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Heavy Alcohol Use in Early Adulthood as a Function of Childhood ADHD: Developmentally-Specific Mediation by Social Impairment and Delinquency

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Abstract

Frequent heavy drinking in early adulthood, particularly prior to age 21, is associated with multiple health and legal consequences including continued problems with drinking later into adulthood. Children with Attention-Deficit/Hyperactivity Disorder (ADHD) are at risk of alcohol use disorder in adulthood, but little is known about their frequency of underage drinking as young adults or about mediational pathways that might contribute to this risky outcome. The current study used data from the Pittsburgh ADHD Longitudinal Study (PALS) to test social impairment and delinquency pathways from childhood ADHD to heavy drinking in early adulthood for individuals with (n=148) and without (n=117) childhood ADHD. Although ADHD did not predict heavy drinking, indirect mediating effects in opposing directions were found. A delinquency pathway from childhood ADHD to *increased* heavy drinking included adolescent and subsequently adult delinquent behavior. A social impairment pathway from childhood ADHD to *decreased* heavy drinking included adolescent, but not adult, social impairment. These findings help to explain the heterogeneity of results for alcohol use among individuals with ADHD and suggest that common ADHD-related impairments may operate differently from each other and distinctly across developmental periods.

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Alcohol use typically begins in adolescence and increases in frequency until it reaches its peak prevalence in the United States between ages 18 and 25 (Jackson, Sher, Gotham, & Wood, 2001; Muthen & Muthen, 2000). Excessive use of alcohol, including the consumption of five or more drinks within two hours (four or more for women), is widespread among young adults including underage drinkers (less than 21 years of age). Findings from the 2010 National Survey on Drug Use and Health (SAMHSA, 2012) indicated that 37.3% of 20 year olds reported binge drinking in the past 30 days, and 13.7% reported past month "heavy drinking" (binge drinking on at least five days out of the past 30). Although excessive underage drinking is more frequent among college students (NSDUH Report, 2003), it also reaches its peak during this age range for the general population, and it is associated with multiple negative consequences such as injury and participation in other risk behaviors (Hingson, Zha, & Weitzman, 2009; NSDUH Report, 2003; 2006). Excessive alcohol use is also associated with increased risk of alcohol disorder (Knight et al., 2002; Merline, O'Malley, Schulenberg, Bachman, & Johnston, 2004). Consequently, identification of vulnerable subgroups of the population has been an important research agenda in the effort to understand alcoholism risk and to develop targeted interventions.

Children with Attention-Deficit/Hyperactivity Disorder (ADHD) have been found, in metaanalytic reviews (Charach, Yeung, Climans, & Lillie, 2011; Lee, Humphreys, Flory, Liu, & Glass, 2011), to have increased risk of alcohol disorders (abuse or dependence) in adulthood. The magnitude of the risk is modest, with an aggregated odds ratio of 1.74 across 11 studies of children with ADHD followed longitudinally into adulthood (Lee et al., 2011). Prospective longitudinal studies have supported this association in clinical and epidemiologic samples (Wilens et al., 2011; Elkins et al., 2007; Knop et al., 2009; Sihvola et al., 2011) although exceptions exist (e.g., Fischer et al., 2002; Klein et al., 2012; Lambert & Hartsough, 1998). Studies of the link between ADHD and problem drinking have increased in recent years due to the overlap between the core symptoms of the disorder and the personality and temperament traits implicated in alcoholism etiology (Chassin, Colder, Hussong, & Sher, 2013; Molina & Pelham, in press; Sher, 1991; Tarter, Kirisci, Habeych, Reynolds, & Vanyukov, 2004; Tarter, Kirisci, Feske, & Vanyukov, 2007; Zucker, 2006). However, despite this strong conceptual link, longitudinal studies of childhood ADHD have tended to emphasize alcohol disorders instead of alcohol consumption indices, such as heavy drinking frequency that is known to predict later alcohol disorders (O'Neill, Parra, & Sher, 2001). This increased risk of continued use and later problems is especially relevant for young adults who, like many children with ADHD (Biederman et al., 2008; Kuriyan et al., 2013; Mannuzza, Klein, Bessler, Malloy & Hynes, 1997), do not attend college (Merline et al., 2004).

In one of the few studies to examine heavy drinking frequency as an outcome of childhood ADHD, we previously reported high rates of binge drinking for young adults in the Pittsburgh ADHD Longitudinal Study (PALS) regardless of their ADHD history (Molina, Pelham, Gnagy, Thompson, & Marshal, 2007). With an average sample-wide frequency of binge drinking 34 times in the past year (approximately two of every three weeks), we failed to find ADHD group differences in this outcome. However, binge drinking rates were significantly more frequent among the younger 15–17 year old adolescents in the PALS.

Many studies have shown long-term prognostic differences between adolescents who do and do not begin to drink heavily at a young age, suggesting increased risk of alcohol disorder for this population in addition to other negative outcomes (e.g., Chassin, Pitts, & Prost, 2002; McCarty et al., 2004). Thus, these findings from the initial follow-up interview of the PALS suggest that despite the typicality of heavy drinking by all adults in their early 20s, there may be meaningful differences in the ontogeny of heavy drinking between adults with and without ADHD histories, and these differences may be clinically and prognostically meaningful. In the current study we followed the PALS adolescents to the oldest underage drinking endpoint (age 20) to examine their rates of heavy drinking in early adulthood and to investigate potential mediating pathways that could lead to prognostic differences regarding long-term consequences of heavy drinking.

Delinquency Pathway

Alcoholism researchers have, for many years, recognized the important contribution made by "deviance proneness" to the development of alcohol and other substance disorders in adolescence (Donovan, Jessor, & Costa, 1999; Jessor & Jessor, 1977) and in adulthood (Sher, 1991). Tendencies toward delinquency, sometimes diagnosed categorically as conduct disorder, and excessive drinking may reflect common genetic influences (Edwards & Kendler, 2012; Slutske et al., 1998) as well as socialized beliefs, attitudes, and behaviors favorable to both types of outcomes (Chassin et al., 2013; Tarter et al., 2011). Dispositional tendencies related to ADHD, such as impulsivity, are also established risk factors for conduct problems and alcohol or other substance use in adolescents and young adults (Caspi, Moffitt, Newman, & Silva, 1996; Martel et al., 2009; Tarter et al., 2012). Similar to findings from non-referred samples (Brook, Brook, Zhang & Koppel, 2010), we recently found support for a "delinquency pathway" from childhood ADHD to late adolescent alcohol use frequency. Childhood ADHD increased risk of adolescent delinquency which in turn was associated with more frequent drinking at age 17 (Molina et al., 2012). These findings suggested the important possibility that, for children with ADHD, underage drinking in adulthood is part of the etiologic chain of events that characterizes antisocial alcoholism and includes a poor prognosis (Zucker, Donovan, Masten, Mattson, & Moss, 2008). If so, then a pathway from childhood ADHD to heavy drinking in adulthood should include an association with delinquent behaviors in adulthood and in adolescence, with a mediational pathway that includes both. We tested this possibility in the current study.

Social Impairment Pathways

It is well-established that children with ADHD have social difficulties beginning at a young age (McQuade & Hoza, 2008; Pelham & Bender, 1982) that are also present in adolescence and adulthood. For example, longitudinal studies of children with ADHD report fewer friends and more peer rejection in adolescence (Bagwell, Molina, Pelham, & Hoza, 2001), fewer friends in adulthood (Barkley, Fischer, Smallish, & Fletcher, 2006; Belendiuk, 2013; Weiss & Hechtman, 1993), and more parent-rated social problems in adulthood (Barkley et al., 2006). One study reported that boys with ADHD and parent-rated social disability have significantly higher rates of later alcohol disorders than boys with ADHD alone and comparison children without ADHD (Greene, Biederman, Sienna, Garcia-Jetton, & Faraone,

1997), although social disability included relationship difficulties with parents and siblings as well as peers. Given the high prevalence of peer problems for individuals with ADHD, more research is needed on the relation between this specific aspect of impairment and alcohol use. This need is particularly important given the prominence of peer social influence and selection processes in discussions of alcohol use in both adolescence (Curran, Stice, & Chassin, 1997; Hussong, 2002) and in early adulthood (Wood, Read, Mitchell, & Brand, 2004).

We recently found support for a "social deviance" pathway to adolescent alcohol use for children with ADHD (Molina et al., 2012). Specifically, adolescents with childhood ADHD had more problems in relationships with same-aged peers, as rated by parents, which were then associated with higher levels of delinquent behavior and, subsequently, higher levels of adolescent alcohol use. At the same time, social impairment appeared to "protect" against drinking in a different mediating pathway in which childhood ADHD predicted more social impairment which, in turn, predicted less drinking. We offered the speculation that these opposing pathways may reflect the heterogeneity of social impairment in ADHD (McQuade & Hoza, 2008). For example, social isolation, withdrawal, and/or social anxiety (Mikami, Ransone, & Calhoun, 2011) might decrease drinking in adolescence when alcohol use primarily occurs in social contexts (Pedersen, LaBrie, & Lac, 2008). However, childhood ADHD also predicts adolescent friendships characterized by less conventional activity involvement (Bagwell et al., 2001) and greater use and tolerance of alcohol and other drugs (Marshal, Molina, & Pelham, 2003) and these factors contribute more strongly to substance use in the presence of comorbid behavior problems (Marshal & Molina, 2006). It is unclear if these same associations would be found in adulthood given the widely accepted use of alcohol in the early 20s and the associated increased opportunities for drinking (Hussong, Jones, Stein, Baucom, & Boeding, 2011). To our knowledge, there are no studies of children with ADHD that have specifically examined peer social impairment longitudinally and in concert with delinquency.

The Current Study

In the current study, we tested delinquency and social impairment pathways to underage heavy drinking in early adulthood. Because of the longitudinal design of the PALS, we were able to test these pathways prospectively from childhood ADHD through adolescence into early adulthood (age 20). By doing so, we incorporated tests of stability in these mediating variables to determine whether social impairment and delinquency influences in adulthood extended from their earlier presentations in adolescence. Moreover, our analyses were carried out in the structural equation modeling framework which included several important features: 1) mediational paths involving social impairment were examined simultaneously with delinquency which allowed tests of their relative and unique effects; 2) prior alcohol use was controlled to test whether these mediational pathways contributed to heavy drinking in early adulthood over and beyond the contribution of drinking in the teen years; 3) post-secondary school attendance, known to be lower in the ADHD than in the non-ADHD group in this sample (Kuriyan et al., 2013) and in others (e.g., Barkley et al., 2006; Mannuzza et al., 1997), and known to be associated with increased heavy drinking in early adulthood (NSDUH Report, 2003), was controlled to examine influences on drinking above and

beyond this important contextual factor. The current study represented the first attempt to test these pathways longitudinally, to ascertain their contributions to heavy drinking in early adulthood, in a prospectively followed sample of children with and without ADHD.

Method

Participants

ADHD group—Participants with childhood ADHD were recruited from a pool of 516 study-eligible participants, diagnosed with DSM-III-R or DSM-IV ADHD in childhood and treated at the Attention Deficit Disorder clinic at the Western Psychiatric Institute and Clinic (WPIC) in Pittsburgh, PA, from 1987 to 1996 (APA, 1994). Of the 516, 493 were recontacted an average of 8.35 years later (SD = 2.79) to participate in annual interviews for the Pittsburgh ADHD Longitudinal Study (PALS). Of those contacted, 364 (70.5 %) enrolled in the follow-up study. At the first follow-up interview, participants with ADHD histories ranged in age from 11 to 28 with 99% falling between 11 and 25 years of age. They were admitted to the follow-up study on a rolling basis between the years 1999–2003 and completed their first follow-up interview immediately upon enrollment.

All adolescents and young adults with childhood ADHD 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 when indicated (Pelham, Fabiano, Gnagy, Greiner, & Hoza, 2005). Diagnostic information for the participants with ADHD histories was collected at initial referral to the clinic in childhood (baseline) using parent and teacher DSM-III-R and DSM-IV symptom ratings scales (DBD; Pelham, Gnagy, Greenslade, & Milich, 1992) and a semi-structured diagnostic interview administered to parents by a doctoral-level clinician. The interview consisted of the DSM-III-R or DSM-IV descriptors for ADHD, ODD, and CD with supplemental probe questions regarding situational and severity factors as well as functional impairment. It also included queries about other comorbidities to determine whether additional assessment was needed. Following DSM guidelines, diagnoses of ADHD, ODD (47%), and CD (36%) were made if a sufficient number of symptoms and clinically-significant impairment was endorsed (considering information from both parents and teachers) to result in diagnosis. Two doctoral-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 participants with ADHD histories was assessed in childhood (baseline) and included a full-scale IO < 80, a history of seizures, neurological problems, pervasive developmental disorder, schizophrenia, and/or other psychotic or organic mental disorders.

Participants in the follow-up study were compared with the eligible individuals who did not enroll in the follow-up study on demographic and diagnostic variables collected at baseline. Only one of 14 comparisons was statistically significant (p < .05): PALS participants had a slightly lower average CD symptom rating at baseline as indicated by a composite of parent and teacher ratings (participants M = 0.43, non-participants M = 0.53).

Non-ADHD group—Two hundred forty participants without ADHD were recruited for the PALS from the greater Pittsburgh community between 1999 and 2001. These individuals were recruited from several sources including pediatric practices in Allegheny County (40.8%), advertisements in local newspapers (27.5%), local universities and colleges (20.8%), and other methods (10.9%), such as Pittsburgh Public Schools and word of mouth. Non-ADHD participant recruitment lagged three months behind the ADHD group enrollment in order to facilitate efforts to obtain demographic similarity (discussed below). A telephone screening interview was administered to parents of potential non-ADHD participants to gather basic demographic characteristics, history of diagnosis or treatment for ADHD and other behavior problems, presence of exclusionary criteria as previously listed for the ADHD group, and a checklist of ADHD symptoms. Young adults (aged 18+) also provided self-report of ADHD symptoms. ADHD symptoms were counted as present if reported by either the parent or the young adult. Non-ADHD individuals meeting DSM-III-R criteria for ADHD, either currently or historically, were immediately excluded from study consideration. Based on parent report with the DBD, none of the non-ADHD group participants had conduct disorder in childhood.

If a potential non-ADHD participant passed the initial phone screen, senior research staff members met to determine whether he/she was demographically appropriate for the study. Each potential non-ADHD participant was examined on four demographic characteristics: 1) age, 2) gender, 3) race, and 4) parent-education level. A non-ADHD participant was deemed study- eligible if his/her enrollment increased the non-ADHD group's demographic similarity to the participants diagnosed with ADHD. At the end of the recruitment process, the two groups were equivalent on the four demographic variables noted above.

Subsample for the current study—Data were selected from the first 13 annual interviews of the PALS for any participants who were between 14 and 20 years old at any of the interviews. Thus, this subsample of the PALS, selected on the basis of age, primarily consists of the younger PALS participants followed longitudinally to age 20. This sampling resulted in 265 participants (117 Non-ADHD; 148 ADHD participants; see Table 1). There were no statistically significant differences between the ADHD and non-ADHD participants on gender, $\chi^2(1) = .02$, ns, racial/ethnic minority, $\chi^2(1) = .28$, ns, frequency of alcohol use in the past year at age 17, t(243) = -.04, ns, or time spent living at home during young adulthood, t(263) = -.63, ns. However, highest parental education was lower, $\chi^2(2) = 10.40$, p < .01, parental psychopathology was higher, t(263) = -6.46, p < .001, and proportion of years in post-secondary education was lower (for the young adults), t(263) = 6.45, p < .001in the ADHD than in the non-ADHD group. Younger teens were excluded from the analyses due to their smaller numbers and low rates of drinking (Molina et al., 2007). As in Molina and colleagues (2012), variables were modeled by age rather than by year of annual interview as recommended when age varies considerably within the sample in a given year or "wave" (Bollen & Curran, 2006).

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 post-baccalaureate 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 DHHS Certificate of Confidentiality.

Measures

Heavy drinking—A structured paper-and-pencil substance use questionnaire (SUQ; Molina et al., 2007), administered annually in the PALS, was used to assess alcohol use. The SUQ is an adaptation of existing measures, including the Health Behavior Questionnaire (Jessor, Donovan, & Costa, 1989) and the National Household Survey of Drug Abuse interview (NHSDA, 1992). Pertinent to the current study are the two SUQ items assessing frequency of binge drinking in the past 12 months ("In the past 12 months, how often did you drink five or more drinks when you were drinking?") and frequency of drunkenness in the past 12 months ("In the past 12 months, how often have you gotten drunk or very, very high on alcohol?", with an average correlation between these two items of .92, p < .001. Participant responses ranged from 0 "not at all" to 11 "several times a day." Responses to these two items were averaged at age 20. Heavy drinking of either type occurred at least once/week for 19% (50/265) of the sample at age 20.

Social functioning—Parents annually provided ratings of the target individual'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 psychometric properties for children, adolescents, and young adults (Fabiano et al., 2006; Sibley et al., 2012a/2012b). 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." As published in Molina et al. (2012), average social impairment in adolescence ranged from .22 to 5.13. Social impairment at age 20 was modeled as a manifest variable, with scores ranging from 0 to 6 for the full sample.

Delinquent behavior—Delinquency data were collected annually in the PALS 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, young adults, and their parents and inquired about past year occurrence of 37 delinquent acts. After the age of 18, items regarding status offenses (e.g., school truancy) were no longer included. An item regarding public drunkenness was also removed due to overlap with drinking behavior outcomes. The full SRD was not administered to adolescents under the age of 18 at the first annual follow-up; for these participants, 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 self report; and DBD, parent, teacher, and self report. A proportion score was calculated for each participant 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. Delinquency at age 20 was modeled as a manifest variable, with scores ranging from 0 to .47 for the full sample.

Covariates—Seven covariates were tested for inclusion in the analyses. These included frequency of alcohol use at age 17, gender, race/ethnicity, socioeconomic advantage, parental psychopathology, and two variables about education and living arrangements between ages 18 and 20. Post-secondary education between these ages was calculated as the proportion of years that the young adult reported being enrolled in any form of post-secondary education on a questionnaire assessing educational experiences since the last interview (Kuriyan et al., 2013); living arrangement was the proportion of these years that the young adult reported living at home when completing an annual demographics questionnaire. Socioeconomic advantage, parent psychopathology (for construction of these variables see Table 1; also Molina et al., 2012), and gender were tested in initial models but they were subsequently excluded due to their lack of statistically significant effects.

Analytic Overview

We first tested the association between childhood ADHD (no=0; yes=1) and heavy drinking at age 20. Multiple regression was used to test whether childhood ADHD contributed to age 20 heavy drinking frequency after inclusion of the covariates described above.

Our primary hypotheses regarding social impairment and delinquency pathways to early adult drinking were tested in a structural equation model using Mplus 7.0 (Muthén & Muthén, 2012). Mediational pathways between childhood ADHD and heavy alcohol use at age 20 through delinquency, in adolescence and at age 20, and social impairment, in adolescence and at age 20, were examined in the model. This allowed a test of whether childhood ADHD was associated with heavy alcohol use at age 20 through continued delinquency and social impairment from adolescence to young adulthood. A path from adolescent social impairment to delinquency at age 20 was also estimated to examine the potential contribution of social impairment on later delinquency above and beyond prior delinquency. As in Molina et al. (2012), adolescent delinquency and social impairment were modeled as intercept-only latent factors to estimate the stable overall level of each construct from the repeated measures between ages 14 and 17. Delinquency and social impairment at age 20 were modeled as manifest variables to understand the co-occurrence of these factors at the peak of underage drinking. Including both adolescent and adult measures of these variables also allowed for estimating age specific unique contributions on alcohol use outcome (e.g., unique contribution of adolescent social impairment over and beyond the concurrent impairment at age 20. All covariates were included in the initial model and incrementally removed based on lack of statistically significant effects on age 20 heavy drinking. Significance tests for mediated effects were conducted using the delta method implemented in Mplus, where mediated effects are obtained by the product of the path coefficients between the variables in the mediational pathways and the statistical

significance is tested under the normality assumption of mediated effects. Following recent recommendations (e.g., MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002; Rucker, Preacher, Tormala, & Petty, 2011) and a growing body of research, our decision to test mediation pathways was not contingent on demonstration of a statistically significant relation between childhood ADHD and age 20 alcohol use (especially because of anticipated opposite directions of the relations by social impairment and delinquency).

Model fit was determined using conventional fit statistics (chi-squared, root mean square error of approximation [RMSEA], and comparative fitness index [CFI]). Maximum likelihood estimation with robust standard errors estimation was utilized to take into account the non-normality of the heavy alcohol use variable.

Results

Preliminary Results

Consistent with our previous report for the PALS participants who were already adults at their first annual interview (Molina et al., 2007), the regression results indicated no statistically significant relation between childhood ADHD and age 20 heavy drinking, $\beta = -0.05$, p = 0.44. Among the covariates, more frequent age 17 drinking, $\beta = 0.55$, p < 0.001, less living at home, $\beta = -0.11$, p = 0.04, and more post-secondary education, $\beta = 0.18$, p = 0.002 predicted more frequent heavy drinking at age 20. Predictions from the remaining covariates failed to attain statistical significance. The overall model R^2 was 0.40.

Mediation by Social Impairment and Delinquency

The results of the mediation model are shown in Figure 1. The model fit the data adequately, $\chi^2(21) = 58.71$, p < .001; RMSEA = .06; CFI = .92. As previously reported in Molina et al. (2012), childhood ADHD significantly predicted adolescent social impairment, $\beta = .57$, p < .001, and adolescent delinquency, $\beta = .26$, p < .001, and these indicators of adolescent functioning were significantly correlated, r = .24, p < .001 after controlling for childhood ADHD. Childhood ADHD also significantly predicted social impairment at age 20, $\beta = .24$, p < .001 over and above adolescent social impairment, but not delinquency at age 20, $\beta = -$. 02, p = .77 after controlling for adolescent delinquency. Adolescent social impairment predicted age 20 social impairment, β = .44, p < .001, and less frequent age 20 heavy alcohol use, $\beta = -.14$, p = .03. Adolescent delinquency predicted age 20 delinquency, $\beta = .21$, p = .2103, but not age 20 heavy alcohol use, $\beta = .10$, p = .29. Age 20 delinquency and social impairment were significantly correlated, r = .16, p = .02, after controlling for childhood ADHD and adolescent delinquency and social impairment. Finally, age 20 delinquency was associated with age 20 heavy alcohol use, $\beta = .27$, p < .001, but age 20 social impairment was not associated with age 20 heavy alcohol use, $\beta = -.03$, p = .67. Forty-two percent of the variance in heavy drinking was explained.

Two statistically significant mediational pathways resulted. In one, childhood ADHD predicted more social impairment in adolescence which in turn predicted less frequent heavy alcohol use at age 20, β = -.08, p = .04, 95% CI = -.16 to -.01. In the other, childhood ADHD predicted more delinquency in adolescence which in turn predicted more

delinquency at age 20. Age 20 delinquency was then associated with more frequent heavy alcohol use at age 20. This indirect effect was also statistically significant, β = .02, p = .04, 95% CI = .001 to .03.

Among the covariates, alcohol use at age 17 positively predicted heavy drinking at age 20, β = .43, p < .001, over and above the other variables in the model. Caucasian participants reported more frequent heavy alcohol use at age 20 than participants of other racial/ethnic backgrounds, β = .11, p = .03. Participants who reported living at home a greater proportion of ages 18 to 20 reported less frequent heavy drinking at age 20, β = -.13, p = .01. Finally, a higher proportion of years in school between ages 18 and 20 predicted more frequent heavy drinking at age 20, β = .13, p < .01.

Discussion

We tested social impairment, social deviance, and delinquency pathways to heavy drinking in early adulthood in a longitudinally followed sample of children with, and without, ADHD. We did not find ADHD group differences in frequency of heavy drinking in early adulthood and thus replicated our prior findings with the older subset of the PALS participants (Molina et al., 2007). However, indirect and opposing pathways to heavy drinking from childhood ADHD were supported. One pathway, largely expected from the existing literature (e.g., Brook et al., 2010; Knop et al., 2009; Molina et al., 2012), indicated progression to heavy drinking following from adolescent and, subsequently, young adult delinquent behavior. A second pathway suggested a suppression effect (Mackinnon, Cheong, & Pirlott, 2012; Rucker et al., 2011) such that childhood ADHD led to adolescent social impairment which led to *decreased* heavy drinking. Age 20 social impairment and heavy drinking were unrelated, suggesting some diminution of the social impairment protective effect in adulthood. These findings provide new information regarding potentially important yet opposing impairment-related pathways to heavy drinking in early adulthood for children with ADHD.

Our replicated finding that childhood ADHD does not predict heavy drinking frequency in early adulthood appears to be at odds with some prior findings of increased alcohol use in adolescence by children with ADHD (Barkley, Fischer, Edelbrock, & Smallish, 1990; Molina & Pelham, 2003; Molina et al., 2007; Molina et al., 2013) or an earlier age of first use (Elkins, McGue, & Iacono, 2007; Milberger, Biederman, Faraone, Wilens, & Chu, 1997; Molina et al., 2013). These earlier signs of increased drinking are well-established risk factors for excessive and problematic drinking in adulthood (Chassin et al., 2002; McCarty et al., 2004). Thus, it is somewhat surprising that we have twice failed to identify ADHD group differences in heavy drinking in early adulthood. We suspect that these developmentally specific findings, which parallel to some extent the overall modest ADHD-related risk of alcohol disorder described in the literature (Lee et al., 2011), reflect important heterogeneity in the ADHD clinical presentation, associated features, and course over time that translate to risk and resilience in this population.

Our findings confirmed the importance of deviance-proneness (Donovan et al., 1999; Sher, 1991) for ADHD-related drinking vulnerability by using a developmental model that

considered the presence of delinquent behavior through adolescence and concurrently in adulthood. Rather than controlling for conduct disorder diagnosis, as analyzed in many studies, our approach modeled the dimensional and adolescent/adult form of this variable to more powerfully test its mediating role (e.g., Brook et al., 2010). Although co-occurring conduct problems/CD are widely established as important for the development of alcohol use vulnerability in ADHD (Edwards & Kendler, 2012; Flory & Lynam, 2003; Molina & Pelham, 2003; Molina et al., 2007; Tuithof, ten Have, van den Brink, Vollebergh, & Graaf, 2012), prior studies have often failed to consider the developmental unfolding of these problems in the ADHD population by controlling for concurrent conduct disorder rather than studying its contribution developmentally. The former approach also risks overcontrolling for variance common to the two disorders (e.g., impulsivity) that contributes to alcoholism vulnerability (Derefinko & Pelham, in press). Along these lines, we previously found inconsistent prediction to alcohol outcomes from childhood CD in the current sample (Molina et al., 2007), presumably due to the tendency for delinquent behaviors to increase with age (Sibley et al., 2011; Barnes, Welte, & Hoffman, 2002). Others have reported a similar cascade of unfolding risks from disinhibited temperament within a sample at high risk by virtue of parental alcoholism and antisociality (Martel et al., 2009). In common among these types of high risk samples are undercontrolled temperament profiles in childhood that, in interaction with other psychosocial and environmental factors, lead to deviant behaviors that include drinking (Chassin et al., 2013; Sher, 1991). Our findings indicate that this pathway is still applicable in early adulthood, for a subset of devianceprone children with ADHD, even when heavy drinking becomes highly normative (SAMHSA, 2012) and may only be developmentally limited for some (perhaps those without ADHD histories) (Sher, 1991; Zucker, 2006). We cannot rule out the likely possibility of bidirectional associations between delinquency and heavy drinking at age 20 (e.g., Barnes et al., 2002), but the inclusion of adolescent delinquency in the mediational pathway is strong evidence that it partially contributes prospectively.

Interpersonal difficulties are widespread among people affected by ADHD (McQuade Hoza, 2008), and our findings with regard to alcohol suggest that accounting for them, in tandem with delinquency pathways, may help to explain the heterogeneity of the ADHD-related alcoholism risk. We found suppression of heavy drinking risk from social impairment in adolescence: children with ADHD were significantly more likely to be rated by their mothers as having relationship problems with other same-aged people, and this social difficulty predicted less frequent heavy drinking at age 20, above and beyond age 17 drinking frequency. Thus, these findings extended our report of the same result in adolescence (Molina et al., 2012) prospectively to early adulthood when drinking contexts have changed. Importantly, this protective effect disappeared when delinquency was dropped from the model in a post-hoc analysis which speaks directly to the heterogeneity of social functioning in ADHD. Specific aspects of interpersonal tendencies related to delinquency may increase, and others (e.g., social isolation; withdrawal; social anxiety) may decrease, heavy drinking risk. Models of ADHD risk may be aided by separating these tendencies into those that result in greater, versus lesser, affiliation with peers who drink. This is especially the case given the largely socially mediated context of alcohol consumption in early adulthood (Pedersen et al., 2008; Wood et al., 2004). It is additionally

quite interesting that, despite the significant stability of social impairment from adolescence to adulthood, that adult social impairment was not correlated with heavy drinking frequency. Other studies have shown that heavy drinking in early adulthood is largely predicted by adolescent and not concurrent risk factors (Sher & Rutledge, 2007). However, we offer an additional interpretation based on our findings that heavy drinking was significantly associated with post-secondary school attendance and living away from home. These factors, known from other samples to be strongly associated with heavy drinking in early adulthood (NSDUH Report, 2003), may simply be more important. Alternatively, our measurement of social impairment, despite its apparent stability and predictive utility, may need to draw from additional reporters in adulthood. It is based on a single parent-reported item that may fail to assess nuances of social functioning important for heavy drinking, such as number of heavy drinking peers and time spent together, and quality or reciprocity of these relationships.

Our model was constrained to include only social functioning and delinquency because we were specifically interested in the longitudinal effects of these mediators that are so commonly associated with ADHD. We previously found support for an "academic deviance" pathway to adolescent drinking in which lower GPAs in the ADHD group were related to delinquency and subsequently alcohol use (Molina et al., 2012). Developmental substance use theorists have long suggested that failure to engage in activities that promote future goal attainment, such as academic success, create vulnerability (Brook, Brook, Gordon, Whiteman, & Cohen, 1990; Jessor & Jessor, 1977). As expected, the non-ADHD group was more likely to attend post-secondary education, as we previously demonstrated for the older PALS participants (Kuriyan et al., 2013), indicating that lower academic achievement continues into early adulthood. Given findings that alcohol problems are more likely to follow from heavy drinking in non-college adults (Merline et al., 2004), future research would benefit from examining academic trajectories that extend from childhood through adolescence into adulthood. Such analyses may uncover potential benefits of targeting academic performance throughout the high risk periods of alcohol entry for individuals with ADHD. Our findings may also be affected by other unmeasured mediators and moderators such as aspects of parental involvement that are known to exert protracted effects on drinking even into adulthood (Guo, Hawkins, Hill, & Abbott, 2001; Raudino, Fergusson, & Horwood, 2013).

Although the majority of children with ADHD continue to experience statistically deviant (non-normative) levels of symptomatology into adulthood (Barkley, Fischer, Smallish, & Fletcher, 2002; Sibley et al., 2012b), heterogeneity exists (Barkley, Murphy, & Fischer, 2008; Sibley et al., 2012b), and several studies have shown that symptom persistence contributes to more alcohol use (Barkley et al., 2008; Chang et al., 2012; Molina & Pelham, 2003; Molina et al., 2012). For pragmatic purposes, our model focused on two key domains of impairment for ADHD, but our inclusion of adults for whom ADHD symptoms had dissipated may have partially diluted the ADHD-drinking association (although many of these children were probably captured in the delinquency pathway). We also did not separately model the two dimensions of ADHD, inattention and impulsivity-hyperactivity, because our interest was in testing pathways from childhood ADHD to underage heavy drinking rather than explaining variability within the ADHD group. Future research should

extend the growing body of literature investigating this distinction within ADHD that, measured as subtypes of ADHD is highly unstable over time (Wilcutt et al., 2012), but which might be effectively studied longitudinally with repeated measures (Molina & Pelham, in press). This approach would also incorporate questions of persistence/desistance. We did not include medication treatment commonly used for children with ADHD. In prior analyses, we found no association between stimulant medication (by far the most common psychoactive drug used), and substance use (Pelham, 2008). A recent meta- analysis (Humphreys, Eng, & Lee, 2013) and comprehensive tests of the association with a large multi-site sample (Molina et al., 2013) also failed to detect associations. Finally, our sample was predominantly male and we were therefore unable to test whether our findings were equally applicable to females.

In summary, our findings provide, with prospectively gathered data for children with ADHD, evidence of two opposing pathways to heavy drinking frequency in early adulthood. Evidence of a delinquency pathway suggests the potential importance of forestalling entry into adolescent peer groups that socialize around delinquent behavior and alcohol use. Refining the measurement of social functioning to identify specific interpersonal tendencies that promote affiliation with drinking peers, known to be elevated for adolescents with ADHD histories (Marshal et al., 2003), may clarify these associations. Integration of evidence-based treatments for ADHD (Pelham & Fabiano, 2008) and adolescent substance abuse (Waldron & Turner, 2008) may hold promise in this regard. More detailed measurement of social functioning, to include collateral ratings by partners and adult friends, and refinement of the various constructs inherent in social functioning, may clarify targets of treatment in adulthood. Finally, future model testing will benefit from consideration of the wide range of additional psychosocial and environmental variables that emerge as potentially important to consider when literatures on the etiology of ADHD and substance abuse are juxtaposed (Molina & Pelham, in press).

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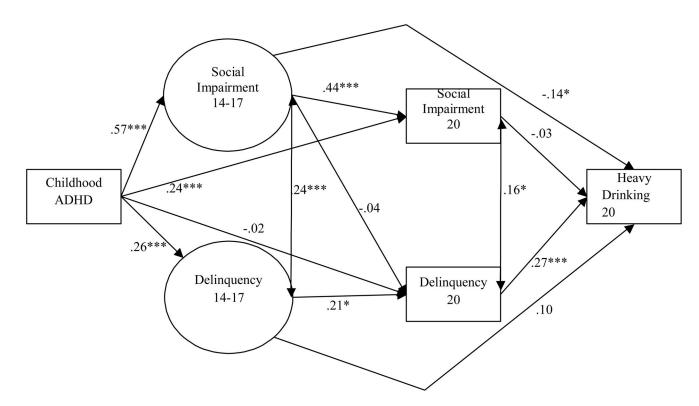


Figure 1. Social impairment and delinquency as mediators of childhood ADHD and young adult heavy alcohol use. The reported path coefficients are standardized. Alcohol use at age 17, race/ethnicity, proportion of time living at home from 18 to 20, and proportion of time enrolled in school from 18 to 20 were controlled for in the model.

 $\chi^2 = 58.71$, df = 21, p < .001

RMSEA = .06, CFI = .92

Coefficients are standardized.

 $^{+}p < .10, *p < .05, **p < .01, ***p < .001$

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Descriptive statistics by ADHD groups

	Non-ADHD $(n = 117)$	ADHD $(n = 148)$
Gender, Male	108 (92.3)	136 (91.9)
Race/Ethnicity		
Caucasian	96 (82.1)	116 (78.4)
Other	21 (17.9)	30 (20.3)
Socioeconomic Advantage	Advantage	
Low	2 (1.7)	9 (6.1)
Medium	28 (23.9)	56 (37.8)
High	87 (74.4)	83 (56.1)
Parental Psychopathology	pathology	
0	62 (53.0)	29 (19.6)
1	29 (24.8)	41 (27.7)
2	15 (12.8)	33 (22.3)
3+	11 (9.4)	45 (30.4)
Years Living at l	Years Living at Home at Ages 18–20	
0	11 (9.4)	8 (5.4)
1	26 (22.2)	37 (25.0)
2	33 (28.2)	40 (27.0)
3	47 (40.2)	63 (42.6)
Years Enrolled in	Years Enrolled in School at Ages 18–20	
0	2 (1.7)	17 (11.5)
1	6 (7.7)	37 (25.0)
2	26 (22.2)	44 (29.7)
	80 (68.4)	50 (33.8)

Mediating Variables (Means and Standard Deviations)

	Non-ADHD $(n = 117)$	ADHD $(n = 148)$
Adolescent Social Impairment	.18 (.60)	2.23 (1.68)
Age 20 Social Impairment	.14 (.52)	2.20 (1.88)

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Deviations)
Standard
Means and
Variables (
Mediating

Non-ADHD $(n = 117)$ ADHD $(n = 148)$.03 (.04)	.04 (.06)	
N	Adolescent Delinquency	Age 20 Delinquency	

Alcohol Variables (Means and Standard Deviations)

	Non-ADHD $(n = 117)$	ADHD $(n = 148)$
Frequency of Alcohol Use in the Past 12 Months at Age 17	1.68 (1.91)	1.69 (2.42)
Percentage Reporting Weekly Drinking at Age 17	4.30	9.50
Frequency of Heavy Alcohol Use at Age 20	3.37 (2.54)	2.48 (2.60)
Percentage Reporting Weekly Heavy Drinking at Age 20	21.40	14.30

1997, 1998; Selzer et al., 1975). For social impairment, 0 = no problem and 6 = extreme problem. For delinquency, score = proportion of delinquency items endorsed. For frequency of alcohol use at age 17 Notes. For socioeconomic advantage, low = single parent with high school education or less education, medium = single parent with more than high school education or married parent with high school or less education, high = married parent with more than high school education. For parent psychopathology, sum score of absence (0) or presence (1) of mother's and father's alcohol use disorder, antisocial personality disorder, or depression based on parent report on the Structured Clinical Interview for the DSM-IV Axis I and Axis II disorders and Short Michigan Alcoholism Screening Test (First et al., and heavy alcohol use at age 20, 0 = not at all, 1 = 1-3 times, 2 = 4-7 times, 3 = 8-11 times, 4 = once a month, ... 11 = several times a day.