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Antisocial Alcoholism in Parents of Adolescents and Young Adults with Childhood ADHD

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Abstract

Objective—Test the hypothesis that alcoholism, including antisocial alcoholism, is more prevalent among mothers and fathers of children with versus without ADHD.

Method—Mothers (312 ADHD group, 235 non-ADHD group) and fathers (291 ADHD group, 227 non-ADHD group) in the Pittsburgh ADHD Longitudinal Study were interviewed along with their adolescent and young adult offspring.

Results—Maternal and paternal alcoholism, with and without comorbid antisociality, was more prevalent in the ADHD group. Paternal alcoholism without antisociality was only marginally higher for probands after controlling for paternal ADHD. Offspring conduct disorder comorbidity was associated with parental antisociality but not parental antisocial alcoholism.

Conclusions—Our findings that 44% of proband fathers and 25% of proband mothers experienced alcohol problems with or without antisociality, are further evidence of increased alcoholism prevalence in families affected by ADHD. Maternal alcoholism and antisociality are prominent contributors to this family-level vulnerability. These findings indicate the need to assess long-term offspring outcomes as a function of parental alcohol and externalizing comorbidities, and perhaps other indicators of parental alcoholism phenotype, as familial vulnerability unfolds across development.

Several decades of research have identified a robust relation between parent alcoholism and offspring externalizing behavior (Pelham & Lang, 1993; Sher, 1991; Waldron, Martin, & Heath, 2009; Zucker, Heitzeg, & Nigg, 2011). Genetic, and to a lesser extent environmental (e.g., child-rearing, peer socialization), factors have been supported as contributory (Haber, Jacob, & Heath, 2005; Hicks, Foster, Iacono, and McGue, 2013; Kendler, Ohlsson, Sundquist, & Sundquist, 2016). Attention-Deficit/Hyperactivity Disorder (ADHD), which

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includes the highly heritable core symptom of impulsivity and is an early precursor to conduct and alcohol-related problems (Barkley, 2015; Lee et al., 2011), should be associated with parental alcoholism. However, a limited number of studies have tested the specific relation between parental alcoholism and offspring Attention Deficit/Hyperactivity Disorder (ADHD). Using Swedish population-based registries of children and adults with ADHD, Skoglunch and colleagues found that 6.5% had parents with alcohol use disorders versus only three percent of controls (Skoglunch, Chen, Franck, Lichtenstein, & Larsson, 2015). Rates of parental alcohol problems based on clinic (Barkley, Fischer, Edelbrock, & Smallish, 1990; Biederman, Faroane, Keenan, Benjamin, Krifcher, Moore, & Tsuang,1992) and community (August, Realmuto, Tamara, & Hektner, 1999) samples are higher. Barkley and colleagues (1990) found that 34.2% of biological fathers of children with ADHD had alcohol abuse histories, compared with 14.1% of controls. Biederman and colleagues (1992) reported higher rates of alcohol dependence in first-degree relatives of children with ADHD (18%) than in controls (11%). August and colleagues (1999) found that 65%–85% of children with ADHD had a parent with a DSM-IV alcoholism diagnosis as compared to 48% of controls. However, despite these reports, other studies have failed to detect higher levels of alcoholism in biological relatives of children clinic-diagnosed with ADHD (Milberger, Faraone, Biederman, Chu, & Wilens, 1998; Biederman et al., 2008; Cadoret & Stewart, 1991; Lahey, Piacentini, McBurnett, Stone, Hartdagen, & Hynd, 1988).

Little research on this topic (the association between childhood ADHD and parental alcoholism) has directly considered the well-known heterogeneity of alcoholism and its impact on the association with ADHD. Alcoholism is variable on multiple dimensions including age of onset, severity, chronicity, and mental health comorbidities (Babor, Hesselbrock, Meyer, & Shoemaker, 1994). Such heterogeneity may account for the variable expression of behavior disorders among offspring, as not all children of alcoholic parents develop behavior problems. Some researchers suggest that far less than half of children of alcoholics (COAs) develop these difficulties which might be connected to the heterogeneity of parent alcoholism (Sher, 1991) and concomitant psychobiological underpinnings (Zucker et al., 2011). Thus, the extent to which alcoholism has a deleterious effect on offspring may not be determined by parent's alcohol use disorder (AUD) per se, but by the pattern of risk factors, including parental alcoholism comorbidity, that are associated with onset and severity of course.

Antisocial Personality Disorder (ASPD) is one of the most common phenotypic comorbidities of alcoholism to have been associated with early onset and a chronic severe course. ASPD is part of the externalizing behavior continuum and is associated with elevated rates of offspring psychopathology including ADHD (Herndon & Iacono, 2005). Beauchaine and McNulty (2013) argue that highly heritable trait impulsivity, a core symptom of ADHD, may be the earliest visible indicator of risk for conduct problems, ASPD, and substance abuse. In line with this thinking, the combination of parent alcoholism and ASPD (antisocial alcoholism) is a particularly strong indicator of offspring risk for such maladaptive outcomes that may be due to greater severity of parental psychopathology, polygenetic risk for undercontrolled behaviors, and an especially disadvantageous rearing environment for the child. Zucker, Ellis, and Fitzgerald (1993) found that antisocial alcoholism was associated with earlier onset, higher overall levels of comorbid alcohol symptoms, and a wider variety

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of comorbid alcohol symptoms in alcoholic fathers, and suggest that aggregating parental risk in this way has increased prognostic value for offspring behavior problems. In their study, sons of antisocial alcoholics were more likely to develop behavior problems than were sons of non-antisocial alcoholics and sons of non-alcoholics (Wong, Zucker, Puttler, & Fitzgerald, 1999). Studies of children with ADHD has corroborated the association between parent ASPD and child ADHD, showing higher levels of familial ASPD for children with than for children without ADHD (Biederman et al., 1992) and higher rates of parent antisocial behaviors such as police contacts or arrests, fights and assaults for children with than for children without ADHD (Barkley et al., 1990). The extent to which alcoholism, which is widely known to co-occur with ASPD in adulthood, was accounting for these findings was not reported. Determining whether parental alcohol problems occur at higher rates in families affected by ADHD, relative to the co-occurrence of antisociality, will aide future research efforts focused on distinct causal mechanisms associated with these phenotypes.

To our knowledge, one study has tested the specific relation between childhood ADHD diagnosis and parent antisocial-alcoholism. Using the well-known multi-site Collaborative Study on Genetics of Alcoholism sample, offspring ADHD was examined for its association with alcoholism among parents treated for alcohol dependence (Kuperman, Schlosser, Lidral, & Reich, 1999). Compared with children of non-alcoholics (5.9%), risk for offspring ADHD was significantly higher in children of alcoholic only families (13.2%) and higher still in children of alcoholic parents with ASPD (19.0%). However, only parental alcoholism (not comorbid antisociality) was associated with offspring ADHD when tested in a multivariate model. Thus, these initial data, confined to the study of treatment-seeking alcoholic adults, suggest an association between parental alcoholism and offspring ADHD but leave open the question of the importance of comorbid antisociality among parents. More recently, Chronis and her colleagues found that paternal antisociality and not recent drunkenness was associated with child conduct problems in a sample of children with ADHD (LeMoine, Romirowsky, Woods, & Chronis-Tuscano, 2015). Father drunkenness, antisociality, and ADHD symptoms were correlated, but without a comparison sample of children without ADHD, their joint prediction of childhood ADHD could not be evaluated.

Another important question that has been minimally evaluated is whether or not the gender of the alcoholic parent explains part of the variability in the association between parent alcoholism and offspring disorders such as ADHD. There is evidence that maternal alcoholism is associated with offspring externalizing disorders such as ADHD (e.g., Knopik et al., 2006) and substance use disorders (Chassin, Curran, Hussong, & Colder, 1996). Knopik and her colleagues (2006, 2009) used genetically-informed twin study designs to assess the association between parental alcohol dependence and offspring ADHD using children-of-female- (Knopik et al., 2006) and male-twin designs (Knopik et al., 2009). Increased risk of offspring ADHD associated with maternal alcohol dependence was supported in both studies, but only the latter controlled for maternal ADHD which was a partial confounder (Knopik et al., 2009). In a smaller community sample of children in greater Pittsburgh, Pennsylvania (not selected for psychopathology), alcoholism in first and second degree biological relatives was associated with male, but not female, offspring externalizing behavior (Molina, Donovan, & Belendiuk, 2010); parental ADHD was not

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controlled but mother's general mental health and not alcohol use was a statistically significant mediator. Others have concluded that the literature is mixed with respect to gender differentiated pathways to alcoholism (Zucker et al., 2011) and the complexity of these associations and need for additional research has been nicely detailed (Knopik et al., 2009). Finally, it is unclear whether maternal *antisocial* alcoholism per se is responsible for offspring behavior problems. Thus, the primary goal of this paper is to examine the associations between maternal and paternal alcoholism and antisocial alcoholism with offspring ADHD diagnosis. We will control for parental ADHD to understand the extent to which these familial associations include, or are independent of, ADHD among the parents.

The Pittsburgh ADHD Longitudinal Study (PALS) is a longitudinal follow-up study of elementary school-aged children comprehensively diagnosed with DSM-III-R or DSM-IV ADHD and followed prospectively through adolescence and early adulthood (e.g., Molina, Pelham, Gnagy, Thompson, & Marshal, 2007; Molina, Walther, Cheong, Pedersen, Gnagy, & Pelham, 2014). The PALS includes interviews with parents and provides an opportunity to test the hypothesis that antisocial alcoholism accounts for a significant proportion of alcoholism prevalence among parents of children diagnosed with ADHD. The study, having collected interview data with mothers and with fathers, also provides the opportunity to test the hypothesis that this association occurs among mothers, as well as fathers of children with and without ADHD, and that proband offspring with conduct problems (ADHD plus CD) will have the highest rates of parental antisocial alcoholism.

Method

Overview

The PALS includes 364 children diagnosed with DSM-III-R or DSM-IV ADHD and followed longitudinally through adolescence into early adulthood (probands) and 240 demographically similar individuals without ADHD recruited and followed for comparison purposes (controls). The PALS methods are described briefly and the reader is referred elsewhere for additional details (Molina et al., 2007).

Participants

ADHD group—Children with ADHD were diagnosed at the ADD Clinic at the Western Psychiatric Institute and Clinic in Pittsburgh, PA during the years 1987–1996. They were eligible for longitudinal follow-up on the basis of 1) their ADHD diagnosis in childhood, 2) participation in an 8-week summer treatment program for children with ADHD (Pelham & Hoza, 1996), and 3) additional inclusion/exclusion criteria described below. Diagnostic information was collected in childhood using several sources, including the parent and teacher DBD Rating Scale that assesses the DSM-III-R and DSM-IV symptoms of the disruptive behavior disorders (Pelham, Gnagy, Greenslade, & Milich, 1992). Parents completed a semi-structured diagnostic interview with PhD level clinicians consisting of the DSM-III-R or DSM-IV descriptors for ADHD, ODD and CD, with supplemental probe questions regarding situational and severity factors. Following DSM guidelines, diagnoses were made if a sufficient number of symptoms was endorsed (considering information from both parents and teachers) to result in diagnosis. At least two Ph.D.-level clinicians

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independently reviewed all ratings and interviews to confirm the DSM diagnoses. Exclusionary criteria also assessed in childhood included: a full scale IQ less than 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. Mean age at initial evaluation was 9.40 years, S.D. = 2.27 years; ADHD group participants were contacted for follow-up an average of 8.35 years later, S.D. = 2.79. Participation rate was 70.5% (364/516) and only 1/14 comparisons between participants and nonparticipants was statistically significant (mean CD symptom rating, Cohen's d=.30). Data for the current study were taken from the first follow-up visit when probands averaged 17.74 years of age (SD=3.38). Most were boys (89.6%) and Caucasian (84.6%). Most minorities were African American (11.0% of probands).

Non-ADHD group—Participants without ADHD were recruited from the Pittsburgh area for their demographic similarity to the ADHD group at follow-up (age within one year, sex, race, parental education). They were recruited on a rolling basis to create ADHD/non-ADHD groups with equal proportions of each demographic characteristic. Participants without ADHD were recruited via several large pediatric practices in Allegheny County (40.8%), advertisements in local newspapers and the university hospital staff newsletter (27.5%), local universities and colleges (20.8%), and other methods (e.g., word of mouth). A telephone screening interview administered to parents gathered basic demographic characteristics, history of diagnosis and treatment for ADHD and other behavior problems, presence of exclusionary criteria as previously listed for probands, and a checklist of ADHD symptoms. Individuals who met DSM-III-R criteria for ADHD -- either currently or historically -- were excluded. The two groups did not differ in age, sex, race, or parent education. A higher percentage of ADHD than non-ADHD group participants were from single parent households, 33.2% vs. 23.6%, $p < .05$, ADHD group parents' incomes were lower, M = $63K vs. $76K, $p < .01$, and more probands than controls had been adopted, 8.0% vs. 0.4%, $p < .01$.

Subsample for the current study—The primary goal of the current study was to compare rates of biological parent alcoholism and antisocial alcoholism between the ADHD and nonADHD groups. Thus, a subsample of the larger PALS sample was used for the current study to ensure that alcoholism and ASPD information described biological parents only. After excluding families for which no biological parent data were available and families for whom only partial data were available, the subsample size for analysis included 312 ADHD group mothers, 291 ADHD group fathers, 235 non-ADHD group mothers, and 227 non-ADHD group fathers. These numbers reflected 90% (n=547/604) of the mothers and 86% (n=518/604) of the fathers of participants in the larger PALS sample. Average ages were 45.6 (SD = 5.7) for ADHD group mothers, 49.5 (6.6) for ADHD group fathers, 46.3 (SD = 5.4) for nonADHD group mothers, and 49.4 (SD = 6.6) for nonADHD group fathers.

Interview Procedures

Follow-up interviews with parents and offspring were completed in the ADD Program offices by post-baccalaureate research staff. Interviewers were not blind to recruitment source (i.e., presence or absence of ADHD in childhood), but they were trained to avoid bias

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in data collection. Informed consent was obtained and all participants were assured confidentiality of all disclosed material except in cases of impending danger or harm to self or others (reinforced with a DHHS Certificate of Confidentiality).

Measures

Parent alcoholism—A lifetime history of alcohol problems in the biological parents was assessed using the Structured Clinical Interview for the DSM-IV with parents (SCID-IV; First et al., 1998). This interview, which has excellent reliability and validity and reflected the version of the DSM in use at the time of data collection, yielded lifetime diagnoses of alcohol abuse or dependence for mothers and for fathers. In the absence of direct interview, alcohol problems of the non-interviewed biological parent were measured with spousal report on the Short Michigan Alcoholism Screening Test (SMAST; Selzer, Vinokur, & van Rooijen, 1975). For the SMAST, alcoholism was considered present upon endorsement of three or more alcohol-related problems or one of three diagnostic items indicating receipt of treatment or help for drinking (Selzer et al., 1975).

Parent's antisocial personality disorder (ASPD)—ASPD was assessed using the SCID-II ASPD Module (SCID-II; Spitzer, Williams, & Gibbon, 1987) administered to biological parents about themselves and about the other biological parent. ASPD diagnoses were assigned if either biological parent's report met diagnostic criteria. Childhood CD was not required for diagnosis of ASPD in adulthood due to concerns about retrospective recall of spouses' childhood behaviors, and because empirical evidence suggests that this criterion is not needed to capture impairing ASPD and associated offspring risk for psychiatric problems (Compton, Conway, Stinson, Colliver, & Grant, 2006).

Parental ADHD—Parental ADHD was included in the analyses to control for its possible role in the association between parental alcoholism and offspring ADHD. Parents were assigned a lifetime ADHD diagnosis if they endorsed at least one of the following during their first wave of participation in the study: (1) six or more *childhood* symptoms of hyperactivity/impulsivity or inattention on the Disruptive Behavior Disorders Rating Scale (Pelham et al. 1992), reporting retrospectively; (2) four or more *current* symptoms of hyperactivity/impulsivity or inattention (Barkley, unpublished measure) that included the DSM-IV ADHD symptoms; or (3) a self-reported lifetime diagnosis of ADHD. The lower threshold for symptoms in adulthood, relative to the DSM-5 threshold of five or more symptoms, was based on recent findings with the PALS dataset and an effort to be over-, rather than under-, inclusive when testing this variable as a possible covariate (Sibley et al., 2012).

Proband conduct disorder (CD)—At follow-up, a diagnosis of CD in the past year for adolescents was made using a combination of parent, teacher, and self-report on the DBD adapted for adolescents, and parent and self-report on the DISC-IV (Shaffer et al., 2000). If each symptom of CD was endorsed by any rater, it was counted and adolescents who exceeded DSM-IV symptom cutoffs were diagnosed with CD. For young adults (18+), CD diagnosis was approximated using selected items from the Self-Reported Delinquency Questionnaire (Elliott, Huizinga, & Ageton, 1985) and the SCID-II ASPD Module (SCID-II;

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Spitzer et al., 1987) administered to self and parents. From these measures, young adults who endorsed two or more of 9 items that overlapped with CD diagnostic criteria were assigned a CD diagnosis. (A lower threshold of two, rather than three, CD symptoms was used for diagnosis to adjust for the smaller number of CD symptoms available for this age range.)

Results

Bivariate Regression Analyses Comparing ADHD and nonADHD Groups

Rates of parental alcoholism, ASPD, and parental antisocial-alcoholism are reported in Table 1 for mothers and for fathers, separately for the ADHD and nonADHD groups, and separately for probands with and without CD at follow-up. Using bivariate analyses, rates of maternal alcoholism (with or without ASPD) were higher in the ADHD (25%, 79/312) versus nonADHD group (13%, 31/235; x^2 (1, N = 547) = 12.3, $p < .01$), and rates of paternal alcoholism were higher in the ADHD (44%, 128/291) versus nonADHD group (28%, 63/227; x^2 (1, N = 518) = 14.4, $p < .01$). Also, rates of maternal antisocial alcoholism were higher in the ADHD (7%, 23/312) versus nonADHD group (1%, 3/235; x^2 (1, N = 545) = 11.1, $p < .01$), and rates of paternal antisocial alcoholism were higher in the ADHD (27%, 79/291) versus nonADHD group (14%, 31/227 x^2 (1, N = 518) = 13.9, $p < .01$). ADHD group fathers had the highest rate of antisocial alcoholism (27%), nearly double the rate for nonADHD group fathers (14%), and nearly four times the rate for ADHD group mothers (7%).

Bivariate Analyses Comparing ADHD Groups with and without Offspring CD Comorbidity

For ADHD group mothers, rates of alcoholism were not significantly different between those with (28%; 22/80) versus without offspring with CD (25%; 56/228), x^2 (1, N = 308) = 0.27. This was also the case for ADHD group fathers: rates of alcoholism were not significantly different between those with (47%; 35/75) versus without offspring with CD (43%; 92/214), x^2 (1, N = 289) = .0.30). Rates of maternal antisocial alcoholism were comparable for offspring with and without comorbid CD (5% vs. 8%, respectively). Rates of paternal antisocial alcoholism were also similar for offspring with and without comorbid CD (32% vs. 25%).

Multivariate Logistic Regression Analyses Comparing ADHD and nonADHD Groups

A series of planned pairwise contrasts (SPSS GLM) were performed to examine whether parent alcoholism with and without ASPD were each independently associated with offspring ADHD. First, alcoholism only, ASPD only, and antisocial alcoholism were compared with no psychopathology. Then, antisocial alcoholism was compared to alcoholism only and to ASPD only. Offspring ADHD (no/yes) was the binary dependent variable. A similar set of pairwise comparisons were conducted within the ADHD group to examine the associations of the parent diagnoses with offspring ADHD plus CD diagnosis. We estimated all models controlling for parental ADHD, which accounted for 18.4% of the proband mothers with alcoholism and 18.7% of the proband fathers with alcoholism, versus none of the nonADHD mothers with alcoholism and 1% of the nonADHD fathers with alcoholism.

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Results are shown in Table 2. They show that, controlling for parental ADHD, mothers of offspring with ADHD were more likely than mothers of offspring without ADHD to have alcoholism, with or without ASPD. They were also more likely to have ASPD without alcoholism. They were not, however, more likely to have antisocial alcoholism than either disorder alone. Findings for fathers were the same with the exception that likelihood of alcoholism alone was only marginally elevated for fathers of offspring with ADHD.

Multivariate Logistic Regression Analyses Comparing ADHD Groups with and without Offspring CD Comorbidity

Conducted within the ADHD group only, these same pairwise contrasts resulted in fewer associations. Mothers of probands with CD were more likely to have ASPD than mothers of probands without CD, and mothers of probands with CD were more likely to have ASPD than antisocial alcoholism compared to mothers of probands without CD. Fathers of probands with CD were also more likely to have ASPD than fathers of probands without CD. Fathers of probands with CD were only marginally more likely to have antisocial alcoholism than fathers of probands without CD.

Discussion

The results of this study are consistent with a long line of studies demonstrating associations between parent alcoholism and offspring externalizing behaviors and support our hypothesis that alcoholism is more prevalent among parents of children with, versus without, childhood ADHD. The current findings extend prior research by demonstrating the associations for a large sample of children rigorously diagnosed with ADHD in childhood and for mothers and fathers separately. Forty-four percent of the fathers and 25% of the mothers of children with ADHD, versus 28% of the fathers and 13% of the mothers of children without ADHD, experienced a problem with alcohol at some time in their lives. These group differences were statistically significant and based on large sample sizes relative to most studies of this question in the literature (about 300 ADHD and just over 200 nonADHD families in our study versus under 200 per group in other widely cited clinic samples). Although our percentages appear high relative to some prior reports (e.g., Barkley et al., 1990; Biederman et al., 1992), they are lower than others (August et al., 1999) and lower than might be expected based on national data collected within our window of study (42% of adult men and 19.5% of adult women had a DSM-IV lifetime alcohol use disorder in the 2001–2002 National Epidemiologic Survey on Alcohol and Related Conditions, NESARC, Hasin et al., 2007). Thus, our rates for index parents are high but not out of line with those expected in the lifetime of adults in the United States. Our methods did not permit distinctions between lifetime and current diagnoses nor abuse versus dependence; such distinctions can lower rates dramatically and also hint at the likely possibility that alcohol problems pre-dated offspring ADHD in many of these families. For example, only 3.8% of mid-aged adults (30–44 years old) in the NESARC survey were diagnosable with alcohol dependence (versus abuse) in the past year (Hasin et al., 2007). Taken together, our findings and those of others (e.g., August et al., 1999; Barkley et al., 1990; Biederman et al., 1992; Skoglunch et al., 2015) provide further evidence for the importance of screening for a positive family history of alcohol problems among individuals being assessed for ADHD, and this risk should

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include maternal history despite the generally higher rate of alcohol problems among men versus women more generally (Hasin et al., 2007). As articulated within a developmental psychopathology perspective of the ontogeny of externalizing disorders (Beauchaine & McNulty, 2013), although at any given assessment a patient's parents may not be actively experiencing alcohol-related difficulties, inherited, prenatal, and other environmental liabilities toward alcoholism may still be at play. Moreover, although trends are shifting, mothers continue to assume much of the parenting responsibilities (Yogman et al., 2016), and the possibility of maternal alcohol use contributing to environmental risk should be considered.

Our findings are partially in line with studies suggesting a genetic link between parent alcoholism and offspring ADHD (Knopik et al., 2005, 2006) that may be particularly prominent in fathers. After controlling for paternal ADHD, the association for fathers was only marginally significant for alcoholism without antisociality and statistically significant for antisocial alcoholism. In fact, 62% of the 128 proband fathers with alcohol problems had comorbid antisociality. Thus, we may be seeing what Knopik and colleagues describe as cross-generational genetic covariation (Knopik et al., 2009) in which parents transmit genetic vulnerability to offspring for multiple disorders that include alcoholism, ADHD, and antisociality. Investigators have written about familial risk for alcoholism being transmitted via multiple mechanisms reflecting the heterogeneity of alcoholism more generally (Zucker et al., 2011). Our findings support the hypothesis that antisocial alcoholism may be particularly important in the familial transmission of risk for an aggregated vulnerability to antisocial alcoholism and ADHD among males. Twenty-seven percent of the probands had a father with antisocial alcoholism (the highest parental diagnostic subgroup across analyses for mothers and for fathers).

Our hypothesis that parental antisocial alcoholism would be most prevalent for probands with CD was not supported. Five percent of the probands with CD had an antisocial alcoholic mother versus eight percent of the probands without CD. Among fathers, substantial minorities of offspring were captured in both sides of this comparison and the difference was only marginally significant. Thirty-two percent of the probands with CD had antisocial alcoholic fathers versus 25% of the probands without CD. One possible explanation for our unexpected findings, aside from small cell sizes, may lie in the contribution of child-rearing to the ultimate behavioral (i.e., conduct-related) outcome of the child. Although this serious parental comorbidity should theoretically lead to the worst offspring outcome (Zucker et al., 1993), relatively high divorce rates in our sample (Wymbs et al., 2008) and small numbers of antisocial mothers may have led to the removal of negative socialization effects by the most impaired parents (fathers in particular). We note, however, that ASPD among parents – among either fathers or mothers – was a statistically significant correlate with proband CD as an adolescent or young adult. These findings confirm and extend those of LeMoine et al. (2015) to mothers, by showing an association between parental (both mother and father) antisociality and child conduct problem comorbidity in offspring ADHD.

We were especially interested in effects pertaining to gender of parent and hypothesized that mothers (like fathers) of children with ADHD would also have higher rates of alcoholism

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than mothers of children without ADHD. The results suggest that maternal (not just paternal) alcoholism and ASPD might be important factors in the transmission of risk for externalizing disorders. While the vast majority of literature to date has focused on paternal alcoholism risk, partly due to the lower rates of alcoholism among women compared to men, our findings encourage ongoing consideration of maternal alcoholism as a risk factor for offspring adjustment. There is evidence to suggest that maternal alcoholism is associated with offspring substance use disorders (e.g., Chassin et al., 1996) and externalizing disorders such as ADHD (e.g., Knopik et al., 2006). There is also evidence to suggest that psychosocial mediators such as adolescent stress mediate the association between maternal alcoholism and offspring substance use (Chassin et al., 1996), and that this mediated pathway (for maternal or paternal alcoholism) may be stronger in offspring with ADHD than in controls (Marshal et al., 2007). Our data indicated a nearly two-fold higher likelihood of maternal alcoholism among mothers of children with ADHD than among mothers of children without ADHD (25% versus 13%, respectively), indicating the possibility (which remains to be tested) that maternal alcoholism partly represents a path of vulnerability toward alcoholism among some children with ADHD. Indeed, we previously found that children trained to enact behaviors characteristic of children with externalizing disorders (uncooperative, distractible, etc.) cause elevated levels of stress in, and alcohol consumption by, mothers in a laboratory setting (Pelham et al, 1997). Maternal alcohol consumption also exacerbated maladaptive parenting (Lang, Pelham, Atkeson, & Murphy, 1999). Parenting may be another important mediator of maternal (as well as paternal) alcoholism in families with a child with ADHD. This is an important hypothesis to test in future longitudinal analyses, as well as conducting an exploration of the potential mediators and moderators of maternal alcoholism among youth with ADHD in relation to the long-term adult outcomes of ADHD persistence, antisociality, and alcoholism among offspring.

In sum, this study provides evidence of an association between parental alcoholism, antisocial alcoholism, and offspring ADHD, for fathers and for mothers. There are some limitations to our findings including the clinic-referred nature of the sample and the timing of our assessments. We were not able to study the course of parental alcoholism or ADHD prospectively which may ultimately provide more detailed and veridical information about alcoholism and ADHD chronicity over time and within families. Our assessment of offspring outcome was limited to CD at initial follow-up for a wide age range. Examination of offspring outcome beyond late adolescence and early adulthood, to mid-adulthood when developmentally limited expressions of behavior have subsided, and chronic alcohol problems are most apparent, will be important. Future studies that include a larger proportion of female offspring with ADHD, and that examine mediators and moderators of the association between parents' antisocial alcoholism and all offspring outcomes into adulthood, will improve understanding of the ADHD-alcohol link in families. In the meantime, the results of the current study highlight the importance of considering multi-layered risk within families that may include some combination of ADHD, alcoholism, and antisociality, for both males and females.

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Table 1

Rates of antisocial alcoholism in biological parents of children with/without childhood ADHD and by CD comorbidity at follow-up.

	NonADHD Group	ADHD Group	ADHD Probands Without CD	ADHD Probands With CD
Mothers # (%)				
No Alcoholism or ASPD	198 (84)	211 (68)	162 (71)	49 (61)
Alcoholism Only	28 (12)	56 (18)	37 (16)	18 (22)
ASPD Only	6 (3)	22 (7)	10 (4)	9 (11)
Both Alcoholism and ASPD	3 (1)	23 (7)	19 (8)	4 (5)
Total	235	312	228	80
Fathers # (%)				
No Alcoholism or ASPD	142 (63)	124 (43)	100 (47)	24 (32)
Alcoholism Only	32 (15)	49 (17)	38 (18)	11 (15)
ASPD Only	22 (10)	39 (13)	22 (10)	16 (21)
Both Alcoholism and ASPD	31 (14)	79 (27)	54 (25)	24 (32)
Total	227	291	214	75

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Table 2

Relation between maternal and paternal antisocial alcoholism and offspring ADHD and CD among offspring with ADHD.

	ADHD Group (No/Yes) Full Sample		Offspring CD (No/Yes) Within ADHD Group	
	Contrast Estimate (Std error)	**p**	**Contrast Estimate (Std error)**	**p**
Mothers				
Alcoholism Only vs No Dx	.129 (.06)	.007	.080 (.07)	NS
Antisocial Only vs No Dx	.259 (.10)	.007	.209 (11)	.05
Antisocial Alcoholism v No Dx	.317 (.10)	.002	.100 (.10)	NS
Alcoholism Only vs Antisocial Alcoholism	−.158 (.11)	NS	.180 (.11)	NS
Antisocial Alcoholism vs. Antisocial Only	.058 (.13)	NS	.309 (.14)	.027
Fathers				
Alcoholism Only vs No Dx	.114 (.06)	.067	.001 (.08)	NS
Antisocial Only vs No Dx	.170 (.07)	.014	.234 (.08)	.005
Antisocial Alcoholism v No Dx	.227 (.06)	.000	.104 (.06)	.10
Alcoholism Only vs Antisocial Alcoholism	−.112 (.07)	NS	.118 (.09)	NS
Antisocial Alcoholism vs. Antisocial Only	.057 (.08)	NS	.129 (.09)	NS

Note. Total n's for maternal models were 526 for the ADHD group analysis and 297 for the offspring CD analysis (within ADHD group), and for paternal models they were 505 and 290, respectively. Models were estimated controlling for parental ADHD, offspring race/ethnicity and gender, family SES (parent education), and presence of one or two biological parents currently in the family. With the exception of parental ADHD, none of the covariates were significant and did not significantly change the interpretation of the results and they were therefore trimmed from the final models.