# Characteristics of the Sibling Relationship that Predict Sibling Similarity in Adolescent Alcohol Use: Exploring Potential Shared Environmental Effects

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**Dedication**

This research is dedicated to everyone out there that has had problems with alcohol and substance use, particularly those who faced these problems in the early and formidable years of adolescence. It is also dedicated to all siblings, who are no doubt important in the socialization of children and adolescents. I especially dedicate this to my sister and brother, Shari and Joe Samek, for their protection, loyalty, and understanding.

**Abstract**

Using a genetically informative sibling-pair design (*N* = 613), three studies were conducted to extend knowledge on how siblings influence adolescent alcohol use. Study 1 utilized latent class analysis to determine if patterns of sibling similarity in alcohol use could be detected. The 3-class model was the best fitting for Wave 1, and the 4-class model was the best fitting for Wave 2. Study 2 established the predictive validity of Study 1 by testing characteristics of the sibling relationship (closeness, conflict, communication) at Wave 1 as predictors of sibling similarity in alcohol use patterns at Wave 2. Communication and conflict predicted the 4-class model, but closeness did not. Study 3 utilized Cholesky decomposition in order to decompose the genetic and environmental contributions to the correlation between mom, dad, and sibling involvement in their association with adolescent alcohol use. There were few significant findings, potentially due to power issues. Altogether, this research demonstrates sibling influences on adolescent alcohol use and provides guidelines for future research in my general program of research.

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**Study 1: Classifying Patterns of Sibling Similarity in Adolescent Alcohol Use**

In the United States, adolescent alcohol use remains highly prevalent. It is estimated that 72% of high school students have had at least one drink, 41% currently use alcohol, and 24% have had 5 or more drinks in 24 hours (Center for Disease Control and Prevention; CDC, 2010). While experimentation with alcohol is somewhat normative in the United States (Newcomb & Bentler, 1989), early and frequent alcohol use in adolescence is associated with a host of problems. For example, it is estimated that 21% of high school students use alcohol for the first time before the age of 13 (CDC, 2010) and that youth who start drinking before age 15 are five times more likely to develop alcohol abuse or dependence later in life compared to those who begin drinking later (Hingson, Heeren & Winter, 2006; Office of Applied Studies, 2004).

Siblings are often similar in their alcohol and substance use (de Leeuw, Scholte, Sargent, Vermulst & Engels, 2010; Fagan & Najman, 2005; McGue, Sharma & Benson, 1996; Rende, Slomkowski, Lloyd-Richardson & Niaura, 2005; Rowe & Gulley, 1992), suggesting a possible causal link to sibling similarity in alcohol use. Siblings are often more similar in their substance use compared to parents and children (Fagan & Najman, 2005; Needle, McCubbin, Wilson, Reineck, Lazar, & Mederer, 1986; Rowe & Gulley, 1992). Furthermore, previous research has demonstrated that the correlation between parents and their children in regards to alcohol use may be largely due to shared genes, while the correlation between siblings may be largely explained by shared social environment (McGue et al., 1996). These findings suggest that something about sibling context is leading to their similarity in alcohol use that could potentially be targeted for intervention or prevention.

**Methods of Studying Sibling Similarity**

Studies that examine sibling similarity in substance use typically utilize a correlational method of analysis (Conger & Rueter, 1996; McGue et al., 1996; Rende et al. 2005; Rowe & Gulley, 1992; Samek & Rueter, 2011). For example, correlations for same-sex siblings have been reported as .36 for alcohol use (McGue et al., 1996), and as high as .66 and .84 for same-sex siblings that have mutual friends (.66 for boys, .84 for girls; Rowe & Gulley, 1992). This research demonstrates that siblings tend to be similar; however, it remains unclear if groups of siblings can be detected based on their similarity in alcohol use. For example, there may be unique groups of siblings that reflect similarity in *low* levels of alcohol use versus similarity in *high* levels of alcohol use. Distinguishing such groups and analyzing the potential differential predictors of groups may be clinically significant to later intervention and prevention work. In this study, I aim to explicitly test whether differential patterns can be detected by utilizing latent class analysis (LCA).

LCA is a specific type of structural equation modeling that is used to uncover unobserved heterogeneity and determine meaningful groups of people that have similarity on some measured response (McCutcheon, 1987; Nyland, Asparouhov, & Muthen, 2007). LCA determines whether groups of cases can be distinguished on some (observed) response to be part of a general latent class (i.e., the multiple observed cases are reduced to single classes). LCA is considered to be an improved cluster analysis because it uses the concept of statistical likelihood of class membership (i.e., probability) rather than absolute membership (i.e., *k*-means method; Francis, 2006).

**Rationale for Analyzing Multiple Patterns of Sibling Similarity**

There is reason to believe there is a detectable difference between low and high patterns of sibling similarity in alcohol use. One hypothesized class of siblings could be described as a pattern of high alcohol use amongst both adolescent siblings. This hypothesis is supported by the fact that a large number of youth currently use alcohol (41%; CDC, 2010). On the other hand, the majority of youth (59%) do not use alcohol (CDC, 2010). Therefore, it is possible that a second class of siblings could be described as a pattern of relatively low alcohol use amongst both siblings. Additionally, older adolescents are more likely to use alcohol compared to younger adolescents (Johnston, O'Malley, Bachman, & Schulenberg, 2011). Therefore, it is reasonable to assume a third class of siblings may reflect high use amongst elder siblings, but low use amongst younger siblings. If multiple patterns of sibling similarity in alcohol use are detected by LCA, results may have clinical relevance after further testing is done to validate such patterns. The aim of this study was to lay the foundation for later validation studies by first describing whether such patterns can indeed be detected.

Although I know of no studies that have examined patterns of alcohol use amongst adolescent siblings, there are a few studies that have examined sibling patterns in other outcomes, such as relationship types (e.g., Gold, 1989; Stewart, Kozak, Tingley, Goddard, Blake, & Cassel, 2005), and similarity and difference in art and athletics (Whiteman, McHale, & Crouter, 2007). Moreover, some of these studies have found differential predictors for classes. Whiteman et al.'s study found that characteristics of relationship quality differentially predicted similarity versus difference in artistic and athletic interests. These results indicate the importance of analyzing specific patterns in the sibling relationship in order to better understand what may predict those patterns.

Finally, research has shown that siblings are more similar in their substance use if they are close in age, and to a lesser extent if they are the same-sex, or genetically similar (McGue et al., 1996; Rowe & Gulley, 1992; Slomkowski et al., 2001). Therefore these variables were used as covariates in study analyses.

### Study 1 Summary

In order to extend knowledge on sibling influences on adolescent alcohol use, this study examined whether patterns of sibling similarity in adolescent alcohol use could be detected using LCA. It was expected that a 3-class model would be statistically and theoretically superior to a 1-, 2-, 4-, and 5- class model, describing sibling pairs who are (a) similar in *high* alcohol use, (b) similar in *low* alcohol use, and (c) siblings with *high* use by elder siblings and *low* use by younger siblings. All analyses were controlled for sibling age difference, sex composition (same versus opposite), and genetic similarity (genetically versus not genetically related). Two waves of data were analyzed to examine potential age cohort effects. Findings will provide implications for alternate ways of investigating sibling similarity in substance use patterns.

## Method

### Data Source

Data from the Sibling Interaction and Behavior Study (SIBS; McGue, Keyes, Sharma, Elkins, Legrand, Johnson, & Iacono, 2007) were used to conduct Studies 1, 2, and 3. The SIBS is a genetically informed, longitudinal study (Wave 1 data collected in 1998, Wave 2 data collected 3.5 years later), based at the University of Minnesota, Minneapolis – Saint Paul.

At Wave 1, a total of 617 families participated. Families had to have at least two children within five years of age. In 285 families, both of those children were adopted and were not genetically related to his/her parents or each other (ADOPT group). In 208 families, both children were the biological offspring of his/her parents (BIO group). Finally, in 124 families, one child was adopted and not genetically related to his/her parents, and one child was the biological offspring of his/her parents (ADOPT-BIO group). Adoptive families were first identified through three large adoption agencies. Adoptive family eligibility included having an adopted child between the ages of 11 and 21 who had been permanently placed into the adoptive home prior to age 2 ($M_{\text{age}}$ = 4.7 months, *SD* = 3.4 months), and a second adolescent in the home who was not biologically related to the adopted adolescent. Biological families were then identified using publicly available birth certificates. These families were targeted based on matching age and sex of target adopted children. Participation rates between non-adoptive (57%) and adoptive (63%) families were not significantly different and comparisons of parents' occupation, education, marital status, and DSM IV disorders in children at Wave 1 showed that the study sample was generally representative of the Twin Cities metro region (McGue et al., 2007). On average, 56% of participating parents were college-educated (44% nonadoptive parents, 61% adoptive parents).

At Wave 2 (3.5 years later), four families were deemed ineligible because either one of the children in the family (a) had a developmental delay, (b) had died, or (c) was adopted but biologically related to their adopted sibling, leaving a total of 613 eligible families (those 4 ineligible families were removed from Wave 1 and 2 analyses). Ninety-four percent of those eligible families participated at Wave 2. The majority of parents were married at Wave 1 (91.1%) and Wave 2 (89.4%). Consistent with the demographics of the area, 96% of parents were Caucasian. Additional information about SIBS is available in McGue et al., 2007.

**Participants**

A total of 1,158 adolescents were used for the present analyses. Out of the 1,158 adolescents, 502 were non-adopted and 656 were adopted adolescents. Among adoptees, 484 were internationally adopted (Asian: 89%; female: 61%), and 172 were domestic adoptions (Caucasian: 77%; female: 42%). For younger siblings, $M_{age}$ at Wave 1 was 13.8 years ($SD$ = 1.6), at Wave 2 $M_{age}$ = 17.1 years ($SD$ = 1.8). For elder siblings, $M_{age}$ at Wave 1 was 16.1 years ($SD$ = 1.5), at Wave 2 $M_{age}$ = 19.4 years ($SD$ = 1.7). The average age difference in sibling pairs was 2.3 years ($SD$ = .89). Sex composition of the sibling pairs is as follows: 25.6% brothers, 35.1% sisters, 23.5% younger sisters – elder brothers, and 15.8% younger brothers – elder sisters.

**Procedure**

At Wave 1, participating families came to the research lab for about a day long assessment. Multiple assessments were completed, including diagnostic interviews and self-report surveys. Wave 2 procedures mirrored Wave 1; however, only one parent (usually the mother) came to the lab with both children. Family members were paid $50 for participating in Wave 1, and $100 for Wave 2.

**Measures**

**Covariates.** Family adoption status was coded so that 1 represented families with siblings that were both adopted (*ADOPT-ADOPT*), 2 represented families with siblings that were both full biological offspring of their parents (*BIO-BIO*), and 3 represented families where one sibling was adopted, and the other was the biological offspring of their parents (*ADOPT-BIO*). Sibling genetic similarity was coded so that 1 = *genetically related* (BIO-BIO siblings), and 2 = *genetically unrelated* (ADOPT-ADOPT and ADOPT-BIO siblings). Sibling age difference was assessed as the absolute value in difference between sibling ages. Sibling sex composition was coded so that 1 = *same sex*, 2 = *mixed sex*.

**Alcohol use.** Adolescent Alcohol use was measured using the *Computerized Substance Use Assessment (CSA*; based on Christiansen & Goldman, 1983; Schafer & Brown, 1991), which was completed by both siblings, at both Waves 1 and 2. Siblings were asked if they have ever used alcohol (without parent's consent, *yes* = 1, *no* = 0), frequency of alcohol use in the last 12 months (answered on a scale of 0 (*Never*) to 9 (*Every day or nearly every day*)), ever been intoxicated (1= *yes* = 0, *no* = 0), 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 in the past 12 months (0 (*0 drinks*) to 10 (*10+ drinks*)). These measures were used as indicators of a latent class variable for each time point.

**Missing Data Analysis**

As described in Table 1.1, the proposed study measures have little missing data. For example, across Wave 1 measures, nearly 100% of the data is complete, and at least 3 quarters of the data is complete at Wave 2. Missing data at Wave 2 is due to participant non-participation (~5% of elders and youngers) or lack of completing computerized assessment (~16% for both elders and youngers). Chi-Square analyses revealed no statistically significant differences between those who dropped out versus stayed in the study at Wave 2 across adoptive status, ethnicity, and sex. Chi-square analyses also revealed no statistically significant differences between those missing self-reported alcohol data or those with complete data at Wave 2. Missing data were handled using Full Information Maximum Likelihood (FIML), which has proven superior in simulation studies as accurately representing the sample data compared to listwise, pairwise deletion, similar response pattern, and mean imputation (Enders & Bandalos, 2009, Johnson & Young, 2011).

**Analysis Plan**

In order to detect patterns of sibling similarity in alcohol use based on study measures, I used LCA. Using MPLUS, 6.0 (Muthén & Muthén, 1998-2010), I compared 1-, 2-, 3-, 4-, and 5-classes of sibling alcohol use using both elder and younger sibling reports of alcohol use at both Waves 1 and 2 (see Figure 1.1). Classes were detected based on all measures of alcohol use described above. I used a combination of theoretical and statistical criteria to determine the best fit. Statistical criteria included examining the

Akaike Information Criterion (AIC), the adjusted Bayesian Information Criterion (BIC), and the Lo-Mendell-Rubin adjusted LRT (Nylund, Asparouhov, & Muthén, 2007). A large decrease in the BIC and AIC values when the class number was increased indicates a better model fit for the model with the additional class. The BIC has a larger penalty than the AIC for increasing model parameters. The null hypothesis for the LRT test is that a model with one less class is a better fitting model. Theoretical criteria include making sense of the mean loadings on each of the observed classes (expected: high means across indicators for both siblings in one class, low means in another class, and a class with high means amongst elders, and low means amongst youngers, relative to sample means overall), as well as the significance of covariates (matching previous research).

## Results

### Wave 1 Sibling Alcohol Use Patterns Results

Table 1.2 describes model fit and summary statistics for the 1-, 2-, 3-, 4-, and 5-class models at Wave 1. Altogether, the 3-class model was the best statistical and theoretical fit for Wave 1.

Compared to the 1-class model, the 2-class model had a substantial drop in AIC and adjusted BIC values ($\Delta$AIC = 5,114.00, $\Delta$BIC = 5,091.63), as well as a significant adjusted LRT result, $p < .0001$, suggesting the 2-class model is a better statistical fit than the 1-class model. In this 2-class model, 20% of siblings were placed in the high alcohol use by both siblings class (HIGH-HIGH), and 80% in the low alcohol use by both siblings class (LOW-LOW). It should be noted here that the LOW-LOW alcohol use class classified both adolescents with lower levels of alcohol use compared to the overall sample, as well as adolescents who altogether abstain from alcohol use.

I hypothesized the 3-class model would be the best fitting statistical and theoretical model. Indeed, fit statistics show improved fit over the 2-class model, $\Delta$AIC = 3,075.06, $\Delta$BIC = 3,051.42, adjusted LRT $p < .05$. As hypothesized, there was a class of LOW-LOW alcohol use (76%), a class of HIGH-HIGH alcohol use (7%), and a class depicting high alcohol use amongst elders and low alcohol use among youngers (16%; eHIGH-yLOW). Compared to the HIGH-HIGH (the HIGH-HIGH alcohol use was the reference group), the eHIGH-yLOW class was significantly predicted by sibling age difference ($t = 2.55$, $p < .03$), such that for every unit increase in age difference, siblings have 1.7 times the odds of being placed in the eHIGH-yLOW alcohol use class.

Compared to the 3-class model, the 4-class model showed little in statistical fit, $\Delta$AIC = 973.76, $\Delta$BIC = 950.14, adjusted LRT $p = .65$. In this 4-class model, the majority of siblings were classified in the LOW-LOW class (76%). Sixteen percent of the sample was classified in the eHIGH-yLOW class. Finally, there was an equal split of proportions placed in the classes reflecting HIGH-HIGH with little theoretical difference (see Table 1.2, class 2 versus class 3, each 4% of the sample). Compared to the eHIGH-yLOW (Class 4), siblings in both the 2$^{nd}$ and 3$^{rd}$ classes were more likely to be close in age (class 2: $t = -1.95$, $p = .05$, *OR* = .57, *Inverse OR* =1.75; class 4: $t = .2.07$, $p < .05$, *OR* = .59, *Inverse OR* = 1.69). These same results for these two classes further justifies little theoretical distinction and relevance for a 4-class model of sibling similarity in alcohol use at Wave 1.

Finally, the 5-class model had even worse fit than the 4-class model, ΔAIC = 383.01, ΔBIC = 359.38, adjusted LRT $p = .76$. Altogether, results indicate the 3-class model is the best statistical and theoretically fitting model, supporting the expected hypothesis.

**Wave 2 Sibling Alcohol Use Patterns Results**

Table 1.3 describes model fit and summary statistics for the 1-, 2-, 3-, 4-, and 5-class models at Wave 2. Unlike the Wave 1 analysis, the Wave 2 analysis provided the best statistical and theoretical fit for the 4-class model of sibling similarity in alcohol use.

Compared to the 1-class model, the 2-class model had a substantial drop in AIC and adjusted BIC values (ΔAIC = 5,133.31, ΔBIC = 5,112.17), as well as a significant adjusted LRT result, $p < .001$, suggesting the 2-class model is a better statistical fit than the 1-class model. In this 2-class model, 31% of the sample was classified in the class depicting high alcohol use amongst both siblings (HIGH-HIGH), and 69% were classified in the class depicting low alcohol use amongst both siblings (LOW-LOW). Compared to the HIGH-HIGH class, the LOW-LOW class was significantly predicted by sibling age difference ($t = 3.28$, $p < .001$), and sibling genetic similarity ($t = -1.95$, $p = .05$). For every unit increase in age difference, siblings were 1.45 times more likely to be placed in the LOW-LOW class compared to the HIGH-HIGH class. On the other hand, genetically related siblings were 1.47 times more likely to be placed in the HIGH-HIGH class compared to the LOW-LOW class.

I hypothesized the 3-class model would be the best fitting statistical and theoretical model. Indeed, fit statistics show improved fit over the 2-class model, ΔAIC =

1,671.61, ΔBIC = 1,655.43, adjusted LRT $p < .03$. As hypothesized, there was a class of LOW-LOW (31%), a class of HIGH-HIGH (34%), and a class depicting high use amongst elder siblings and low use amongst younger siblings (eHIGH-yLOW; 35%). Compared to the HIGH-HIGH class, the eHIGH-yLOW was significant predicted by sibling age difference ($t = 3.24$, $p < .001$). For every unit increase in sibling age difference, siblings were 1.57 times more likely to be placed in the eHIGH-yLOW class.

However, the 4-class model was an even better statistical fitting model than the 3-class model at Wave 2, ΔAIC = 1,076.41, ΔBIC = 1,056.52, adjusted LRT $p < .001$. The difference with this model compared to the 3-class model is that an additional class depicting “high” alcohol use amongst younger siblings and “low” alcohol use amongst elder siblings was detected (eLOW-yHIGH; 7% of the sample). It makes theoretical sense that a class such as this emerged given the nature of this sample at Wave 2 consisting of older adolescents (younger sibling $M_{age}$ at Wave 2 was 17.1, elder sibling $M_{age}$ at Wave 2 was 19.4), as compared to younger adolescents at Wave 1 (i.e., ~13 to 16 years old).

In the 4-class model, siblings in the HIGH-HIGH class were more likely to be genetically related compared to the LOW-LOW class ($t = 2.08$, $p < .05$, $OR = 1.69$), the eHIGH-yLOW class ($t = 1.98$, $p < .05$, $OR = 1.75$), and were trending to be more likely to be genetically related compared to the eLOW-yHIGH class ($t = 1.67$, $p < .10$, $OR = 1.92$). Moreover, compared to the HIGH-HIGH class, sibling age difference was associated with the eHIGH-yLOW class ($t = 2.33$, $p < .03$), such that for each unit increase in age difference, siblings were 1.45 times more likely to be placed in the eHIGH-yLOW class.

Finally, although the 5-class model showed some model fit improvements, the change in AIC and the adjusted BIC were quite small, and the adjusted LRT *p*-value, although significant at $p < .05$, was increasing in nature. Moreover, the class descriptions were theoretically irrelevant when comparing the 4$^{th}$ and 5$^{th}$ class as they both depicted "high" alcohol use amongst both elder and younger siblings with little theoretical difference.

Altogether, for Wave 2, the 4-class model appears to be the best fitting statistical and theoretical model compared to 1 through 5 class models. This contradicts study hypotheses and results found for Wave 1, potentially due to the increased age of younger siblings at Wave 2.

**Post-Hoc Analysis**

Previous analyses examined whether latent classes could be predicted by whether or not the siblings were genetically versus not genetically related. *Ex-post-facto*, I examined the possibility that latent classes would be predicted by groups of siblings who were classified by family adoptive status (1=ADOPT-ADOPT, 2 = BIO-BIO, 3 = ADOPT-BIO). This possible association is supported by previous research has found that adoptees tend to have greater adjustment problems compared to non-adoptees (Juffer & van IJzendoorn, 2005; Keyes, Sharma, Elkins, Iacono, & McGue, 2008). Results showed no significant association between this grouping and the 3-class model of alcohol use at Wave 1, or between this grouping and the 4-class model of alcohol use at Wave 2.

# Discussion

Previous research has long demonstrated a strong, positive correlation between siblings on adolescent alcohol and substance use (e.g., McGue et al., 1996; Rende et al. 2005; Samek & Rueter, 2011). This study used an alternate way of measuring sibling similarity in adolescent alcohol use by using latent class analysis (LCA). This was done in order to lay the foundation for future research to examine if predictors differentially predicted sibling similarity in alcohol use groups, which may be useful in prevention and intervention work. Before examining the possibility of alternate predictors, it was first necessary to determine whether patterns of adolescent sibling similarity in alcohol use could indeed be detected. This study found evidence to support a 3-class model of sibling similarity in alcohol use in early adolescence, and a 4-class model of sibling similarity in late adolescence.

## Early versus Late Adolescence

Wave 1 results implicated a 3-class model of sibling similarity in alcohol use as being the best statistical and theoretical fit in early adolescence, when younger siblings were on average 13 ½ years old and elder siblings were roughly 16 years old. As hypothesized, and in comparison to sample means, there was a class representing “low to abstinent” use amongst both elder and younger siblings, a class representing relatively “high” use amongst both elder and younger siblings, and a class representing relatively “high” use amongst elder siblings and “low to abstinent” use amongst younger siblings. By the nature of elder siblings being older than younger siblings, and that alcohol use increases with adolescent age (Johnston et al., 2011), this result was not surprising.

On the other hand, Wave 2 results implicated a 4-class model of sibling similarity in alcohol use as being the best statistical and theoretical fit. In addition to a class representing "high" use amongst both siblings and a class representing "low" use amongst both siblings, there were also two classes representing differential patterns of use amongst siblings. There was a class demonstrating "high" use amongst elders and "low to abstinent" use among younger siblings (eHIGH-yLOW, like Wave 1), but also, a smaller proportion of siblings classified in a class where younger siblings had "high" use and elder siblings had "low to abstinent" use (7%, eLOW-yHIGH). In order to make sense of these findings, I will next explore how covariates predicted differential sibling alcohol use patterns.

**Sibling Age Difference, Sex Composition, and Genetic Similarity**

Previous research has found siblings are more similar in various outcomes if they are close in age (Buchanan, McGue, Keyes, & Iacono, 2009; McGue et al., 1996; Trim, Leuthe, & Chassin, 2006), and indeed, this study replicated these findings for both Wave 1 and Wave 2 analyses. Siblings further apart in age were more likely to be placed in the class reflecting differential patterns of alcohol use (elder having greater alcohol use than the younger), compared to siblings that both had similar levels of "high" alcohol use.

Sibling sex composition did not significantly predict either Wave 1 or Wave 2 class models. Previous research has found some support for same sex siblings being more similar in substance use (McGue et al., 1996; Rowe, Rodgers & Meseck-Bushey, 1992), although other research has not demonstrated such an effect (Samek & Rueter, 2011; Slomkoski et al., 2005). At this point, it remains unclear if sibling sex composition consistently influences adolescent alcohol use behaviors. This study's findings suggest that siblings are often similar in their adolescent alcohol use regardless of their sex composition.

Finally, at Wave 2, siblings that were genetically related were more likely to be placed in the class reflecting "high" use by both siblings, compared to every other class. This may reflect a potential genetic contribution towards high alcohol use in late adolescence. This fits with previous research that has implicated the heritability of alcohol and substance use (Hopfer, Crowly, & Hewitt, 2003; Han, McGue & Iacono, 1999; Prescott, Madden, & Stallings, 2006), as well as documenting that genetic influence of alcohol and substance use becomes stronger as adolescents become older (Kendler, Schmitt, Aggen & Prescott, 2008).

Previous research has been mixed in terms of the association between sibling genetic similarity and similarity in substance use (McGue et al., 1996; Samek & Rueter, 2011). Earlier research tended to note slightly stronger associations amongst genetically related siblings, but ones that were not significantly different from genetically unrelated siblings. One reason for the lack of findings in earlier research may be due to measurement and analytic plan. Rather than using a continuous measure of alcohol use in a correlational analysis, further distinction was made by using LCA in distinguishing between low and high alcohol use groups.

Additionally, post-hoc analyses showed that this result was not affected by collapsing the two groups of genetically unrelated siblings together (ADOPT-ADOPT, ADOPT-BIO); adoptees were not more likely to be similar in high alcohol use compared to non-adoptees, which previous research might suggest (Juffer & van IJzendoorn, 2005; Keyes et al., 2008); rather, genetically related siblings were more likely to be similar in their alcohol use compared to genetically unrelated siblings. This further supports the heritability explanation.

**Future Research**

This study was one of the first to demonstrate patterns of sibling similarity in adolescent alcohol use could be detected using LCA. Covariates showed some validity of detected patterns, however future research is needed to replicate and extend these initial findings. A logical next step in this program of research is to "fine- tune" our understanding of what predicts patterns of similarity in alcohol use amongst adolescent siblings. This will give us a better understanding of sibling influences on adolescent alcohol use, which can later inform prevention and intervention work in order to reduce or demote alcohol use amongst adolescence.

For example, we know from previous research that sibling closeness and warmth helps explain the sibling similarity in substance use (Rende et al., 2005), and that this occurs largely through shared environmental mechanisms when siblings feel close (Slomkowski et al., 2005), yet we do not know how closeness may differentially predict *low* versus *high* substance use classes. We also know from previous research that there is a positive correlation between sibling conflict and adjustment outcomes such as depression and externalizing problems (Pike, McGuire, Hetherington, Resiss, & Plomin, 1996), but again, we do not know how conflict may differentially predict different patterns of sibling alcohol use.

Additional research that would be useful would tackle what predicts the change in sibling similarity in alcohol use classes from early to late adolescence. This could be done through the use of transition analysis. Finally, a similar LCA approach could be used to examine sibling similarity in other domains, such as other types of substance use (e.g., stimulants, marijuana) or adjustment outcomes (externalizing, depression). Analytic plans such as this have been conducted in regard to similarity in artistic and athletic abilities (Whiteman, McHale, & Crouter, 2007), as well as classifications of sibling relationship types (Gold, 1989; Stewart et al., 2005).

**Limitations**

Study findings can be generalized to the population it was sampled from. The sample was representative in terms of parents education, income, and ethnicity compared to the Midwestern population it was sampled from. However, adolescents in this sample were a higher proportion of internationally adopted, Asian females. The majority of siblings were from 2-parent, White, heterosexual married families. Further research is needed to replicate findings across a variety of sibling pair types (e.g., step-, blended), and family structures (e.g, single) in order to determine if findings may apply to these groups. Also, in this study, genetic similarity was measured in terms of adoption status. Other variations of genetic similarity (such as monozygotic versus dizygotic twins, step versus half siblings) were not examined here.

**Conclusion**

In summary, this study found evidence for alternate patterns of sibling similarity in adolescent alcohol use, possibly dependent on adolescent age. A 3-class model depicting “high” use by both siblings in one class, “low to abstinent” use by both siblings in another class, and a third class depicting “high” use by elders and “low to abstinent” use by youngers seems particularly applicable in younger adolescence (ages 13 to 16). However, a 4-class model following the same description above with one additional class depicting “high” use among younger siblings and “low to abstinent” use by elder siblings appears to be more appropriate in examining siblings in late adolescence (ages 17 to 19). These results were the first step in a logical procession of steps to lead to a better understanding of sibling socializing influences on adolescent substance use.

## Study 2: Establishing the Predictive Validity of Patterns of Sibling Similarity in Adolescent Alcohol Use

Adolescent siblings are often similar in their alcohol and substance use (Fagan & Najman, 2005; McGue, Sharma & Benson, 1996; Rende, Slomkowski, Lloyd-Richardson & Niaura, 2005; Rowe & Gulley, 1992). Providing support for principles of social learning theory, some evidence suggests siblings are even more similar if they close are in age (McGue et al., 1996), and to a lesser extent if they are the same sex (Rowe & Gulley, 1992), or are genetically similar (McGue, Sharma, & Benson, 1996). To date, there is less evidence on social-psychological influences that explain variance in sibling similarity in alcohol use; however, several studies have found that sibling closeness perceptions, warmth, and positivity appear to be key (Pike, McGuire, Hetherington, Reiss, & Plomin, 1996; Rende, Slomkowski, Rende, Lloyd-Richardson & Niaura, 2005), while other studies have not (Samek & Rueter, 2011).

Previous research (Study 1) has detected multiple patterns of sibling similarity in alcohol use. In this research, there were distinct groups of reflecting patterns of sibling similarity in alcohol use. There was one group reflecting relatively “high” use amongst both siblings (HIGH-HIGH), another group reflecting relatively “low” use amongst both siblings (LOW-LOW), and two final groups reflecting differential patterns of use amongst both siblings (eHIGH-yLOW, eLOW-yHIGH). In order to build on research examining the social-psychological influences on sibling similarity in alcohol use, this study tests whether characteristics of sibling relationship quality (closeness, conflict, and communication) predict sibling similarity in alcohol use patterns at a later time point, while controlling for sibling shared genetics (genetically versus not genetically related), sex composition (same versus mixed), and age difference.

**Theoretical Frameworks**

Several theoretical frameworks offer insight on which characteristics of the sibling relationship may influence sibling similarity in adolescent alcohol use. This study is guided by social learning and attachment theories, as well as behavioral genetics literature on alcohol use.

According to social learning theory (Bandura, 1977), children acquire attitudes and learn behaviors through two key mechanisms: observation and reinforcement. Family members are key role models in children's learning. In adolescence, elder siblings may play a particularly influential role model for younger siblings to observe various behaviors, as well as the positive and negative reinforcement the older sibling receives as a result of partaking in such behaviors. For example, an older sibling's alcohol using behavior may be one of the first non-adult alcohol using experiences a younger sibling observes, which likely influences a younger sibling's alcohol use expectancies.

Moreover, an elder sibling may be a more influential role model to the younger sibling if the sibling relationship is warm and loving, if they are the same sex, and close in age (Bandura, 1969; Whiteman, McHale, & Soli, 2011). Younger siblings may be more likely to identify with their elder sibling under these conditions. On the other hand, if the sibling relationship is coercive and aggressive, negative interactions may escalate rapidly (Patterson, 1984). Therefore, a key construct to examine in furthering our understanding of why siblings are similar in their alcohol use is that of sibling relationship quality in general. This concerns characteristics of the sibling relationship that are warm and loving (e.g., perceptions of closeness and warmth, general patterns of communication), as well as perceptions of conflict and negativity.

Attachment theory (Bowlby, 1969) is also useful in considering the psycho-social influences of sibling similarity in alcohol use. In general, this theory posits children form important social bonds in family relationships, which they rely on during times of stress and sadness. Sibling relationships, in particular, offer an important attachment bond because they share the unique family environment and have experienced major family events. Such examples might include co-experience of family stress such as parental divorce or separation (Kim, McHale, Osgood, & Crouter, 2006; Voorpostel & Blieszner, 2008). According to attachment theory, older sibling alcohol use may have a highly important effect on younger sibling alcohol use given their unique emotional bond. Like social learning theory, key theoretical constructs from attachment theory that might explain sibling influences on adolescent alcohol use concern general relationship quality (closeness, communication, conflict).

Finally, it is useful to discuss the general behavioral genetics framework when researching alcohol use. According to this view, every trait (e.g., alcohol use) is caused by characteristics of both genes and the environment. In fact, there is evidence that alcohol use is genetically influenced, particularly when alcohol use reaches levels of dependence or abuse (Heath et al., 1997; True et al., 1999). In adolescence and in terms of initial alcohol use, however, there appears to be less of a genetic effect and more of a shared environment effect (for a review, see Hopfer, 2003). In general, the shared environment refers to anything that causes sibling similarity in alcohol use that is not due to genetics (Burt, 2009), sometimes referred to as the family environment. In line with social learning and attachment theories, sibling relationship quality appears to be key source of shared environmental influence, which I review next.

**Sibling Relationship Quality and Sibling Similarity**

There is mixed evidence on whether sibling closeness plays a key social-psychological role in explaining sibling similarity in adolescent adjustment. Using a behavioral genetic design, Slomkowski and colleagues (2005) found that when siblings were high in social connection, sibling similarity in tobacco use was explained in large part by the shared environment. This effect held even when controlling for peer and parental smoking, and results have been replicated elsewhere (Rende et al., 2005). Yet, recent research has found that sibling closeness perceptions (measured independent of siblings sharing the same friends) did not explain sibling similarity in substance use; rather, when younger siblings felt close to their elder sibling, they were all together less likely to report substance use (Samek & Rueter, 2011). These mixed findings suggest further research is needed to examine the effect of sibling closeness as a contributor to sibling similarity in alcohol use.

In addition to closeness, previous research has shown conflict plays a key social-psychological role in explaining sibling similarity in adolescent adjustment. Pike and colleagues (1996) found that the correlations between family member negativity, depression, and antisocial behavior were explained in part by the shared environment. This was especially true for siblings such that 24 – 37% of the correlation was explained by shared environmental effects (mothers: 5-16%; fathers: 17-19%). At this time, it is unclear whether family member negativity will better explain differential patterns of sibling similarity alcohol use.

In addition to closeness and conflict, communication has been found to be a key variable in explaining other adjustment outcomes in adolescence (e.g., Steinberg, 2001). Like perceptions of closeness and warmth, being able to communicate with a sibling may foster close emotional bonds which influence observational reinforcement. Indeed, previous research has demonstrated that sibling self-disclosure is associated with warmth in the relationship (Furman & Burhmester, 1985; 1992; Howe, Aquan-Assee, Burkowski, Lehoux, & Rinaldi, 2001). While conceptually unique, perceptions of closeness, conflict, and communication are often correlated (Furman & Burhmester, 1985).  A simultaneous examination of closeness, communication, and conflict is needed to tease out potential similarities or differences in predicting patterns of sibling similarity in alcohol use.

**Methodological Advances**

This study utilizes measures of both observed and self-reported sibling relationship quality to better understand how siblings influence one another in adolescent alcohol use. As mentioned, previous research has demonstrated a connection between sibling relationship quality and adolescent problematic outcomes (Rende et al., 2005; Samek & Rueter, 2011; Slomkowski et al., 2005), yet no research to date has examined characteristics of observed sibling relationship quality in association with adolescent alcohol use. An analysis of observed relationship quality in comparison to self-reported perceptions allows a more thorough investigation in the relationship between behaviors and perception in association with adolescent alcohol use.

This study was also unique in its measurement of sibling similarity in alcohol use. Previous research often relied on a correlational approach (Conger & Rueter, 1996; McGue et al., 1996; Rende et al. 2005; Rowe & Gulley, 1992; Samek & Rueter, 2011;

Slomkowski et al., 2001, 2005). This study utilized latent class analysis (LCA) in its measurement of sibling similarity in alcohol use, as recent research (Study 1) has shown theoretical and statistical justification for an analysis of classes of sibling similarity in alcohol use. A necessary next step from this recent research is to establish the predictive validity of these classes by examining characteristics of sibling relationship quality as predictors. Previous research has found a connection between sibling relationship quality and adolescent outcomes (Rende et al., 2005, Samek & Rueter, 2011, Slomkowskit et al., 2005). Yet, there is no research to date that examines how sibling relationship quality may differentially predict *low* versus *high* alcohol use classes.

### Study 2 Summary

Altogether, this research replicates and extends earlier research (Rende et al., 2005, Samek & Rueter, 2011, Slomkowski et al., 2005, Study 1) in order to better understand the social-psychological influences of sibling relationships. The primary objective of this study was to predict patterns of sibling alcohol use based on several self-reported and observed sibling relationship quality variables (closeness, conflict, communication), while controlling for fixed sibling contextual variables as (e.g., age difference, sex composition, genetic similarity).

## Method

### Data Source

Data from Sibling Interaction and Behavior Study (SIBS, McGue, Keyes, Sharma, Elkins, Legrand, Johnson, & Iacono, 2007) was again utilized for Study 2. The same participants used in Study 1 are again used in Study 2.

### Procedure

In addition to the procedure outlined in Study 1, observed interaction scales from Wave 1 were also utilized. At Wave 1, participating family members included mother, father, elder, and younger sibling. All video-recorded interactions occurred in a room decorated to look like a dining/living room, with family members seated around a table. Family members were aware they were being recorded; however, the video camera was inconspicuously placed in a bookcase. In the first family task, families were presented with an ambiguous image (Rorschach inkblot) and asked to come to consensus about what it represented. In the second family task, families were presented with a moral dilemma (Kohlberg, 1981). Specifically, families read a story about a man whose wife had been diagnosed with a fatal disease. The only drug that could save her life was unaffordable. Families were asked to decide whether the man should steal this drug for his wife, as well as whether he should steal this drug for a stranger in need.

**Measures**

The same concepts of *sibling closeness*, *communication*, and *conflict* were measured as latent factors using observed and self-reported scales using Wave 1 data. Observed scales reflected one sibling's behavior toward another sibling. Self-reported scales reflected one sibling's perceptions about their relationship with their sibling.

Reliability (inter-item, inter-rater) information for the utilized scales are described in Table 2.1. Fit statistics for latent factors are described in Table 2.2. Standard interpretation of fit statistics is as follows: factor loadings represent the degree to which each indicator is correlated with the overall latent factor. Loadings of at least .30 are considered acceptable (Kline, 2005); however, loadings .70 or above are generally preferred. The RMSEA penalizes for increasing parameters; an index of 0 indicates best fit. A standard interpretation is that the model indicates good fit if the RMSEA $\leq .05$ (Kline, 2005). The SRMR is the standardized difference between the observed predicted values, a value $\leq .08$ is considered good fit (Hu & Bentler, 1998).

**Sibling Observed Scales.** Observational scales were adapted from the *Iowa Family Interaction Scales* (Melby et al., 1998). Trained observers globally rated each family member's behavior towards each other family member using a scale ranging from 1 (*not at all characteristic of the person*) to 9 *(mainly characteristic of the person*). All observers received approximately 100 hours of training and were required to pass written tests before independent coding. Observers also attended bi-monthly meetings for continued training. Reliability was assessed by double coding 25% of tapes. Double coded ratings were compared using intraclass correlations (ICC; Shrout & Fleiss, 1979; Suen & Ary, 1989). All ICCs for observable scales reported here had $p$'s < .01 (see Table 2.1).

First, *sibling closeness* was assessed as a latent factor (for each sibling uniquely) indicated by the following scales: Warmth (defined as the degree to which each sibling expressed liking, appreciation, praise, care, concern, or support for the other sibling), and Relationship Quality (defined as the observer's evaluation of the quality of the sibling relationship, with a low score representing an unhappy, emotionally unsatisfying, or brittle relationship, and a high score representing a warm, open, happy, and emotionally satisfying relationship).

A second overall factor of *sibling conflict* was assessed as indicated by two scales: Hostility (defined as the degree to which each sibling displayed hostile, angry, critical, disapproving and/or rejecting behavior toward the other sibling's behavior (actions), appearance, or state), and Angry Coercion (defined as a specific form of hostility that assessed degree to which each sibling achieves goals, attempts to control or change the behavior or opinions of the other sibling, or attempts in a hostile manner to get the other sibling to do what the focal wants).

Finally, a third overall factor of *sibling communication* was assessed as indicated by two scales: Communication (defined as the extent to which each sibling conveys in a neutral or positive manner his/her needs and wants, rules and regulations, as well as clearly expressed information and ideas that may be useful to others), and Listener Responsiveness (defined as the degree to which each sibling attends to, shows interest in, acknowledges, and validates the verbalizations of the other person through the use of nonverbal backchannels and verbal assents).

**Sibling Self-Report Scales.** In addition to these observed scales, self-reported characteristics of sibling relationship quality were also assessed using the *Sibling Relationship Questionnaire* (SRQ; Furman & Buhrmester, 1985). The SRQ asks adolescents to rate interactions with the other sibling on a scale of 1 (Hardly at all) to 5 (EXTREMELY much). Latent factors of perceived *sibling closeness* were assessed separately for elder and younger siblings using the following scales: affect (3-items for each sib, e.g., “How much is there a strong feeling of affection (love) between you and this sibling?”); and nurturance (3 items, e.g., “How much do you help/does this sibling help you/ with things you can’t do by yourself?”); and admiration (3 items, e.g. “How much do you/does this sibling/ admire and respect this sibling/you?”). Please see Table 2.1 for reliability of all self-reported scales.

The following scales were used to assess latent factors of *sibling conflict*, separately for elder and younger siblings: quarrelling (3-items for each sib, e.g., "How much do you and this sibling argue with each other?"); antagonism (3-items for each sib, e.g., "How much do you and this sibling bug and pick on each other in mean ways?"); and competition (3-items for each sib, e.g., "How much do you and this sibling try to do things better than each other?").

Finally, the following scales were used to assess latent factors of *sibling communication* for elder and younger sibling separately: intimacy (3-items for each sib, e.g., "How much do you and this sibling tell each other everything?"); and prosocial behavior (3-items of each sib, e.g., "How much do both you and your sibling share with each other?").

**Patterns of Sibling Similarity in Alcohol Use.** Each sibling completed a self-report survey of their own alcohol use via the *Computerized Substance Use Assessment (CSA*; based on Christiansen & Goldman, 1983; Schafer & Brown, 1991). Wave 2 data was utilized here. Siblings were asked if they have ever used alcohol (without parent's consent, *yes* = 1, *no* = 0), ever been intoxicated (*yes* = 1, *no* =0), frequency of alcohol use in the last 12 months (0 (*Never*) to 9 (*Every day or nearly every day*)), 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 in the past 12 months (0 (*0 drinks*) to 10 (*10+ drinks*)). These variables were used as indicators of a latent class variable.

**Demographic Variables.** Age was assessed based on the date of assessment and reported birthdays. Adolescent sex was coded so that 1 = *male*, 2 = *female*. Adoptive status was coded so that 1 = *adopted*, 2 = *non-adopted*. Sibling age difference was assessed as the absolute value in difference amongst sibling ages. Sibling sex composition was coded so that 1 = *same sex*, 2 = *mixed sex*. Genetic similarity amongst siblings was coded so that 1 = *genetically related*, 2 = *genetically unrelated*.

**Missing Data Analysis**

As described in Table 2.2, the proposed study measures have little missing data. Across Wave 1 predictors, nearly 100% of the data is complete. Across Wave 2 outcomes (alcohol use self-reports), nearly 80% of the data was completed. Lack of data was due to participant non-participation (~5% of elders and youngers) or a failure to complete computerized assessment (~16% for both elders and youngers). Chi-Square analyses revealed no statistically significant differences between those who dropped out versus stayed in the study at Wave 2 across adoptive status, ethnicity, and sex. Chi-square analyses also revealed no statistically significant differences on these demographics between those missing self-reported alcohol data and those with complete data at Wave 2. Missing data were handled using Full Information Maximum Likelihood (FIML), shown to be superior in accurately representing the sample data compared to other missing value handling in simulation studies (Enders & Bandalos, 2009, Johnson & Young, 2011).

**Analysis Plan**

Using MPLUS, 6.0, patterns of sibling alcohol use were predicted based on the self-reported and observed variables described above via structural equation modeling. Specifically, mixture modeling was used by specifying ALGORITHM=INTEGRATION and INTEGRATION=MONTECARLO. Means were set for each latent class based on Study 1's result. All predictors were correlated (see Table 2.3). All tests controlled for sibling sex composition, age difference, and genetic similarity. To reduce model complexity, first, factors were estimated and outputted as factor scores, then merged back into the data file and specified as predictors. The final analysis was conducted using the previously created factor scores. Finally, to compare all classes, 4 different models were run using each of the classes as alternate reference for all tests.

## Results

**Preliminary Analyses.** Table 2.2 describes the descriptive statistics for the observed and self-reported sibling relationship quality latent factors. Elder siblings reported greater perceived closeness and communication with their younger siblings when they were female (closeness: $t = 5.65$, $\beta = .24$, $p < .001$; communication: $t = 4.88$, $\beta = .24$, $p < .001$). Elder siblings tended to report more conflict with their younger siblings when they were younger in age ($t = -3.06$, $\beta = -.13$, $p < .01$), and male ($t = -3.68$, $\beta = -.16$, $p < .001$). Both siblings tended to report better communication when they were older (elder $t = 2.07$, $\beta = .08$, $p < .05$; younger $t = 3.32$, $\beta = .15$, $p < .01$). Younger siblings reported better communication with their elder sibling if they were non-adopted ($t = 2.04$, $\beta = .10$, $p < .05$). Correlations amongst the continuous predictors are presented in Table 2.3.

**Class Descriptions.** LCA results that were reported in Study 1 and based on Wave 2 alcohol reports were utilized in the present study. In Study 1, 1- through 5-class models of sibling similarity in alcohol use were compared. The 4-class model had superior fit (AIC, adjusted BIC, LRT test) compared to the other models. Sibling age difference, sex composition, and genetic similarity were entered as covariates. Thirty-three percent of siblings were classified as belonging to the relatively “low” use amongst both siblings class (LOW-LOW), 26% were classified in the class reflecting "high" use amongst both siblings (HIGH-HIGH), 34% in the "high" use amongst elder but "low" use amongst younger sibling class (eHIGH-yLOW), and 7% classified in the "low" use amongst elders but "high" use amongst younger sibling class (eLOW-yHIGH; see Table 1.3, Study 1).

There were significant associations between the 4-class model and two out of the three covariates. Siblings in the HIGH-HIGH class were significantly more likely to be genetically related than siblings in the LOW-LOW class ($t$ = 1.99, $p$ < .04, $OR$ = 1.66), the eHIGH-yLOW class ($t$ = 2.01, $p$ < .05, $OR$ = 1.70), and were trending to be more likely to be genetically related compared the eLOW-yHIGH class ($t$ = 1.84, $p$ = .07, $OR$ = 2.08). Finally, sibling age difference was associated with the 4-class model. Siblings in the eHIGH-yLOW class were likely to be further apart in age compared to the other differential alcohol use class (eLOW-yHIGH class, $t$ = 2.65, $p$ < .01, $OR$ = 1.79), and siblings in the HIGH-HIGH class ($t$ = 2.24, $p$ < .03, $OR$ = 1.42).

**Associations between Sibling Relationship Quality and 4-Class Sibling Alcohol Model**. Table 2.4 shows means of observed and self-reported latent factors (closeness, communication, and conflict) across the 4-class alcohol model. Overall, closeness (either observed or self-reported) was not associated with the 4-class model, but communication and conflict were associated.

***Communication.*** Siblings in the HIGH-HIGH class were more likely to display greater communication ($t$ = 1.98, $p$ < .05, $OR$ = 1.74), and elder siblings perceived greater communication ($t$ = 3.31, $p$ < .01, $OR$ = 1.20) compared to siblings in the eHIGH-yLOW class. Following this pattern, there was a marginal association between elder siblings self-reported communication and the HIGH-HIGH class ($t$ = 1.82, $p$ = .07, $OR$ = 1.11) compared to siblings in the LOW-LOW class. Younger siblings in the eHIGH-yLOW class were also trending to be more likely to perceive better communication ($t$ = 1.63, $p$ = .10, $OR$ = 1.12) compared to siblings in the LOW-LOW class. Altogether, these results suggest that siblings similar in high levels of alcohol use have greater levels of general communication compared to siblings similar in low or differential patterns of alcohol use.

***Conflict.*** Elder siblings in the LOW-LOW class were more likely to perceive conflict compared to siblings in the eLOW-yHIGH class, $t$ = 2.37, $p$ < .03, $OR$ = 1.21. On the other hand, siblings in the eLOW-yHIGH were more likely to display more conflict ($t$ = 1.99, $p$ < .05, $OR$ = 1.21), and were trending to display better observed communication ($t$ = 1.89, $p$ = .06, $OR$ = 1.95), compared to the LOW-LOW class. These results suggests that, while elder siblings perceive low conflict in their sibling relationship when they have low levels of alcohol use, greater levels of conflict and communication are actually observed when younger siblings have high levels of alcohol use.

Furthermore, siblings in the eLOW-yHIGH class were also more likely to display greater conflict ($t$ = 2.39, $p$ < .03, $OR$ = 1.26) and communication ($t$ = 2.64, $p$ < .01, $OR$ = 2.55) compared to the other differential sibling class (eHIGH-yLOW). Moreover, siblings in the eLOW-yHIGH class were trending to display greater conflict compared to siblings in the HIGH-HIGH class ($t$ = 1.65, $p$ = .10, $OR$ = 1.08). Finally, elder siblings in the eHIGH-yLOW class were more likely to perceive conflict compared to the other differential sibling alcohol use class (eLOW-yHIGH; $t$ = 2.33, $p$ < .03, $OR$ = 1.20). These results suggest siblings different in their alcohol use tend to have more conflict. When younger siblings use more than their elder siblings, there tends to also be more communication with that conflict.

## Discussion

This study demonstrated an association between characteristics of sibling relationship quality and patterns of sibling similarity in adolescent alcohol use across two time points in adolescence. Controlling for age difference, sex composition, and genetic relatedness, this study found that levels of conflict and communication helped explain patterns of similarity and difference in alcohol use amongst adolescents. These results add to a body of research demonstrating sibling influences on substance use, as well as extend that research by comparing theoretically distinct sibling relationship quality characteristics in their relation to sibling similarity in alcohol use.

### Covariates: Age Differences, Sex Composition, Genetic Relatedness

As reported in Study 1, siblings who were genetically related were more likely to be placed in the class reflecting "high" use by both siblings (HIGH-HIGH), compared to other classes (LOW-LOW, eHIGH-yLOW, eLOW-yHIGH). This association may reflect a genetic contribution towards high levels of alcohol use in late adolescence. Previous research has found a modest to moderate heritability of substance use in adolescence (Hopfer, Crowly, & Hewitt, 2003; Han, McGue & Iacono, 1999; Prescott, Madden, & Stallings, 2006). On the other hand, previous research suggests adopted adolescents tend to have a greater likelihood of adjustment problems compared to non-adopted adolescents (Juffer & van IJzendoorn, 2005; Keyes et al., 2008). Post-hoc analyses showed that this result was not affected by collapsing the two groups of genetically unrelated siblings into one group (ADOPT-ADOPT, ADOPT-BIO); adopted siblings were not more likely to be similar in high alcohol use compared to sibling pairs that were different in their adoption status.

Following previous research demonstrating a negative association between sibling age difference with sibling similarity in various outcomes (Buchanan, McGue, Keyes, & Iacono, 2009; McGue et al., 1996; Trim, Leuthe, & Chassin, 2006), this study demonstrated that siblings were more likely to be placed in the class reflecting differential patterns of alcohol use (elder having greater alcohol use than the younger), compared to siblings placed in a "high" alcohol use by both siblings class in both early and late adolescence. Finally, sibling sex composition (same versus opposite sex) did not significantly predict either sibling similarity classes like previous research has found (McGue et al.,1996; Rowe, Rodgers & Meseck-Bushey, 1992), but that other research has not found (Samek & Rueter, 2011; Slomkoski et al., 2005). In this study, consistent findings across same versus mixed sex siblings point to the important effects siblings have on one another regardless of their sex composition.

## Predictors: Sibling Relationship Quality

**Communication.** What was new to this study was the examination of characteristics of sibling relationship quality as predictors of sibling similarity in alcohol use classes. A key finding of this study was for sibling pairs where both adolescents had high levels of alcohol use (HIGH-HIGH): they were likely to have better communication (perceived and observed) compared to siblings in the differential pattern of similarity in alcohol use where elder siblings used more alcohol than their younger siblings (eHIGH-yLOW). Moreover, siblings in the HIGH-HIGH class were marginally more likely to have better communication than siblings in the class reflecting relatively low alcohol use amongst both siblings (LOW-LOW, $p$ = .07). Previous studies have found evidence of an association between perceptions of sibling social closeness and patterns of sibling similarity in substance use that utilized a correlational rather than latent class approach (Rende et al., 2005; Slomkowski et al., 2005).

Following social learning theory, this finding may support the claim that siblings are more similar in their behaviors if their relationship is "close;" however, this finding could be a first step to help clarify what is theoretically meant by "close." In this study, having a warm and loving relationship was not significantly associated with sibling similarity in alcohol use classes, but communication was. On the other hand, both concepts (close, communication) were highly correlated, especially in terms of self-report (see Table 2.3). This correlation fits with previous research demonstrating this association (Buhrmester & Furman, 1987; Howe et al., 2001). In terms of analyzing sibling similarity in alcohol use via alcohol use patterns, it may be that self-disclosure and open communication are influential (perhaps through reinforcement of ideas and expectations), rather than general perceptions of emotional closeness alone. Replication and further exploration of these concepts is needed to make this distinction, particularly because of low effect sizes and some marginal associations.

These findings may have clinical relevance in order to reduce rates of adolescent alcohol use. This might include monitoring the relationship quality and level of communication of adolescents with siblings, as well as assessing the amount of time and interests they may share. For example, if siblings are particularly close in their communication and comfort with another, these findings provide some evidence for targeting both siblings in interventions.

**Conflict.** An additional major finding of this study concerns sibling conflict. Even though abstinent to low alcohol using elder siblings *perceived* low levels of conflict with their relatively high alcohol using younger siblings, greater levels of *observed* conflict and communication were actually observed in their sibling interactions compared to siblings who both had low levels of alcohol use (eLOW-yHIGH compared to LOW-LOW). It is important to note that elder siblings in the other differential pattern of alcohol use (eHIGH-yLOW) also perceived more conflict in their sibling relationships compared to the other differential class (eLOW-yHIGH). These findings lead me to believe that sibling conflict tends to be greater in sibling pairs with differential patterns of alcohol use in particular.

One potential explanation for this finding is that something about the individual (high alcohol using) adolescent is driving this association. For example, these adolescents may be having more conflict with their siblings and potentially others at large because of having a more difficult temperament or personality to begin with (Brody, 1998). Having a difficult temperament may have helped drive these adolescents to engage in more conflict with others as well as various adjustment outcomes, including the use of more alcohol. This follows previous research that has long demonstrated the associations between sibling conflict and various adjustment problems (e.g., Stocker, Burwell, & Briggs, 2002; Gamble, Yu, & Kuehn, 2011), between sibling and parent conflict (Rinaldi & Howe, 2003), as well as between sibling temperament and conflict (Stoneman & Brody, 1993).

Finally, both siblings in the low to abstinent alcohol use pattern (LOW-LOW) perceived greater sibling conflict compared to siblings where there was low use by the elder, but relatively high alcohol use by the younger (eLOW-yHIGH). Indeed, previous research has found that siblings tend to report very different things as contributing to their overall conflict (Dunn & Plomin, 1990; McGuire, Manke, Eftekhari, & Dunn, 2000), and differential alcohol use may be one of those factors. That is, when siblings are similar in low alcohol use, something else may be driving levels of increased sibling conflict. Of course, sibling conflict is not always a bad thing. It is through conflict with others that children learn about negotiation, persuasion, and engage in conflict resolution skills (Whiteman, McHale, & Soli, 2011), as well as foster social and emotional competence (Bedford, Volling, & Smith, 2000). Moreover, conflict is appropriate and normal in adolescence when teens are exploring identity and concerning issues of individuation (Raffaelli, 1992).

**Limitations and Future Directions**

The main limitation of this study concerns the generalizability of sample findings. The sample was representative in terms of parents' education, income, and ethnicity compared to the population it was sampled from. However, there were a higher proportion of adolescents that were internationally adopted, Asian females compared to the general population. The majority of siblings were from 2-parent, heterosexual married families. Additionally, this study analyzed patterns of sibling similarity in alcohol use. It is unclear whether such patterns could be replicated for other substances, such as tobacco or marijuana, or whether these patterns have the same predictors found here (communication, conflict). Replication of study findings across a variety of samples and a variety of substances is needed to demonstrate the reliability of this study's findings.

As a reminder, siblings in the “high” alcohol use by both elder and younger pattern (HIGH-HIGH) were more likely to be genetically related compared to the three other sibling alcohol use patterns. This may be reflecting the heritability for alcohol use amongst heavy users (e.g., Han, McGue & Iacono, 1999). With this, an important future research question is whether the associations between sibling relationship quality variables (communication, conflict, closeness) and patterns of sibling alcohol use are explained through primarily genetic versus environmental effects. The behavioral genetics methodology, specifically Cholesky decomposition (Neale & Cardon, 1992), can be used to partial out effects between predictors and outcomes. A logical next step in this program of research is to conduct such an analysis.

**Conclusion**

This study demonstrated a longitudinal association between characteristics of sibling relationship quality and patterns of sibling similarity in adolescent alcohol use. Above and beyond “fixed” effects of shared genetics, sex, and age, the overall level of sibling conflict and communication were significant predictors of patterns of similarity and difference in alcohol use amongst adolescents. Communication may be more important in explaining why siblings are similar with high levels of alcohol use compared to differential levels (eHIGH-yLOW) and potential low levels (LOW-LOW) of alcohol use. Conflict may be important in explaining sibling differences in alcohol use. When the effects of communication and conflict are included, there appears to be little effect for sibling emotional closeness predicting patterns of sibling similarity in alcohol use.

## Study 3: Family Involvement and Adolescent Alcohol use: Potential Sources of Shared Environmental Influence

Adolescent alcohol use remains highly prevalent and is associated with several problematic outcomes such as physical assault and suicide (Bonnie & O'Connell, 2004; Miller, Naimi, Brewer & Jones, 2007; U.S. Department of Health, 2007). Previous research and theory have shown that siblings are important socializing agents of early alcohol use, perhaps even more so than parents (Fagan & Najman, 2005; McGue & Iacono, 2009). Understanding why adolescent siblings are similar or different in their alcohol use is important because it can give us some insight on what contributes to adolescent alcohol use.

In this paper, a behavioral genetics methodology was used to better understand genetic and environmental sources of variance that contribute to sibling similarity in adolescent alcohol use. Requiring a genetically-informed design, this methodology partials out sibling similarity due to *shared genetic effects* (similarity due to having shared genes), *shared environmental effects* (similarity not due to genetics, e.g., having the same rearing environment), and nonshared environmental effects (*sibling differences*). Because of this genetic and environmental partialling, results can provide evidence for variables that have strong potential for environmental intervention.

As a family social scientist, I am particularly interested in variables that explain a significant proportion of *shared environment* estimates for two reasons. First, even though we cannot change genetic predispositions or other fixed predictors (e.g., sex) to reduce the likelihood of substance use, it is especially useful to understand what specific aspects of the family environment are associated with adolescent substance use so that we have evidence for environmental prevention and intervention. Second, shared environment estimates tend to have moderate to strong effects on adolescent substance use (e.g., Hopfer, Crowley & Kewitt, 2003; McGue, Elkins & Iacono, 2000; McGue et al., 1996), suggesting research is needed to determine what specifically is happening within the shared environment to influence such outcomes.

Previous research has found a correlation between family involvement and child adjustment problems (Amato & Fowler, 2002; Flouri, 2007; Flouri & Buchanan, 2004), such as alcohol use (Nash, McQueen, & Bray, 2005). Building on this research, this study aims to decompose the correlation between family involvement (mom, dad, sibling) and adolescent alcohol use into genetic and environmental influences. Findings may highlight family process variables (mom, dad, sibling involvement) that may be useful to environmentally target in adolescent substance use interventions.

**Background on Behavioral Genetics**

From a behavioral genetics perspective, three latent variance components contribute to the total variance in any trait or outcome: additive genetic, shared environmental and nonshared environmental effects. Additive genetic effects (*A*) account for similarity between siblings for having the same biological parents. Shared environmental effects (*C*) account for similarity between siblings not due to genetics (e.g., this could be due to having the same family environment, neighborhood, friends, school, etc). Finally, nonshared environment effects (*E*) account for differences between siblings (e.g., unique peer groups, differential parental treatment), as well as measurement error (Burt, 2009). In order to partial out these variance components, a genetically informed sibling sample is necessary. In this paper, I compare (a) siblings who are adopted and are not genetically related to each other or their parents (meaning they share 0% of additive genes), to (b) siblings who are the biological offspring of their parents (meaning they share 50% of their additive genes).

**Moving Towards a Theory of the Environment.** By itself, quantitative behavioral genetics is not meant to be a causal theory (Rutter, 2002). Rather, it is only a method to estimate general contributions of gene and environment effects. The theory behind the method is roughly based in the diathesis-stress model (Barlow & Durand, 2005)**.** This model posits that a combination of biological and environmental influences lead to the development of various behavioral outcomes, including alcohol use. Specifically, a person's "diathesis" or genetic predisposition is activated under certain circumstances of environmental stress. In order to fully understand the etiology of adolescent alcohol use, we need to consider and examine both genetic and environmental effects.

Altogether, the behavioral genetic methodology (also known as Falconer, biometric or A-C-E modeling) has led to advancement in testing and furthering our understanding of the contribution of the "genome" (i.e., through analyses of specific genetic makers via advancements in molecular genetics). However, there has been a lack in furthering our understanding of the contribution of the "envirome" (Anthony, 2001; i.e., through analyses of environmental variables controlling for genetics). As family social scientists or family professionals, we may be able to significantly contribute to the understanding of the envirome by utilizing the behavioral genetics methodology as a way to at least theoretically control genetic effects in study designs.

***Towards a theory of the shared environment.*** Specifically, it is useful for family researchers to consider what may contribute to shared (versus nonshared) environmental effects. Burt, McGue, Krueger, and Iacono (2007) described several reasons to do so. First, shared environmental estimates are not as small as was once thought (Plomin & Daniels, 1987), in particular for children and adolescents (e.g., Burt, 2009; Hopfer, Crowley, & Hewitt, 2003; McGue, Elkins, & Iacono, 2000). For example, McGue and colleagues (2000) found that shared environment effects were substantial for adolescent substance use, generally explaining 40-60% of the variance in adolescent substance use. Koopmans & Boomsma (1996) found that shared environmental effects explain 58-88% of the variance in whether adolescents ever use alcohol. Substantial shared environmental effects for general frequency of alcohol use have been reported elsewhere (McGue et al., 1996; Rose Dick, Viken, Pulkkinen, & Kaprio, 2001).

Replicated research shows the association between parent-child relationship quality and adolescent delinquency is at least in part due to the shared environment (Burt et al., 2007; Pike et al., 1996), implying that strengthening parent-child relationship quality may in some part reduce the likelihood of adolescent maladjustment. However, Burt et al. found that only 2.8% of the total variation in adolescent delinquency was explained by parent-child relationship quality out of roughly 15% of the total variance of the shared environment. This suggests that additional characteristics of the shared environment need to be identified and tested in order to move towards a theory of the shared environment within a behavioral genetics framework.

***The importance of sibling effects in explaining the shared environment.*** Sibling effects have been shown to be stronger than those of parents in explaining shared environmental variance in adolescent substance use (McGue & Iacono, 2009) and various other adjustment outcomes (Bussell, Neiderhiser, Pike, Plomin, Simmens, Howe, Hetherington, Carroll & Reiss, 1999; Feinberg, Neiderhiser & Hetherington, 2001; Pike et al., 1996). These findings point to the importance of siblings in explaining shared environmental effects on adolescent alcohol use. A logical next step is to better understand what it is about the shared environment that is influencing alcohol use directly; decomposing the correlation between adolescent alcohol use and family processes will meet this demand. For example, Pike and colleagues (1996) found that the correlations between (a) family member negativity and depression and (b) family member negativity and antisocial behavior were both explained in part by the shared environment, especially for siblings (24 – 37% of the correlation was explained by shared environmental effects), compared to mothers (5-16%) or fathers (17-19%).

In addition, Slomkowski and colleagues (2005) found support for the findings that when siblings were high in social connection, sibling similarity in tobacco use was explained in large part by the shared environment. This effect held even when controlling for peer and parental smoking. Conversely, other research has found that sibling closeness perceptions (measured independent of siblings sharing the same friends) did not explain sibling similarity in substance use; rather, when younger siblings felt close to their elder sibling, they were all together less likely to report substance use (Samek & Rueter, 2011). This was true across sibling pairs that were genetically related and genetically unrelated, implying important sibling influences of sibling closeness regardless of biological relationships. These mixed findings suggest further replication and additional exploration of adolescent outcomes (i.e., alcohol use) is needed to better understand the socializing effects of sibling relationships.

**Summary: Study 3**

The goal of this study was to utilize Cholesky decomposition (Neale & Cardon, 1992) in order to partial out the covariance between mother, father, sibling involvement, and adolescent alcohol use. An increased understanding of the genetic and environmental contributions to adolescent alcohol use should lead to a better understanding of which family variables (mother, father, sibling) could then be targeted for environmental prevention and intervention work.

## Method

**Data Source, Participants, and Procedure**

Data from Sibling Interaction and Behavior Study (SIBS, McGue, Keyes, Sharma, Elkins, Legrand, Johnson, & Iacono, 2007) was again utilized for Study 3. The same participants and procedures used in Study 1 and Study 2 were again used in Study 3.

**Measures**

**Adolescent alcohol use** (Wave 2) was measured as a latent variable using indicators from the *Computerized Substance Use Assessment* (CSA) (based on Christiansen & Goldman, 1983; Schafer & Brown, 1991). Siblings were asked if they have ever used alcohol (without parent's consent, *yes* = 1, *no* = 0), ever been intoxicated (*yes* = 1, *no* = 0), frequency of alcohol use in the last 12 months (0 (*Never*) to 9 (*Every day or nearly every day*)), 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 in the past 12 months (0 (*0 drinks*) to 10 (*10+ drinks*)). For both elder and younger, α = .89. All dichotomous indicators were specified as such in the latent variable modeling. All items were used, so if adolescents never used alcohol, remaining items were recoded to 0. Indicators were strongly correlated with the overall latent factor indicating strong reliability; all loadings were greater than or equal to .90.

**Parent involvement** (Wave 1) was measured as a latent variable using adolescent reports from the *Parent Environment Questionnaire* (PEQ; Elkins & McGue, 1997). The PEQ asks adolescents to rate items that describe their relationships with their parents on a scale of 1 (*Very True*) to 4 (*Very False*). Twelve items were used to assess latent factors for both perceptions of involvement with mothers and fathers, separately. Example items include "I talk about my problems and concerns with my parent," "My parent does not seem to know much about how I am doing in school," and "I don't feel very close to my parent." Items were recoded so that a higher value reflected less involvement. α's ranged from .81 to .88 for elder and younger reports on mother and father. Indicators were adequately correlated with the overall latent factors indicating good reliability; loadings ranged from .50 to .71 for elder sibling report, and .32 to .69 for younger sibling report.

**Sibling Involvement** (Wave 1) was measured as a latent variable using the *Sibling Relationship Questionnaire* (SRQ; Furman & Buhrmester, 1985). The SRQ asked adolescents to rate interactions with the other sibling on a scale of 1 (*Hardly at all*) to 5 (*EXTREMELY much*). Three items from the Companionship scale were used to assess overall sibling involvement. These items were (a) "Some siblings play around and have fun with each other a lot, while other siblings play around and have fun with each other a little, how much do you and this sibling play around and have fun with each other?"; (b) "How much do you and this sibling go places and do things together?"; and, (c) "Some kids spend lots of time with their siblings, while others don't spend so much. How much free time do you and this sibling spend together?" Items were recoded so that a higher value indicated less involvement. For elder, $\alpha = .85$; for younger $\alpha = .86$. Indicators were adequately correlated with the overall latent factors indicating strong reliability; loadings ranged from .79 to .88.

**Covariates.** Adolescent sex (1 = *male*, 2 = *female*), age (date of assessment – birthdate), and ethnicity (1 = *White*, 0 = *African American, Asian, Hispanic, Mixed/Other*) were used as covariates in study analyses.

**Missing Data**

As described in Table 3.1, the proposed study measures have little missing data. For example, across Wave 1 measures, nearly 100% of the data were complete, and over three-quarters of the data were complete for Wave 2 alcohol use reports. Missing data at Wave 2 were due to participant non-participation (~5%) or lack of completing assessments (~16% for both elders and youngers). Chi-Square analyses revealed no statistically significant differences for these demographics between those who dropped out versus stayed in the study at Wave 2, or between those missing self-reported alcohol data or those with complete alcohol data for these variables across adoptive status, ethnicity (White versus African American, Asian, Mixed, Other ethnicities), and sex. Missing data were handled using Full Information Maximum Likelihood (FIML), which has proven superior in accurately representing the sample data compared to listwise or pairwise deletion, similar response pattern, and mean imputation in simulation studies (Enders & Bandalos, 2009; Johnson & Younger, 2011).

**Analysis Plan**

Independent and dependent variables were first computed and exported as factor scores using MPLUS, 6.0 (Muthén & Muthén, 1998-2010). Following the analysis plan of Burt et al., (2007) and consistent with previous research (Deater-Deckard & Plomin, 1999), demographic variables of age, sex, and ethnicity were regressed out of all latent variables. Next, correlations amongst study variables were analyzed. If correlations were greater amongst BIO siblings as compared to ADOPT/ADOPT-BIO siblings, this suggested a genetic influence on adolescent alcohol use. If correlations amongst BIO siblings were about the same as the correlations amongst ADOPT/ADOPT-BIO siblings, this suggested a shared environmental influence on adolescent alcohol use. If correlations amongst all siblings were weak to non-existent, this suggests substantial nonshared environmental effects.

After these preliminary analyses, a Cholesky decomposition structural equation model (SEM) was then estimated using Mx (Neale, 2006). In this type of modeling, parameters of additive genetic (A), shared environmental (C), and nonshared environmental (E) are tested as contributors to the covariance between study variables (mother, father, sibling involvement, alcohol use). The additive genetic path for the BIO group was set to .5 (since they share about half of their additive genes), and .0 for the ADOPT and ADOPT-BIO groups (since they share 0 of their additive genes). The shared environment path was set to 1.0 for all three groups because of the equal environments assumption.

Figure 3.1 illustrates the Cholesky model. The order of variables entered has a defined hierarchical meaning (Loehlin, 1996). Within this framework, children usually form their first close relationships with their parents, predominately the mother first, then other family members. Therefore, variables were entered following this logic. As described in Figure 3.1, the total variance in adolescent alcohol use is decomposed into (1) components attributed to genetic (a41, a42, a43), shared environmental (c41, c42, c43), and nonshared environmental effects (not shown for clarity, e41, e42, e43), as well as the (2) residual effects of additive genetics (a44), shared environmental (c44), and nonshared environment (not shown for clarity, e44). Using Mx, the full ACE model was estimated. The -2 times the loglikelihood (-2 lnL) value was then compared with the more restrictive models (AE, CE, and E models) by using the Chi-Square difference test.

Standardized path estimates for the best fitting model were then squared to estimate the proportion of variance accounted for. For example, to determine the total proportion of variance explained by the shared environment in adolescent alcohol use, all c paths leading to alcohol use were standardized, squared, and summed, including the residuals (c41, c42, c43, c44). To determine the total proportion of variance explained by the shared environment in alcohol use explained by these family variables, the paths c41, c42, and c43 were squared and summed and divided by all squared and summed c paths (c41, c42, c43, c44). Ninety-five percent confidence intervals for each path were obtained in order to determine the statistical significance of each path (i.e., if the paths did not cross zero, then significant).

## Results

### Preliminary Analyses

As described in the analysis plan, adolescent sex, age, and ethnicity were regressed out of all phenotypes. Table 3.2 describes how these covariates are associated with study variables. The only variable ethnicity was associated with was younger sibling perceived involvement with elder sibling ($\beta = -.10$, $p < .05$), suggesting that African-American, Asian, Hispanic, Mixed/Other younger siblings perceived greater involvement with their older siblings. In general, older adolescents were more likely to use alcohol than younger adolescents. Younger sibling males were more likely to use alcohol than younger sibling females ($\beta = -.34$, $p < .05$). In general, females and adolescents younger in age were more likely to report greater involvement with parents and siblings than males and older adolescents.

Table 3.3 shows correlations amongst study variables for the entire sample (adjusted for age, sex, and ethnicity). Most variables were significantly correlated at $p < .01$. Table 3.4 shows correlations for study variables across sibling pair type (BIO, ADOPT, ADOPT-BIO) and sex composition (same versus opposite). In general, correlations were stronger amongst BIO siblings as compared to ADOPT-ADOPT and ADOPT-BIO. This pattern suggests the variables used are at least partly genetic in origin. Correlations were stronger amongst same compared to opposite sex siblings. To increase power, however, both same and opposite sex siblings were used in SEM analyses.

**Cholesky Decomposition**

Univariate genetic and environmental contributions to study variables are described in Table 3.5. Shared environmental influences on mom and dad were minimal, while they were moderate to strong on sibling involvement. There were moderate additive genetic influences on all study variables and substantial nonshared environmental influences, as well.

Fit indices comparing the full Cholesky (ACE) model to the more restricted models are presented in Table 3.6. As indicated by significantly worse fit via the Chi-squared tests and associated *p*-values, as well as minimal change in the AIC and sample-sized adjusted BIC values, the best fitting model was the full Cholesky ACE. This indicates important genetic, shared, and nonshared environmental influences on both the variance and covariance of study variables.

On the other hand, there were few statistically significant paths in the full ACE model, as indicated by the 95% confidence intervals crossing zero in Figure 3.2. The standard interpretation to path coefficients is as follows, but caution should be noted due to the few significant paths. Paths are squared and summed to compute the percent of variance accounted for (e.g., the additive genetic variance in adolescent alcohol use = $.20^2 + .20^2 + .06^2 + .41^2 = .25$).

Results revealed that shared environmental influences contributed substantially to family involvement and adolescent alcohol use. The shared environmental contributions to family involvement accounted for 23% ($.10^2 + -.08^2 + .46^2 + .00^2 = .23$) of the total variance in adolescent reported alcohol use, or roughly 100% of the total shared environmental influence ($.10^2 + -.08^2 + .46^2 = .23$; .23/total shared environmental variance of .23 = 1.0). Moreover, 92% of the total shared environmental variance in alcohol use was explained by sibling involvement variable alone ($.46^2$= .21/.23). These findings indicated that shared environmental influences on alcohol use are moderate, and largely explained by sibling, rather than parent, involvement.

To a lesser extent, the influence of family involvement on adolescent alcohol use was also explained by additive genetics. Four percent of the total additive genetic variance could be explained by family involvement variables ($. 20^2 + .20^2 + .06^2 = .04$).

Following this pattern,

family involvement explained only 5% of the total non-shared environmental variance in adolescent alcohol use ($.18^2 + -.00^2 + .14^2 = .05$). These findings suggest variables other than family involvement are influencing adolescent alcohol use via additive genetics and the non-shared environment.

## Discussion

The goal of this study was to determine whether the association between family involvement (mom, dad, sibling) and adolescent alcohol use was primarily due to genetic versus environmental influences. However, there were few significant findings as indicated by the 95% confidence intervals crossing zero. This is most likely due to power issues. First, the correlations between predictors (mom, dad, sibling involvement) and alcohol use were low to begin with (see Table 3.3). Specifically, the correlations between elder sibling alcohol use and parent involvement were slight, and were stronger between younger sibling alcohol use and parent involvement. The correlations were also minimal between sibling involvement and adolescent alcohol use. Moreover, as described in Table 3.4, correlations were stronger amongst same versus opposite sex siblings, and were stronger amongst ADOPT-ADOPT as compared to ADOPT-BIO pairs, suggesting that selecting sub-samples of same sex or ADOPT only as the genetically unrelated comparison are needed to improve the model and potentially lead to more significant paths.

In a post-hoc analysis using only same-sex siblings, there were comparable findings to the model utilizing all sibling pairs (i.e., lack of significant paths). Again, this is most likely related to power. Only 128 siblings were genetically related and the same sex, and only 244 genetically unrelated siblings were the same sex. These cell sizes are too small for the number of parameters measured. Similar results were obtained utilizing only ADOPT-ADOPT and BIO-BIO comparisons (ADOPT-BIO excluded). The main implication of study findings is that future research utilizing Cholesky decomposition that are using a comparable sample size, most likely need to decompose variables with a stronger correlation in order to see significant effects.

Another potential reason for lack of significant paths may be due to high collinearity amongst predictor variables (mom, dad, sibling involvement), such that the first variable entered controlled for the total correlation amongst the genetic and environmental latent variables. Indeed, Table 3.3 shows correlations in the .8 range for adolescent ratings of mother and father involvement, and correlations in the .2 range between sibling and parent involvement. In further post-hoc analyses, variables were entered in alternate orders (dad involvement first, then mom, then sib, or sib involvement first, than mom, than dad). In any case, the same pattern of results was found; there were no significant associations between the predictors (mom, dad, sibling involvement) or the outcome (adolescent alcohol use) in any of the models.

**Future Research**

As mentioned, future research utilizing the Cholesky decomposition method should utilize either (a) larger samples with modest to moderate correlations, or (b) decompose correlations that are stronger than the initial correlations found here (i.e., are moderate to strong). A review of the literature sheds light on the potential family variables that may have stronger correlations with adolescent alcohol use. For example, a recent study conducted by van den Eijnden, van de Mheen, Vet, & Vermulst (2011) tested parents' alcohol use, availability of alcohol in the home, parental rule settings, and frequency and quality of communication as predictors of alcohol related problems at a later time point. These researchers found perceived alcohol availability was correlated to both alcohol use ($r$ = .50) and alcohol related problems ($r$ = .59) two years later; however, it is unclear whether the association between this characteristic of parenting and alcohol is best explained by genetic or environmental factors. These moderate to strong correlations are good candidates for a future research paper utilizing the cholesky decomposition method.

Following this logic, it would be useful to examine variables such as sibling facilitation of alcohol use in relation to adolescent alcohol use, as well as whether this correlation could best be explained by shared environmental factors. Previous research supporting this logic has found that sibling substance use has a greater influence on adolescent substance use compared to parent substance use (Fagan & Najman, 2005) and that perceptions of sibling alcohol use alone are associated with increased adolescent alcohol use (D'Amico & Fromme, 1997). Moreover, previous research has found that sibling facilitation mediates the relationship between older and younger drug use (McGue & Iacono, 2009), and that sibling modeling of alcohol use is associated with increased adolescent alcohol use (Ary, Tildesley, Hops, & Andrews, 1993). Based on this body of research, a useful research paper would seek to decompose correlations between variables such as perceived sibling alcohol use, sibling facilitation of alcohol use, sibling modeling of alcohol use, etc., in their association with adolescent alcohol use.

Of course, there are a myriad of other family predictors that have been shown to have moderate to strong associations with adolescent alcohol use that would also be useful to examine, including parental monitoring and discipline (Gossrau-Breen, Kuntsche, & Gmel, 2010; Latendresse, Rose, Viken, Pulkkinen, Kaprio, & Dick, 2008; Nash, McQueen, & Bray, 2005), religious involvement (Vakalahi, 2002), and parental divorce (Thompson, Lizardi, Keyes, & Hazin, 2008), amongst others. Future research should continue to examine a variety of predictors in order to better understand the full etiology of adolescent alcohol use.

Finally, Cholesky decomposition may be better utilized in analyzing other outcomes concerning adolescent development, such as internalizing or externalizing problems. For example, previous research (Burt et al., 2007) found stronger correlations between parent involvement and child externalizing than those found in this study between parent involvement and adolescent alcohol use. Following this, Pike et al. (1996) found stronger correlations between predictor variables of mother, father, and sibling negativity with their outcome variables of antisocial behavior (in the .60 range) and depressive symptoms (in the .30 range) than the initial correlations found in this study. Therefore, it may be that these theoretical concepts are more strongly related to externalizing and internalizing outcomes rather than adolescent alcohol use. Altogether, this study demonstrates the importance of preliminary analyses before conducting Cholesky decomposition.

**Conclusion**

The main conclusion based on this study's findings is that more research is needed to better understand how family predictors influence adolescent alcohol use via genetic or environmental mechanisms. The lack of findings in this study is most likely due to power issues, including correlations too low to decompose to begin with, as well as correlations that differ in magnitude depending on sub-sample selection. A logical next step in this program of research is to decompose correlations that have moderate to strong correlations, including the potentially strong correlation amongst alcohol availability in the home, sibling facilitation of alcohol use, and adolescent alcohol use. If findings show significant shared environmental influences on this association, implications would be to focus on characteristics of the sibling relationship (such as their facilitation of alcohol) in prevention and treatment of adolescents with alcohol use problems.

## General Discussion

For this dissertation, I took on three studies to further knowledge on sibling influences on adolescent alcohol use. All three studies utilized the same data source – the Sibling Interaction and Behavior Study (SIBS; McGue et al., 2007). SIBS produced data on over 600 sibling pairs and across sibling types that vary in their genetic relatedness (ADOPT, BIO, and ADOPT-BIO). Two waves of data were utilized across all three studies. The general discussion reviews key findings, discusses general implications, and comments on future directions in my general research program.

In Study 1, an alternate measurement of sibling similarity in alcohol use was assessed. The basic correlational approach (1-class model) was compared to 2-, 3-, 4-, and 5- class models of sibling similarity in alcohol use across two time points. The main implications of Study 1 findings are that indeed, multiple patterns of sibling similarity in alcohol use can be detected. There were both theoretical and statistical evidence to support this alternate measurement of sibling similarity; the 3-class model was the best fitting model in early adolescence, and the 4-class model was the best fitting model in later adolescence. Results show that utilizing latent class analysis (LCA) may be particularly useful in research that seeks to better understand the dynamics of sibling relationships in terms of substance use and other adjustment outcomes. In order to better understand what may lead to sibling similarity in alcohol use, it is useful to distinguish between sibling groups that are similar in *low* versus *high* alcohol use, as well as between groups representing differential patterns of alcohol use. Determining predictors of these unique groups may shed light on areas that may be important in reducing overall rates of adolescent alcohol use.

Further supporting this argument, Study 2 established the predictive validity of sibling similarity in alcohol use patterns. Sibling relationship quality differentially predicted the four latent classes at Wave 2; sibling communication and conflict were significantly associated, but closeness was not. The main implications of Study 2 findings build on those of Study 1. Future research in the field of sibling influences in adolescence may find it useful to utilize LCA to better understand the sibling dynamics in relation to adjustment outcomes such as alcohol and substance use. Collapsing sibling dynamics by using a correlational method may hide capabilities of what may predict unique patterns of sibling similarity in adjustment outcomes. A better understanding of what predicts these sibling groups may be useful for clinicians and future intervention and prevention work in the field of adolescent alcohol and substance use. For example, it may be useful to monitor characteristics of sibling relationship quality for those in adolescent substance use treatment. It may be that bringing the sibling into the treatment with the adolescent will be useful in their treatment. Future research is needed to replicate and extend Study 2 findings in order to build a case for such intervention studies.

Finally, Study 3 sought to decompose the correlations amongst mom, dad, sibling involvement, and adolescent alcohol use by examining the contribution of both genetics and the environment. This was done in order to determine which variables had significant associations via the shared environment, which could then be targeted for environmental intervention and prevention work. While results appeared to initially support study hypotheses, there were few significant paths found when examining the 95% confidence intervals in Study 3 results. It was concluded that the lack of findings was most likely due to power issues, including the low correlations amongst family involvement and adolescent alcohol use to begin with, as well as the stronger correlations between same-sex pairs, and non-significant correlations between opposite-sex, ADOPT-BIO pairs.

In general, I learned a great deal conducting Study 3 analyses. I learned that correlations need to be much stronger in order to be decomposed to begin with. After reviewing the literature comparing different predictors of adolescent alcohol use, I determined one hopeful area of future research would be to decompose the correlation between sibling facilitation of alcohol use and adolescent alcohol use, as previous research has shown this relationship to have a high correlation (D'Amico & Fromme, 1997; Fagan & Najman, 2005; McGue & Iacono, 2009; Tildesley et al., 1993). Even with this high correlation, I know of no research to date that has decomposed these correlations.

A logical next step in my program of research is to continue to utilize Cholesky decomposition in analyses predicting individual adolescent alcohol use and other adjustment outcomes, as well as to continue examining sibling influences on adolescent alcohol use through a dyadic sibling framework (e.g., by using LCA, through models of moderation and mediation).

At the individual level, I intend to accumulate information that can be used to target adolescents for prevention and intervention of alcohol use by determining covariance that can be largely explained by the shared environment. I aim to discover that the shared environment is largely shaped by the sibling context, and provide arguments for closely monitoring and evaluating the sibling context at the therapy and prevention levels.

At the dyad level, I aim to learn more about the importance of contextual factors in explaining sibling similarity in alcohol and substance use (e.g., age difference, communication patterns) in order to better understand and evaluate sibling influences on adolescent substance use. This will be done through analyses utilizing moderators, mediators (Samek & Rueter, 2011), and alternate measures of sibling similarity and influences via LCA (Study 1 and 2). In both cases, the long-term goal of this research program is to have applicable findings for adolescents and their families that are seeking substance use treatment. In this way, I aim to continue my research on siblings by using an interdisciplinary approach. I will continue using methods from both fields of family science and behavioral genetics in order to produce research that has implications for family research, prevention/intervention professionals, and families themselves.

**Table 1.1. Descriptive Statistics for Study 1 Variables**

| | *M* | *SD* | Range | % Complete |
|---|---|---|---|---|
| Wave 1 | | | | |
| Elder Sibling | | | | |
| Ever use alcohol | .38 | .49 | 0 – 1 | 99.5 |
| Alcohol frequency | 1.16 | 1.86 | 0 – 7 | 99.5 |
| Ever been intoxicated | .25 | .44 | 0 – 1 | 99.5 |
| Intoxication frequency | .76 | 1.58 | 0 – 7 | 99.5 |
| Drank to get drunk | .82 | 1.58 | 0 – 5 | 99.5 |
| Max drinks | 1.70 | 3.03 | 0 – 9 | 99.5 |
| Younger Sibling | | | | |
| Ever use alcohol | .18 | .39 | 0 – 1 | 99.5 |
| Alcohol frequency | .45 | 1.19 | 0 – 7 | 99.5 |
| Ever been intoxicated | .08 | .28 | 0 – 1 | 99.5 |
| Intoxication frequency | .26 | .99 | 0 – 7 | 99.5 |
| Drank to get drunk | .30 | 1.07 | 0 – 5 | 99.5 |
| Max drinks | .65 | 1.98 | 0 – 9 | 99.5 |
| Wave 2 | | | | |
| Elder Sibling | | | | |
| Ever use alcohol | .76 | .43 | 0 – 1 | 77.7 |
| Alcohol frequency | 3.32 | 2.51 | 0 – 9 | 77.7 |
| Ever been intoxicated | .65 | .48 | 0 – 1 | 77.7 |
| Intoxication frequency | 2.49 | 2.36 | 0 – 9 | 77.7 |

| | | | | |
|---|---|---|---|---|
| Drank to get drunk | 2.41 | 2.05 | 0 – 5 | 77.7 |
| Max drinks | 5.05 | 3.78 | 0 – 9 | 77.7 |
| Younger Sibling | | | | |
| Ever use alcohol | .54 | .50 | 0 – 1 | 80.9 |
| Alcohol frequency | 1.89 | 2.22 | 0 – 9 | 80.9 |
| Ever been intoxicated | .39 | .49 | 0 – 1 | 80.9 |
| Intoxication frequency | 1.35 | 2.04 | 0 – 7 | 80.9 |
| Drank to get drunk | 1.40 | 1.95 | 0 – 5 | 80.9 |
| Max drinks | 3.13 | 3.76 | 0 – 9 | 80.9 |

**Table 1.2. Comparison of 1-, 2-, 3-, 4- and 5-class sibling alcohol use patterns models using Wave 1 Reports (*N* = 613)**

| | 1-class | 2-class | | 3-class[b] | | | 4-class[b] | | | | 5-class | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Means | 1 | 2 | 1[b] | 2 | 3 | 1 | 2[b] | 3[b] | 4 | 1 | 2 | 3 | 4 | 5 |
| Elder Sib | | | | | | | | | | | | | | | |
| Ever alc | .38 | .22 | **1.0** | **1.0** | .21 | **.78** | .21 | **.66** | **.91** | **1.0** | **1.0** | .15 | .44 | **1.0** | **1.0** |
| Freq alc | 1.16 | .36 | **4.24** | **4.22** | .34 | **2.96** | .34 | **2.60** | **3.38** | **4.22** | **2.61** | .19 | .83 | **4.60** | **4.40** |
| Ever intox | .25 | .06 | **1.0** | **1.0** | .05 | **.73** | .05 | **.61** | **.87** | **1.0** | **.43** | .04 | .39 | **1.0** | **1.0** |
| Intox freq | .76 | .07 | **3.42** | **3.36** | .06 | **2.34** | .06 | **1.91** | **2.84** | **3.35** | **.67** | .05 | .50 | **3.79** | **3.60** |
| Drunk | .82 | .07 | **3.72** | **3.75** | .06 | **2.31** | .06 | **1.97** | **2.74** | **3.74** | **.67** | .05 | .50 | **3.73** | **4.04** |
| Maxdrink | 1.71 | .30 | **7.15** | **7.13** | .28 | **4.61** | .28 | **3.72** | **5.67** | **7.12** | **4.28** | .05 | .78 | **7.31** | **7.36** |
| Younger Sib | | | | | | | | | | | | | | | |
| Ever alc | .18 | .12 | **.42** | .25 | .09 | **1.0** | .09 | **1.0** | **1.0** | .24 | .30 | .08 | **1.0** | **1.0** | .24 |
| Freq alc | .45 | .27 | **1.18** | .44 | .14 | **3.78** | .14 | **4.87** | **2.55** | .43 | .57 | .12 | **4.11** | **3.60** | .41 |

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Ever intox | .08 | .04 | **.28** | .06 | .00 | **1.0** | .00 | **1.0** | **1.0** | .05 | .05 | .00 | **1.0** | **1.0** | .05 |
| Intox freq | .26 | .13 | **.77** | .06 | .00 | **3.46** | .00 | **4.78** | **1.96** | .05 | .07 | .00 | **4.06** | **3.08** | .05 |
| Drunk | .30 | .13 | **.94** | .06 | .00 | **3.98** | .00 | **4.22** | **3.60** | .05 | .07 | .00 | **4.00** | **4.04** | .05 |
| Maxdrink | .65 | .31 | **1.99** | .55 | .10 | **6.73** | .10 | **7.48** | **6.04** | .47 | .69 | .07 | **6.72** | **6.84** | .78 |
| % in class | 1.0 | .80 | .20 | .16 | .76 | .07 | .76 | .04 | .04 | .16 | .07 | .71 | .03 | .04 | .14 |
| # of params | 27 | 44 | | 63 | | | 82 | | | | 101 | | | | |
| AIC | 24692 | 19578.20 | | 16503.14 | | | 15529.38 | | | | 15146.37 | | | | |
| Adjusted BIC | 24724 | 19632.912 | | 16581.488 | | | 15631.35 | | | | 15271.97 | | | | |
| Entropy | -- | .99 | | .99 | | | 1.0 | | | | .99 | | | | |
| LRT *p* | -- | < .0001 | | .045 | | | .65 | | | | .76 | | | | |

*NOTE:* Numbers in bold indicate high alcohol, use relative to sample averages. AIC refers to the Akaike Information Criterion, BIC refers to Bayesian Information Criterion. [a]Sex composition (same versus mixed), [b]age difference, and [c]genetic similarity (related vs. not) were treated as covariates, an & denotes significance at the p <= .05 level (last class was the reference class in 2-5 class models)

**Table 1.3. Comparison of 1-, 2-, 3-, 4- and 5-class sibling alcohol use patterns models using Wave 2 Reports (*N* = 613)**

| | 1-class | 2-class[bc] | | 3-class[b] | | | 4-class[bc] | | | | 5-class[ab] | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Means | 1[bc] | 2 | 1 | 2[b] | 3 | 1[c] | 2 | 3[bc] | 4 | 1 | 2[b] | 3[a] | 4 | 5 |
| Elder Sib | | | | | | | | | | | | | | | |
| Ever alc | .76 | .67 | **.93** | .35 | **1.0** | **.93** | .36 | .58 | **1.0** | **1.0** | .36 | **1.0** | .56 | **1.0** | **1.0** |
| Freq alc | 3.32 | 2.69 | **4.52** | .77 | **4.78** | **4.46** | .80 | 1.23 | **4.79** | **5.25** | .80 | **4.76** | 1.16 | **5.45** | **5.11** |
| Ever intox | .65 | .54 | **.85** | .11 | **1.0** | **.85** | .12 | .19 | **1.0** | **1.0** | .13 | **1.0** | 1.0 | **1.0** | **1.0** |
| Intox freq | 2.49 | 1.92 | **3.60** | .12 | **3.89** | **3.52** | .15 | .24 | **3.89** | **4.34** | .15 | **3.87** | .14 | **4.70** | **4.24** |
| Drunk | 2.41 | 1.96 | **3.28** | .13 | **3.93** | **3.22** | .15 | .24 | **3.95** | **3.95** | .15 | **3.94** | .14 | **3.89** | **3.96** |
| Maxdrink | 5.05 | 4.13 | **6.84** | .74 | **7.75** | **6.78** | .79 | 1.34 | **7.80** | **8.07** | .80 | **7.79** | 1.10 | **7.95** | **8.09** |
| Younger Sib | | | | | | | | | | | | | | | |
| Ever alc | .54 | .30 | **1.0** | .18 | .44 | **1.0** | .15 | **1.0** | .45 | **1.0** | .15 | .43 | **.14** | **1.0** | **1.0** |
| Freq alc | 1.89 | .61 | **4.40** | .27 | .96 | **4.45** | .22 | **3.57** | .98 | **4.62** | .22 | .93 | **3.88** | **2.89** | **5.44** |

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Ever intox | .39 | .08 | **1.0** | .06 | .11 | **1.0** | .03 | **1.0** | .10 | **1.0** | .03 | .08 | **1.0** | **1.0** | **1.0** |
| Intox freq | 1.35 | .09 | **3.81** | .09 | .12 | **3.86** | .03 | **2.88** | .11 | **4.10** | .03 | .08 | **3.21** | **1.94** | **5.16** |
| Drunk | 1.40 | .09 | **3.93** | .11 | .12 | **3.98** | .03 | **3.17** | .11 | **4.17** | .03 | .08 | **3.42** | **3.23** | **4.46** |
| Maxdrink | 3.13 | .68 | **7.89** | .27 | 1.18 | **7.95** | .17 | **6.81** | 1.18 | **8.12** | .17 | 1.05 | **6.94** | **7.20** | **8.43** |
| % in class | 1.0 | .69 | .31 | .31 | .35 | .34 | .33 | .07 | .34 | .26 | .32 | .32 | .07 | .11 | .17 |
| # of params | 27 | 44 | | 57 | | | 73 | | | | 89 | | | | |
| AIC | 25536 | 20403.06 | | 18731.45 | | | 17655.04 | | | | 17115.68 | | | | |
| Adjusted BIC | 25569 | 20457.77 | | 18802.34 | | | 17745.82 | | | | 17226.36 | | | | |
| Entropy | -- | .84 | | .81 | | | .82 | | | | .82 | | | | |
| LRT *p* | -- | < .0001 | | .012 | | | < .001 | | | | .04 | | | | |

*NOTE:* Numbers in bold indicate high alcohol use, relative to sample averages. AIC refers to the Akaike Information Criterion, BIC refers to Bayesian Information Criterion. [a]Sex composition (same versus mixed), [b]age difference, and [c]genetic similarity (related vs. not) were treated as covariates, an & denotes significance at the p <= .05 level (last class was the reference class in 2-5 class models)

**Table 2.1. Overview of Sibling Relationship Quality Measures for Study 2**

| | Characteristics of Relationship Quality | Reliability |
|---|---|---|
| **Observed** | | ICCs |
| Closeness | Warmth *(2 indicators)* | .44 - .83 |
| | Relationship Quality *(1 indicator)* | .84 |
| Conflict | Hostility *(2 indicators)* | .78 - .88 |
| | Anger Coercion *(2 indicators)* | .66 - .87 |
| Communication | Communication *(2 indicators)* | .68 – .72 |
| | Listener Responsiveness *(2 indicators)* | .68 - .72 |
| **Self-Reported** | | α |
| Closeness | Affect *(3 items)* | .88 - .90 |
| | Nurturance *(3 items)* | .85 - .87 |
| | Admiration *(3 items)* | .91 - .93 |
| Conflict | Quarreling *(3 items)* | .88 - .91 |
| | Antagonism *(3 items)* | .70 - .74 |
| | Competition *(3 items)* | .86 - .90 |
| Communication | Intimacy *(3 items)* | .90 - .91 |
| | Prosocial Behavioral *(3 items)* | .75 - .78 |

*NOTE:* ICC refers to intra-class correlation coefficient

**Table 2.2. Overview of Fit and Descriptive Statistics for Relationship Quality Latent Factors for Study 2**

| | Number of Indicators | Standardized Factor Loadings | RMSEA | 90% CI for RMSEA | SRMR | *M* | *SD* | *Range* | % Complete |
|---|---|---|---|---|---|---|---|---|---|
| **Observed** | | | | | | | | | |
| Sibling Closeness | 3 | .66 - .84 | .00 | .00 - .04 | .01 | .26 | 1.35 | -2.34 – 4.41 | 98.4 |
| Sibling Conflict | 4 | .43 - .94 | .32 | .30 – 34 | .11 | -.00 | 2.10 | -2.80 – 5.38 | 98.4 |
| Sibling Communication | 4 | .54 - .67 | .07 | .05 - .10 | .03 | .06 | .48 | -.88 – 2.21 | 98.4 |
| **Self-Reported** | | | | | | | | | |
| Elder Closeness | 3 | .72 - .91 | .11 | .08 - .14 | .03 | 2.57 | 2.14 | -4.21 – 6.95 | 95.9 |
| Younger Closeness | 3 | .70 - .88 | .08 | .06 - .11 | .03 | 1.81 | 2.05 | -5.01 – 6.07 | 98.2 |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Elder Conflict | 3 | .57 - .92 | .11 | .08 - .14 | .04 | -4.91 | 2.38 | -10.32 – 1.34 | 95.8 |
| Younger Conflict | 3 | .46 - .88 | .10 | .07 - .13 | .04 | -1.20 | 2.25 | -6.36 – 5.42 | 98.0 |
| Elder Communication | 2 | .64 – 1.00 | .03 | .00 - .09 | .01 | 5.10 | 3.09 | -.85 – 13.22 | 95.9 |
| Younger Communication | 2 | .77 - .88 | .04 | .00 - .10 | .01 | 5.02 | 2.57 | -1.13 – 11.90 | 98.2 |

*NOTE:* RMSEA refers to the Root Mean Square Error of Approximation, SRMR refers to the Standardized Root Mean Square Residual

**Table 2.3. Correlations amongst Continuous Study 2 Predictors**

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|
| 1. Sibling observed closeness | -- | | | | | | | | |
| Sibling self-report closeness | -- | -- | | | | | | | |
| 2. Elder report | .33 | -- | | | | | | | |
| 3. Younger report | .31 | .44 | -- | | | | | | |
| 4. Sibling observed conflict | -.27 | -.16 | -.11 | -- | | | | | |
| Sibling self-report conflict | -- | -- | -- | -- | -- | | | | |
| 5. Elder report | -.19 | -.37 | -.26 | .22 | -- | | | | |
| 6. Younger report | -.19 | -.26 | -.26 | .16 | .37 | -- | | | |
| 7. Sibling observed communication | .24 | .14 | .13 | -.02 † | -.10 | -.06 † | -- | | |
| Sibling self-report communication | -- | -- | -- | -- | -- | -- | -- | -- | |
| 8. Elder report | .26 | .60 | .34 | -.09 | -.19 | -.18 | .10 | -- | |
| 9. Younger report | .27 | .39 | .71 | -.05 † | -.21 | -.20 | .13 | .52 | -- |

*NOTE:* All correlations are significant at p < .05, unless noted with †, then *ns*

**Table 2.4. Sibling Relationship Quality Means (Wave 1) across 4-class Sibling Alcohol Model (Wave 2)**

| | **LOW-LOW (*n* =192)** | **eLOW-yHIGH (*n* =48)** | **eHIGH-yLOW (*n* =209)** | **HIGH-HIGH (*n* =164)** |
|---|---|---|---|---|
| **Observed** | | | | |
| Sibling Closeness | .47 | .21 | .08 | .27 |
| Sibling Conflict | -.06[b] | .98[acd] | -.26[b] | .12[b] |
| Sibling Communication | .07[b] | .24[ac] | -.05[bd] | .14[c] |
| **Self-Reported** | | | | |
| Elder Closeness | 2.7 | 2.28 | 2.32 | 2.83 |
| Younger Closeness | 1.8 | 2.08 | 1.74 | 1.83 |
| Elder Conflict | -4.8[b] | -5.25[ac] | -4.77[b] | -5.15 |
| Younger Conflict | -1.21 | -.89 | -1.38 | -1.04 |
| Elder Communication | 5.1[d] | 5.16[d] | 4.32 | 7.17[ac] |
| Younger Communication | 4.77[c] | 5.74 | 4.93[a] | 5.32 |

*NOTE:* [a] = LOW-LOW (low levels of alcohol by both siblings), [b] = eLOW-yHIGH (youngers > elders), [c] = eHIGH-yLOW (elders > youngers), [d] = HIGH-HIGH (high levels of alcohol use by both siblings). Superscript denotes significant differences across groups (some marginal).

**Table 3.1. Descriptive Statistics for Study 3 Variables**

| | *M* | *SD* | *Range* | % Complete |
|---|---|---|---|---|
| Elder Sibling | | | | |
| Mother involvement (Wave 1) | .58 | .56 | -.24 – 2.77 | 95.4 |
| Father Involvement (Wave 1) | 1.06 | .56 | .18 – 3.04 | 94.6 |
| Sibling Involvement (Wave 1) | .32 | .71 | -1.51 – 1.90 | 95.8 |
| Alcohol Use (Wave 2) | 5.10 | 1.08 | 3.44 – 7.21 | 77.7 |
| Younger Sibling | | | | |
| Mother involvement (Wave 1) | .27 | .36 | -.24 – 1.55 | 97.6 |
| Father Involvement (Wave 1) | .70 | .41 | .07 – 2.12 | 97.1 |
| Sibling Involvement (Wave 1) | .32 | .76 | -1.40 – 1.99 | 98.0 |
| Alcohol Use (Wave 2) | 4.46 | 1.08 | 3.39 – 6.88 | 80.9 |

**Table 3.2. Standardized Beta Weights of the Association between Age, Sex, Ethnicity and Study 3 Variables**

| | Age | Sex | Ethnicity |
|---|---|---|---|
| Elder Sibling | | | |
| Mother Involvement | .13** | -.13** | -.04 |
| Father Involvement | .17*** | -.01 | -.00 |
| Sibling Involvement | .10* | -.22*** | -.06 |
| Adolescent Alcohol Use | .31*** | -.06 | .06 |
| Younger Sibling | | | |
| Mother Involvement | .17*** | -.23*** | -.07 |
| Father Involvement | .23*** | -.13** | -.04 |
| Sibling Involvement | -.06 | .00 | -.10* |
| Adolescent Alcohol Use | .50*** | -.08* | .09* |

*NOTE:* Sex is coded (1 = male, 2 = female); ethnicity is coded (1 = White, 0 = African-American, Asian, Mixed, Other)

*** $p < .001$

** $p < .01$

* $p < .05$

**Table 3.3. Correlations amongst Study 3 Variables**

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|
| Elder Sibling | | | | | | | | |
| 1. Mom Involvement | - | | | | | | | |
| 2. Dad Involvement | .81 | - | | | | | | |
| 3. Sibling Involvement | .43 | .37 | - | | | | | |
| 4. Alcohol Use | .16 | .15 | .12 | - | | | | |
| Younger Sibling | | | | | | | | |
| 5. Mom Involvement | .18 | .15 | .14 | .17 | - | | | |
| 6. Dad Involvement | .17 | .26 | .18 | .15 | .77 | - | | |
| 7. Sibling Involvement | .24 | .19 | .52 | .08* | .27 | .29 | - | |
| 8. Alcohol Use | .12 | .14 | .06ǂ | .39 | .24 | .24 | .08* | - |

*NOTE:* All correlations are significant at $p < .01$ unless * $p < .10$, or ǂ $p > .10$

**Table 3.4. Correlations of Study 3 Variables across Biological, Adopted, and Adopted-Biological Sibling Pairs**

| | BIO | | | ADOPT | | | ADOPT-BIO | | |
|---|---|---|---|---|---|---|---|---|---|
| | Total | Same sex | Opposite sex | Total | Same sex | Opposite sex | Total | Same sex | Opposite sex |
| Mom Involvement | .24** | .34** | .11 | .14** | .22** | .04 | .17 Ϯ | .23* | -.10 |
| Dad Involvement | .38** | .41** | .34** | .19** | .17* | .22** | .02 | .07 | -.20 |
| Sibling Involvement | .60** | .65** | .48** | .47** | .49** | .37** | .49** | .48** | .32 |
| Alcohol Use | .46** | .50** | .38** | .35** | .38** | .31** | .38** | .44** | .13 |

*NOTE:* BIO = Full biological siblings, ADOPT = Adopted, genetically unrelated siblings, ADOPT-BIO = Sibling pair where one is adopted, one is biological offspring of parents (siblings are genetically unrelated to one another)

** $p < .01$

* $p < .05$

Ϯ $p > .10$

**Table 3.5. Univariate Genetic and Environmental Contributions to Family Involvement and Adolescent Alcohol Use**

| | Mom Involvement | Dad Involvement | Sibling Involvement | Adolescent Alcohol Use |
|---|---|---|---|---|
| A | .22 (.03, .46) | .35 (.11, .58) | .33 (.13, .53) | .25 (.01, .56) |
| C | .05 (.02, .11) | .06 (.02, .13) | .46 (.38, .53) | .23 (.12, .33) |
| E | .73 (.49, .91) | .59 (.37, .81) | .21 (.06, .38) | .53 (.27, .76) |

*NOTE:* This table denotes % of variance explained by additive genetic (A), shared environmental (C), and nonshared environmental (E) effects (column's add to 1.0, i.e., 100%). Confidence intervals are shown in parentheses.

**Table 3.6. Comparison of Model Fit Indices**

| Model | -2 lnL | *df* | $\chi^2$ | *p*-value | *AIC* | *BIC* |
|---|---|---|---|---|---|---|
| ACE | 7,324.99 | 4480 | -- | -- | -1635.01 | -3603.10 |
| CE | 7,345.71 | 4490 | 20.72 | .02 | -1634.29 | -3608.96 |
| AE | 7,462.77 | 4490 | 137.78 | <.0001 | -1517.23 | -3550.43 |
| E | 7,594.89 | 4500 | 269.90 | <.0001 | -1405.11 | -6508.65 |

*NOTE*: -2 lnL refers to -2 times the loglikelihood, AIC refers to the Akaike Information Criterion, BIC refers to Bayesian Information Criterion.

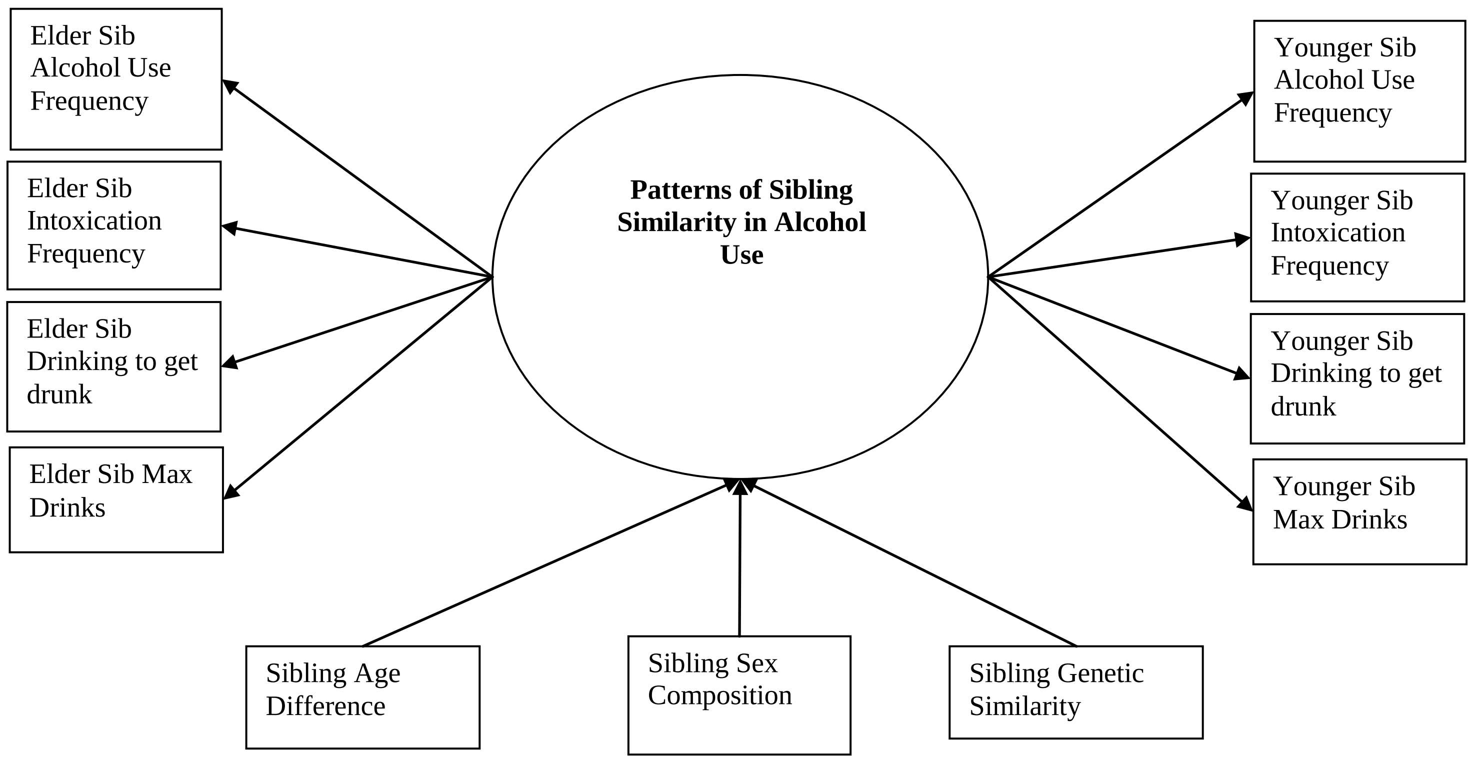


**Figure 1.1 Latent Variable Plan for Study 1.** Patterns of sibling similarity in alcohol use was measured as a latent factor, indicated by both elder and younger reports of alcohol use.  Covariates included sibling age difference, sex (same versus opposite), and genetic similarity (genetically versus not genetically related).

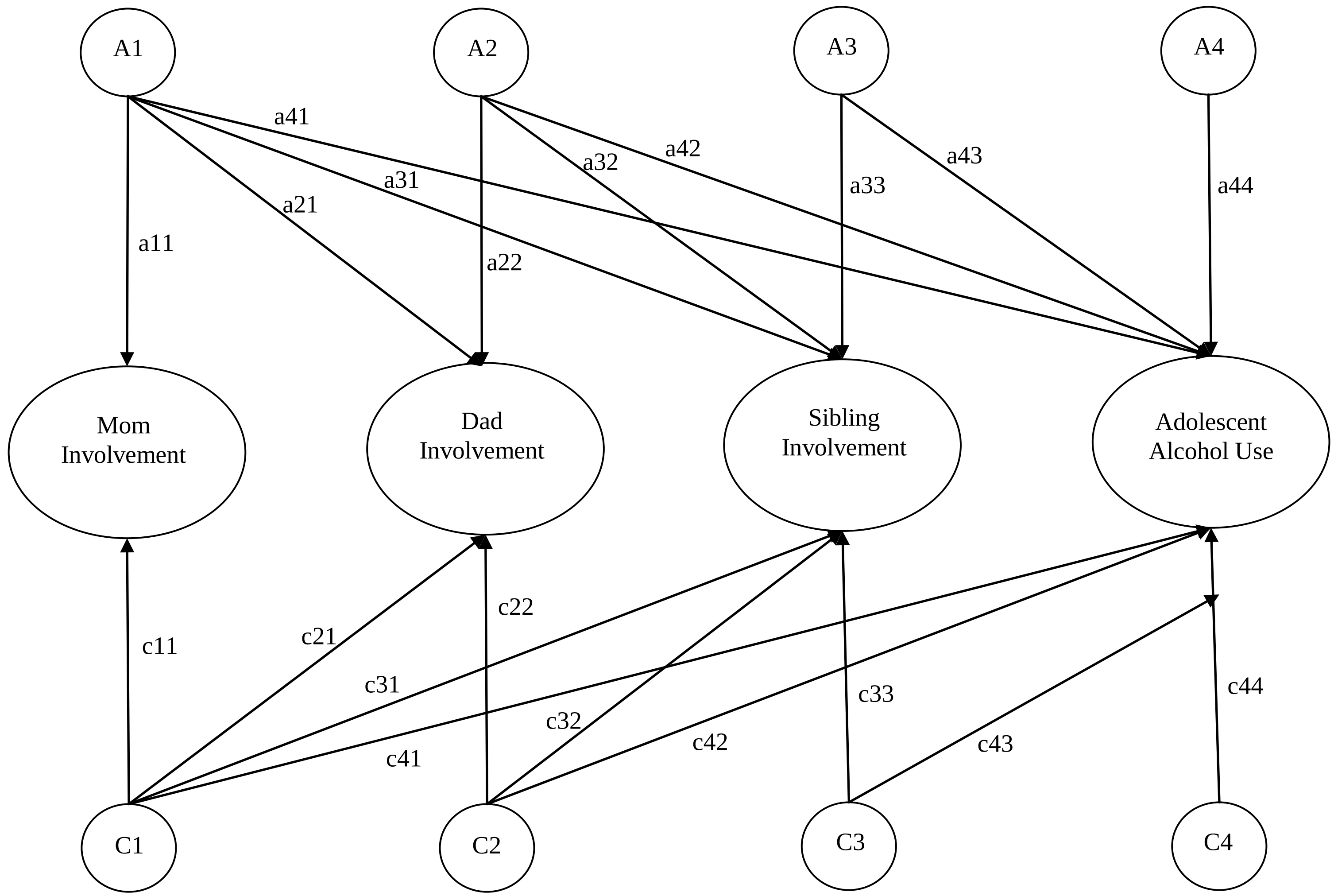


**Figure 3.1. Conceptual Model for Study 3.** This model represents the path diagram of the Cholesky decomposition model. Variance of each phenotypes (Mom, Dad, Sibling involvement, Adolescent Alcohol Use) is partialled out into additive genetic

effects (A1, A2, A3, A4), shared environmental effects (C1, C2, C3, C4), and non-shared environmental effects. Though in the model, non-shared environmental effects (E1, E2, E3, E4) are not represented here to promote ease of presentation. Paths, represented by lowercase letters follow by two numbers (e.g., a11, a21) are squared to estimate the proportion of variance accounted for.

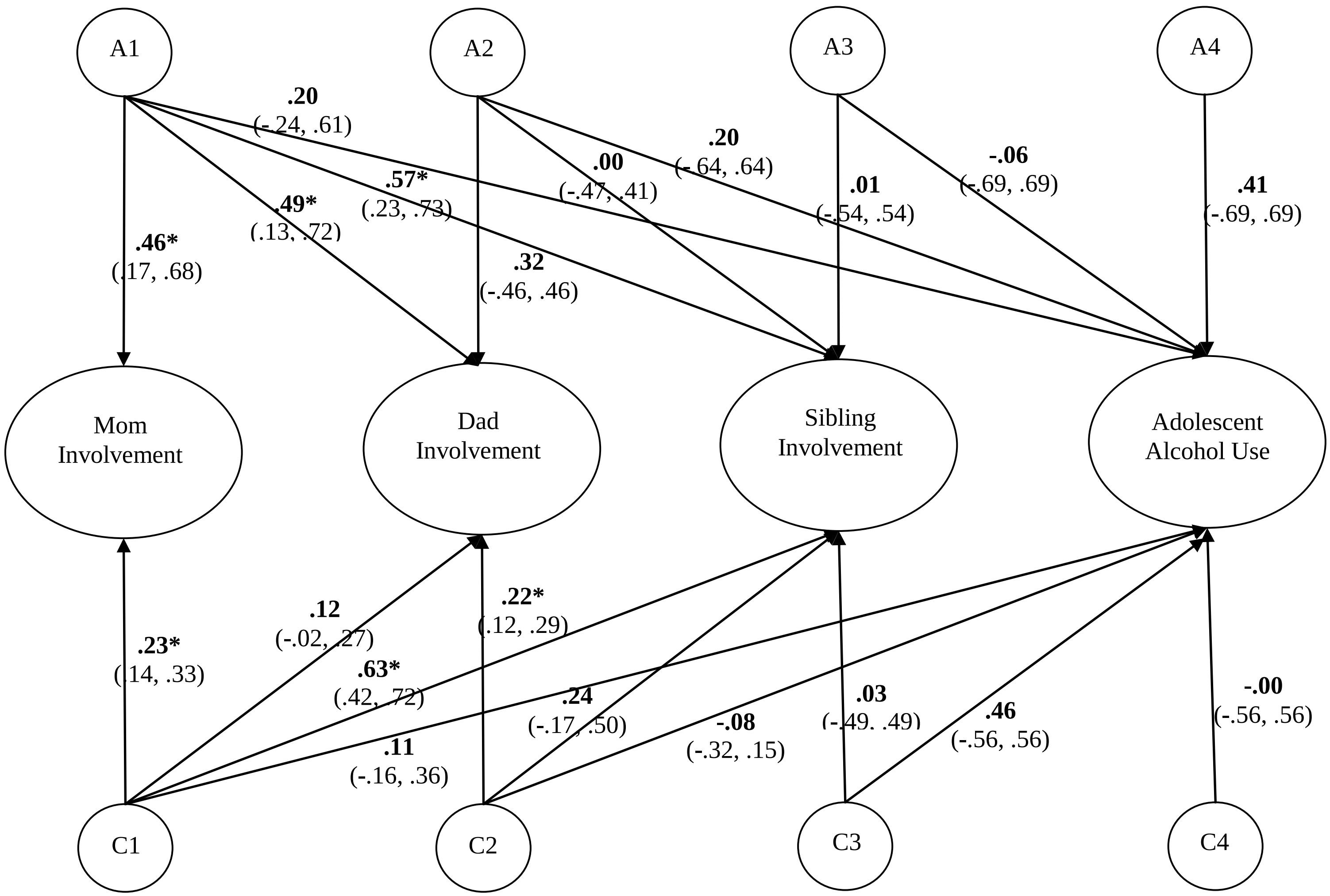
A1
A2
A3
A4
.20
(-.24, .61)
.20
(-.64, .64)
.00
(-.47, .41)
.57*
(.23, .73)
.49*
(.13, .72)
.46*
(.17, .68)
.32
(-.46, .46)
.01
(-.54, .54)
-.06
(-.69, .69)
.41
(-.69, .69)
Mom
Involvement
Dad
Involvement
Sibling
Involvement
Adolescent
Alcohol Use
.23*
(.14, .33)
.12
(-.02, .27)
.22*
(.12, .29)
.63*
(.42, .72)
.24
(-.17, .50)
.11
(-.16, .36)
-.08
(-.32, .15)
.03
(-.49, .49)
.46
(-.56, .56)
-.00
(-.56, .56)
C1
C2
C3
C4

**Figure 3.2. Standardized Path Estimates.** The variance within and the covariance amongst study variables are decomposed into genetic (A), shared environmental (C), and non-shared environmental I components. The standardized non-shared environmental estimates in the model are not shown for clarity (e11 = .86*, e21 =.66*, e31 = -.09, e41 =.18, e22 =.40*, e32 = .10, e42 = -.00, e33 =.44*, e34 = -.14, e44 = .69). Ninety-five percent confidence intervals are also presented; those that do not overlap zero indicate a significant path (also indicated by * $p < .05$). As a reminder, involvement variables were reverse scored prior to analyses.

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