Delinquency questionnaire (SRD; Elliott, Huizinga, & Ageton, 1985). The SRD provides a continuous measure of delinquency that is a more comprehensive assessment of conduct problems than is a CD symptom checklist or diagnosis. The SRD was administered to adolescents and parents and inquired about past year occurrence of 37 delinquent acts. The full SRD was not administered at the first annual follow-up; items were supplemented from any positive reports on the CD module of the Diagnostic Interview Schedule for Children for *DSM–IV* (Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000), parent and adolescent report; and DBD, parent, teacher, and adolescent report. A proportion score was calculated for each adolescent at each wave of assessment to reflect the proportion of independent delinquent acts endorsed as occurring in

the past year (range = 0 to .78). The variable was multiplied by 10 to render its scale similar to other variables for ease of model estimation.

ADHD symptoms. ADHD symptoms in adolescence were measured with the DBD adapted for *DSM–IV* and completed annually by parents and teachers (Pelham, Gnagy, Greenslade, & Milich, 1992). The DBD is similar in structure and content to other widely used ADHD symptom rating scales and has demonstrated convergent and discriminant validity (Molina, Smith, & Pelham, 2001; Pelham, Gnagy, Greenslade, & Milich, 1992; Pillow, Pelham, Hoza, Molina, & Stultz, 1998). The DBD includes, in addition to symptoms of ODD and CD, the 18 *DSM–IV* symptoms of ADHD. Responses ranged from 0, *not at all,* to 3, *pretty much.* Teacher data were available for 88% of the sample. Parent and teacher data were combined by taking the higher of the two reporter's responses for each item and then averaging across the 18 items. Internal consistency of this scale was high, average $\alpha = .96$.

Covariates. In addition to demographic covariates described earlier (sex and race/ethnicity), two additional variables were included as covariates in the mediational models because of their associations with childhood ADHD and their expected influences on the mediational processes being modeled. Socioeconomic advantage was calculated based on the parents' education and marital status as *low = 0* (single parent with high school or less education), *medium = 1* (single parent with more than high school education or married parent with high school or less education), or *high = 2* (married parent with more than high school education). Parental psychopathology was calculated as the sum of mother's and father's AUD, antisocial personality disorder (ASPD), and depression (major depressive disorder, dysthymia, or depressive disorder Not Otherwise Specified), each individually scored as *absent* (0) or *present* (1). Parental AUD, depression, and ASPD were based on parent report on the Structured Clinical Interview for the *DSM–IV* Axis I and Axis II disorders (First, Gibbon, Spitzer, Williams, & Benjamin, 1997; First, Spitzer, Gibbon, & Williams, 1998). Diagnoses of AUD were supplemented with the Short Michigan Alcoholism Screening Test (Selzer, Vinokur, & van Rooijen, 1975) administered to parents about themselves and about the other biological parent. ASPD questions were also asked about the other parent.

Analysis Overview

We tested our hypotheses using latent growth curve modeling in MPlus Ver 4.2 (Muthén & Muthén, 2005). Three models reflected our questions: (1) the relation between childhood ADHD and age 17 level and growth rate in adolescent alcohol use; (2) parental knowledge as moderator of the association between childhood ADHD and age 17 level and growth rate in alcohol use; and (3) the relative and unique effects of hypothesized symptom- and impairment-related mediational pathways under conditions of less versus more parental knowledge.

For Question 1, unconditional growth models were first tested to determine whether a linear or nonlinear growth pattern best fit the alcohol use data from ages 14 to 17. In the linear growth curve model, the factor loadings on the alcohol use slope factor were specified as -3, -2, -1, and 0 for ages 14, 15, 16, and 17, respectively, to estimate the level of alcohol use at age 17 by the intercept factor. Nonlinear models were also tested, where the

levels of alcohol use at later time points were freely estimated (i.e., specifying the loadings on the slope factor as 0, 1, *, and * for ages 14, 15, 16, and 17, respectively) to examine whether the estimated loadings significantly departed from a linear trajectory. These analyses were followed by regression of the growth factors (intercept and slope of alcohol use) on childhood ADHD (0 = non-ADHD, 1 = ADHD). This model tested our first hypothesis that mean level of drinking at age 17 and rate of growth in drinking across ages 14 and 17 would be greater for the ADHD probands than for the non-ADHD comparison youth.

For Question 2, the growth model from Question 1 was tested in a multiple group framework using a median split on parental knowledge as the grouping variable (i.e., testing an interaction between childhood ADHD and parental knowledge in association with alcohol use). Although this subgrouping approach is generally less desirable statistically than creating product terms to test an interaction, it was necessary in order to avoid overly complicated moderated-mediation models for question three. The multiple group model simultaneously estimated the relations between childhood ADHD and the alcohol growth factors (intercept and slope) across the two parental knowledge subgroups. All parameters were freely estimated across subgroups to allow differences in parameter estimates across the two subgroups.

To create the subgrouping variable, parental knowledge scores were first examined for age-related changes. As there were none, the mean level across the 4-year age span was modeled with an intercept-only latent variable model. Each adolescent's factor score was saved for subgrouping. Those with scores greater than the total sample median (3.77) of the intercept factor were classified as higher knowledge (M = 4.19; knew about their teen's whereabouts and friends most of the time, n = 142), and the rest were classified as lower knowledge (M = 3.30; knew some of the time, n = 141). Qualitatively similar parental knowledge subgroups (i.e., higher knowledge = parental awareness most or all of the time) have been used elsewhere to prospectively predict alcohol use among nonreferred adolescents (Beck, Boyle, & Boekeloo, 2004). In the current sample, the average score was lower for those with (M = 3.69; SD = .60) than without ADHD histories (M = 3.86; SD = .49), t(281) = 2.899, p < .01; d = .30, and is reflected in a larger proportion of the ADHD than non-ADHD cases falling into the "lower knowledge" subgroup (see Table 1) In the sample overall, the distribution of knowledge scores was highly similar to those observed for other large samples of nonreferred similarly aged adolescents (Beck et al., 2004; Fletcher, Steinberg, & Williams-Wheeler, 2004). The scores were normally distributed, ranging from 1.98 to 4.78, and the mean was just shy of "most of the time," M = 3.75, SD = .57.

For Question 3, mediational pathways between childhood ADHD and the adolescent alcohol use growth factors were added to the multiple group model in Question 2. We added the hypothesized mediators (social impairment, ADHD symptoms in adolescence, GPA, and delinquent behavior) and all background covariates to the previous multiple group model. This allowed a test of whether these mediational pathways differed by the parental knowledge subgroups. As a preliminary step, to decide whether or not to model mediators as parallel growth processes with alcohol use, we first examined whether each mediator exhibited linear change with increasing age (none did). Thus, we modeled these variables in the intercept-only latent factor models to estimate the

overall level of each construct from the repeated measures between ages 14 and 17. The estimated intercept factor scores from each of the four intercept-only models were used as the mediators in the growth models to improve measurement accuracy and to reduce model complexity. The factor score determinacy coefficients were greater than .80 for all of the constructs (average determinacy across four constructs = .87), indicating high correlations between the estimated and the true factor scores. Significance tests for mediated effects were conducted using the method developed by Sobel (1982) and replicated with Bootstrap mediation testing (MacKinnon, 2008). These tests indicate whether a chain of individual path coefficients are statistically significant as a mediational pathway from predictor to outcome through other variables (e.g., childhood ADHD to alcohol slope through adolescent ADHD symptoms).

In all cases, model fit was determined using conventional fit statistics (chi-squared, root mean square error of approximation [RMSEA], and comparative fitness index [CFI]). We used full information maximum likelihood estimation to handle the missing data arising from rearranging the data according to age. We used maximum likelihood estimation with robust standard errors estimation to take into account the non-normality of the delinquency and alcohol use variables (skew range 1.2 to 3.4). We also used the bootstrapping method (Efron, 2000) in Mplus to validate parameter estimates in the presence of non-normal distributions of study variables.

Results

Alcohol Use as a Function of Childhood ADHD

The comparisons of model fit for alcohol growth revealed that the linear model fit the data best, $\chi^2(6)$ = 8.20, ns; RMSEA = .04; CFI = .96. The mean and the variance of the growth rate (slope) factor were statistically significant, indicating that alcohol use frequency on average increased with age, M = .45, SE = .06, p < .001, and the participants in the full sample showed significant variability in the growth rates, variance = .32, SE = .11, p < .01. Alcohol use level at age 17 was significantly greater than zero, M = 1.82, SE = .15, p < .001, and there was statistically significant variability in frequency of drinking at this age, variance = 3.57, SE = .51, p < .001.

When the intercept and slope of alcohol trajectory were regressed on childhood ADHD, the model fit the data well, $\chi^2(8)$ = 10.25, ns; RMSEA = .03; CFI = .97. The associations between childhood ADHD and alcohol use at age 17, B = .38, SE = .29, ns, and between childhood ADHD and alcohol slope, B = .03, SE = .11, ns, were not statistically significant, indicating that the level of alcohol use at age 17 and the growth rate did not differ between the ADHD proband and the non-ADHD comparison groups. These results were not different when we controlled for the demographic covariates.

Parenting as Moderator of the Childhood ADHD–Alcohol Growth Association

The multiple group model fit the data adequately, $\chi^2(25)$ = 39.97, p < .054; RMSEA = .07; CFI = .92. For the lower parental knowledge subgroup, childhood ADHD significantly

Table 1

Descriptive Statistics by ADHD and Parental Knowledge Subgroup

	Covariate variables (frequencies and percentages)[a]			
	Non-ADHD (*n* = 120)		ADHD (*n* = 163)	
	Lower parental knowledge (*n* = 50)	Higher parental knowledge (*n* = 70)	Lower parental knowledge (*n* = 91)	Higher parental knowledge (*n* = 72)
Gender				
Male	47 (94.0)	64 (91.4)	86 (94.5)	64 (88.9)
Female	3 (6.0)	6 (8.6)	4 (4.4)	8 (11.1)
Race/ethnicity				
Caucasian	42 (84.0)	57 (81.4)	71 (78.0)	55 (76.4)
Other	8 (16.0)	13 (18.6)	18 (19.8)	16 (22.2)
Socioeconomic advantage				
Low	0 (0)	2 (2.9)	7 (8.4)	2 (2.8)
Medium	14 (29.2)	14 (20.0)	34 (41.0)	25 (35.2)
High	34 (70.8)	54 (77.1)	42 (50.6)	44 (62.0)
Parental psychopathology				
0	21 (42.0)	42 (60.0)	18 (19.8)	17 (23.6)
1	13 (26.0)	16 (22.9)	20 (22.0)	22 (30.6)
2	7 (14.0)	8 (11.4)	24 (26.4)	15 (20.8)
3	7 (14.0)	3 (4.3)	20 (22.0)	9 (12.5)
4	2 (4.0)	1 (1.4)	4 (4.4)	8 (11.1)
5	0	0	5 (5.5)	1 (1.4)
6	0	0	0	0

	Mediating variables (means and standard deviations)[b]			
	Non-ADHD (*n* = 120)		ADHD (*n* = 163)	
	Lower parental knowledge (*n* = 50)	Higher parental knowledge (*n* = 70)	Lower parental knowledge (*n* = 91)	Higher parental knowledge (*n* = 72)
Social impairment	.62 (.55)	.49 (.43)	1.93 (1.18)	2.12 (1.27)
ADHD symptoms	.90 (.38)	.78 (.32)	1.58 (.44)	1.57 (.50)
Grade point average	81.6 (6.94)	84.1 (5.08)	75.1 (7.02)	78.8 (5.92)
Delinquency	.05 (.004)	.04 (.005)	.13 (.009)	.06 (.005)

	Alcohol use at each age (means and standard deviations)[b]			
	Non-ADHD (*n* = 120)		ADHD (*n* = 163)	
	Lower parental knowledge (*n* = 50)	Higher parental knowledge (*n* = 70)	Lower parental knowledge (*n* = 91)	Higher parental knowledge (*n* = 72)
Age 14	.47 (1.07)	.22 (.55)	.93 (1.96)	.15 (.56)
Age 15	.85 (1.69)	.59 (1.19)	1.55 (2.06)	.26 (.62)
Age 16	1.25 (1.56)	1.03 (1.73)	2.15 (2.43)	.55 (1.40)
Age 17	2.21 (1.83)	1.63 (2.01)	2.75 (2.94)	.81 (1.39)

Note. For social impairment, 0 = *no problem* and 6 = *extreme problem*. For ADHD symptoms, 0 = *not at all* and 3 = *pretty much*. For grade point average, 90% = A, 80% = B, etc. For delinquency, score = proportion of delinquency items endorsed. The scale units of grade point average and delinquency were transformed for the analyses as described in the measures section. For alcohol use, 0 = *not at all*, 1 = *1–3 times*, 2 = *4–7 times*, 3 = *8–11 times*, … 11 = several times a day.
[a] Frequency and percentage of frequency for each ADHD/parental knowledge subgroup. [b] Means and standard deviations for each ADHD/parental knowledge subgroup.

predicted the alcohol intercept at age 17, B = .86, *SE* = .42, *p* < .05, but childhood ADHD did not significantly predict alcohol slope, B = .05, *SE* = .22, *ns*. For the higher parental knowledge subgroup, childhood ADHD was not significantly related to the alcohol intercept, B = −.43, *SE* = .31, *ns*, nor to the alcohol slope, B = −.11, *SE* = .10, *ns*. When the path coefficients between childhood ADHD and the alcohol intercepts were forced to be equal across parenting subgroups,

model fit deteriorated, *p* < .001, demonstrating a statistically significant interaction between childhood ADHD and parental knowledge in association with alcohol use frequency at age 17. The means for the slopes and the variances of the intercept and slope factors were not significantly different across subgroups. The changes in alcohol use across ages are graphed for both parental knowledge subgroups in Figure 1, where the childhood ADHD effect on drinking frequency at age 17 (but not slope) is
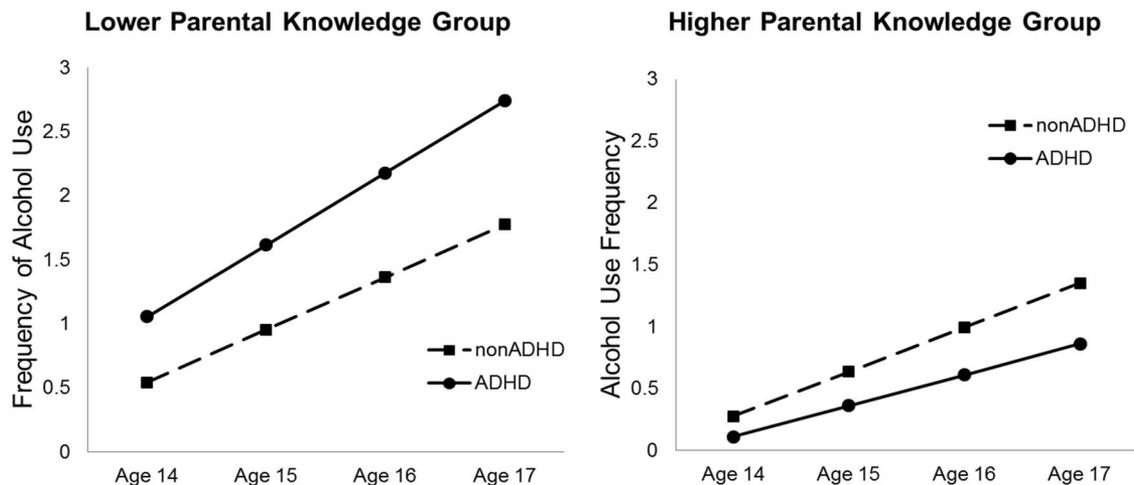
Figure 1. Estimated trajectory of alcohol use frequency by ADHD history and parental knowledge subgroup. The R^2 values for the intercept and slope factors were .15 and .06, respectively, for the lower knowledge group and .15 and .16, respectively, for the higher knowledge group.

visible for the lower parental knowledge subgroup but not for the higher parental knowledge subgroup. (Although the average frequency of alcohol use appeared lower for ADHD than non-ADHD in the higher parental knowledge subgroup, the differences was not statistically significant.)

Mediational Model

The multiple group mediation model is shown in Figure 2. As described earlier, adolescent GPA, social impairment, delinquent behavior, and ADHD symptoms, measured between the ages of 14 and 17, were tested as mediators of the relation between childhood ADHD diagnosis and adolescent alcohol growth for the parental knowledge subgroups. The model fit the data well, $\chi^2(101) = 89.37$, *ns*; RMSEA = .00; CFI = 1.00, with large proportions of variance accounted for in the intercept and slope factors. Childhood ADHD was associated with social impairment, adolescent ADHD symptoms, and GPA independent of parental knowledge level. Other direct paths tended to be specific to parental knowledge subgroup. We set nonsignificant path coefficients to zero and there were trivial differences in the model fit. Means and standard deviations for all included variables, separately by ADHD and the parental knowledge subgroups, are shown in Table 1. The results of the significance tests for mediated effects are shown in Table 2. These results show the presence of multiple mediational pathways from childhood ADHD to adolescent alcohol frequency slope and intercept.

Lower parental knowledge subgroup. Two mediational pathways through social impairment to alcohol frequency at age 17 were found. In one, childhood ADHD predicted more social impairment which in turn related *negatively* to alcohol use frequency at age 17. In the other, childhood ADHD predicted more social impairment, which in turn related positively to delinquency, which in turn related positively to alcohol use frequency at age 17. Two mediational pathways through adolescent ADHD symptoms were found. One was to alcohol frequency at age 17 and the other was to alcohol slope: children with ADHD whose symptoms persisted reported more frequent drinking by age 17, and their drinking frequency escalated more rapidly with age. A mediational pathway through GPA and delinquency was found. Childhood ADHD predicted lower GPAs in adolescence, which in turn related negatively to delinquency (lower GPA, more delinquency), which in turn related positively to alcohol use frequency at age 17. A mediational pathway through delinquency was found. Childhood ADHD predicted higher delinquency in adolescence, which in turn related positively to alcohol use frequency at age 17.

Higher parental knowledge subgroup. No mediational pathways through social impairment or GPA were found. A mediational pathway through adolescent ADHD symptoms and delinquency was found such that childhood ADHD predicted ADHD symptoms in adolescence, which in turn related positively to delinquency and to alcohol frequency at age 17. One inverse direct association between childhood ADHD and alcohol use at age 17 was found, such that childhood ADHD predicted lower frequency of alcohol use at age 17, independent of the mediators (consistent with the nonsignificant pattern found in Model 2, Figure 1). No other mediational pathways reached conventional statistical significance (i.e., $p < .05$) (see Table 2).

The four direct paths that were statistically significant in both parental knowledge subgroups (childhood ADHD to social impairment, childhood ADHD to adolescent ADHD symptoms, childhood ADHD to GPA, and delinquency to alcohol frequency at age 17) were individually tested for equivalence by constraining them to be equal across subgroups and examining change in model fit. There were no statistically significant changes in the model chi-squared values. Thus, these direct paths were not significantly different between parental knowledge subgroups.

Discussion

To our knowledge, this was the first longitudinal study of children with ADHD that used latent growth curve modeling to examine age-related increases in adolescent alcohol use, and alcohol use frequency at age 17, as a function of childhood ADHD.
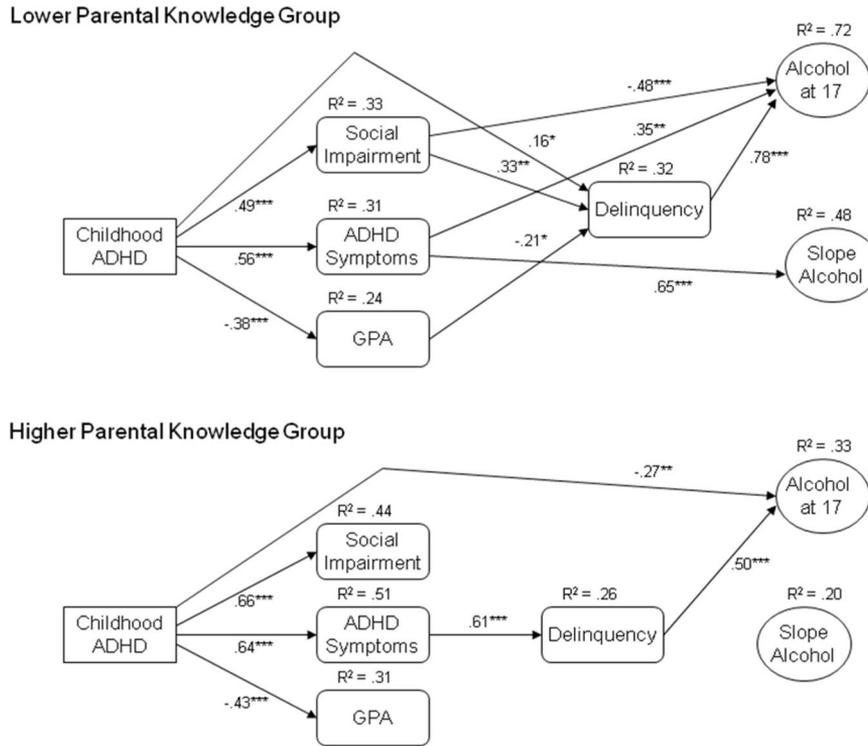
Figure 2. Mediation model for growth in alcohol use from ages 14 to 17. Path coefficients are standardized. Nonsignificant paths are omitted from the figure. Background covariates (e.g., socioeconomic advantage) and covariances among the residual variances of social impairment, ADHD symptoms, and GPA were included in the model estimation, but they are omitted from the figure for ease of presentation. Significant associations with covariates may be obtained from the first author. The covariance between the residual variances of the growth factors (i.e., intercept and growth rate [slope] factors) was not statistically significant in the lower parental knowledge group, whereas it was significant in the higher parental knowledge group (B = .53, *SE* = .20, *p* <.01). $^{*} p < .05$, $^{**} p < .01$, $^{***} p < .001$.

Contrary to expectation, our results did not support a main effect of childhood ADHD diagnosis on these alcohol outcomes. However, when parental knowledge of the teen's friendships, activities, and whereabouts was below the sample median, alcohol use by age 17 was higher for the ADHD than for the non-ADHD group. Multiple mediational pathways were found, in the presence of low parental knowledge in particular, that highlighted the importance of symptom persistence and functioning in domains secondary to ADHD for the development of alcohol use. Above-median parental knowledge nullified the association between childhood ADHD and later alcohol use, supporting moderation of the childhood ADHD association with drinking frequency by a key parenting variable widely studied in the adolescent alcohol use literature and taught in the ADHD parent-training literature (Pelham & Fabiano,

Table 2

Mediated Effects by Parental Knowledge Subgroups

Mediational paths	Lower parental knowledge	Higher parental knowledge
ADHD → social impairment → intercept of alcohol frequency	−.81 (.21)***	
ADHD → social impairment → delinquency → intercept of alcohol frequency	.44 (.15)**	
ADHD → ADHD symptoms → intercept of alcohol frequency	.70 (.26)**	
ADHD → ADHD symptoms → alcohol slope	.34 (.11)**	
ADHD → GPA → delinquency → intercept of alcohol frequency	.22 (.10)*	
ADHD → delinquency → intercept of alcohol frequency	.45 (.22)*	
ADHD → ADHD symptoms → delinquency → intercept of alcohol frequency		.60 (.19)**

Note. Only mediational paths reaching $p < .05$ significance are reported. Reported statistics reflect the mediated effects obtained by the product of the path coefficients (Sobel, 1982) in the chain of associations. These tests were replicated with bootstrap mediation tests, and the results were essentially identical.
$^{*} p < .05$. $^{**} p < .01$. $^{***} p < .001$.

2008). These findings of moderated mediation, demonstrated above and beyond parental psychopathology and socioeconomic background, provide new information about etiologic and clinically relevant pathways to alcoholism risk in children with ADHD.

The absence of a main effect for childhood ADHD and adolescent alcohol use was surprising given our prior findings of more frequent heavy drinking, drunkenness, and AUD symptoms for adolescents with versus without childhood ADHD (Molina & Pelham, 2003; Molina, Pelham et al., 2007). We attempted to model heavy drinking frequency to test whether a more deviant level of alcohol consumption would yield the same group differences we have reported in the past, but the skewed distribution of the variable combined with the analytic demands of the growth modeling resulted in poor model fit. Thus, we were not able to test whether these findings would hold for heavy drinking/problematic drinking outcomes. Alternatively, our findings may reflect the generally modest and inconsistent relation between childhood ADHD and alcohol use that has been seen in the literature to date (Lee et al., 2011; Molina, 2011). Our results show that a key moderator and several mediators help to explain conditions under which ADHD vulnerability to alcoholism may be most strongly expressed.

Our findings regarding parental knowledge as moderator of the ADHD–alcohol association point to the potentially important protective role of continued parental supervision through high school-aged adolescence. Many studies have documented associations between this parenting behavior and delinquency/substance use in teens, but the current study indicates its direct relevance for alcohol use by adolescents with ADHD histories. Parenting practices, including the degree to which parents attend to and monitor their children, have long been targeted in the evidence-based treatment of ADHD in children and adolescents (Chronis, Chacko, Fabiano, Wymbs, & Pelham, 2004). Our finding is particularly noteworthy in light of the normative trend for parental monitoring to decrease throughout adolescence (Barnes et al., 2006). Unfortunately, reduced monitoring may be ill-advised for adolescents with ADHD. Improved parenting of children with ADHD leads to better functioning in multiple domains of impairment and conduct (Chronis et al., 2004). We did not find better social skills or GPA for ADHD in the presence of higher parental knowledge, but we did find that the associations between these variables and drinking was dampened when parents knew "most of the time" what their teens were doing and with whom. Thus, vigilant parenting of teens with ADHD may thwart the socialization influences that typically surround teen drinking. Successful parental monitoring may also be a marker of effective parenting more generally that includes clear limit-setting and consistent rule enforcement. These parenting strategies have been shown in multiple studies to be interrelated and to predict healthy adolescent adjustment more generally (Dishion & McMahon, 1998; Patterson & Fisher, 2002).

Parent efforts to monitor, however, reflect only one side of the parent-teen dynamic. A recent literature recognizes that parental awareness of their teens' activities and whereabouts may reflect teen disclosure in the context of a warm and supportive relationship rather than active parental effort to supervise (Kerr, Stattin, & Burk, 2010; Stattin & Kerr, 2000). This is an important alternative interpretation of our findings. However, from our first follow-up study of children with ADHD, we found that parental knowledge and not quality of parent-teen relationship was correlated with

heavy alcohol use (Walther et al., 2012). In fact, these and other findings about parenting in the adolescent substance use literature (e.g., Barnes et al., 2000, 2006) prompted us to study parental knowledge instead of a more in-depth examination of multifaceted parenting. For adolescents with ADHD histories, whose relationships with parents are often conflictual (Barkley, Fischer, Edelbrock, & Smallish, 1991; Walther et al., 2012), active parental monitoring efforts may have greater salutary impact than for teens not affected by ADHD. Ultimately, it may be the case that a combination of active parental monitoring and teen disclosure results in the healthiest and lowest risk outcome for adolescents including teens with ADHD histories.

We found a mediational pathway to drinking through school performance that was significant in the presence of lower parental knowledge. Grades earned in school were lower for teens with ADHD histories, lower grades were associated with more delinquency, and more delinquency was associated with more frequent alcohol consumption by age 17. Although correlational, this "academic-deviance pathway" underscores for children with ADHD the potential importance of school performance and parental knowledge, above and beyond persistence of ADHD symptoms. The findings also expand our understanding of ADHD-related alcohol risk beyond CD (herein captured as delinquency) to emphasize the import of school performance. This point is particularly important because stimulant medication, the most readily available treatment for ADHD, decreases ADHD symptoms and disruptive behavior in childhood, but it has a limited impact on long-term academic achievement in children with ADHD (Loe & Feldman, 2007). At least one study of stimulant treatment for ADHD teens in an academic setting shows that medication acutely improves multiple aspects of academic performance (Evans et al., 2001). However, despite these salutary acute effects, the great majority of children with ADHD stop taking medication beyond the elementary school-aged years (Barkley, Fischer, Smallish, & Fletcher, 2003; Molina et al., 2009). Thus, interventions are needed that either result in sustained medication treatment that results in benefits throughout adolescence and/or that use psychoeducational and psychosocial techniques that directly target GPA-related skills (e.g., study and organizational skills, homework completion). Such efforts might specifically include methods appropriate for attention-challenged teens (Evans, Schultz, DeMars, & Davis, 2011; Jitendra, DuPaul, Someki, & Tresco, 2008; Sibley et al., 2011).

We found both expected and (somewhat) unexpected associations, in opposing directions, for the pathways that included social impairment. The predicted pathway, from childhood ADHD to alcohol use through social impairment and delinquency, was partially supported, in the lower parental knowledge subgroup, for drinking frequency at age 17. Thus, to the extent that social difficulties that result in peer rejection are accompanied by delinquent activities such as skipping school, damaging or stealing property, and fighting, ADHD-related risk for drinking is increased. This "social deviance pathway," like the "academic deviance pathway," also broadens our understanding of ADHD-related risk beyond CD comorbidity (a narrow diagnostic version of the delinquency behavior spectrum that has been strongly associated with substance use risk). Whereas previous research has tended to focus solely on the role of CD in ADHD-related risk, the current findings allow speculation that enduring social deficits

may propel children with ADHD into antisocial networks that eventually include alcohol consumption.

However, social functioning difficulties for children with ADHD are heterogenous (McQuade & Hoza, 2008; Pelham & Bender, 1982), which may help to explain the negative association that we observed between social impairment and age 17 alcohol use outside (independent) of the delinquency pathway. Some of the typical peer problems suffered by children with ADHD, such as few or no friends, may shield children with ADHD from exposure to the social arenas where teen drinking occurs. Having such poor social skills that one is ignored by as opposed to actively rejected by peers may be associated with different outcomes, as the developmental literature on peer relationships suggests (Bierman, 2004). At the risk of oversimplifying, peer functioning problems for children with ADHD tend to be described as "rejected" or "neglected" and this distinction, infrequently considered, may be important for substance use risk. Our sample may have a mixture of these social profiles. Our planned analyses with this sample in adulthood, when we have social network data that provides more detailed information about friendships, will aide further testing of these ideas.

Persistence of ADHD symptoms predicted alcohol use at age 17 (intercept) and escalating alcohol use (slope). These associations were found in the presence of lower parental knowledge and independent of any associations with delinquency. We found a cross-sectional version of this association in our first follow-up of children with ADHD (Molina & Pelham, 2003) and others have reported it (Barkley, Murphy, & Fischer, 2008; Knop et al., 2009) but not with simultaneous tests of social and academic impairment. These findings suggest the possibility of other, possibly unaccounted for, pathways to alcohol use for teens with persisting symptomatology. Childhood ADHD is known for variable persistence over time; this may reflect distinct biological underpinnings with implications for alcohol use. Studies of differential response to alcohol for disinhibited adults presumably measure traits that have persisted from childhood and that may be similar to the chronic cognitive and behavioral deficits experienced by those with persisting ADHD. In fact, a recent laboratory study found that adulthood ADHD is associated with more disinhibition following alcohol consumption (Weafer, Fillmore, & Milich, 2009). If this speculation is true, it might also help to explain the presence of the ADHD symptom–delinquency pathway in the presence of higher parental knowledge. Thus, although this speculation remains to be tested, our findings encourage research on additional mechanisms of risk and pathways to alcohol beyond peer relations and academic functioning that may be unique to the neurocognitive mechanisms that produce both ADHD symptoms and alcoholism vulnerability.

When considered as a whole, our results help to explain the inconsistent associations between childhood ADHD and adolescent alcohol use that have characterized the literature (Derefinko & Pelham, in press; Lee et al., 2011; Molina, 2011). Children with ADHD do not appear to be universally at-risk for adolescent alcohol use and, in fact, may have dramatically different risk levels that are affected by parenting behavior (or by child-to-parent disclosure) and the child's specific social, academic, and behavioral deficits. Thus, by including additional mediating and moderating variables in our statistical models, a variant of the suppression effect (MacKinnon, Krull, & Lockwood, 2000) was observed

such that ADHD is predisposing of alcohol use in one context (low parental awareness, social difficulties with peers, tendency toward deviance) but ironically protective in another (high parental awareness, social difficulties with peers). These results may explain why, when analyzed as a group, children with ADHD show inconsistent risk for alcohol use in adolescence. As mentioned earlier, sample characteristics (this being a clinic-referred sample) and operationalization of the alcohol use variable may also drive inconsistent findings across studies. Finally, as not all participants were followed for the full 4 years of adolescence that were modeled, replication of these results with other samples and with even more complete longitudinal data will be important. Our findings suggest the possibility that parenting behavior moderates ADHD-related alcohol use risk, and they encourage additional efforts to increase the armamentarium of intervention possibilities for this underserved adolescent population. They also point to the necessity of considering functioning levels beyond ADHD symptoms and deviance proneness through the inclusion of indicators of impairment that have a direct bearing on the deviance-alcoholism pathway (social rejection; poor performance in school). Given the importance of these domains of functioning for other outcomes (e.g., adolescent depression experienced by aggressive children; Patterson & Capaldi, 1990), it appears crucial that both theoretical models and related interventions directly target these areas of functioning.

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