2011

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Brief Report of a Test of Differential Alcohol Risk Using Sibling Attributions of Paternal Alcoholism*

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ABSTRACT. Objective: Parental alcoholism is generally found to be a strong predictor of alcohol misuse. Although the majority of siblings agree on the presence of parental alcohol issues, there is a significant minority who do not. Method: The current study analyzed sibling data from the 1979 National Longitudinal Survey of Youth using multilevel modeling, which accounts for the nested structure of the data. These analyses permitted a test of whether (a) identifying one’s father as an alcoholic predicted greater risk of alcohol problems, (b) being from a family whose siblings did not all agree on the presence of paternal alcoholism increased the likelihood of alcohol problems, and (c) risk of alcohol misuse significantly differed among individuals from families in which there was familial disagreement about paternal alcoholism. Results: Results show that individuals who identified their father as an alcoholic were themselves more likely to have alcohol issues as compared with individuals both within and between families who did not identify their father as an alcoholic. Risk was similar for individuals in families in which there was disagreement about paternal alcoholism compared with families in which everyone agreed on the presence of paternal alcoholism. Moreover, there was not a significant interaction between familial alcoholism attributions and familial disagreement. Conclusions: Findings indicate that in the case of child reports of paternal alcoholism, the increased risk of alcohol problems holds true regardless of the accuracy of an individual’s assessment. These results may be not only because of the impact of paternal alcoholism on a person’s alcohol misuse but also because of a person’s alcohol problems potentially influencing his or her perceptions of familial alcohol-related behaviors. (J Stud Alcohol Drugs, 72, 1037–1040, 2011)

ALCOHOL MISUSE IS ASSOCIATED WITH myriad negative health outcomes, including markedly increased rates of accidental death (Rehm et al., 2001; Smith et al., 1999), cancer (Boffetta and Hashibe, 2006; Pöschl and Seitz, 2004), and suicide (Andreasson et al., 1988; Sher, 2006). Additionally, alcoholism tends to run in families, with both maternal and paternal alcoholism being significant predictors of children’s substance use, even after accounting for other related factors such as the child’s stress, sociability, and peer substance use (Chassin et al., 1993). Research on alcoholism frequently uses family member reports when determining rates of alcoholism in a family; however, there is not always consensus among family members as to whether a family member exhibits problematic drinking behavior (Andreasen et al., 1977; Crews and Sher, 1992; Prescott et al., 2005; Slutske et al., 1996).

Our goal in the present research was to examine whether attributions of paternal alcohol use were predictive of differential risk of alcohol problems within and between families and whether this effect was moderated by familial disagreement about paternal alcoholism. Real disagreement among family members is of interest clinically because it may result in problems mounting or sustaining a family-based alcohol intervention effort. Moreover, disagreement about parental alcoholism among siblings is meaningful beyond considerations of accuracy and intervention motivations because there may also be differential levels of risk for siblings identifying a parent as an alcoholic compared with those who do not. Given the ubiquity of studies administering measures of retrospective parental alcoholism and current alcohol use, it is important to understand the biases inherent in self-reported familial alcoholism. It is possible that the reported link between parental alcoholism and a person’s own alcohol issues is not entirely a function of genetic and behavioral influences but is also influenced by an individual’s psychological need to explain the etiology of current issues with alcohol.

Originally proposed by Heider (1958), attribution theory is a highly relevant theoretical perspective for why and how individuals draw inferences for explaining health-related behaviors and conditions. Specifically, when individuals are injured, fall ill, or struggle with some sort of chronic disease such as alcoholism, they will often attempt to regain a sense of control over their situation by developing a story or theory as to why the event has occurred (e.g., Schulz and Decker, 1985; Taylor, 1983), with one common strategy being to place the blame on others (Tennen and Affleck, 1990). When faced with one’s own problematic drinking, a person may be more likely to identify other family members as alcoholics,
Examinining sibling reports of familial alcoholism provides a useful test of whether individuals with alcohol problems are in fact more likely to perceive a parent as an alcoholic. If it is the case that individuals with alcohol problems are inclined to project their alcohol problems onto their parents, then risk will be greater for those who identify their parents as an alcoholic and whose siblings do not. Alternatively, if those who do not identify a parent as an alcoholic and who have siblings who do are at greater risk for alcohol issues, then the cause of the disagreement may be more likely because of underreporting of parental alcoholism than projection. In brief, the overreporting and underreporting of parental alcoholism are both possible attribution errors, although we hypothesize the causes are somewhat different (i.e., overreporting is likely a result of projection, whereas underreporting is likely because of denial and/or a lack of full knowledge of the parent’s behavior).

The current analyses were conducted using the 1988 wave of the 1979 National Longitudinal Survey of Youth (NLSY79). Given the nested nature of the data (i.e., siblings nested within family), multilevel modeling was used, which simultaneously accounts for both individual-level (Level 1) and between-family (Level 2) differences (Raudenbush and Bryk, 2002). This approach therefore allows us to simultaneously examine individual-level variables (e.g., identifying one’s father as an alcoholic), family-level variables (e.g., familial disagreement about the presence of paternal alcoholism), and cross-level interactions (e.g., attribution of paternal alcoholism by familial disagreement). In a simulation study of sibling data on parental alcohol use, Krull (2007) determined that multilevel modeling achieved a power value of .80 with approximately 227 families of 410 nonindependent individuals. The NLSY79 data set contained 1,331 sibling sets comprising 3,125 respondents, indicating more than adequate statistical power to test the effects of interest.

Data from families in which there were two or more sibling reports on paternal alcoholism were analyzed to determine whether there were significant differences in alcohol misuse between those who identified their father as an alcoholic and those who did not (Level 1). In addition, we examined whether individuals from families in which there was disagreement about paternal alcoholism were more at risk for alcohol misuse (Level 2) as well as whether risk was greater for those who identified their father as an alcoholic and who had one or more siblings who did not (Level 1 × Level 2 interaction). Demographic variables such as age, race, and gender (all Level 1) were also tested to control for their potential effects in the model.

Method

The NLSY79 is a longitudinal study of a nationally representative sample of men and women who were 14–22 years old at the time of their first interview in 1979. The overall data set includes all youths living in surveyed households who were born between 1957 and 1964, yielding an initial sample of 12,686 individuals in 2,826 families. The sampling procedure was designed to include a disproportionate number of racial minorities and poverty households (Bureau of Labor Statistics, 2009). The data set used for the current analyses comprised families for whom two or more siblings reported on familial alcoholism in 1988 and whose data could be linked (N = 1,331). Seventy-two percent (n = 952) of the families had two siblings, 23% (n = 309) had three siblings, and the remainder (n = 70) had four to six siblings. Seventy-nine percent of the sibling sets agreed that their father was not an alcoholic. Of the families in which one or more siblings indicated paternal alcoholism, approximately 40% (n = 114) were in agreement, whereas the remainder (n = 168) had some level of disagreement. Only 2% of the total sample identified their mother as an alcoholic; therefore, further analyses comparing sibling reports for maternal alcoholism were not possible.

Alcohol misuse was assessed using a version of the well-validated CAGE questionnaire (Ewing, 1984; O’Brien, 2008), which some research indicates is a more reliable measure of alcoholism than even some laboratory-administered biochemical tests (Girela et al., 1994). The 1988 NLSY79 survey asked 12 CAGE-type items (see Table 1). Concordant
with CAGE scoring practices, answering positively to two or more of these items resulted in a positive alcoholism score for an individual. Directly after answering the CAGE items, respondents were asked to identify any close relatives who have “been alcoholics or problem drinkers at any time in their lives” (National Opinion Research Center, 1988, p. 173). Because the outcome of interest (likely alcohol misuse) was dichotomous, models were estimated using the PROC GLIMMIX procedure in SAS 9.2 (SAS Institute Inc., Cary, NC). Given that the data were primarily dyadic, only the intercept was treated as a random effect (see Kenny et al., 2002). All continuous variables were grand mean centered to facilitate interpretation of the estimates. Dichotomous variables were dummy-coded using 0 and 1. Because multilevel generalized linear models use a quasi-likelihood estimation strategy, only the Wald statistic was used to determine whether an effect should be kept in the final model. The model-building approach used a modified version of the bottom-up approach advocated by Hox (2010).

Results

The intraclass correlation, computed assuming a threshold model (Snijders and Bosker, 1999), was .098. This indicates that 9.8% of the variance for alcohol misuse was at Level 2, suggesting a substantial degree of family-level effects. Being male \( (b = 0.52, p < .001) \), unmarried \( (b = 0.55, p < .0001) \), and having lower educational attainment \( (b = 0.11, p < .001) \) were all significantly associated with an increased risk of alcohol problems. Racial category and urban/rural environment were not significant predictors of alcohol misuse \( (ps > .05) \). Younger age and lower income were individually predictive of greater alcohol misuse, although the former became marginally significant \( (p = .07) \) and the latter became nonsignificant \( (p = .16) \) when entered with the other significant predictors. Both age and income were therefore dropped from the final model.

The hypothesis of interest, which was based on projection and attribution theories, posits that individuals who identify their father as an alcoholic, regardless of the accuracy of that attribution, will have higher rates of alcohol problems. Results from the multilevel modeling supported this supposition \( (b = 0.89, p < .0001) \). That is, after controlling for significant demographic factors, those who identified their father as an alcoholic, regardless of familial agreement, had an alcohol misuse rate of 27.3%, which was more than double the rate of 13.4% for those not identifying their father as an alcoholic. Disagreement about paternal alcoholism was not significantly predictive of alcohol misuse, nor was the interaction between identifying one’s father as an alcoholic and familial agreement \( (ps > .05) \).

Discussion

The purpose of the analyses was to determine whether identifying one’s father as an alcoholic, regardless of accuracy or familial consensus, can be taken prima facie as a risk indicator of alcohol misuse. The results support this view: Those who identified their father as an alcoholic, regardless of familial consensus, were more likely to exhibit problematic drinking. Familial disagreement was not predictive of alcohol issues, nor was the interaction between familial disagreement and identifying one’s father as an alcoholic. These findings indicate that the risk of alcohol misuse for individuals who identified their father as an alcoholic and who had siblings who did not identify their father as an alcoholic did not differ from the risk of alcohol misuse for individuals whose siblings all agreed that their father was an alcoholic. The results suggest that the association between the attribution of paternal alcoholism and alcohol problems may be in part because of problem drinkers seeking to explain the genesis of their own alcohol issues.

A limitation of this data set, and therefore of our analysis, is that objective measures of paternal alcohol use were
not available. This means that it cannot be definitively determined that cases of familial disagreement are mainly because of overreporting of paternal alcoholism. Underreporting seems less likely given that there was neither a main effect for familial disagreement nor an interaction between familial disagreement and a paternal alcoholism attribution. Also, it is possible that when siblings disagree about a parent’s alcoholism, in some cases they each may be correct in that they have different memories and experiences because of changes in the parent’s drinking behavior over time. Future research would benefit from a comparison of the predictive utility of objective measures of familial alcoholism with family member reports. These data would allow a direct examination of whether familial estimates are more likely to be overestimates, underestimates, or differentially accurate as a function of age and experiential differences. In the meantime, the research outlined herein clearly demonstrates that, from a clinical perspective, an attribution of parental alcoholism is a fundamentally important risk indicator of alcohol problems, regardless of its accuracy.

Acknowledgments

The authors thank Phil Costanzo and Phil Cook for their input on the study concept; David A. Kenny, Madeline Carrig, and Krista Ranby for advice on the analyses; and Rose Wilson for her assistance with the reference citations.

References