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Peer Deviance, Alcohol Expectancies, and Adolescent Alcohol Use: Explaining Shared and Nonshared Environmental Effects Using an Adoptive Sibling Pair Design

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Abstract

Previous research suggests adolescent alcohol use is largely influenced by environmental factors, yet little is known about the specific nature of this influence. We hypothesized that peer deviance and alcohol expectancies would be sources of environmental influence because both have been consistently and strongly correlated with adolescent alcohol use. The sample included 206 genetically related and 407 genetically unrelated sibling pairs assessed in mid-to-late adolescence. The heritability of adolescent alcohol use (e.g., frequency, quantity last 12 months) was minimal and not significantly different from zero. The associations among peer deviance, alcohol expectancies, and alcohol use were primarily due to shared environmental factors. Of special note, alcohol expectancies also significantly explained nonshared environmental influence on alcohol use. This study is one of few that have identified specific environmental variants of adolescent alcohol use while controlling for genetic influence.

Keywords

Adolescence; Alcohol Expectancies; Alcohol Use; Nonshared Environment; Peer Deviance; Shared Environment

In the United States, adolescent alcohol use remains highly prevalent (Center for Disease Control and Prevention; CDC, 2010). While experimentation with alcohol in adolescence is normative (Newcomb & Bentler, 1989), early and frequent alcohol use is associated with various adjustment problems, including school and social problems, suicide, memory problems, and abnormal changes in brain development (Bonnie & O'Connell, 2004; Miller et al., 2007). Research has shown adolescent alcohol use is moderately heritable (see Hopfer, Crowly & Hewett, 2003 or Lynskey, Agrawal, & Health, 2010 for a review). Nonetheless, a substantial portion of variance in adolescent alcohol and substance use is explained by environmental contributions, both shared and nonshared. For example, in a sample of adolescent twins, Han, McGue, & Iacono (1999) reported that shared environmental influences (c^2) explained 46% of the variation in lifetime alcohol use (ever drinking), and that 63% of the common variance among lifetime tobacco, alcohol, and substance use was accounted for by the shared environment. Also demonstrating the importance of genetic (a^2) and nonshared factors (e^2), Han and colleagues reported 23% of the covariance among different types of substance use was genetic, and 14% was due to nonshared environmental factors. Substantial shared environmental effects for ever drinking in adolescence have been reported elsewhere (Rose et al., 2001) and moderate shared environmental contributions have been found for substance use disorders (Buchanan et al.,

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2009; McGue, Elkins, & Iacono, 2000), number of substances used (Walden et al., 2004), and quantity and frequency of drinking (Rende et al., 2005).

Although research has demonstrated large environmental effects on alcohol and substance use, there are only a few behavioral genetic studies that have sought to identify the specific environmental variants that underlie these overall effects. For example, Baker, Maes, & Kendler (2012) reported a large shared environmental effect on alcohol use (62%), but less than 15% of this variance was explained by the variables they examined (household substance use and parental attitudes towards substance use). Similarly, Latendresse et al. (2010) reported that parent knowledge and warmth accounted for 6–32% of the shared environmental variance on adolescent alcohol use. More research is clearly needed to identify the specific environmental variants that influence adolescent alcohol use in order to better understand the etiology of alcohol use and develop appropriate prevention and intervention programs for adolescents. Using a sample of adoptive and non-adoptive siblings assessed in mid-to-late adolescence, we examined two characteristics that have been strongly associated with adolescent alcohol use: peer deviance and alcohol expectancies.

Peer Deviance

Having friends who use alcohol and other drugs is one of the strongest correlates of adolescent alcohol and substance use (Adrados, 1995; Bahr, Hoffman, & Yang, 2005; Ellickson et al., 2004; Simons-Mortons et al., 2004). For example, Curran, Stice, & Chassin (1997) reported correlations in the .60 range between peer and adolescent alcohol use at three different time points in adolescence. Research has shown that peer deviance is moderately heritable, with estimates of about 16% in early adolescence (Walden et al., 2004) and ranging from 30% to 55% in late adolescence (Agrawal et al., 2010; Fowler et al., 2007; Kendler et al., 2007). This association likely reflects a gene-environment correlation (*rGE*) process (Scarr & McCartney, 1983), such that adolescents seek friends with similar genetic predispositions (active *rGE*), or that adolescents' friends are drawn to them because of their shared, genetically influenced traits and interests (evocative *rGE*), including their openness to drinking and using other substances. Even though adolescent peer choice is heritable, the effect peers have on their behaviors may still be environmental in nature (Rutter & Silberg, 2002).

In fact, there is some evidence that the correlation between peer deviance and substance use may be partially mediated by environmental factors, at least in adolescence (Fowler et al., 2007; Gillespie et al. 2009; Walden et al., 2004). Using a twin-study of 14 year-olds, Walden et al. (2004) found the association between peer deviance and early substance use explained 76% of the total shared environmental variation in substance use. Further, Gillespie et al. (2009) found that shared environmental effects accounted for 73% of the covariance between cannabis use and peer deviance at age 15–17, but declined at older ages. Using a discordant monozygotic twin analysis, Burt, McGue, & Iacono (2009) found nonshared environmental influence on the relationship between peer deviance and adolescent externalizing behaviors from ages 14 to 17. With this evidence of environmental influence accounting for the relationship between peer deviance and the adolescent adjustment outcomes reviewed here (substance, cannabis use, externalizing), it seems likely that peer deviance will also explain environmental influence on adolescent alcohol use.

Alcohol Expectancies

Like peer deviance, alcohol expectancies, are also highly associated with alcohol use (Christiansen et al., 1989; Sher et al., 1996). Alcohol expectancies refer to beliefs regarding positive and negative effects of alcohol; for example, whether alcohol enhances social behavior or deteriorates cognitive and behavioral functioning. Thus, if adolescents think

drinking helps them to feel more outgoing, or that it enhances a celebration, then they are more likely to drink because of their positive expectations about drinking. On the other hand, if adolescents perceive drinking to be associated with negative outcomes, such as embarrassing or debilitating behavior, they may be less likely to drink then if they did not hold these negative expectations.

As with peer deviance, there is evidence for genetic and environmental influences on alcohol expectancies. For example, using a sample of adult twins (M_{age} = 31 years), Vernon et al. (1996) demonstrated moderate additive genetic and shared environmental contributions to alcohol expectancies across a variety of measures (estimates of a^2 ranged from 0 to 36%; estimates of c^2 ranged from 0 to 40%), and substantial nonshared environmental influences, ranging from 55 to 72%. In a study of 14 to 22 year-old twins, Slutske et al. (2002) reported moderate shared environmental influences for expectancies related to affect regulation (e.g., “Drinking helps people feel less shy”) and performance enhancement (e.g., “Drinking helps people think better “). In this same paper, Slutske et al. reported an average shared environmental estimate of 31% for affect regulation (mean estimated a^2 = 0%, mean estimated e^2 = 69%) and an average shared environmental estimate of 5% for performance enhancement (mean estimated a^2 = 21%, mean estimated e^2 = 74%), across age cohorts. It is useful to note that across these two studies there were substantial nonshared influences, suggesting that environmental factors contribute to within twin pair differences in alcohol expectancies.

There is little research on the relative contribution of genetic and environmental effects on the covariation between drinking expectancies and alcohol use. Agrawal et al. (2008) examined the relative contributions of each to alcohol expectancies across groups of alcohol-using versus non-alcohol-using adult women. They found that shared environmental effects were greater for alcohol-using women, and genetic effects were greater for non-alcohol-using women, at least for some types of expectancies (e.g., cognitive behavior impairment). Although this study provided some evidence that shared environmental variance is important to individual differences in alcohol expectancies, it did not examine the degree to which shared environmental effects account for the covariance between expectancies and alcohol use. Prescott et al. (2004) also found genetic influence on the relationship between motivations to drink and alcohol abuse/dependence in an adult sample of twins. Given the adolescent age of the sample used in the present study, we expected significant shared environmental contributions to the covariance between both alcohol expectancies and alcohol use and alcohol expectancies and peer deviance.

Summary

There is substantial shared and nonshared environmental influence on adolescent substance use (Buchannan et al., 2009; Han et al., 1999; Rose et al., 2001; Walden et al., 2004). There is ample evidence that peer deviance, alcohol expectancies, and adolescent alcohol use are highly correlated (e.g. Barnow et al., 2004; Curran et al., 1997; Zamboanga et al., 2009), perhaps due to shared environmental influences (Gillespie et al., 2009; Walden et al., 2004). This is the first paper to decompose the genetic and environmental covariance among these variables using a genetically informed sibling pair design. A multivariate Cholesky decomposition was utilized. Given the adolescent age of our sample, we expected moderate shared environmental influences would be evident for all three variables.

Method

Participants

Data from the first follow-up assessment of adolescents in the Sibling Interaction and Behavior Study (SIBS; McGue et al., 2007) were used to address study aims. The SIBS study was designed to investigate shared environmental effects on substance use and related psychopathology in 617 adoptive and non-adoptive families. Data collection for the intake and first follow-up assessment began in 1998 and 2003, respectively. Families with adopted children were first recruited through three large adoption agencies. Adoptive family eligibility included having an adopted child between the ages of 11 and 21 years who had been permanently placed into the adoptive home prior to age 2 years (M_{age} = 4.7 months, SD = 3.4 months; 96% were placed before they were 1-year-old), and a second adolescent in the home who was not biologically related to the adopted adolescent and within five years of age. Families with biologically related children were recruited to match the adoptive sample in terms of age and sex through publicly available birth certificates. Participation rates for non-adoptive (57%) and adoptive (63%) families were not significantly different. Comparisons of parents' education and marital status to 2000 US Census data showed that the sample of non-adoptive families was generally representative of Minnesota families with at least two children living at home (McGue et al., 2007). Furthermore, a brief phone interview was administered to 73% of non-participating families. There were no significant differences in parent education, occupation, marital status, and parent-reported behavioral disorders in their children (substance use, attention-deficit-hyperactivity disorder, depression) for adoptive families. The only difference for non-adoptive families was that participating mothers were more likely to be college educated than non-participating mothers (McGue et al., 2007). Altogether, these results suggest that any effects of participation bias are likely to be minimal.

The impact of restriction of range in the family environments of adopted adolescents was analyzed in this sample (McGue et al., 2007). While adoptive families experienced a reduction in the variance associated with variables tapping parent disinhibitory psychopathology and socio-economic status, this restriction in range had no effect on adoptive-sibling correlations for delinquency, drug use, or IQ, supporting the use of SIBS in estimating genetic and environmental influence.

The vast majority of families (94%) returned at the follow-up visit 3.5 years after the study intake assessment. Follow-up data was used for the present study. A total of 1,158 eligible adolescents (502 non-adopted and 656 adopted) came to the follow-up visit. Among adoptees, the majority (n = 484) were internationally adopted (Asian: 89%; female: 61%; domestic adoptions, n = 172; Caucasian: 77%; female: 42%). The average age difference in sibling pairs was 2.3 years (SD = .89; younger siblings M_{age} = 17.1 years (SD = 1.8), elder siblings M_{age} = 19.4 years (SD = 1.7). A total of 206 sibling pairs were genetically related, full biological siblings, and a total of 407 sibling pairs were genetically unrelated (made up of at least one adopted sibling). Sixty-one percent of sibling pairs were same-sex and 80% were same-ethnicity. There was no significant differences in the proportion of same-sex siblings in the genetically related (62%) and genetically unrelated (60%) groups; χ^2 = .27, p = .60. For more information about SIBS, please see McGue et al., (2007).

Procedure

At the follow-up assessment, participating families (adolescent siblings and one parent, usually the mother) came to the research lab for a half-day assessment. After consenting to participate, multiple assessments were completed, including diagnostic interviews and self-

report surveys. For those who were unable to come to the lab for assessment, phone interviews were conducted (14% of adolescent participants).

Measures

Adolescent alcohol use—Alcohol use was assessed in a private setting using the *Computerized Substance Use Assessment* (CSA, Han et al., 1999). Adolescents answered items pertaining to the frequency of their drug and alcohol use and their expectancies regarding use. Items used to assess adolescent alcohol use were: ever used alcohol (1 =*Yes*, 0 = *No*), frequency of alcohol use in the last 12 months (0= *Never* to 9 = *Every day or nearly every day*), ever been intoxicated (1 =*Yes*, 0 = *No*), frequency of being drunk in the last 12 months (0 = *Never* to 9 = *Every day or nearly every day*), how often they drank enough to get drunk in the last 12 months (0 = *Never* to 5 = *Nearly every time or every time*), and finally, number of maximum drinks consumed at one time in the past 12 months (0 = *0 drinks* to 10 = *10+ drinks*). The reliability of these items in a summed score was excellent (α = .89). The alcohol use measure was constructed by using these items as indicators in the estimation of a latent factor score of adolescent alcohol use, as described in the analysis plan below.

Peer deviance—Peer quality was assessed using items from the *Friends* self-report survey concerning adolescent's relationships with peers (Burt, McGue, & Iacono, 2009; Walden et al., 2004). Adolescents rated friendships on a scale of 1 *(All my friends are like that*) to 4 *(None of my friends are like that*). Peer deviance was measured by 8 items, for example, "My friends get into trouble with the police," "My friends drink alcohol or beer" (α = .86). Items were coded so that a higher score indicated more deviant friends and were used as indicators in the estimation of a latent factor score of peer deviance.

Positive alcohol expectancies—Expectancies were assessed using items from the CSA (based on Christiansen & Goldman, 1983; Schafer & Brown, 1991). Alcohol expectancies were measured by 17 items, all assessing the positive qualities of drinking. Example items include, "Drinking is a good way to celebrate occasions," "Drinking can make you feel less shy" (α = .94). Adolescents rated their expectations about alcohol use on a scale of 1 (*Strongly Agree*) to 4 (*Strongly Disagree*). All items were coded so that a higher score indicated more lenient attitudes toward alcohol use and were used as indicators in the estimation of a latent factor score of alcohol expectancies.

Missing Data

Over three quarters of the data were complete for alcohol use and alcohol expectancy reports, and over 90% of data was complete for peer deviance reports. Missing data were a result of non-participation (~5%) or participation via phone interview which precluded the option to use the computerized assessment (~14%). Therefore, adolescents who did not come to the lab had missing data on alcohol use and alcohol expectancies. Whether they came to the lab or interviewed by phone, peer deviance was assessed. Missing data were handled using Full Information Maximum Likelihood (FIML; Enders & Bandalos, 2001; Johnson & Younger, 2011).

Chi-Square analyses revealed there were no statistically significant differences between those who participated versus those who did not, or between those who completed the computerized alcohol use assessment and those who did not, on key demographic variables (adoptive status, sex, ethnicity). Furthermore, independent *t*-tests and chi-square analyses comparing the 6 alcohol use items at intake across those who did or did not participate at the follow-up, showed no significant differences across items for the younger siblings, and one significant difference for elder siblings: compared to those who participated at both time

points, elder siblings who visited at the follow-up had significantly higher mean scores for how often they drank enough to get drunk in the last 12 months (answered on a scale of 0 = *Never*, 1 = *Not in the last 12 months*, 2 = *Almost Never*, 3 = *Less than half the time*, 4 = *About half the time*, 5 = E*very time or nearly every time*; elder siblings that visited at follow-up: $M = .85$, $SD = 1.60$; elder siblings that did not visit at follow-up: $M = .35$, $SD = 1.10$, $t(608) = 1.77$, $p = .02$). This mean difference was small in magnitude (less than half of one *SD*) and would not be significant after a Bonferroni correction for multiple testing.

Analysis Plan

First, latent factor scores for study phenotypes (peer deviance, alcohol expectancies, and alcohol use) were estimated and exported from MPLUS, 6.12 (Muthén & Muthén, 1998–2012). All items from the scales described above were used as indicators of the latent factors. Categorical indicators (e.g., ever intoxicated) were specified as such in the modeling. Adolescent sex, age, and ethnicity (1 = *White*, 0 = *African-, Asian-, Hispanic-American, Other*) were regressed out of all three latent factor scores. The MPLUS COMPLEX specification was used so that individual data was clustered by family in latent factor score estimation. We analyzed the skewness and kurtosis of all latent factor phenotypes to evaluate whether they met assumptions of normality. For all three outcomes, standardized skewness was < .5 and standardized kurtosis was < 2.0; values well below the thresholds of 3 and 10, respectively, for which Kline (2005) has suggested structural equation analyses are not markedly affected by deviations from normality.

Cholesky decomposition was accomplished using Mx (Neale & Cardon, 1992). Figure 1 depicts the full Cholesky decomposition (ACE), where the total variance and covariance among peer deviance, alcohol expectancies, and adolescent alcohol use are decomposed into that which is attributable to additive genetic (A), shared environmental (C), and nonshared environmental influences (E). Additive genetic variance (A) refers to genetic explanations for why siblings are similar. Shared environment variance (C), also known as the common environment, refers to anything that makes siblings similar other than genes. Finally, nonshared environmental variance (E) refers to anything that makes siblings different, other than genes, and also includes measurement error. While not shown in Figure 1, the additive genetic path between siblings for the genetically related sibling group was set to .5 (since they share, on average, half of the additive genetic effect), and .0 for the genetically unrelated sibling group (since they share none of the additive genetic effect). The shared environment path was set to 1.0 for both groups because these models assume that adopted and non-adopted siblings share their rearing environment to a similar degree.

The ordering of the variables does have conceptual relevance; peer deviance was entered first because we hypothesize that peers influence both adolescent's perceptions and expectations about alcohol use as well as their use (although we tested for alternate ordering in post-hoc analyses and found similar results). The primary goal of this paper was to examine whether peer deviance and alcohol expectancies accounted for substantial environmental influence on alcohol use. To test this, we calculated the proportion of genetic and environmental variance explained based on squaring the standardized path estimates and dividing by the total genetic, shared environment, or nonshared environment variance. For example, to determine the total proportion of shared environmental variance in alcohol use accounted for by peer deviance and alcohol expectancies, the sum of all the c path leading the drinking outcome (see Figure 1; these include c31, c32), except the residual (c33), were squared and summed, and then divided by the sum of all c squared paths including the residual.

Results

Table 1 describes the overall levels of alcohol use for younger and elder siblings. As expected given their respective ages, elder siblings drank more than younger siblings. For example, 76% of elder siblings had ever drunk alcohol (without parents' permission) in comparison to about 54% of younger siblings. On average, elder siblings reported having about 5 drinks on average when they drank the most while younger siblings reported ~3 drinks. The large standard deviations for each of the quantitative variables document considerable variability in adolescent drinking levels.

As expected from the internal consistency reliability for each measure, standardized factor loadings for each phenotype were moderate to strong in magnitude. Loadings for each item ranged from .91 to .99 for alcohol use (M = .94, SD = .04, α = .89), from .46 to .81 for peer deviance (M = .66, SD = .12, α = .86), and .46 to .82 for alcohol expectancies (M = .70, SD = .11, α = .94). Table 2 provides correlations and descriptive statistics for adolescent alcohol use, peer deviance, and alcohol expectancies. In general, all study variables were moderately and significantly correlated within both the elder and younger sibling samples.

The standardized path estimates from the full ACE Cholesky are presented in Figure 2. The primary goal of this paper was to evaluate the proportion of genetic and environmental variance in adolescent alcohol use that was explained by the genetic and environmental variance in peer deviance and alcohol expectancies. Table 3 provides these calculations and confidence intervals. As shown in the first three columns, there were moderate shared environmental (19–35%) and substantial nonshared environmental influences (42–65%) on the three phenotypes. The heritability of peer deviance (36%) was slightly greater in magnitude compared to alcohol expectancies (16%) and alcohol use (21%). The lower bound of the 95% confidence interval (CI) of the genetic influence on alcohol use was zero, suggesting the heritability of alcohol use was not significantly different from zero.

The right-most three columns in Table 3 show the percentage of genetic and environmental variance in alcohol use explained by peer deviance, alcohol expectancies, and peer deviance and alcohol expectancies combined. While peer deviance explained 56% and alcohol expectancies explained 44% of the genetic variance in alcohol use, these proportions were not statistically significant, as indicated by the lower bound of the 95% CI crossing zero (because the heritability of alcohol use itself was not significantly different from zero). Significant shared and nonshared environmental effects were found for peer deviance and alcohol expectancies. Peer deviance alone significantly explained 82% (95% CIs: .55, 1.0) of the shared environmental variance on alcohol use, and alcohol expectancies significantly explained 7% (95% CIs: .001, .41) of the nonshared environmental variance. Together, peer deviance and alcohol expectancies explained 92% (95% CIs: .68, 1.0) of the shared environmental variance and 22% (95% CIs: .01, .48) of the nonshared environmental variance in alcohol use.

To evaluate the possibility that adolescents may choose friends based on their pre-existing ideas about alcohol use, we conducted post-hoc analyses in which we reordered the latent factors so that alcohol expectancies were entered first, followed by peer deviance and alcohol use. In comparison to the original full ACE Cholesky, the results showed that no matter which variable was entered first (alcohol expectancies or peer deviance), the first variable accounted for most of the shared environmental variance in alcohol use, and the second variable did not have a significant shared environmental covariance path with alcohol use. However, the decomposition with peer deviance entered first accounted for more shared environmental variance in alcohol use than the reordered decomposition (peer deviance accounted for 82% compared to alcohol expectancies accounting for 69% of the

shared environmental variance in alcohol use). In both instances, any significant nonshared environmental covariance with alcohol use was only found for alcohol expectancies and alcohol use, and this outcome did not depend on which variable was entered first.

Altogether, the results indicated that (a) adolescent peer deviance was moderately heritable, (b) adolescent alcohol expectancies was minimally heritable, (c) the heritability of adolescent alcohol use as measured by frequency and quantity did not differ significantly from zero, (d) moderate shared environmental and substantial nonshared environmental variance were evident for all three variables, (e) the covariance between peer deviance, alcohol expectancies, and adolescent alcohol use was primarily due to shared environmental factors, and (f) the covariance between alcohol expectancies and adolescent alcohol use was in part due to nonshared environmental factors.

Discussion

In order to extend research on environmental influences on adolescent alcohol use and to determine whether there was support for environmental explanations for the associations among peer deviance, alcohol expectancies, and adolescent alcohol use (Kuther, 2002; Zamboanga et al., 2009), this study used an adoptive and non-adoptive sibling design including individuals assessed in mid-to-late adolescence. Results suggested that the associations between peer deviance, alcohol expectancies, and adolescent alcohol use were primarily environmental in nature. We found substantial shared and nonshared environmental effects of peer deviance on adolescent alcohol use. This replicated and extended results found in studies of twins by Walden et al. (2004) who reported shared environmental influence on the relationship between peer deviance and substance use more broadly defined, and also Burt, McGue, & Iacono (2009) who reported nonshared environmental influence on the covariance between peer deviance and adolescent externalizing behaviors.

Environmental Influence on Adolescent Alcohol use

Following previous research (Han, McGue, & Iacono, 1999; Hopfer, Crowly & Hewett, 2003; Koopsman & Boomsma, 1996), we found moderate shared and nonshared environmental influence on a general measure of adolescent of alcohol use (c^2 =.35, e^2 = . 44), and a non-significant heritability estimate (α^2 = . 21). This general measure covered ever using alcohol, ever getting intoxicated, as well as frequency and quantity of drinking behaviors. In general, measures of alcohol initiation, frequency, and intoxication tend to have greater shared environmental contributions than additive genetic contributions in adolescent samples, which is the reverse of the pattern seen with DSM-based diagnostic measures which show greater genetic than shared environmental effects (Hopfer, Crowly & Hewitt, 2003; Rhee et al., 2003). Moreover, adolescent samples tend to demonstrate greater shared environmental contributions to alcohol and substance use (Maes et al., 1999; McGue et al., 2000) compared to adult samples (Kendler et al., 1999; McGue et al., 1999). Our failure to find significant heritable effects on adolescent alcohol use is thus consistent with the existing literature in that, in the aggregate, indicate at most a small genetic influence on adolescent alcohol use. Also, SIBS was designed to have greater power to detect small shared environmental effects in comparison to power to detect moderate heritable effects by containing approximately twice the number of genetically unrelated siblings than related siblings. Therefore, failure to identify a small heritability as significant is consistent with expectations given the SIBS study design (Buchanan et al., 2009).

Environmental Influences of Peer Deviance and Alcohol Expectancies with Adolescent Alcohol Use

Peer deviance was heritable in this study ($a^2 = .36$), as it has been consistently reported by others (e.g., Agrawal et al., 2010; Fowler et al., 2007; Kendler et al., 2007). This heritability can be explained, at least in part, through gene-environmental correlation (Scarr & McCartney, 1982). For example, adolescents seek out friends based on their similar genetically predisposed traits and interests (like openness to drinking). Indeed, there was significant additive genetic covariance between peer deviance and alcohol expectancies, supporting this notion. However, the influence of peer deviance on alcohol use was predominately explained by shared environmental mechanisms. Specifically, peer deviance explained 43% of the shared environmental influence on alcohol expectancies and 82% of the shared environmental influence on alcohol use.

The shared environment influence of peer deviance on adolescent alcohol use is unlike findings reported by Prescott et al. (2004), who found genetic influence on the relationship between motivations to drink and alcohol abuse/dependence in an adult sample of twins. This is most likely due to the age difference between Prescott's adult sample and our adolescent sample, the difference in the (albeit, related) constructs of alcohol expectancies and motives, and also the difference in how alcohol behavior was assessed in the two studies (diagnosis versus general patterns of use).

Given the difficulty of identifying specific nonshared environmental variables influencing adolescent alcohol use (for a meta-analysis, see Turkheimer & Waldron, 2000), the fact that we accounted for a modest proportion of nonshared environmental effects in addition to shared environmental effects was noteworthy. Turkheimer & Waldron speculated that nonshared environmental influences are too idiosyncratic, personalized, or unstable to detect, or may largely reflect measurement error. Here, however, we found alcohol expectancies significantly explained 7% of the nonshared environmental influence on adolescent alcohol use and together, peer deviance and alcohol expectancies significantly explained 22% of the nonshared environmental variance in alcohol use. These effects are large compared to those reported in other studies aimed at detecting specific nonshared environmental effects on deviant behavior. For instance, Pike et al. (1996) reported 1–2% of the nonshared environmental influence in antisocial behavior was due to family member relationship quality. This comparison raises the possibility that peers may be a greater source of nonshared environmental influence on problem behavior compared to family characteristics. However, considering that the heritability of peer deviance increases with age (Kendler et al., 2007), it remains unclear whether peer deviance would continue to have a nonshared environmental influence in adulthood.

Limitations

There are several limitations to the present study. First, although we demonstrated significant associations among peer deviance, expectancies, and alcohol use, the data were cross-sectional. There is a need for longitudinal analyses to further support the direction of these effects. Second, we do not know how these results would extend to more disordered alcohol use; our measure was broadly defined in terms of quantity and frequency of use. Measures of problem drinking tend to be more heritable than measures of alcohol use (Rhee et al., 2003), which likely would affect results. Third, peer deviance was measured in terms of adolescent self-report. It is unclear how these associations might be affected by utilizing peer report directly (although previous research shows similar validity of both measures, Iannotti & Bush, 1992). Also, not all adolescents in our sample had used alcohol by the follow-up assessment (see Table 1). Alcohol use variables were coded to zero for this group, resulting in inflation of the internal consistency of the estimated latent factor.

The equal environment assumption (EEA) remains critical in all biometric analyses. The EEA assumes that siblings in adoptive and non-adoptive families have rearing environments that are not significantly different from one another. Importantly, whereas biologically related siblings had the same biological mother, adopted sibling pairs had different maternal gestational environments. Furthermore, adopted siblings might vary in ethnicity while non-adoptive siblings were the same ethnicity. Such differences might inflate heritability estimates. The fact that we found the heritability of adolescent alcohol use to be not significantly different from zero, and that the heritability of peer deviance that we found follow previous findings (Agrawal et al., 2010; Fowler et al., 2007; Kendler et al., 2007) help to ensure that such differences in adoptive and non-adoptive families do not likely impact study findings.

In the genetically unrelated sibling group, we compared sibling correlations for study phenotypes across same-ethnicity ($n = 282$) and mixed-ethnicity siblings ($n = 125$) and found no significant differences. We also found no significant differences in sibling correlations across White-only siblings ($n = 78$) and Asian-only siblings ($n = 177$) in this group, suggesting that little impact of shared ethnicity on variance component estimates. Finally, even though adoptive parents tended to have higher SES, more education, and less disinhibitory psychopathology than non-adoptive parents, this restriction in range did not influence the adoptive sibling correlation in substance use and related externalizing behaviors (McGue et al., 2007). Together, this suggests that environmental differences between adoptive and non-adoptive families had minimal impact on the sibling correlations studied here. Nonetheless, replication of these results in other genetically informed samples would be helpful to confirm study findings.

Conclusion

This study demonstrated substantial shared environmental influence of peer deviance on adolescent alcohol use and modest nonshared environmental influence of alcohol expectancies on adolescent alcohol use. This research adds to a growing body of research identifying specific environmental factors associated with adolescent substance use (Baker et al., 2012; Gillespie et al., 2009; Latendresse et al., 2010; Walden et al., 2004), and shows peer deviance and alcohol expectancies appear to influence adolescent alcohol use primarily through environmental mechanisms. However, more research is needed to better understand environmental influences on adolescent substance use and related adjustment problems. Longitudinal research is helpful to further infer the direction of influence among these variables. Variables to target in future research include those specific to adolescent social networks, such as peers, and other strong correlates of adolescent alcohol use, including parenting (Latendresse et al., 2010), and sibling factors (Samek & Rueter, 2011; Slomkowski et al., 2005).

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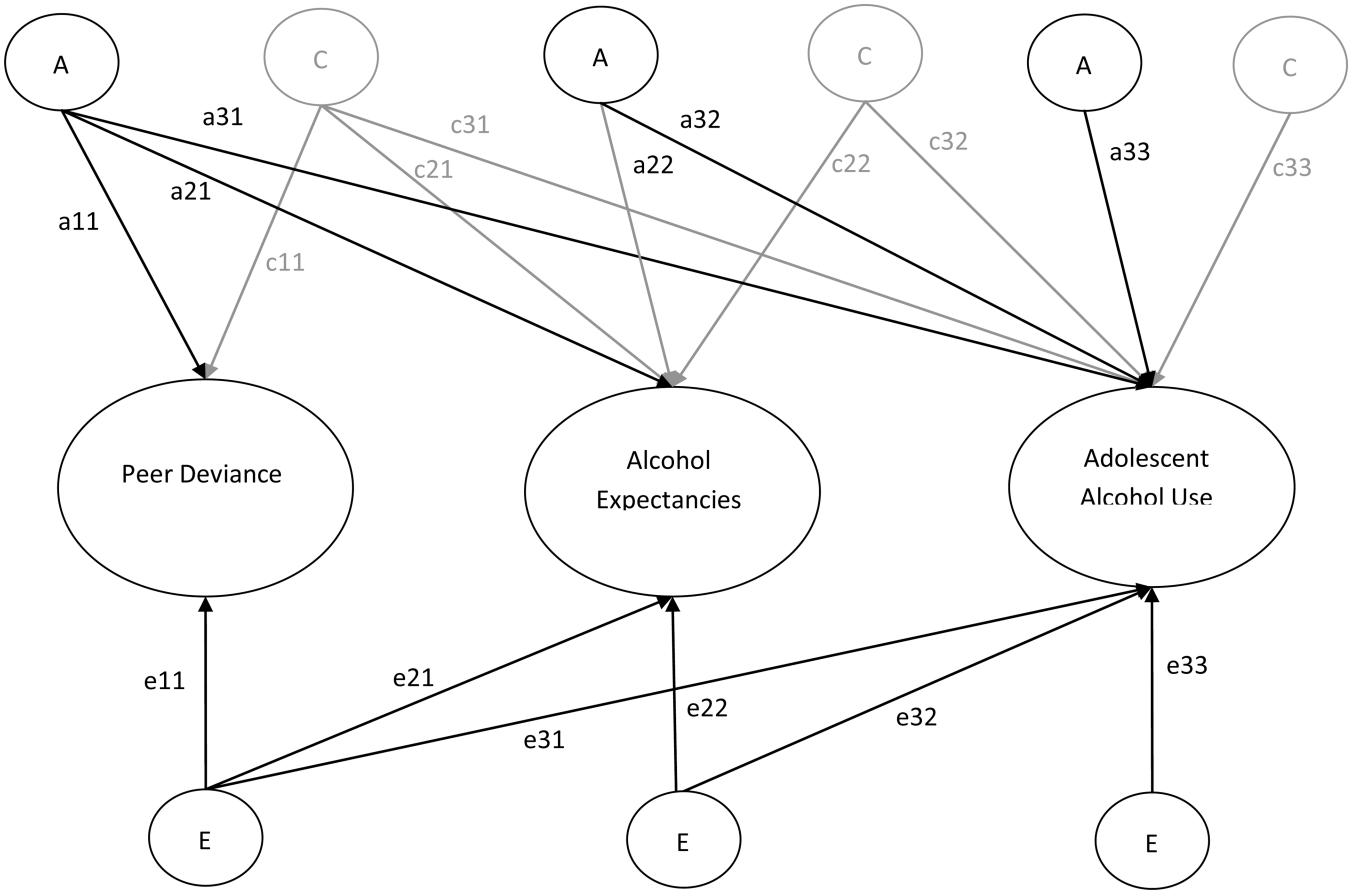

Figure 1. Path Diagram for the Full Cholesky Decomposition

Variance of each phenotype (Peer Deviance, Alcohol Expectancies, Adolescent Alcohol Use) is decomposed into additive genetic effects (A1, A2, A3), shared environmental effects (C1, C2, C3), and nonshared environmental effects (E1, E2, E3). C paths and labels are shown in gray to ease the clarity of presentation. Paths, represented by lowercase letters followed by two numbers (e.g., a11, a21, a31) are squared to estimate the proportion of variance accounted for. For example, squaring and summing paths c31, c32, and c33 calculates the total shared environmental variance in adolescent alcohol use.

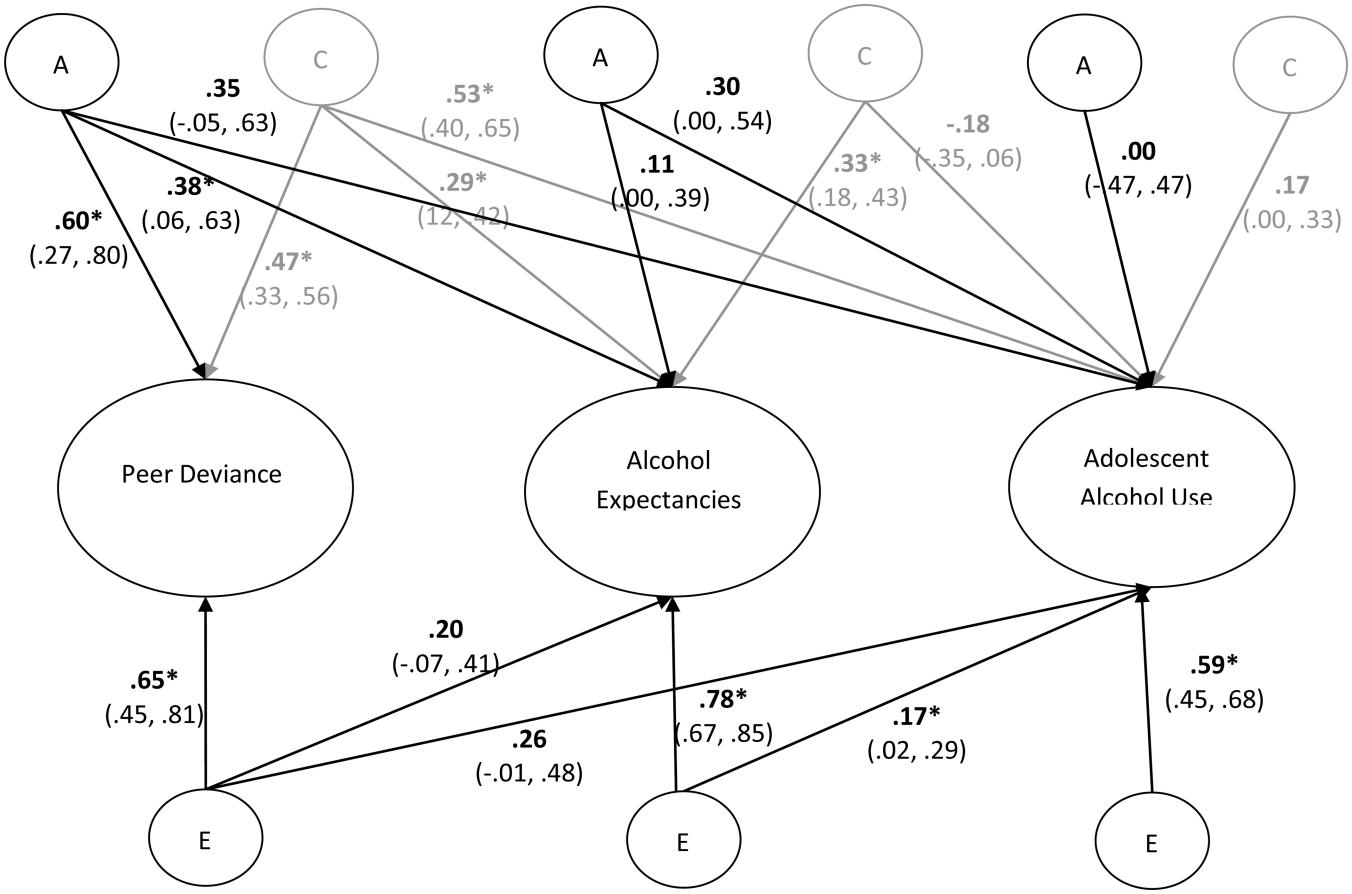

Figure 2. Standardized Path Coefficients for the Full ACE Cholesky Decomposition

The variance within and the covariance amongst study variables are decomposed into genetic (A), shared environmental (C) and nonshared environmental components (E). C paths and labels are shown in gray to ease the clarity of presentation. Ninety-five percent confidence intervals (CIs) are also presented. Significant paths are indicated by CIs not crossing zero and also by * $p < .05$.

Table 1

Mean levels of Adolescent Alcohol Use (*N*= 613 Sibling Pairs)

	Range	Elder Sibling M_{age} = 19.4 (*SD* = 1.7)	Younger Sibling M_{age} = 17.1 (*SD* = 1.8)
Ever used alcohol	0 – 1	.76 (.43)	.54 (.50)
Frequency of alcohol use[a]	0 – 9	3.32 (2.51)	1.89 (2.22)
Ever intoxicated	0 – 1	.65 (.48)	.39 (.49)
Frequency of intoxications[a]	0 – 9	2.49 (2.36)	1.35 (2.04)
How often drink to get drunk[b]	0 – 5	2.41 (2.05)	1.40 (1.95)
Max drinks in one sitting[c]	0 – 10	5.05 (3.78)	3.13 (3.76)

NOTE: Standard deviations are shown in parentheses.

[a]Frequency items answered on a scale of 0 = *Never* to 9 = *Every day or nearly every day*

[b]how often they drank to get drunk answered on a scale of 0 = *Never* to 5 = *Nearly every time or every time*

[c]max drinks answered on a scale of 0 = *0 drinks* to 10 = *10+ drinks*.

Table 2

Age, Sex, and Ethnicity Corrected Correlations and Descriptive Statistics of Study Data (N = 613 Eligible Sibling Pairs)

	1	2	3	4	5	*M*	*SD*	% Complete
Elder Sibling								
1. Peer deviance	--					.69	.37	90.5
2. Alcohol Expectancies	.42 (.35, .50)	--				1.43	.45	75.7
3. Alcohol Use	.58 (.52, .64)	.53 (.46, .59)	--			5.11	1.08	77.7
Younger Sibling								
4. Peer deviance	.29 (.21, .36)	.21 (.13, .30)	.31 (.23, .39)	--		.58	.42	92.0
5. Alcohol Expectancies	.14 (.05, .22)	.23 (.14, .31)	.27 (.19, .36)	.53 (.47, .59)	--	1.23	.50	81.4
6. Alcohol Use	.25 (.17, .34)	.21 (.12, .30)	.39 (.31, .47)	.66 (.60, .70)	.59 (.53, .65)	4.47	1.09	80.9

NOTE: All correlations were significant at $p < .01$. 95% Confidence Intervals (CIs) for all correlations are presented in parentheses.

Table 3

Total Genetic and Environmental Contributions to Peer Deviance, Alcohol Expectancies, and Alcohol Use

	% Variance			% of ACE Variance in Alcohol Use accounted for by Peer Deviance	% of ACE Variance in Alcohol Use accounted for by Alcohol Expectancies	% of ACE Variance in Alcohol Use accounted for by Peer Deviance and Alcohol Expectancies
	Peer Deviance	**Alcohol Expectancies**	**Adolescent Alcohol Use**			
A	$a11^2$	$a21^2 + a22^2$	$a31^2 + a32^2 + a33^2$	$(a31^2 +)/(a31^2 + a32^2 + a33^2$	$(a32^2)/(a31^2 + a32^2 + a33^2$	$(a31^2 + a32^2/(a31^2 + a32^2 + a33^2$
	.36*	**.16***	.21	.56	.44	1.0
	(.07, .65)	(.01, .41)	(.00, .49)	(.00, 1.0)	(.00, 1.0)	(.00, 1.0)
C	$c11^2$	$c21^2 + c22^2$	$c31^2 + c32^2 + c33^2$	$(c31^2)/ (c31^2 + c32^2 + c33^2$	$(c32^2)/ (c31^2 + c32^2 + c33^2$	$(c31^2 + c32^2)/ (c31^2 + c32^2 + c33^2$
	.21*	**.19***	**.35***	**.82***	.10	**.92***
	(.11, .32)	(.09, .29)	(.24, .44)	(.55, 1.0)	(.00, .36)	(.68, 1.0)
E	$e11^2$	$e21^2 + e22^2$	$e31^2 + e32^2 + e33^2$	$(e31^2)/ (e31^2 + e32^2 + e33^2$	$(e32^2)/ (e31^2 + e32^2 + e33^2$	$(e31^2 + e32^2)/(e31^2 + e32^2 + e33^2$
	.43*	**.65***	**.44***	.15	**.07***	**.22***
	(.21, .66)	(.45, .85)	(.22, .65)	(.00, .41)	(.001, .41)	(.01, .48)

NOTE: A, C, and E represent genetic, shared environmental, and non-shared environmental influences. Please see Figure 1 for a reminder of path labels and Figure 2 for the accompanying standardized estimates from the Cholesky decomposition. Standardized coefficients and 95% confidence are presented in parentheses. Significant proportions of variance explained are denoted by the 95% confidence intervals not crossing 0; these estimates are bolded for ease of presentation. The first three columns represent the proportions of genetic and environmental variance within each variable; each column sums to 100% (allowing for rounding error). The final three columns represent that proportion of genetic, shared, and non-shared environmental variance in adolescent alcohol use that is accounted for by peer deviance and alcohol expectancies.