

EFFECTS OF SIBSHIP SIZE AND COMPOSITION ON YOUNGER BROTHERS'
AND SISTERS' ALCOHOL USE INITIATION: FINDINGS FROM AN AUSTRALIAN
TWIN SAMPLE

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Master of Arts

by
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The undersigned, appointed by the dean of the Graduate School, have examined the thesis entitled

EFFECTS OF SIBSHIP SIZE AND COMPOSITION ON YOUNGER BROTHERS'
AND SISTERS' ALCOHOL USE INITIATION: FINDINGS FROM AN AUSTRALIAN
TWIN SAMPLE

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ABSTRACT

The effects of sibship size and structure on delinquency are well established, but despite strong links between delinquency and alcohol use, the contribution of these factors to drinking behaviors remains largely unexplored. The current study investigated the impact of sibship size and composition on younger brothers' and sisters' ages of drinking and intoxication onset. Large sibship size was hypothesized to facilitate earlier onset in both males and females, and having many older brothers was hypothesized to predict earlier drinking in males and later drinking in females. These hypotheses were tested through a series of statistical investigations performed on information collected from a large Australian twin sample. Results indicated that sibship size and composition effects are strongest when older siblings are close in age. In addition, close in age siblings exerted the strongest effects on drinking when: (1) respondents were from homes of divorce; and (2) they did not have a paternal history of alcohol problems. Potential mechanisms behind these effects and their implications for prevention and intervention are discussed.

Chapter 1: Introduction

Large family size is widely found to predict delinquency (Brownfield & Sorenson, 1994; Farrington, 1996; Farrington, 2005; Robins, 1966), and longitudinal studies in particular provide evidence for increased risk. In the Cambridge Study of Delinquent Development, one of the most important predictors of conviction rate up to age 32 was large family size (Farrington, 1993; Farrington, 2005). Similar risk was observed in adolescents in the Pittsburgh and Oregon Youth Studies and the Nottingham study (Farrington & Loeber, 2000; Newson, Newson, & Adams, 1993). It is also notable that while sibship size contributes to delinquency risk, the two are not linearly related. Lauritsen (1993) observed that while levels of offending in adolescents from one- and two-child homes were comparable, rates in families of three to five children were much greater. Hypotheses for why large family size predicts delinquency include a “contagion” model, in which large families increase the risk of exposure to delinquent siblings (Robins et al., 1975), poor parental supervision in large families (Farrington, 1996), and familial disruption (Farrington, 2005; Brownfield & Sorenson, 1994).

Because sibship size is a function of composition (number of males and females in the sibship), it is important to investigate how composition may account for the effect of family size. Indeed, some studies have found that the relation between family size and delinquency is explained by the number of brothers in the sibship (Lauritsen, 1993;

Loeber & Stouthamer-Loeber, 1986). Having many brothers has been shown to predict delinquency in boys (Lauritsen, 1993; Loeber & Stouthamer-Loeber, 1986; Reiss & Farrington, 1991), and Lauritsen (1993) found a different effect for girls, in which rates of delinquency decreased as number of brothers increased. However, research on the influence of brothers on female delinquency has produced mixed results. Jones, Offord, & Abrams (1980) found that brothers potentiated antisocial behavior in boys, but not in girls. Others have also found stronger sibling effects for brothers than sisters (Farrington, 1996). Therefore, clarifying the effect of older brothers on female risk behaviors is an important research goal.

Other factors may both underlie and interact with sibling type and number to explain their effects on adolescent delinquency. For example, similarity in offending may stem from strong sibling bonds (Rowe & Gulley, 1992), recruitment to co-offend in families with many brothers (Reiss & Farrington, 1991), and shared family and environmental effects (Rowe & Farrington, 1997; Rowe, Rodgers, & Meseck-Bushey, 1992). Older sibling behaviors in particular have been shown to predict younger sibling delinquency, largely through modeling (Loeber & Stouthamer-Loeber, 1986; Fagan & Najman, 2003; Slomkowski, Rende, Conger, Simons, & Conger, 2001). Behavioral and attitudinal influences may therefore partially explain sibship size and composition effects.

Substantial evidence exists for a link between externalizing behavior disorders and substance use. While the direction of causality is unclear, delinquency has been found to correlate strongly with numerous measures of alcohol use, including past year weekly drinking and frequency of intoxication (French & Maclean, 2006; Webb, Bray,

Getz, & Adams, 2002). In addition to co-occurring with alcohol use, conduct disorder (CD) and alcohol abuse are frequently comorbid (Hall, 2009; Kessler, Crum, Warner, Nelson, Schulenberg, & Anthony, 1997; Merikangas et al., 1998). This is due possibly to shared genetic risk (Iacono, Carlson, Taylor, Elkins, & McGue, 1999; Slutske et al., 1998) and shared family environmental factors (True et al., 1999). In addition, while childhood conduct problems do not place individuals at much risk for alcohol abuse, delinquency that continues into adolescence increases individuals' likelihood of developing alcoholism (Hawkins, Catalano, & Miller, 1992; King, Iacono, & McGue, 2004). CD has also been shown not only to co-occur with alcohol use and alcohol use disorders, but also to predict adolescent drinking (Disney, Elkins, McGue, & Iacono, 1999), and vice versa. Of particular interest to this study are findings that early age of drinking onset is associated with adolescent delinquency (Barnes, Welte, & Hoffman, 2002; Zhang, Wieczorek, & Welte, 1997). Also of interest are findings that family members' deviance can promote adolescents' alcohol use (Stormshak, Comeau, & Shepard, 2004).

Although research on the influence of sibship size and composition on drinking initiation is limited, studies have found that sibling attitudes and behaviors about alcohol are highly influential during adolescence (Ary, Tildesley, Hops, & Andrews, 1993; McGue, Sharma, & Benson, 1996; Scholte, Poelen, Willemsen, Boomsma, & Engels, 2008). In particular, findings regarding older sibling effects align with facilitative influences identified in the delinquency literature. Older siblings may provide their younger siblings with alcohol (Mayer, Forster, Murray, & Wagenaar, 1998; Needle,

McCubbin, Wilson, Reineck, Lazar, & Mederer, 1986; Wagenaar, Finnegan, Wolfson, Anstine, Williams, & Perry, 1993; Wagenaar, Toomey, Murray, Short, Wolfson, & Jones-Webb, 1996). In addition, individuals whose older siblings use alcohol are more likely to do so (Gfroerer, 1987; Van Der Vorst, Engels, Meeus, Dekovic, & Leeuwe, 2007), and younger siblings' alcohol use norms are associated with those of their older siblings (Brody, Flor, Hollett-Wright, & McCoy, 1998; Needle, McCubbin, Wilson, Reineck, Lazar, & Mederer, 1986). Older siblings' frequency and quantity of alcohol use predicts younger siblings' alcohol use over time (Trim, Leuthe, & Chassin, 2006; Van Der Vorst, Engels, Meeus, Dekovic, & Leeuwe, 2007), and simply having an older sibling, regardless of his or her drinking behaviors, may increase risk for alcohol use (Ellickson, Tucker, Klein, & McGuigan, 2001). Finally, younger siblings' perceptions of and expectancies about their older siblings' drinking, regardless of their accuracy, strongly shape their drinking behaviors (D'Amico & Fromme, 1997; Needle et al., 1986).

Despite these findings, the influence of sibship size and composition remains largely unexplored. To my knowledge, only two studies have examined the relation between family size and adolescent alcohol consumption (Fergusson, Lynskey, & Horwood, 1994; Little, 1989), and I am aware of only one study that investigated the association between number of older siblings and ages of drinking and intoxication onset (Hellandsjøbu, Watten, Foxcroft, Ingebrigtsen, & Relling, 2002). In addition, researchers have not investigated the influence of multiple older brothers or looked at outcomes in both younger brothers and sisters.

Understanding the importance of sibship composition to ages of drinking and intoxication onset is warranted for several reasons. First, early-life drinking behaviors have been shown to predict later alcohol use disorders (Chou & Pickering, 1992; DeWit, Adlaf, Offord, & Ogborne, 2000; Grant & Dawson, 1997; Grant, Stinson, & Harford, 2001; Hingson, Heeren, & Winter, 2006; Pitkänen, Lyyra, & Pulkkinen, 2005). Second, studies of both human and animal models indicate that age of onset is relevant to brain development, as heavy drinking in adolescence can result in both short- and long-term cognitive deficits. Outcomes include impaired memory formation, impaired decision-making, and diminished intellectual abilities (Monti et al., 2005; Swartzwelder, Wilson, & Tayyeb, 2006; White & Swartzwelder, 2004; Zeigler et al., 2005).

As noted above, delinquency and alcohol use are strongly linked. Given that they have a relation, it was predicted that sibship factors would influence both delinquency and drinking initiation similarly. The current study first aimed to replicate findings regarding the influence of sibship size and composition on delinquency (particularly, to investigate whether large sibships and having many brothers promoted delinquency). The study also extended findings from the delinquency literature to test three hypotheses regarding the influence of sibship size and composition on younger siblings' drinking initiation: (1) Large sibship size will facilitate earlier ages of drinking and intoxication onset in males and females; (2) Having many older brothers will predict earlier ages of drinking and intoxication onset in males; and (3) Having many older brothers will result in later ages of drinking and intoxication onset in females. Because there have been mixed findings regarding the effect of brothers on female delinquency, a goal of this

research was to clarify their influence on alcohol use. The third hypothesis, therefore, stems from the protective effect of brothers that was found in an exemplary study of delinquency based on a large representative national dataset (Lauritsen, 2003). These hypotheses were tested using data from a large epidemiologic database.

Chapter 2: Methods

Sample

The sample consisted of adult twins drawn from the Australian National Twin Register, a database of twin pairs and their relatives maintained by the Australian Twin Registry (ATR). The cohort (ATR Cohort II) consists of 4,268 twin pairs born between 1964 and 1971 (Slutske, Meier, Zhu, Statham, Blaszczyński, & Martin, 2009). Cohort members were assessed in two waves of interviews. The first wave, which included all variables to be used in analyses, is described below. Because members of opposite-sex twin pairs do not report having the same number of brothers and sisters, opposite-sex twins were excluded from analyses. Only same-sex monozygotic (MZ) and dizygotic (DZ) twins were selected ($n = 4,841$; 2,139 males and 2,702 females). See Table 1 for a list of sample characteristics.

Procedures

All members of ATR Cohort II completed two waves of interviews conducted by a team of trained lay-interviewers, with separate interviewers assessing each member of a

twin pair. The first wave (which included all variables to be used in the current study) consisted of a structured psychiatric telephone interview conducted between 1996 and 2000 ($n = 6,265$ twins), during which participants were administered the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA-OZ; see Slutske, Meier, Zhu, Statham, Blaszczynski, & Martin, 2009 for further information on interview procedures and participant demographics).

Measures

Conduct disorder. Conduct Disorder (CD) symptoms were assessed using the SSAGA-OZ and were evaluated here using a 15-item count variable. Respondents were asked if they had ever had any of the 15 symptoms (13 of which had to occur prior to age 18, and two of which had to occur prior to age 15). Because the variable was strongly positively skewed and kurtotic, it was rank-transformed prior to analyses. The skew of the CD variable was 2.63 and 1.07 prior to and following the transformation, respectively. The kurtosis of the variable was 8.63 and 0.02 prior to and following the transformation, respectively. Analyses using rank-transformed data were assessed, however, results from analyses using the raw data are presented in the appendix. The number of CD symptoms ranged from 0 to 10 ($M = 1.20$, $SD = 1.64$) for males and from 0 to 9 for females ($M = 0.39$, $SD = 0.89$). Figure 1 displays the frequency distribution of CD symptoms in the sample. Table 1 displays the prevalence of any CD symptoms in the sample, as well as the number of individuals who met criteria for conduct disorder (endorsed three or more symptoms).

Alcohol use disorder. An 11-item count variable assessing alcohol use disorder (AUD; alcohol abuse and alcohol dependence) symptoms was used to determine the strength of the relationship between AFD, AFI, and AUDs in the sample. Because the variable was strongly positively skewed and kurtotic, it was rank-transformed prior to analyses. The skew of the AUD variable was 2.07 and 0.66 prior to and following the transformation, respectively. The kurtosis of the variable was 4.84 and -0.52 prior to and following the transformation, respectively. The number of symptoms ranged from 0 to 7 for both men and women. The mean number of symptoms for the sample was 1.33 ($SD = 1.91$), and was 1.91 ($SD = 2.18$) and 0.99 ($SD = 1.60$) for men and women, respectively. Figure 2 displays the frequency distribution of AUD symptoms in the sample. Table 1 displays the prevalence of any AUD symptoms in the sample.

Sibship composition. Sibship composition was assessed using sibling information collected as part of the telephone interview conducted between 1996 and 2000 (Slutske, Meier, Zhu, Statham, Blaszczyński, & Martin, 2009). Individuals were asked to report on the number of biological siblings they had, alive and dead (not including their co-twin). Two types of CIAO brother and sister variables were created to better assess the importance of age difference between siblings. To create these variables, age difference was determined from information about sibling birthdates included in a twin-family demographic database maintained at the Queensland Institute of Medical Research (QIMR) Genetic Epidemiology Unit. Sisters were included in analyses to determine whether potential relationships between sibship size (composite) variables and drinking outcomes were driven primarily by the number of males or

females in the sibship. Table 1 displays the sibship composition variables and sample frequencies. Variables included number of:

Full brothers (0, 1, 2, 3 or more)

Full sisters (0, 1, 2, 3 or more)

Full older brothers (0, 1, 2, 3 or more)

Full older sisters (0, 1, 2, 3 or more)

Close in age older (CIAO) brothers (older by less than three years; 0, 1 or 2)

CIAO sisters (older by less than three years; 0, 1 or 2)

CIAO brothers (older by two years or less; 0, 1 or 2)

CIAO sisters (older by two years or less; 0, 1 or 2)

Sibship size. Sibship size was assessed using composite variables. Composite variables were created by summing participant responses for numbers of full brothers and sisters (older and younger), full older brothers and sisters, and CIAO brothers and sisters. Table 1 displays the sibship size variables and sample frequencies. Variables included number of:

Full siblings (0, 1, 2, 3 or more)

Full older siblings (0, 1, 2, 3 or more)

CIAO siblings (older by less than three years; 0, 1 to 3)

CIAO siblings (older by two years or less; 0, 1 or 2)

Both sibship composition and size measures were analyzed as categorical variables. This was done: (1) to account for a positive skew in the data, in which more individuals had a moderate number (i.e., 1 – 3) than a large number (i.e., 5 or more) of

siblings; and (2) to facilitate analyses, such as pair-wise comparisons, that better assessed mean differences in age of onset between individuals with different numbers of siblings.

Figure 3 displays the frequency distribution of full siblings in the sample.

Age of onset of alcohol use. Age of onset was assessed using respondents' reports for age at first drink (AFD) and age at first intoxication (AFI). These measures were included in the SSAGA-OZ (Agrawal et al., 2009), which has demonstrated good reliability and validity across assessments of alcohol abuse and dependence (Bucholz et al., 1994; Hesselbrock, Easton, Bucholz, Schuckit, & Hesselbrock, 1999; Kramer et al., 2009). Lifetime abstainers (1.1% of the sample) were not asked to report on their age at first drink or age at first intoxication. Non-abstainers were asked, "How old were you the first time you had more than just a sip of beer, wine or spirits?" and of the non-abstainers, those who had ever been drunk were asked, "How old were you the first time you got drunk (that is, your speech was slurred or you were unsteady on your feet?"); 5.2% of the sample had never been intoxicated. The measures age at first drink and age at first intoxication have been widely used to assess age of alcohol use onset (DeWit, Adlaf, Offord, & Ogborne, 2000; Donovan, 2007; Hingson, Heeren, Winter, & Wechler, 2003; Hingson, Heeren, Zakocs, Winter, & Wechsler, 2003; McGue, Iacono, Legrand, Malone, & Elkins, 2001; York, Welte, Hirsch, Hoffman, & Barnes, 2006). Responses for AFD ranged from 1 to 47 years and from 2 to 35 years for AFI.

Mean AFD and AFI for the sample were 15.86 years ($SD = 2.67$) and 17.20 years ($SD = 2.69$), respectively. Mean AFD was 16.20 years ($SD = 2.60$ years) and 15.43 years ($SD = 2.68$ years) for women and men, respectively. Mean AFI was 17.61

years ($SD = 2.76$ years) and 16.73 years ($SD = 2.54$ years) for women and men, respectively. Figure 4 displays the cumulative age of onset distributions for AFD and AFI.

The decision was made to perform analyses in two ways: (1) Values above and below three standard deviations from the mean were equated to three standard deviations from the mean. For AFD, values less than or equal to eight years were equated to eight years, and those greater than or equal to 24 years were equated to 24 years. For AFI, values less than or equal to nine years were equated to nine years, and those greater than or equal to 25 years were equated to 25 years. (2) The full range of scores for AFD and AFI were used. Results from analyses using the first method will be discussed; however, results from analyses performed with the full range of drinking and intoxication values are presented in the appendix. Please see the results section for diagnostic analyses and tests of model assumptions that were performed to determine the analytic approach for AFD and AFI.

Retest data on age of drinking and intoxication onset were collected 3.68 years ($SD = 0.39$, range = 1.1 – 4.3) after the first wave of interviews for a small subsample of the twins. These data provide strong evidence for reliability in respondent recall: (1) AFD ($n = 215$): Pearson's $r = .79$, $p < .0001$; (2) AFI ($n = 200$): Pearson's $r = .70$, $p < .0001$. In addition, matched-pairs t-tests show that the mean ages of onset reported at Time 1 and Time 2 for the retest subsample did not significantly differ: (1) AFD: $t(214) = -1.91$, $p = .06$; (2) AFI: $t(199) = -0.33$, $p = .74$. Mean AFD was 15.15 years ($SD = 2.92$) at Time 1 and 15.42 years ($SD = 2.96$) at Time 2. Mean AFI was 16.38 years (SD

= 2.35) at Time 1 and 16.40 years ($SD = 2.97$) at Time 2. Such strong test-retest reliability is particularly compelling considering the “forward telescoping” phenomenon (respondents reporting later ages of substance use onset over time) that has been documented in the literature (Bailey, Flewelling, & Rachal, 1992; Engels, Knibbe, & Drop, 1997; Johnson & Mott, 2001; Labouvie, Bates, & Pandina, 1997; Parra, O’Neill, & Sher, 2003).

Religion. Familial religion has been shown to predict both abstention from and patterns of alcohol use (Degenhardt, Chiu, Sampson, Kessler, & Anthony, 2007; Michalak, Trocki, & Bond, 2007), and Heath & Martin (1988) found that age of onset of alcohol use for individuals from this sample was associated with religious affiliation. In addition, different ideologies regarding contraception and family planning lead certain religions to produce larger families than others (i.e., individuals raised in Catholic families tend to have more siblings; Lehrer, 2009). Religious affiliation of the sample (during their upbringing) was therefore assessed, focusing specifically on individuals whose families identified as: (1) Roman Catholic; (2) Church of England/Anglican; (3) No religion; or (4) Other. These variables were included in analyses to investigate whether potential relationships between sibship size and composition and alcohol were mediated by religion. Religion was coded as both a four-level categorical variable and a set of binary dummy codes. Table 1 depicts the prevalence of the various familial religions in the sample.

Divorce. Research has shown that parental divorce (particularly prior to age 16) influences adolescents’ alcohol use involvement (Bumside, Baer, McLaughlin, &

Pokorny, 1986; Jeynes, 2001; Needle, Su, & Doherty, 1990). Parental divorce was therefore assessed to test whether coming from a family of divorce moderated potential associations between sibship size and composition and alcohol use. The variable was dummy coded to represent presence versus absence of parental divorce. Table 1 displays the prevalence of parental divorce in the sample.

Parental alcohol misuse. Children whose parents abuse alcohol are more likely to start drinking earlier (Hussong, Bauer, & Chassin, 2008; Obot, Wagner, & Anthony, 2001). Excessive paternal and maternal alcohol use was therefore assessed as a potential moderator of associations between sibship size and composition and alcohol use. Participants were asked if their mother and/or father drank any wine, beer, or spirits when they were between six and thirteen years of age. If so, they were asked, "Do you think your mother/father drank too much?" The variables were dummy coded (Yes / No). Table 1 displays the prevalence of excessive paternal and maternal alcohol use in the sample.

Statistical Analyses

All analyses were performed using PROC MIXED procedures in SAS version 9.2. Linear mixed models incorporating a compound symmetry covariance structure were constructed. Data were treated as clustered in all analyses, with the twin pair serving as the cluster. A random intercept term was included to account for the clustered data. Analyses proceeded as follows:

(1) We aimed to replicate findings that having many siblings and many brothers promotes conduct disorder in adolescents (Lauritsen, 1993). Separate models were

constructed for each sibling type, with sibship variables serving as the predictor variables and conduct disorder serving as the criterion.

(2) We aimed to draw on previous findings linking age at first drink, age at first intoxication, and alcohol use problems (Grant & Dawson, 1997; Hingson, Heeren, & Winter, 2006), and to provide evidence for the clinical significance of our findings. To do so, Pearson correlations assessing the relationship between AFD, AFI, and AUD symptoms were calculated.

(3) Building on previous evidence for the comorbidity of delinquency and substance abuse (Hall, 2009; Slutske et al., 1998), we aimed to determine whether AFD, AFI, CD, and AUD symptoms were associated in this sample. To do so, Pearson correlations between all measures were calculated.

(4) We aimed to investigate whether respondents with at least one older brother or sibling started drinking earlier than individuals without any siblings. Sibship variables were therefore dichotomized to represent presence versus absence of a sibling type. A separate model was constructed for each sibling type, with sibship variables serving as the predictor variables and age at first drink serving as the dependent variable. Models were then re-run with age at first intoxication serving as the dependent variable.

(5) We aimed to further investigate whether large sibship size predicts earlier alcohol use onset. A separate model was constructed for each sibship size variable, with age at first drink serving as the dependent variable. Sibship size and sex were included as predictor variables to test for both main effects and interactions. Models were then re-run with age at first intoxication serving as the dependent variable. Least squares means for

each level of the sibling variable were calculated. If significant interactions were found, models were re-run separately by sex.

(6) We aimed to assess whether having many older brothers leads to earlier drinking in males and later drinking in females. A separate model was constructed for each sibship composition variable, with age at first drink serving as the dependent variable. Sibship composition variables and sex were included as predictors to test for both main effects and interactions. Models were then re-run with age at first intoxication serving as the dependent variable. Least squares means for each of level of the sibling variable were calculated. If significant interactions were found, models were re-run separately by sex.

(7) To better assess mean differences in age of onset between individuals with different numbers of siblings or older brothers, Tukey-Kramer-adjusted pair-wise comparisons were performed for models where significant or nearly significant effects of sibship variables were found.

(8) Where significant or nearly significant effects of sibship size and composition variables were detected, models were re-run controlling for familial religion.

(9) Where significant or nearly significant effects of sibship size and composition variables were detected, models were re-run testing for moderation by parental divorce.

(10) Where significant effects of sibship size and composition variables were detected, models were re-run testing for moderation by parental alcohol use problems.

As mentioned above, analyses were performed in two ways: (1) Values of AFD and AFI greater and less than three standard deviations from the mean were equated to

three standard deviations from the mean; and (2) the full range of values were used. Results from analyses using the first method were assessed, however, results from analyses performed using the second method are presented in the appendix.

Ethical Considerations

All possible steps were taken to maintain the confidentiality of respondent data. All participants reside in Australia, and as the present study involved secondary data analyses, no direct contact with respondents took place. This was ensured through de-identification of respondent information. Their identities and responses were distinguished solely by numerical codes pertaining to family membership, twin pair type (MZ vs. DZ), and gender. Only authorized users had access to the dataset, statistical analyses, and results, and no information was copied or sent to a location in which unauthorized users could view or use it.

Chapter 3: Results

Diagnostics and tests of model assumptions. The distributions of age at first drink and age at first intoxication were first examined to assess for normality. The distributions approximated normality somewhat well, but they were relatively leptokurtic. The skew and kurtosis of the distributions were -0.33 and 9.08 for AFD and 0.81 and 5.37 for AFI. Figure 5 displays the frequency distributions for AFD and AFI. While the distributions were not significantly skewed, the kurtosis of the variables was some cause

for concern. To determine whether outlying observations might be contributing to kurtosis (and might affect model results), the distributions of the variables were examined for outliers. Measures of influence (leverage values) were also assessed. Examination of influence plots and leverage values revealed one observation (reported AFD: 47 years) to be significantly influential. This observation was therefore removed. It was also identified as an outlier given that the majority of individuals were between 24 and 36 years of age at the time of their report. One individual was interviewed at age 23, and two at ages 37 and 39, respectively. Removing this observation significantly reduced the kurtosis of the distribution of AFD (kurtosis = 5.76).

The tails of the distributions of AFD and AFI were found to be moderately influential. In addition, examination of normal probability plots revealed that the distributions had slightly heavier tails than would be expected under normality. To reduce the influence of extreme observations, values greater than or equal to three standard deviations above and below the mean were equated to three standard deviations above and below the mean (see methods section). This transformation was successful in significantly reducing the remaining kurtosis of the variables (AFD: kurtosis = 1.51; AFI: kurtosis = 0.43). The skew of AFI was also decreased (skew = -0.45). While the skew of AFD was somewhat increased (skew = -0.84), it was not large enough to be a cause for concern. As mentioned above, analyses using the transformed variables are assessed in this paper. However, results from analyses performed using untransformed variables are presented in the appendix.

Relationship between conduct disorder symptoms, alcohol use onset, and alcohol use disorder. Number of conduct disorder symptoms was correlated with age of drinking and intoxication onset, and consistent with previous findings (Barnes, Welte, & Hoffman, 2002; Zhang, Wieczorek, & Welte, 1997), greater numbers of CD symptoms were associated with earlier ages of alcohol use initiation. These associations were stronger, however, for AFD than for AFI: (1) AFD ($n = 4,683$): Pearson's $r = -0.25, p < .0001$; (2) AFI ($n = 4,683$): Pearson's $r = -0.09, p < .0001$. In addition, consistent with previous research (Merikangas et al., 1998; True et al., 1999), individuals with greater levels of conduct disorder were found to exhibit more AUD symptoms (Pearson's $r(n = 3,444) = 0.27, p < .0001$). A summary of these findings, as well as the rest of the results, is presented in Table 2.

Effects of sibship size on conduct disorder symptoms. There was a significant main effect of sex in predicting conduct disorder symptoms in the sample ($F(1, 2528) = 426.71, p < .0001$) and across all models at the $p < .0001$ level. As expected, men exhibited greater mean number of symptoms ($M = 1.20, SD = 1.64$) than women ($M = 0.39, SD = 0.89$). Consistent with previous research demonstrating a facilitative effect of large families (Brownfield & Sorenson, 1994; Farrington, 2005; Lauritsen, 1993), there was a significant main effect of number of full siblings ($F(3, 2733) = 3.50, p = .02$) and number of full older siblings ($F(3, 2710) = 3.27, p = .02$) in predicting number of conduct disorder symptoms. Analyses revealed that individuals whose siblings were close in age were not at increased risk for CD symptomatology. There was no main effect of number of CIAO siblings (< 3 years older) or number of CIAO siblings (≤ 2

years older). There were no significant interactions with sex for any of the models.

Table 2 in the appendix displays the F -statistics and p -values for all models run with both raw and rank-transformed data.

As noted above, having a large family was a risk factor for delinquency.

Individuals without any full siblings had an average of 0.67 CD symptoms ($SD = 1.33$), while those with one, two, or three or more full siblings had an average of 0.69 ($SD = 1.26$), 0.75 ($SD = 1.32$), and 0.81 ($SD = 1.42$) symptoms, respectively. People without any full older siblings had an average of 0.65 CD symptoms ($SD = 1.20$), while those with one, two, or three or more full older siblings had an average of 0.73 ($SD = 1.33$), 0.80 ($SD = 1.40$), and 0.84 ($SD = 1.46$) symptoms, respectively.

The mean differences in number of conduct disorder symptoms for individuals with different numbers of full siblings and full older siblings were small. However, greater differences were observed when we examined whether the number of individuals meeting criteria for CD (endorsed three or more CD symptoms) differed depending on their number of siblings. Only 41 (8.3%) people without any full siblings met criteria for CD. However, 118 (8.2%), 122 (9.6%), and 159 (10.9%) people with one, two, or three or more full siblings, respectively, met criteria for CD. A similar increase in the prevalence of conduct disorder was observed in relation to number of full older siblings. 104 (7.1%) respondents without any full older siblings met criteria for conduct disorder. 128 (9.7%), 95 (10.7%), and 110 (11.3%) individuals with one, two, or three or more full siblings, respectively, met criteria for CD.

We also investigated the prevalence of male and female conduct disorder among individuals with varying numbers of full siblings and full older siblings. 34 (16.3%) men without any full siblings met criteria for CD. 86 (13.8%), 91 (15.0%), and 135 (21.1%) men with one, two, or three or more full siblings, respectively, met criteria for CD. 7 (2.5%) women without any full siblings met criteria for CD. 32 (3.9%), 31 (4.6%), and 29 (3.4%) women with one, two, or three or more full siblings, respectively, met criteria for CD. Similar to number of full siblings, the prevalence of conduct disorder was greater for males than females across all numbers of full older siblings. 82 (13.4%) men without any full older siblings met criteria for CD. 95 (16.5%), 66 (15.2%), and 95 (22.4%) men with one, two, or three or more full older siblings, respectively, met criteria for CD. By contrast, only 22 (2.6%) women without any full older brothers endorsed three or more conduct disorder symptoms. 33 (4.4%), 29 (6.3%), and 15 (2.7%) women with one, two, or three or more full older siblings, respectively, endorsed three or more CD symptoms.

Effects of sibship composition on conduct disorder symptoms. Similar to sibship size analyses, there was a significant main effect of sex for all sibship composition models at the $p < .0001$ level. Contrary to previous research (Lauritsen, 1993; Reiss & Farrington, 1991), number of full brothers and number of full older brothers did not have a significant effect on levels of conduct problems. Number of full sisters was, unexpectedly, related to CD symptomatology ($F(3, 2714) = 4.62, p < .01$), as was number of full older sisters ($F(3, 2709) = 6.76, p < .001$). Number of CIAO brothers and sisters (< 3 years older) did not influence CD symptomatology, nor did

number of CIAO brothers and sisters (≤ 2 years older). There were no significant interactions with sex for any of the models. Table 2 in the appendix displays the F -statistics and p -values for all models run with both raw and rank-transformed data.

It appears that sisters contributed to the facilitative effects found for total number of full siblings and total number of full older siblings. Respondents without any full sisters had an average of 0.67 CD symptoms ($SD = 1.27$), versus 0.76 ($SD = 1.37$), 0.85 ($SD = 1.43$), and 0.80 ($SD = 1.30$) symptoms for respondents with one, two, or three or more full sisters, respectively. In addition, the mean number of CD symptoms for individuals without any full older sisters was 0.67 ($SD = 1.25$). For individuals with one, two, or three or more full older sisters, the mean number of CD symptoms was 0.79 ($SD = 1.44$), 0.88 ($SD = 1.43$), and 0.89 ($SD = 1.40$), respectively.

In accord with these results were findings that the prevalence of conduct disorder increased with increasing numbers of full sisters and full older sisters. 141 (8.0%) people without any full sisters met criteria for CD. However, 159 (10.7%), 89 (11.8%), and 53 (12.7%) respondents with one, two, or three or more full sisters, respectively, met criteria for CD. A similar increase in the prevalence of conduct disorder was observed in relation to number of full older sisters. 203 (8.0%) people without any full older sisters endorsed three or more CD symptoms. 135 (9.5%), 60 (12.1%), and 42 (10.9%) people with one, two, and three or more full older sisters, respectively, met criteria for CD.

We investigated the prevalence of both male and female conduct disorder in relation to number of full sisters and full older sisters. As expected, males exhibited much higher rates of CD than females across all numbers of full sisters and full older

sisters. 111 (15.0%) men without any full sisters endorsed at least three CD symptoms. 119 (15.0%), 72 (22.6%), and 39 (19.8%) men with one, two, or three or more full sisters endorsed at least three CD symptoms. 30 (3.0%) women without any full sisters met criteria for CD. 40 (4.5%), 15 (3.6%), and 14 (4.8%) women with one, two, or three or more full sisters met criteria for CD. The prevalence of conduct disorder for both sexes was similar in relation to number of full older sisters. 160 (14.4%) men without any full older sisters met criteria for CD. 99 (17.1%), 48 (22.3%), and 34 (23.3%) men with one, two, or three or more full older sisters, respectively, met criteria for CD. 43 (3.0%) women without any full older sisters endorsed three or more conduct disorder symptoms. 36 (5.2%), 12 (4.1%), and 8 (4.3%) women with one, two, or three or more full older sisters, respectively, endorsed three or more CD symptoms.

Relationship between age at first drink, age at first intoxication, and alcohol use disorders. As predicted, AFD was negatively correlated with number of AUD symptoms ($n = 3,602$; Pearson's $r = -0.16$, $p < .0001$). AFI, however, was uncorrelated with number of AUD symptoms ($n = 3,602$; Pearson's $r = 0.03$, $p = .12$).

Sibship size: Effect of having at least one sibling. Models were run looking at the effect of having at least one full sibling, full older sibling, CIAO sibling (< 3 years older), and CIAO sibling (≤ 2 years older) on drinking and intoxication initiation. In regards to AFD, there were no main effects of any of the sibling variables. In regards to AFI, there was a significant main effect of having at least one CIAO sibling (< 3 years older; $F(1, 2305) = 5.37$, $p = .02$).

Having at least one CIAO sibling (< 3 years older) appeared to be a risk factor for early intoxication initiation. Individuals without any CIAO siblings first got drunk at age 16.33 ($SE = 0.08^1$), as opposed to age 16.01 ($SE = 0.12$) for those with at least one CIAO sibling.

Sibship composition: Effect of having at least one brother/sister. Models were run testing for the effect of having at least one full brother, full sister, full older brother, full older sister, CIAO brother (< 3 years older), CIAO sister (< 3 years older), CIAO brother (≤ 2 years older), & CIAO sister (≤ 2 years older) on drinking and intoxication initiation. Having at least one full brother had a nearly significant effect on AFD ($F(1, 2607) = 3.59, p = .06$), as did having at least one CIAO brother (< 3 years older; $F(1, 2191) = 3.75, p = .05$). There were no significant effects of sibship composition variables on AFI.

Having at least one full brother appeared to be somewhat protective: Individuals without any full brothers had their first drink at age 15.66 ($SE = 0.08$), versus age 15.83 ($SE = 0.06$) for those with at least one full brother. Having at least one CIAO brother (< 3 years older) led to earlier drinking initiation: Respondents without any CIAO brothers had their first drink at age 15.60 ($SE = 0.06$), as opposed to age 15.33 ($SE = 0.13$) for people with at least one CIAO brother.

Sibship size: Effects of different numbers of siblings. Models were first run testing for main effects of sibship size variables and sex and interactions with sex. In

¹ Given the unbalanced design, least squares means and associated standard errors (SEs) are reported for all drinking and intoxication means from sibship size and composition analyses. Raw means and associated standard deviations (SDs) are reported in the appendix.

regards to AFD, there was a nearly significant main effect of number of CIAO siblings (< 3 years older; $F(1, 2379) = 3.78, p = .05$). There were no significant main effects of any other sibship size variables and no significant interactions with sex. There was a significant main effect of sex for all models at the $p < .0001$ level.

As indicated by previous analyses, having more CIAO siblings (< 3 years older) appeared to be a risk factor for early drinking. People without any CIAO siblings (< 3 years older) had their first drink at age 15.56 ($SE = 0.06$), as opposed to age 15.34 ($SE = 0.10$) for respondents with between one and three CIAO siblings.

When models were re-run testing for effects on AFI, there was a significant main effect of number of CIAO siblings (< 3 years older; $F(1, 2348) = 5.56, p = .02$). There were no significant main effects of any other sibship variables and no significant interactions with sex. There was a significant main effect of sex for all models at the $p < .0001$ level. Individuals without any CIAO siblings first got drunk at age 16.32 ($SE = 0.08$), while individuals with between one and three CIAO siblings first got drunk at age 16.00 ($SE = 0.12$). Figure 6 displays the mean ages of first drink and first intoxication for individuals without any CIAO siblings compared to those with between one and three CIAO siblings.

Sibship composition: Effects of different numbers of brothers/sisters. Models were first run testing for main effects of sibship composition variables and sex and interactions with sex, with AFD included as the dependent variable. A significant main effect of number of CIAO brothers (< 3 years older) was found ($F(1, 2231) = 4.79, p = .03$). There were no other main effects of sibship composition variables and no

significant interactions with sex. There was a significant main effect of sex for all models at the $p < .0001$ level.

Individuals with CIAO brothers (< 3 years older) were at increased risk for early drinking initiation: People without any CIAO brothers had their first drink at age 15.55 ($SE = 0.06$), while people with either one or two CIAO brothers had their first drink at age 15.24 ($SD = 1.3$). Figure 7 displays the mean age of first drink for individuals without any CIAO brothers compared to those with one or two CIAO brothers.

In regards to AFI, no significant main effects of sibship variables or interactions with sex were found. Interestingly, and in contrast to previous models, there was no significant main effect of sex for any of the models.

Pair-wise comparisons. Sibship variables that were found to have a significant or nearly significant effect on AFD and AFI were number of CIAO siblings (< 3 years older) and number of CIAO brothers (< 3 years older). Given that both of these factors consisted of two levels, it was not necessary to assess pair-wise comparisons, as these analyses yield the same results in regards to mean differences and statistical significance.

Influence of parental divorce. Main effects of divorce and sex and interactions with the two factors in predicting AFD and AFI were assessed. Coming from a family of divorce predicted early drinking onset ($F(1, 3135) = 7.92, p = .005$) and early intoxication onset ($F(1, 3096) = 4.57, p = .03$). There was a main effect of sex for the model predicting AFD ($F(1, 2855) = 36.98, p < .0001$), however, there was no main effect of sex for the model predicting AFI. There were no significant interactions with sex. Individuals from homes of divorce had their first drink at age 15.47 ($SE = 0.12$) and

first got intoxicated at age 16.05 ($SE = 0.15$), compared to ages 15.82 ($SE = 0.05$) and 16.42 ($SE = 0.06$) for individuals from intact families.

We tested for main effects of and interactions with divorce in models where significant or nearly significant effects of sibship variables and/or interactions with sex were previously detected. For AFD, models in which these factors were found to be significant or nearly significant tested for the influence of: (1) Number of CIAO siblings (< 3 years older); and (2) number of CIAO brothers (< 3 years older). In regards to the first model, main effects of number of CIAO siblings ($F(1, 2500) = 7.22, p = .01$), divorce ($F(1, 2203) = 7.57, p = .01$), and sex ($F(1, 2040) = 26.34, p < .0001$) were detected. In addition, there was a significant interaction between number of CIAO siblings and divorce ($F(1, 2602) = 5.34, p = .02$). Re-running the model separately by divorce revealed that, for individuals from intact families, there was no main effect of number of CIAO siblings and no interaction with sex. There was a significant main effect of sex ($F(1, 1584) = 40.84, p < .0001$). For individuals from homes of divorce, however, there was a significant main effect of number of CIAO siblings ($F(1, 258) = 6.90, p = .01$). There was also a significant main effect of sex ($F(1, 211) = 10.47, p = .001$). Individuals from intact families without any CIAO siblings had their first drink at age 15.75 ($SE = 0.06$), approximately the same age as individuals with between one and three CIAO siblings ($M = 15.67, SE = 0.10$). However, the difference in age of onset for individuals from families of divorce was much more substantial. Individuals without any CIAO siblings had their first drink at age 15.65 ($SE = 0.18$), compared to age 14.80 ($SE = 0.27$) for individuals with between one and three CIAO siblings.

In regards to the second model (which included number of CIAO brothers (< 3 years older) as the predictor variable)), there was a significant main effect of divorce ($F(1, 2238) = 4.79, p = .03$) and a significant main effect of sex ($F(1, 2087) = 20.59, p < .0001$), but no significant interactions.

Number of CIAO siblings (< 3 years older) was the only sibship factor previously found to influence AFI. Re-running this model testing for main effects of and interactions with divorce revealed significant main effects of number of CIAO siblings ($F(1, 2382) = 8.04, p < .01$) and divorce ($F(1, 2133) = 7.62, p = .01$), and a significant interaction between the two factors ($F(1, 2472) = 4.71, p = .03$). Testing the model separately by familial divorce status showed that there were no main effects of number of CIAO siblings or sex or interactions with sex for individuals from intact families. For respondents from families of divorce, however, there was a significant main effect of number of CIAO siblings ($F(1, 226) = 9.47, p = .002$). There was no main effect of sex and no interaction. Having CIAO siblings was a significant risk factor for early intoxication: In intact families, individuals with between one and three CIAO siblings started drinking at nearly the same time as people without any CIAO siblings (16.34 years ($SE = 0.12$) vs. 16.49 years ($SE = 0.08$), respectively). In families of divorce, however, individuals without any CIAO siblings first got drunk at age 16.37 ($SE = 0.19$), while people with between one and three CIAO siblings first got drunk nearly a full year earlier (15.31 years ($SE = 0.29$)). Figures 8.a and 8.b display the mean ages of drinking and intoxication onset for individuals with different numbers of CIAO siblings from both intact families and homes of divorce.

Influence of parental alcohol problems. First, the effect of believing one's parent drank excessively on age at first drink and age at first intoxication was tested. In regards to AFD, there was a significant main effect of excessive paternal alcohol use ($F(1, 3646) = 6.60, p = .01$) and a significant main effect of sex ($F(1, 2634) = 42.45, p < .0001$). There was no significant interaction with sex. There was also a significant main effect of excessive maternal alcohol use ($F(1, 2904) = 4.10, p = .04$). While there was no main effect of sex, there was a significant interaction with sex ($F(1, 2904) = 5.74, p = .02$). Running the model separately by sex revealed that believing one's mother drank too much had an effect on AFD for women ($F(1, 1589) = 11.79, p < .001$), but not for men. Individuals who believed their father drank too much had their first drink at age 15.38 ($SE = 0.12$), versus age 15.63 ($SE = 0.06$) for individuals who did not believe their father had a problem. Women who thought their mother drank too much had their first drink a full year earlier than those who did not think their mother had a drinking problem (age 14.95 ($SE = 0.30$) versus age 15.93 ($SE = 0.07$), respectively).

Excessive paternal alcohol use and excessive maternal alcohol use did not have an effect on AFI. There were no significant main effects of or interactions with sex for either of the models. It appears, therefore, that believing one's parent drank too much has a greater impact on the initial drinking experience than on first intoxication.

Main effects of and interactions with parental alcohol use were tested in models where significant or nearly significant effects of sibship variables and/or interactions with sex were previously detected. As described above, for AFD, models in which these factors were found to be significant or nearly significant tested for the influence of: (1)

Number of CIAO siblings (< 3 years older); and (2) number of CIAO brothers (< 3 years older). For the first model, there was a nearly significant main effect of excessive paternal alcohol use ($F(1, 2492) = 3.56, p = .06$) and a significant main effect of sex ($F(1, 1793) = 26.94, p < .0001$). There were no interactions. There was a significant main effect of excessive maternal alcohol use ($F(1, 1981) = 4.55, p = .03$), but no effect of sex and no interactions.

For the second model, there was a significant interaction between number of CIAO brothers and excessive paternal alcohol use ($F(1, 2583) = 5.17, p = .02$). There was also a main effect of sex ($F(1, 1914) = 17.36, p < .0001$). Testing the model separately by excessive paternal use revealed that there was a nearly significant main effect of number of CIAO brothers for individuals who did not believe their father had a drinking problem ($F(1, 1559) = 3.80, p = .05$). However, there was no effect of number of CIAO brothers for people who believed their father had a drinking problem ($F(1, 467) = 1.81, p = .12$). There was no effect of excessive maternal alcohol use and no interactions between any of factors.

Individuals without any CIAO brothers (< 3 years older) who did not believe their father had a drinking problem had their first drink at age 15.75 ($SE = 0.07$). However, in comparison, individuals who did not think their father had a drinking problem with one or two CIAO brothers had their first drink at age 15.43 ($SE = 0.15$). Having many CIAO brothers, therefore, appeared to be a risk factor for early drinking (as was found for the overall sample). However, this was only the case for respondents who did not believe their father drank excessively.

Influence of familial religion. Before testing for mediation by familial religion, relationships between religion and: (1) age at first drink and age at first intoxication; and (2) sibship variables that were previously found to significantly predict AFD and/or AFI were tested. Religion was significantly associated with AFD ($F(3, 2772) = 4.84, p = .002$), but not AFI ($F(3, 2779) = 1.36, p = .25$). Individuals who endorsed having no familial religion started drinking earliest ($M = 15.08, SE = 0.14$), while respondents who endorsed an "other" religion started drinking latest ($M = 15.74, SE = 0.12$). Mean ages for individuals from Anglican and Catholic families were comparable ($M = 15.46, SE = 0.10$ and $M = 15.47, SE = 0.10$, respectively). While religion was associated with AFD, it was not related to number of CIAO siblings (< 3 years older) or number of CIAO brothers (< 3 years older). It was therefore not appropriate to test for mediation. It should be noted, however, that consistent with previous research (Lehrer, 2009), religion was related to overall family size (number of full siblings; $F(3, 1777) = 16.70, p < .0001$). While this difference was somewhat minimal, individuals from Catholic families had the most full siblings ($M = 2.38, SE = .05$). Individuals from Anglican and non-religious families and respondents from families who endorsed other religions did not significantly differ in family size (2.03 full siblings ($SE = 0.04$), 2.05 full siblings ($SE = 0.04$), and 2.04 full siblings ($SE = 0.04$)), respectively.

Chapter 4: Discussion

This study aimed to investigate the impact of sibship size and structure on alcohol use initiation. Drawing on findings from the delinquency literature, we used data from a large sample of Australian twins to test whether family size and number of brothers differentially affected alcohol use in males and females. Findings indicate that sibship size and composition effects are strongest when older siblings are close in age. In addition, CIAO sibling exerted the strongest influence on drinking when: (1) respondents were from homes of divorce; and (2) they did not believe their father had a drinking problem during their youth.

Our results align with findings from the delinquency literature regarding effects of sibship size. Similar to previous studies (Farrington, 1996; Lauritsen, 1993), a facilitative effect of family size was noted, with individuals with more full siblings exhibiting progressively more conduct disorder symptoms. In addition, the number of individuals meeting criteria for CD increased with increasing numbers of full siblings. However, in contrast to previous findings (Lauritsen, 1993; Reiss & Farrington, 1991), the facilitative effect of large families was not due to the number of boys in the sibship. No effect of number of full brothers or full older brothers was found; rather, having many full sisters and many full older sisters was a risk factor for increased conduct disorder symptomatology.

The only types of siblings found to have an effect on respondents' alcohol use initiation were those that were close in age (three years older or less). This is in accord with previous studies noting that sibling similarity in alcohol use decreased with increasing age difference between individuals (Scholte et al., 2007; Trim et al., 2006). The fact that number and type of non-close in age siblings was not found to influence younger sibling drinking indicates that proximal sibling effects likely interact with structural variables to affect drinking behaviors. For instance, adolescents whose siblings are close in age have been found to engage in more shared activities and interact with mutual peer groups (Rowe, Woulbroun, & Gulley, 1994; Trim et al., 2006). Adolescents whose siblings are close in age, therefore, might engage in mutual drinking activities and even experiment with alcohol together for the first time. Older siblings may encourage their younger siblings to drink, similar to the "recruitment to co-offend" observed in the delinquency literature (Reiss & Farrington, 1991). Other previously identified "top down" sibling effects, such as modeling and mentoring of alcohol use norms (Brody et al., 1998; Needle et al., 1986), are likely to operate in this context. These effects are likely magnified when the number of CIAO brothers or siblings increases (similar to the "contagion hypothesis" proposed in the delinquency literature; Robins et al., 1975).

Some have argued that potential sibling effects on substance use may actually be attributable to the effects of mutual friends (particularly in the case of CIAO siblings). While this study did not investigate the effect of shared peer networks, previous work has demonstrated that sibling effects may interact with or be partially accountable to peer influences, but they also function separately. Rowe (1985) found that co-twins' influence

on each others' delinquency operated independently of peer effects. Trim et al. (2006) noted that close-in-age siblings' alcohol use predicted their younger siblings' alcohol use, even after controlling for membership in a shared peer network. It is likely, therefore, that CIAO siblings' modeling and mentoring of alcohol use norms and attitudes interacts with sibship size and composition variables to influence younger siblings' drinking, independent of the effects of mutual social contacts.

Respondents with CIAO siblings from homes of divorce were at heightened risk for early drinking and intoxication initiation. Individuals from intact families with several CIAO siblings were no more likely to start drinking or get intoxicated earlier than people from intact families without any CIAO siblings. In homes of divorce, however, people with between one and three CIAO siblings had their first drink and first got intoxicated nearly a full year earlier than individuals from homes of divorce without any CIAO siblings. It appears, therefore, that CIAO siblings exert the strongest facilitative effects in family environments that contain other risk factors for substance involvement.

Adolescents from families of divorce may use drugs and alcohol to cope with the lack of positive familial relationships that often precede and follow marital disruption. Divorce is particularly challenging when it accompanies the variety of other stressors that manifest in early development (Needle, Su, & Doherty, 1990). How is it that older siblings might facilitate such maladaptive coping behaviors? Abbey and Dallow (2004) found that siblings became closer and relied more on each other for emotional support both during and after parental divorce. Others have shown that sibling relationships may become more "affect intense" following divorce, resulting in high levels of both hostility

and warmth (Sheehan, Darlington, Noller, & Feeney, 2004). It could be, therefore, that increased sibling interaction and support following divorce facilitates engagement in more shared activities, such as drinking. Indeed, greater perceived approval of drug use among siblings, particularly in families of divorce, increases the likelihood that youth will endorse more symptoms of substance use problems (Barrett & Turner, 2005). It could also be that the affect intensity that characterizes sibling relationships in families of divorce induces more stress in adolescents. Increased sibling conflict, as well as the stresses of divorce, may motivate individuals to engage in more maladaptive coping behaviors, which include increased drinking. Changes in family structure dramatically impact sibling dynamics (Barrett & Turner, 2005), and further research is needed to understand how older siblings in families of divorce may confer risk for early alcohol involvement.

Results indicated that parental alcohol problems differentially impacted respondent drinking depending on the gender of the respondent and the parent. For instance, believing one's father drank excessively predicted early alcohol use initiation for the entire sample. However, believing one's mother had a drinking problem was a risk factor for early AFD only among women. Believing one's mother drank excessively was highly facilitative for women, as female respondents who thought their mother had a drinking problem started drinking a full year earlier than those who did not think their mother had a problem. In addition, it was found that having many CIAO brothers predicted earlier alcohol use initiation only among individuals who did not think their father had a drinking problem. These findings are particularly interesting in light of

research regarding sex-of-parent and sex-of-offspring effects on alcoholism. There has been disagreement as to whether paternal alcoholism confers greater risk for offspring AUDs than maternal alcoholism. Various studies have demonstrated an increased influence of paternal versus maternal alcoholism (Lieb, Merikangas, Hofler, Pfister, Isensee, & Wittchen, 2002; Pollock, Schneider, Gabrielli, & Goodwin, 1987). Others, however, have shown no difference in risk between children of alcoholic fathers and those of alcoholic mothers (Kendler, Davis, & Kessler, 1997; Kendler, Neale, Heath, Kessler, & Eaves, 1994). In general, more evidence seems to support a lack of sex-of-parent effects. In addition to these findings, studies generally indicate that male and female children of alcoholics are at comparable risk of developing AUDs (Chassin, Pitts, DeLucia, & Todd, 1999; Slutske et al., 2008).

Given that early drinking onset has been shown to be a marker for future alcohol problems, results from the current study may add to the above-described literature regarding sex-of-parent and sex-of-offspring effects. Generally, the literature does not support differences in risk for AUDs by parent and offspring gender. However, the present findings that paternal alcoholism affected both male and female drinking onset, but maternal alcoholism only influenced drinking in females, indicates that gender effects may manifest themselves in regards to alcohol use initiation. This is interesting in light of arguments that an early age at first drink may serve as a marker of genetic liability for future problems (Grant et al., 2006; Prescott & Kendler, 1999) or as a risk factor for later AUDs (DeWit et al., 2000; Hingson et al., 2006). These results could reflect a true sex difference in risk for early initiation, as previous research has demonstrated that liability

for early alcohol use was significantly more heritable in boys than girls. Liability for early use in women was accounted for more by shared family environmental factors (McGue, Iacono, Legrand, & Elkins, 2001). It could also be that parent and offspring sex moderates the strength of the relationship between parental alcoholism and offspring drinking initiation; however, by the time certain individuals develop disorders, these effects are no longer detected. Such a finding indicates - as is well documented - that while early initiation is much more strongly correlated with future problems than late initiation, it does not guarantee that individuals will develop disorders. If, however, early AFD is a predictor of future problems, tailoring early-life interventions to be sensitive to gender effects may help to delay alcohol involvement.

It is notable that having many CIAO brothers was a risk factor for individuals without a paternal history of alcohol problems, but not for individuals with this history. This is somewhat surprising because: (1) excessive paternal drinking was found to predict early AFD for the entire sample; and (2) analyses demonstrated that having many CIAO siblings predicted earlier drinking in individuals from at-risk families (i.e., homes of divorce). Given that CIAO siblings were previously found to exert stronger effects in homes at risk for problem drinking, but CIAO brothers were not found to do so in combination with other risk factors (i.e., paternal alcoholism), it seems that one type of familial factor may induce greater risk than the other. Perhaps, for instance, the genetic liability conferred by familial alcoholism "outweighs" the environmental effect of siblings. Parental divorce may present some environmental liability for early drinking that is facilitated and magnified by sibling effects such as those discussed above.

However, the genetic risk for early drinking transmitted by paternal alcoholism may leave individuals somewhat “immune” to shared environmental effects such as sibling influences. Indeed, while the effects of parental alcoholism on offspring age at first drink are relatively unexplored, recent analyses using the current sample suggest that: (1) genetic factors play a significant role in determining the timing of first alcohol use and liability for alcohol dependence (AD); and (2) there is substantial genetic overlap in the risk for AD and early drinking initiation (Agrawal et al., 2009; Sartor, Lynskey, Bucholz, Madden, Martin, & Heath, 2009). In addition, Sartor et al. (2009) noted that heritable and unique environmental factors accounted for a much greater proportion of variance in AFD (36% and 49%, respectively) than shared family environmental factors (15%). It should be noted that while Agrawal et al. (2009) found a lower contribution of genetic factors and a greater contribution of shared environment to AFD (9 – 14% and 76 – 80%, respectively), this is likely accounted for by different operationalization of alcohol use variables in the two studies. Findings from the current project seem to support overlapping liability for alcohol problems and early drinking initiation, and may indicate that shared environmental influences, such as sibling effects, are less influential when individuals possess this genetic risk.

It should be noted that genetic and environmental factors have been shown to exert differential effects on substance use over the life course. Specifically, environmental factors tend to have a greater impact on early-life drinking behaviors (i.e., alcohol use initiation), while genetic factors more strongly shape later-life alcohol use phenotypes (i.e., the development of problems; Young-Wolff, Enoch, & Prescott, 2011).

Using a genetically informed design to assess children of alcoholics' AUD status, and whether their risk for future problems is associated with number and type of older siblings, would: (1) elucidate how sibship influences differentially affect alcohol use initiation and problems; and (2) clarify how genetic risk both interacts with and operates separately from these sibship variables to shape alcohol use over the life course.

Effects of both sibship variables and other predictors were generally found to be more robust for age at first drink than for age at first intoxication. For instance, AFD was more strongly related than AFI to number of conduct disorder symptoms and number of AUD symptoms. In addition, number of CIAO brothers was found to predict earlier drinking initiation, but not earlier intoxication initiation, and excessive parental alcohol use was a risk factor for early AFD, but not early AFI. While age at first intoxication is much less investigated than age at first drink, the current findings suggest that AFD is both more subject to heritable and environmental influences and more strongly related to future problems than first drunkenness. This finding, combined with other results from the current study, has significant implications for prevention and intervention. Results indicate that CIAO siblings promote increased risk for alcohol use initiation, particularly in high-risk environments, such as homes of divorce. Sibling attitudes and expectancies about alcohol are likely to interact with structural variables (i.e., number of siblings) to confer the greatest risk for early drinking. It appears, therefore, that family-level interventions should focus particularly on sibship composition, as well as relational variables, such as cohesion and shared activities, and older siblings' attitudes and

expectancies about alcohol. In particular, targeting interventions to precede first alcohol use, rather than first intoxication, is likely to better avert future alcohol use disorders.

The present study presents the first empirical investigation of the effects of sibship size and composition on alcohol use initiation. Findings indicated that sibship size and composition effects were strongest when older siblings were close in age. In addition, CIAO siblings were most facilitative when respondents were from homes of divorce and when they did not have a paternal history of alcohol problems. Results highlight that proximal sibling attitudes, behaviors, and motivations about alcohol likely interact with structural factors to influence younger siblings' drinking. In addition, the differential impact of sibship variables in separate high-risk populations (families of divorce and individuals with a paternal history of alcohol problems) indicates that sibling effects may influence individuals differently depending on their level of genetic versus shared family environmental risk for initiation. Future genetically informed studies of sibship influences on children of alcoholics will help clarify how both genetic and sibling environmental effects differentially affect a variety of alcohol use phenotypes. The present study indicates that prevention and intervention efforts performed at the family level, and introduced before first use of alcohol, are likely to both delay drinking initiation and help prevent future alcohol problems.

One limitation of this study is that respondents' reports on age of onset may be subject to recall bias as a function of their age at interview (24 to 36). To check for potential bias due to retrospective reporting, the correlations between individuals' ages at interview and their reported ages of onset were obtained. The correlations were 0.04 and

0.06 for AFD and AFI, respectively. This indicates that individuals who were older reported slightly later ages of onset; however, the difference between their reports and those of younger individuals were minimal. In general, longitudinal studies that begin in early adolescence can minimize the issue of retrospective bias. They also provide valuable information about the progression of alcohol use over the life course (Grant, Stinson, & Harford, 2001), and research into sibling influences that continue and develop throughout adolescence could strengthen our knowledge of how the effects of sibship size and composition function longitudinally. In addition to recall bias, reports on age at onset may be subject to demand characteristics. Another limitation may be generalizability, as the sample consists largely of heavy drinkers and is mostly Caucasian. The modal age at first drink in this sample (16 years) is notably younger than that of a national sample of American adults (18 years and older; Dawson, Goldstein, Chou, Ruan, & Grant, 2008). Future research that focuses on geographically diverse groups would improve external validity and add to our knowledge of how sibship composition and age of onset both interact with and are influenced by cultural factors. Another limitation of the current study is the use of a twin sample, as the high degree of genetic relatedness and shared experiences between twins may minimize the effects of additional siblings. Future explorations of sibship size and composition that utilize non-twin samples may help define whether and why sibship effects are more robust when individuals do not have a co-twin. Finally, this study focused on the effects of sibship size and composition on alcohol use initiation, regardless of older brothers' age of onset and other alcohol use phenotypes. Given that the present findings suggest that close-in-age-older siblings'

attitudes and behaviors interact with structural effects, future research should examine the alcohol-related cognitions and behaviors of the entire sibship. Assessing siblings' (particularly older brothers') attitudes and behaviors about drinking will help clarify the mechanisms behind their influence on younger brothers' and sisters' alcohol use.

References

- Abbey, C., & Dallos, R. (2004). The experience of the impact of divorce on sibling relationships: A qualitative study. *Clinical Child Psychology and Psychiatry, 9*, 241 - 259.
- Agrawal, A., Sartor, C.E., Lynskey, M.T., Grant, J.D., Pergadia, M.L., Gruzza, R., et al. (2009). Evidence for an interaction between age at first drink and genetic influences on DSM-IV alcohol dependence symptoms. *Alcoholism: Clinical and Experimental Research, 35*, 2047 – 2056.
- Ary, D.V., Tildesley, E., Hops, H., & Andrews, J. (1993). The influence of parent, sibling, and peer modeling and attitudes on adolescent use of alcohol. *The International Journal of the Addictions, 28*, 853 – 880.
- Bailey, S.L., Flewelling, R.L., & Rachal, J.V. (1992). The characterization of inconsistencies in self-reports of alcohol and marijuana use in a longitudinal study of adolescents. *Journal of Studies on Alcohol, 53*, 636 – 647.
- Barnes, G.M., Welte, J.W., & Hoffman, J.H. (2002). Relationship of alcohol use to delinquency and illicit drug use in adolescents: Gender, age, and racial/ethnic differences. *Journal of Drug Issues, 32*, 153 – 178.
- Barrett, A.E., & Turner, R.J. (2005). Family structure and substance use problems in adolescence and early adulthood: Examining explanations for the relationship. *Addiction, 101*, 109 - 120.
- Brody, G.H., Flor, D.L. Hollett-Wright, N., & McCoy, J.K. (1998). Children's development of alcohol use norms: Contribution of parent and sibling norms, children's temperaments, and parent-child discussions. *Journal of Family Psychology, 12*, 209 – 219.
- Bucholz, K.K., Cadoret, R., Cloninger, C.R., Dinwiddie, S.H., Hesselbrock, V.M., Nurnberger, J.I., et al. (1994). A new, semi-structured psychiatric interview for use in genetic linkage studies: A report on the reliability of the SSAGA. *Journal of Studies on Alcohol and Drugs, 55*, 149 – 158.
- Bumside, M.A., Baer, P.E., McLaughlin, R.J., & Pokorny, A.D. (1986). Alcohol use by adolescents in disrupted families. *Alcoholism: Clinical and Experimental Research, 10*, 274 – 278.
- Brownfield, D. & Sorenson, A.M. (1994). Sibship size and sibling delinquency. *Deviant Behavior, 15*, 45 – 61.

- Chassin, L., Pitts, S.C., DeLucia, C., & Todd, M. (1999). A longitudinal study of children of alcoholics: Predicting young adult substance use disorders, anxiety, and depression. *Journal of Abnormal Psychology, 108*, 106 - 119.
- Chou, S.P. & Pickering, R.P. (1992). Early onset of drinking as a risk factor for lifetime alcohol-related problems. *British Journal of Addiction, 87*, 1199 – 1204.
- D’Amico, E.J. & Fromme, K. (1997). Health risk behaviors of adolescent and young adult siblings. *Health Psychology, 16*, 426 – 432.
- Dawson, D.A., Goldstein, R.B., Chou, S.P., Ruan, W.J., & Grant, B.F. (2008). Age at first drink and the first incidence of adult-onset DSM-IV alcohol use disorders. *Alcoholism: Clinical and Experimental Research, 32*, 2149 – 2160.
- Degenhardt, L., Chiu, W.T., Sampson, N., Kessler, R.C., & Anthony, J.C. (2007). Epidemiological patterns of extra-medical drug use in the United States: Evidence from the National Comorbidity Survey Replication, 2001 - 2003. *Drug and Alcohol Dependence, 90*, 210 - 223.
- DeWit, D.J., Adlaf, E.M., Offord, D.R., & Ogborne, A.C. (2000). Age at first alcohol use: A risk factor for the development of alcohol disorders. *American Journal of Psychiatry, 157*, 745 – 750.
- Donovan, J.E. (2007). Really underage drinkers: The epidemiology of children’s alcohol use in the United States. *Prevention Science, 8*, 192 – 205.
- Disney E.R., Elkins, I.J., McGue, M., & Iacono, W.G. (1999). Effects of ADHD, conduct disorder, and gender on substance use and abuse in adolescence. *American Journal of Psychiatry, 156*, 1515 – 1521.
- Ellickson, P.L., Tucker, J.S., Klein, D.J., & McGuigan, K.A. (2001). Prospective risk factors for alcohol misuse in late adolescence. *Journal of Studies on Alcohol and Drugs, 62*, 773 – 782.
- Engels, R.C.M.E., Knibbe, R.A., & Drop, M.J. (1997). Inconsistencies in adolescents’ self-reports of initiation of alcohol and tobacco use. *Addictive Behaviors, 22*, 613 – 623.
- Fagan, A.A. & Najman, J.M. (2003). Sibling influences on adolescent delinquent behaviour: An Australian longitudinal study. *Journal of Adolescence, 26*, 546 – 558.
- Farrington, D.P. (1993). Childhood origins of teenage antisocial behavior and adult social dysfunction. *Journal of the Royal Society of Medicine, 86*, 13 – 17.

- Farrington, D.P. (1996). *The Explanation and Prevention of Youthful Offending*. In J.D. Hawkins (Ed.), *Delinquency and crime: Current theories* (pp. 64 – 148). New York: Cambridge University Press.
- Farrington, D.P. (2005). Childhood origins of antisocial behavior. *Clinical Psychology and Psychotherapy*, *12*, 177 – 190.
- Farrington, D.P. & Loeber, R.L. (2000). Epidemiology of juvenile violence. *Juvenile Violence*, *9*, 733 – 748.
- Fergusson, D.M., Lynskey, M.T., & Horwood, L.J. (1994). Childhood exposure to alcohol and adolescent drinking patterns. *Addiction*, *89*, 1007 – 1016.
- French, M.T. & Maclean, J.C. (2006). Underage alcohol use, delinquency, and criminal activity. *Health Economics*, *15*, 1261 – 1281.
- Gfroerer, J. (1987). Correlation between drug use by teenagers and drug use by older family members. *American Journal of Drug and Alcohol Abuse*, *13*, 95 – 108.
- Grant, B.F. & Dawson, D.A. (1997). Age at onset of alcohol use and its association with DSM-IV alcohol abuse and dependence: Results from the National Longitudinal Alcohol Epidemiologic Survey. *Journal of Substance Abuse*, *9*, 103 – 110.
- Grant, B.F., Stinson, F.S., & Harford, T.C. (2001). Age at onset of alcohol use and DSM-IV alcohol abuse and dependence: A 12-year follow-up. *Journal of Substance Abuse*, *13*, 493 – 504.
- Guo, G. & Wang, J. (2002). The mixed or multilevel model for behavior genetic analysis. *Behavior Genetics*, *32*, 37 – 49.
- Hall, W., Degenhardt, L., & Teesson, M. (2009). Understanding comorbidity between substance use, anxiety and affective disorders: Broadening the research base. *Addictive Behaviors*, *34*, 526 – 530.
- Hawkins, J.D., Catalano, R.F., & Miller, J.Y. (1992). Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance abuse prevention. *Psychological Bulletin*, *112*, 64 – 105.
- Heath, A.C., & Martin, N.G. (1988). Teenage alcohol use in the Australian Twin Register: Genetic and social determinants of starting to drink. *Alcoholism: Clinical and Experimental Research*, *12*, 735 - 741.

- Hellandsjøbu, E.T., Watten, R.G., Foxcroft, D.R., Ingebrigtsen, J.E., & Relling, G. (2002). Teenage alcohol and intoxication debut: The impact of family socialization factors, living area and participation in organized sports. *Alcohol and Alcoholism, 37*, 74 – 80.
- Hesselbrock, M., Easton, C., Bucholz, K.K., Schuckit, M., & Hesselbrock, V. (1999). A validity study of the SSAGA – a comparison with the SCAN. *Addiction, 94*, 1361 – 1370.
- Hingson, R.W., Heeren, T., & Winter, M.R. (2006). Age at drinking onset and alcohol dependence. *Archives of Pediatric and Adolescent Medicine, 160*, 739 – 746.
- Hingson, R.W., Heeren, T., Winter, M.R., & Wechsler, H. (2003). Early age of first drunkenness as a factor in college students' unplanned and unprotected sex attributable to drinking. *Pediatrics, 111*, 34 – 41.
- Hussong, A., Bauer, D., & Chassin, L. (2008). Telescoped trajectories from alcohol initiation to disorder in children of alcoholic parents. *Journal of Abnormal Psychology, 117*, 63 - 78.
- Iacono, W.G., Carlson, S.R., Taylor, J., Elkins, I.J., & McGue, M. (1999). Behavioral disinhibition and the development of substance-use disorders: Findings from the Minnesota Twin Family Study. *Development and Psychopathology, 11*, 869 – 900.
- Jeynes, W.H. (2001). The effects of recent parental divorce on their children's consumption of alcohol. *Journal of Youth and Adolescence, 30*, 305 – 319.
- Johnson, T.P., & Mott, J.A. (2001). The reliability of self-reported age of onset of tobacco, alcohol, and illicit drug use. *Addiction, 96*, 1187 – 1198.
- Jones, M.B., Offord, D.R., & Abrams, N. (1980). Brothers, sisters and antisocial behavior. *British Journal of Psychiatry, 136*, 139 – 145.
- Kendler, K.S., Davis, C.G., & Kessler, R.C. (1997). The familial aggregation of common psychiatric and substance use disorders in the National Comorbidity Survey: A family history study. *British Journal of Psychiatry, 170*, 541 - 548.
- Kendler, K.S., Gardner, C.O., & Prescott, C.A. (1997). Religion, psychopathology, and substance use and abuse: A multimeasure, genetic-epidemiologic study. *American Journal of Psychiatry, 154*, 322 – 239.

- Kendler, K.S., Neale, M.C., Heath, A.C., Kessler, R.C., & Eaves, L.J. (1994). A twin-family study of alcoholism in women. *American Journal of Psychiatry*, *151*, 707 - 715.
- Kessler, R.C., Crum, R.M., Warner, L.A., Nelson, C.B., Schulenberg, J., & Anthony, J.C. (1997). Lifetime co-occurrence of DSM-III-R alcohol abuse and dependence with other psychiatric disorders in the National Comorbidity Survey. *Archives of General Psychiatry*, *54*, 313 – 321.
- King, S.M., Iacono, W.G., & McGue, M. (2004). Childhood externalizing and internalizing psychopathology in the prediction of early substance use. *Addiction*, *99*, 1548 – 1559.
- Knopik, V.S., Heath, A.C., Madden, P.A.F., Bucholz, K.K., Slutske, W.S., Nelson, E.C., et al. (2004). Genetic effects on alcohol dependence risk: Re-evaluating the importance of psychiatric and other heritable risk factors. *Psychological Medicine*, *34*, 1519 – 1530.
- Kramer, J.R., Chan, G., Kuperman, S., Bucholz, K.K., Edenberg, H.J., Schuckit, M.A., et al. (2009). A comparison of diagnoses obtained from in-person and telephone interview, using the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA). *Journal of Studies on Alcohol and Drugs*, *70*, 623 – 627.
- Labouvie, E., Bates, M.E., & Pandina, R.J. (1997). Age of first use: Its reliability and predictive utility. *Journal of Studies on Alcohol*, *58*, 638 – 643.
- Lauritsen, J. (1993). Sibling resemblance in juvenile delinquency: Findings from the National Youth Survey. *Criminology*, *31*, 387 – 409.
- Lehrer, E.L. (2009). *Religion, economics, and demography*. New York, NY: Routledge.
- Lieb, R., Merikangas, K.R., Hofler, M., Pfister, H., Isensee, B., & Wittchen, H.-U. (2002). Parental alcohol use disorders and alcohol use and disorders in offspring: A community study. *Psychological Medicine*, *32*, 63 - 78.
- Little, R.E. (1989). An exploration of the effect of select family socialization variables on teenage alcohol use. *Journal of Alcohol and Drug Education*, *34*, 22 – 28.
- Loeber, R. & Stouthamer-Loeber, M. (1986). Family factors as correlates and predictors of juvenile conduct problems and delinquency. *Crime and Justice*, *7*, 29 – 149.
- Mayer, R.R., Forster, J.L., Murray, D.M., & Wagenaar, A.C. (1998). Social settings and situations of underage drinking. *Journal of Studies on Alcohol*, *59*, 207 – 215.

- McGue, M., Iacono, W.G., Legrand, L.N., & Elkins, I. (2001). Origins and consequences of age at first drink. II. Familial risk and heritability. *Alcoholism: Clinical and Experimental Research*, *25*, 1166 – 1173.
- McGue, M., Iacono, W.G., Legrand, L.N., Malone, S., & Elkins, I. (2001). Origins and consequences of age at first drink. I. Associations with substance-use disorders, disinhibitory behavior and psychopathology, and P3 amplitude. *Alcoholism: Clinical and Experimental Research*, *25*, 1156 – 1165.
- McGue, M., Sharma, A., & Benson, P. (1996). Parent and sibling influences on adolescent alcohol use and misuse: Evidence from a U.S. adoption cohort. *Journal of Studies on Alcohol*, *57*, 8 – 18.
- Mears, D.P., Ploeger, M., & Warr, M. (1998). Explaining the gender gap in delinquency: Peer influence and moral evaluations of behavior. *Journal of Research in Crime and Delinquency*, *35*, 251 – 266.
- Meier, M.H., Slutske, W.S., Heath, A.C., & Martin, N.G. (2009). The role of harsh discipline in explaining sex differences in conduct disorder: A study of opposite-sex twin pairs. *Journal of Abnormal Child Psychology*, *37*, 653 – 664.
- Merikangas, K.R., Mehta, R.L., Molnar, B.E., Walters, E., Swendsen, J.D., Aguilar-Gaziola, S., et al. (1998). Comorbidity of substance use disorders with mood and anxiety disorders: Results of the International Consortium in Psychiatric Epidemiology. *Addictive Behaviors*, *23*, 893 – 907.
- Michalak, L., Trocki, K., & Bond, J. (2007). Religion and alcohol in the U.S. National Survey: How important is religion for abstention and drinking? *Drug and Alcohol Dependence*, *87*, 268 - 280.
- Moffitt, T.E., Caspi, A., Rutter, M., & Silva, P.A. (2001). *Conduct disorder, delinquency, and violence in the Dunedin Longitudinal Study*. Cambridge, UK: Cambridge University Press.
- Monti, P.M., Miranda, Jr., R., Nixon, K., Sher, K.J., Swartzwelder, H.S., Taper, S.F., et al. (2005). Adolescence: Booze, brains, and behavior. *Alcoholism: Clinical and Experimental Research*, *29*, 207 – 220.
- Mukhopadhyay, P.K., An, A.B., Tobias, R.D., & Watts, D.L. (2008). Try, try again: Replication-based variance estimation methods for survey data analysis in SAS 9.2. *SAS Global Forum: Statistics and Data Analysis*.

- Needle, R., McCubbin, H., Wilson, M., Reineck, R., Lazar, A., & Mederer, H. (1986). Interpersonal influences in adolescent drug use – the role of older siblings, parents, and peers. *The International Journal of the Addictions*, 21, 739 – 766.
- Needle, R.H., Su, S.S., & Doherty, W.J. (1990). Divorce, remarriage, and adolescent substance use: A prospective longitudinal study. *Journal of Marriage and the Family*, 52, 157 – 169.
- Newson, J., Newson, E., & Adams, M. (1993). The social origins of delinquency. *Criminal Behavior and Mental Health*, 3, 19 – 29.
- Obot, I.S., Wagner, F.A., & Anthony, J.C. (2001). Early onset and recent drug use among children of parents with alcohol problems: Data from a national epidemiologic survey. *Drug and Alcohol Dependence*, 65, 1 - 8.
- Parra, G.R., O’Neill, S.E., & Sher, K.J. (2003). Reliability of self-reported age of substance involvement onset. *Psychology of Addictive Behaviors*, 17, 211 – 218.
- Pitkänen, T., Lyyra, A., & Pulkkinen, L. (2005). Age of onset of drinking and the use of alcohol in adulthood: A follow-up study from age 8 – 42 for females and males. *Addiction*, 100, 652 – 661.
- Pollock, V.E., Schneider, L.S., Gabrielli, W.F., & Goodwin, D.W. (1987). Sex of parent and offspring in the transmission of alcoholism: a meta-analysis. *The Journal of Nervous and Mental Disease*, 175, 668 - 673.
- Reiss, A.J. & Farrington, D.P. (1991). Advancing knowledge about co-offending: Results from a prospective longitudinal study of London males. *The Journal of Criminal Law & Criminology*, 82, 360 – 395.
- Robins, L.N., West, P.A., & Herjanic, B.L. (1975). Arrests and delinquency in two generations: A study of black urban families and their children. *Journal of Child Psychology and Psychiatry*, 16, 125 – 140.
- Rowe, D.C. (1985). Sibling interaction and self-reported delinquent behavior: A study of 265 twin pairs. *Criminology*, 23, 223 - 240.
- Rowe, D.C. & Farrington, D.P. (1997). The familial transmission of criminal convictions. *Criminology*, 35, 177 – 202.
- Rowe, D.C. & Gulley, B.L. (1992). Sibling effects on substance use and delinquency. *Criminology*, 30, 217 – 234.

- Rowe, D.C., Rodgers, J.L., & Meseck-Bushey, S. (1992). Sibling delinquency and family environment: Shared and unshared influences. *Child Development, 63*, 59 – 67.
- Rowe, D.C., Woulbroun, E.J., & Gulley, B.L. (1994). Peers and friends as nonshared environmental influences. In E.M. Heatherington, D. Reiss, & R. Plomin (Eds.), *Separate social worlds of siblings: The impact of nonshared environment on development* (pp. 159 – 173). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.
- SAS Institute Inc. (2010). *Base SAS 9.2. Procedures Guide: Statistical Procedures, Third Edition*. Cary, NC: SAS Institute Inc.
- Scholte, R.H.J., Poelen, E.A.P., Willemsen, G., Boomsma, D.I., & Engels, R.C.M.E. (2008). Relative risks of adolescent and young adult alcohol use: The role of drinking fathers, mothers, siblings, and friends. *Addictive Behaviors, 33*, 1 – 14.
- Sheehan, G., Darlington, Y., Noller, P., & Feeney, J. (2004). Children's perceptions of their sibling relationships during parental separation and divorce. *Journal of Divorce and Remarriage, 41*, 69 - 94.
- Slomkowski, C., Rende, R., Conger, K.J., Simons, R.L., & Conger, R.D. (2001). Sisters, brothers, and delinquency: Evaluating social influence during early and middle adolescence. *Child Development, 72*, 271 – 283.
- Slutske, W.S., Blaszczyński, A., & Martin, N.G. (2009). Sex differences in the rates of recovery, treatment-seeking, and natural recovery in pathological gambling: Results from an Australian community-based twin survey. *Twin Research and Human Genetics, 12*, 425 – 432.
- Slutske, W.S., D'Onofrio, B.M., Turkheimer, E., Emery, R.E., Harden, K.P., Heath, A.C., et al. (2008). Searching for an environmental effect of parental alcoholism on offspring alcohol use disorder: A genetically informed study of children of alcoholics. *Journal of Abnormal Psychology, 117*, 534 - 551.
- Slutske, W.S., Heath, A.C., Dinwiddie, S.H., Madden, A.F., Bucholz, K.K., Dunne, M.P., et al. (1998). Common genetic risk factors for conduct disorder and alcohol dependence. *Journal of Abnormal Psychology, 107*, 363 – 374.
- Slutske, W.S., Meier, M.H., Zhu, G., Statham, D.J., Blaszczyński, A., & Martin, N.G. (2009). The Australian Twin Study of Gambling (OZ-GAM): Rationale, sample description, predictors of participation, and a first look at sources of individual differences in gambling involvement. *Twin Research and Human Genetics, 12*, 63 – 78.

- Stormshak, E.A., Comeau, C.A., & Shepard, S.A. (2004). The relative contribution of sibling deviance and peer deviance in the prediction of substance use across middle childhood. *Journal of Abnormal Child Psychology*, *32*, 635 – 649.
- Swartzwelder, H.S., Wilson, W.A., & Tayyeb, M.I. (1995). Age-dependent inhibition of long-term potentiation by ethanol in immature versus mature hippocampus. *Alcoholism: Clinical and Experimental Research*, *19*, 1480 - 1485.
- Trim, R.S., Leuthe, E., & Chassin, L. (2006). Sibling influence on alcohol use in a young adult, high-risk sample. *Journal of Studies on Alcohol and Drugs*, *67*, 391 – 398.
- True, W.R., Heath, A.C., Scherrer, J.F., Xian, H., Lin, N., Eisen, S.A., et al. (1999). Interrelationship of genetic and environmental influences on conduct disorder and alcohol and marijuana dependence symptoms. *American Journal of Medical Genetics*, *88*, 391 – 397.
- Van Der Vorst, H., Engels, R.C.M.E., Meeus, W., Dekovic, M., & Van Leeuwe, J. (2007). Similarities and bi-directional influences regarding alcohol consumption in adolescent sibling pairs. *Addictive Behaviors*, *32*, 1814 – 1825.
- Wagenaar, A.C., Finnegan, J.R., Wolfson, M., Anstine, P.S., Williams, C.L., & Perry, C.L. (1993). Where and how adolescents obtain alcohol. *Public Health Reports*, *108*, 459 – 464.
- Wagenaar, A.C., Toomey, T.L., Murray, D.M., Short, B.J., Wolfson, M., & Jones-Webb, R. (1996). Sources of alcohol for underage drinkers. *Journal of Studies on Alcohol*, *57*, 325 – 333.
- Webb, J.A., Bray, J.H., Getz, J.G., & Adams, G. (2002). Gender, perceived parental monitoring, and behavioral adjustment: Influences on adolescent alcohol use. *American Journal of Orthopsychiatry*, *72*, 392 – 400.
- White, A.M., & Swartzwelder, H.S. (2004). Hippocampal functioning during adolescence: A unique target of ethanol effects. *Annals of the New York Academy of Sciences*, *1021*, 206 - 220.
- York, J.L., Welte, J., Hirsch, J., Hoffman, J.H., & Barnes, G. (2006). Association of age at first drink with current alcohol drinking variables in a national general population sample. *Alcoholism: Clinical and Experimental Research*, *28*, 1379 – 1387.
- Young-Wolff, K.C., Enoch, M.A., & Prescott, C.A. (2011). The influence of gene-environment interactions on alcohol consumption and alcohol use disorders: A comprehensive review. *Clinical Psychology Review*, *31*, 800 – 816.

Zeigler, D.W., Wang, C.C., Yoast, R.A., Dickinson, B.D., McCaffree, M.A., Robinowitz, C.B., et al. (2005). The neurocognitive effects of alcohol on adolescents and college students. *Preventive Medicine, 40*, 23 – 32.

Zhang, L., Wieczorek, W.F., & Welte, J.W. (1997). The impact of age of onset of substance use on delinquency. *Journal of Research in Crime and Delinquency, 34*, 253 – 268.

Table 1

Sample Characteristics

	Males		Females		Total	
	n	%	n	%	n	%
<i>Sample Size</i>	2139	44.2	2702	55.8	4841	100
1+ Conduct Disorder Sxs	1096	51.2	631	23.4	1727	35.7
3+ Conduct Disorder Sxs	345	16.1	99	3.7	444	9.2
1+ Alcohol Use Disorder Sxs	977	46.7	944	34.9	1921	39.7
<i>Sibship Composition</i>						
Full Brothers	1356	63.4	1745	64.6	3101	64.0
Full Sisters	1310	61.3	1593	59.0	2903	60.0
Full Older Brothers	1066	49.9	1251	46.3	2317	47.9
Full Older Sisters	941	44.0	1167	43.2	2108	43.6
CIAO Brothers (< 3 yrs.)	229	10.7	326	12.1	555	11.5
CIAO Sisters (< 3 yrs.)	203	9.5	322	11.9	525	10.8
CIAO Brothers (\leq 2 yrs.)	149	7.0	175	6.5	324	6.7
CIAO Sisters (\leq 2 yrs.)	97	4.5	197	7.3	294	6.1
<i>Sibship Size</i>						
Full Siblings	1842	86.1	2325	86.0	4167	86.1
Full Older Siblings	1435	67.1	1755	65.0	3190	65.9
CIAO Siblings (< 3 yrs.)	410	19.2	621	23.0	1031	21.3
CIAO Siblings (\leq 2 yrs.)	246	11.5	372	13.8	618	12.8
<i>Religion</i>						
Roman Catholic	440	20.6	684	25.3	1124	23.2
Church of England/Anglican	448	21.0	577	21.4	1025	21.2
No Religion	211	9.9	270	10.0	481	9.9
Other	391	18.3	557	20.6	948	19.6
Family of Divorce	246	11.5	337	12.5	583	12.0
Paternal Alcohol Problems	306	14.3	455	16.8	761	15.7
Maternal Alcohol Problems	56	2.6	79	2.9	135	2.8

Note: Sample frequencies for sibship size and composition represent the number of individuals with at least one of the specified sibling type. Sxs = symptoms; CIAO = Close in Age Older.

Table 2

Summary of Results

Analysis	Finding(s)
Relationship between CD symptoms and alcohol use onset	More CD symptoms associated with earlier AFD and AFI.
Relationship between CD symptoms and AUD symptoms	More CD symptoms associated with more AUD symptoms.
Effects of sibship size on CD symptoms	<p>Respondents with more full siblings and full older siblings had higher mean levels of CD.</p> <p>Number of individuals who met criteria for CD increased with greater numbers of full siblings and full older siblings.</p> <p>Men exhibited higher rates of conduct disorder across all numbers of full siblings and full older siblings than women.</p> <p>People with more full sisters and full older sisters had higher mean levels of CD.</p>
Effects of sibship composition on CD symptoms	Prevalence of CD increased with increasing numbers of full sisters and full older sisters.
Relationship between AFD, AFI, and AUDs	<p>Men exhibited higher rates of conduct disorder across all numbers of full sisters and full older sisters than women.</p> <p>AFD negatively correlated with number of AUD symptoms.</p>
<i>Sibship size</i> : Effect of having at least one sibling	<p>AFI uncorrelated with number of AUD symptoms</p> <p>No effects of any sibship variables on AFD.</p>
<i>Sibship composition</i> : Effect of having at least one brother/sister	<p>Having at least one CIAO brother (< 3 years older) led to earlier AFI.</p> <p>Having at least one full brother led to later AFD.</p> <p>Having at least one CIAO brother (< 3 years older) predicted earlier AFD.</p> <p>No effect of sibship composition variables on AFI.</p>

Table 2 continued

Analysis	Finding(s)
<i>Sibship size:</i> Effects of different numbers of siblings.	<p>Having many CIAO siblings (< 3 years older) a risk factor for early AFD.</p> <p>Having many CIAO siblings (< 3 years older) a risk factor for early AFI.</p>
<i>Sibship composition:</i> Effects of different numbers of brothers/sisters.	<p>Having many CIAO brothers (< 3 years older) led to earlier AFD.</p> <p>No effect of sibship composition variables on AFI.</p>
Pair-wise comparisons.	Not performed; results would align with previous analyses of two-level CIAO sibling variables.
Influence of parental divorce.	<p>Divorce associated with early AFD and early AFI.</p> <p>Having many CIAO siblings (< 3 years older) a significant risk factor for early AFD and early AFI among respondents from homes of divorce. There was no effect of number of CIAO siblings on AFD or AFI for people from intact homes.</p>
Influence of parental alcohol problems.	<p>Excessive paternal alcohol use a risk factor for early AFD.</p> <p>Excessive maternal alcohol use a risk factor for early drinking initiation among women, but not among men.</p> <p>No effects of excessive paternal or maternal alcohol use on AFI.</p> <p>Having many CIAO brothers (< 3 years older) was a risk factor for early AFD among individuals who did not think their father drank excessively, but not among people who did not hold this belief.</p>

Table 2 continued

Analysis	Finding(s)
Influence of familial religion	<p data-bbox="873 359 1409 388">Religion associated with AFD, but not with AFI.</p> <p data-bbox="873 422 1409 506">People with no familial religion had the earliest AFD, while those raised in an "other" religion had the latest AFD.</p> <p data-bbox="873 539 1409 602">Religion related to family size (number of full siblings).</p> <p data-bbox="873 636 1409 699">Catholic families largest; other familial religions had comparable family sizes.</p> <p data-bbox="873 732 1409 842">Religion not associated with number of CIAO siblings (< 3 years older) or number of CIAO brothers (< 3 years older). Not appropriate to test for mediation.</p>

Note: CIAO = Close in Age Older.

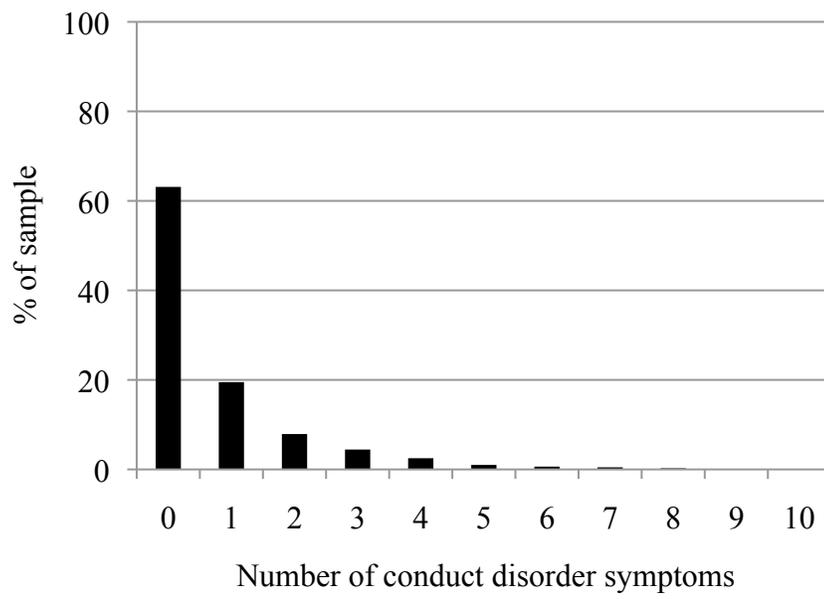


Figure 1. Sample prevalence of conduct disorder symptoms.

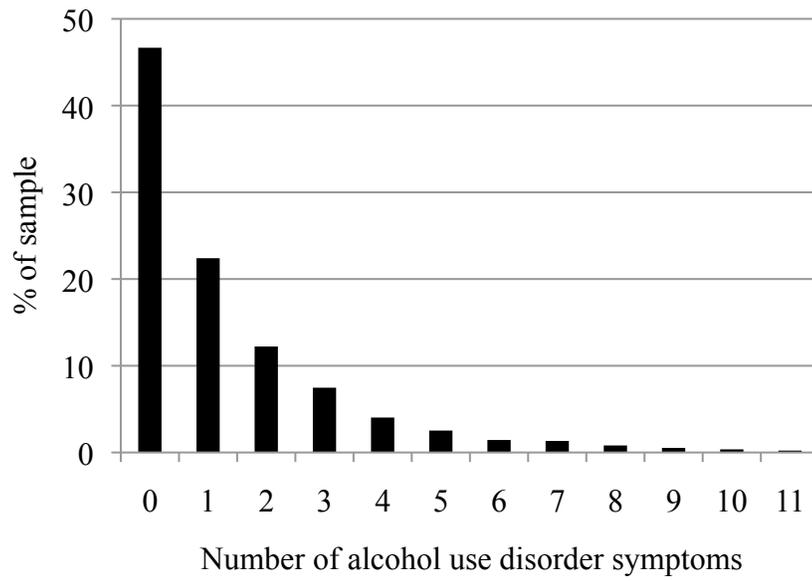


Figure 2. Sample prevalence of alcohol use disorder symptoms.

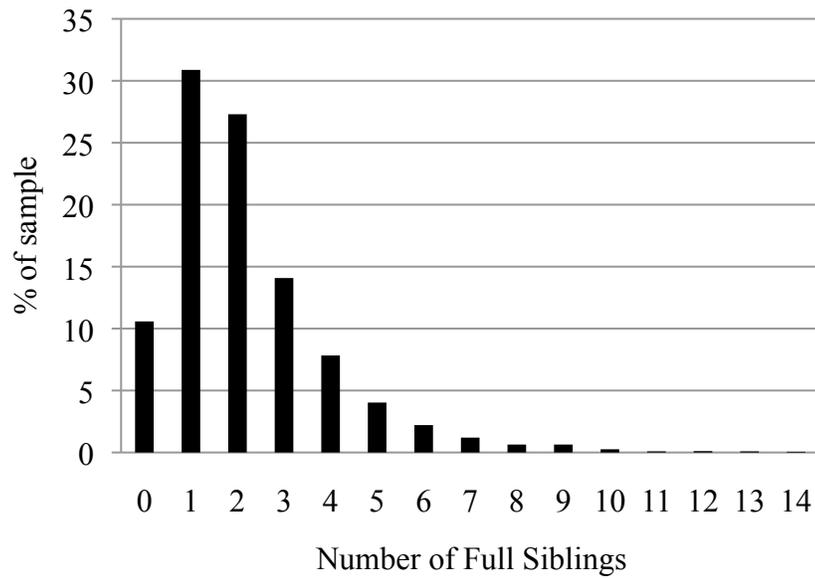


Figure 3. Frequency distribution of number of full siblings in the sample. The frequency represents the number of individuals with the specified number of siblings.

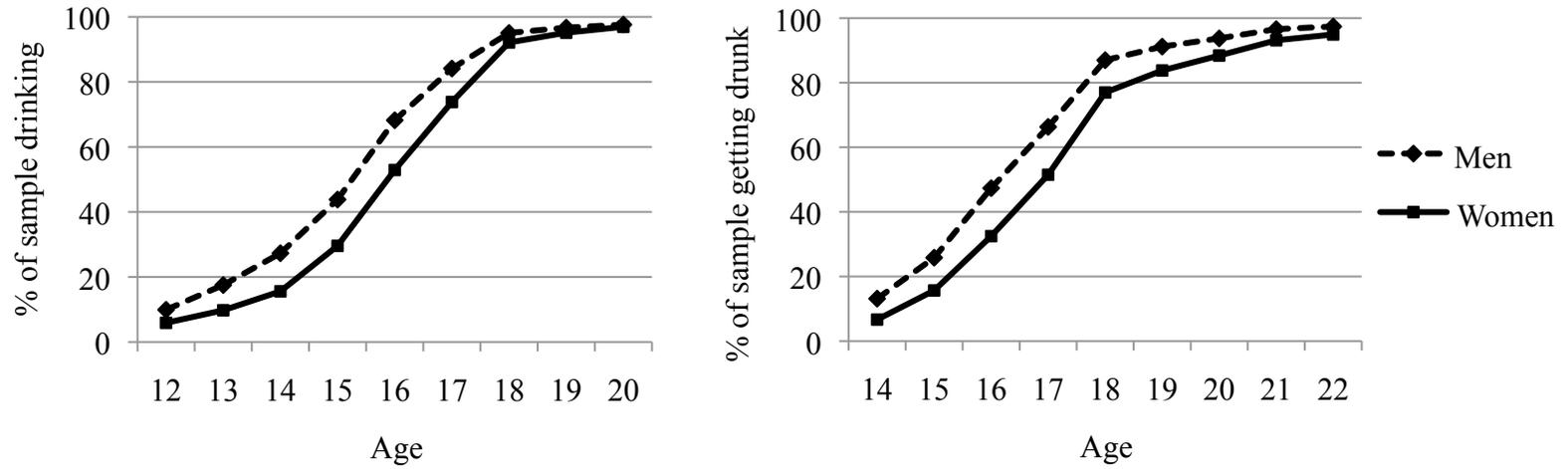


Figure 4. Cumulative age of onset distributions for age at first drink and age at first intoxication. Age at first drink and age at first intoxication are depicted in the left and right panels, respectively.

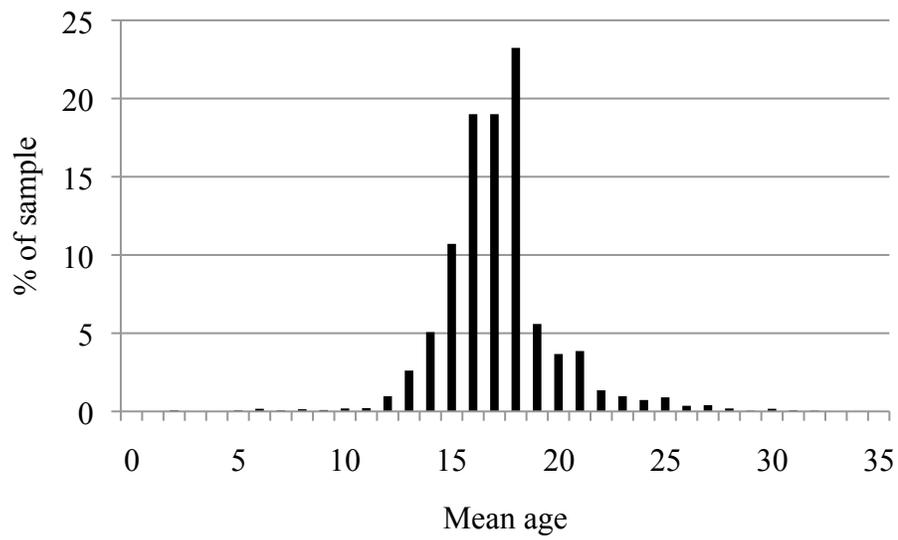
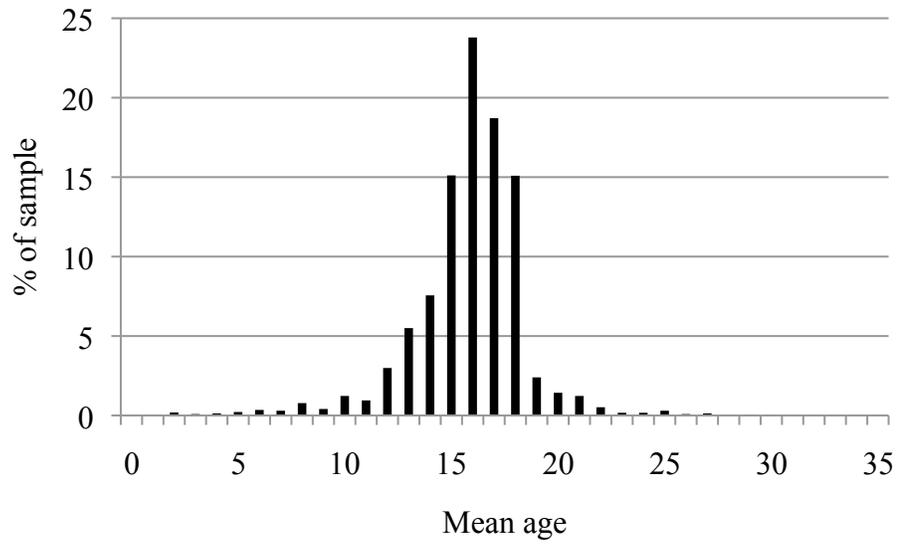


Figure 5. Frequency distributions for age at first drink and age at first intoxication. Age at first drink and age at first intoxication are depicted in the top and bottom panels, respectively.

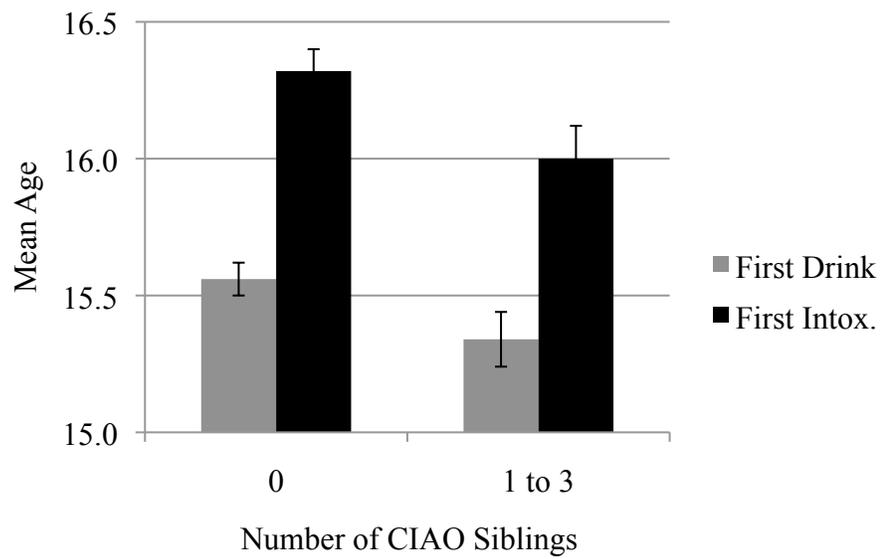


Figure 6. Mean ages at first drink and first intoxication for individuals with 0 and 1 to 3 close in age older siblings (< 3 years older).

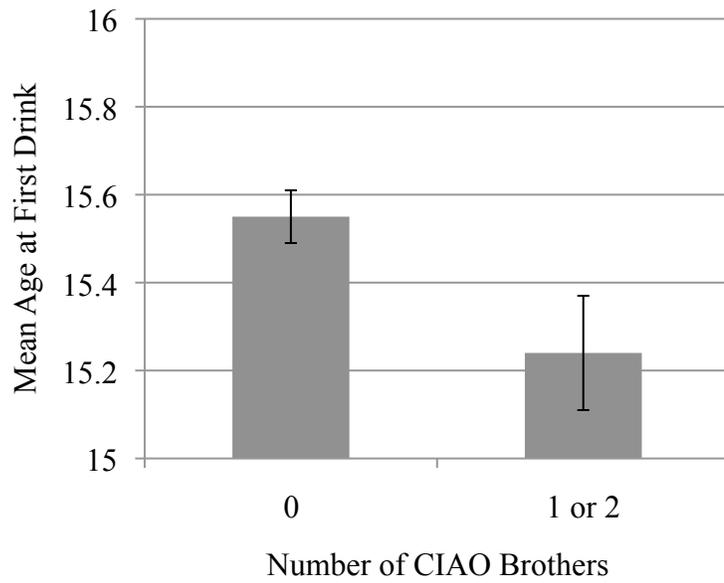


Figure 7. Mean age at first drink for individuals with 0 and 1 or 2 close in age older brothers (< 3 years older).

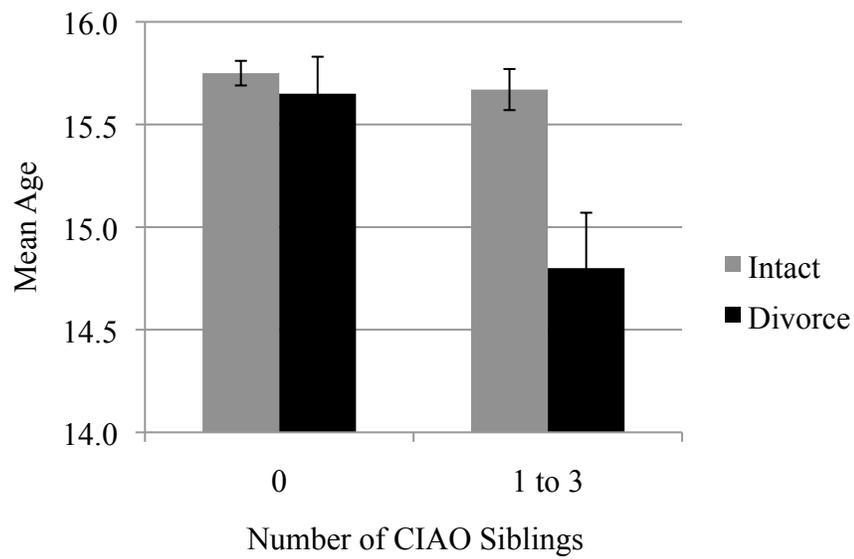


Figure 8.a. Mean age at first drink for individuals with close in age older siblings (< 3 years older) from intact homes and families of divorce.

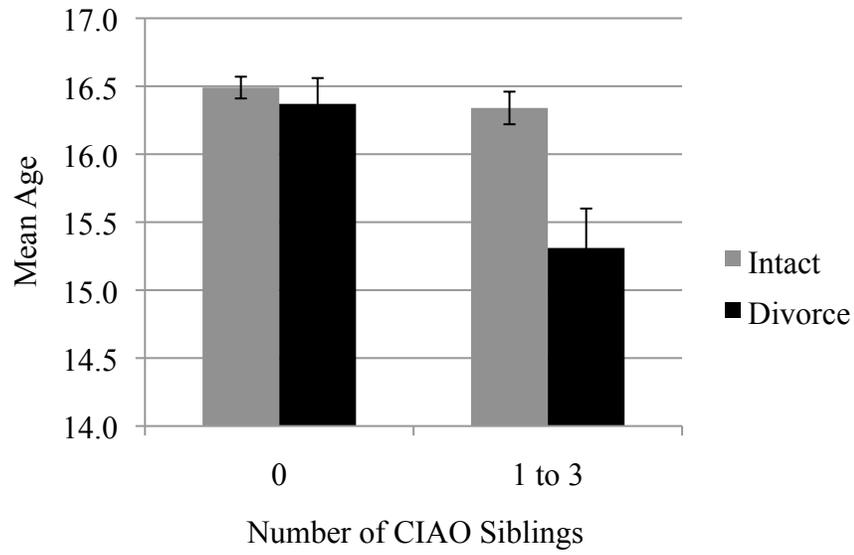


Figure 8.b. Mean age at first intoxication for individuals with close in age older siblings (< 3 years older) from intact homes and families of divorce.

APPENDIX

Table 1.a

Mean Age at First Drink and Age at First Intoxication by Number of Siblings: Sibship Size

Measure	Males			Females			Total		
	N	Mean AFD	Mean AFI	N	Mean AFD	Mean AFI	N	Mean AFD	Mean AFI
<i>Full Siblings</i>									
0	284	15.8 (2.7)	16.3 (3.7)	209	15.3 (3.0)	16.3 (3.2)	439	15.6 (2.8)	16.3 (3.5)
1	817	16.0 (2.4)	16.5 (3.6)	622	15.3 (2.6)	16.1 (3.0)	1439	15.7 (2.5)	16.4 (3.3)
2	667	16.3 (2.4)	16.3 (3.8)	605	15.5 (2.6)	16.5 (2.7)	1272	15.9 (2.5)	16.4 (3.3)
3 or more	841	16.2 (2.6)	16.4 (3.9)	615	15.3 (2.5)	16.4 (2.7)	1456	15.8 (2.6)	16.4 (3.4)
<i>Full Older Siblings</i>									
0	854	16.0 (2.5)	16.5 (3.6)	612	15.4 (2.8)	16.3 (3.1)	1466	15.7 (2.6)	16.4 (3.4)
1	746	16.1 (2.5)	16.3 (3.8)	577	15.2 (2.7)	16.3 (2.9)	1323	15.7 (2.6)	16.3 (3.4)
2	458	16.2 (2.6)	16.2 (3.9)	434	15.5 (2.5)	16.4 (2.6)	892	15.9 (2.6)	16.3 (3.4)
3 or more	551	16.3 (2.4)	16.6 (3.8)	424	15.3 (2.6)	16.4 (2.7)	975	15.9 (2.5)	16.5 (3.4)
<i>CIAO Siblings (< 3 Yrs. Older)</i>									
0	975	15.2 (2.7)	16.2 (2.9)	1275	15.9 (2.7)	16.4 (3.8)	2250	15.6 (2.8)	16.3 (3.4)
1 to 3	410	14.9 (3.0)	15.9 (3.1)	621	15.9 (2.6)	16.1 (3.7)	1031	15.5 (2.8)	16.0 (3.5)
<i>CIAO Siblings (\leq 2 Yrs. Older)</i>									
0	1139	15.2 (2.8)	16.2 (2.9)	1524	15.8 (2.7)	16.3 (3.8)	2663	15.6 (2.8)	16.3 (3.4)
1 or 2	246	14.8 (2.9)	15.9 (3.1)	372	16.0 (2.5)	16.3 (3.5)	618	15.5 (2.7)	16.1 (3.3)

Note: AFD = Age at First Drink; AFI = Age at First Intoxication; CIAO = Close in Age Older. Standard deviations given in parentheses.

Table 1.b

Mean Age at First Drink and Age at First Intoxication by Number of Siblings: Sibship Composition

Measure	Males			Females			Total		
	N	Mean AFD	Mean AFI	N	Mean AFD	Mean AFI	N	Mean AFD	Mean AFI
<i>Full Brothers</i>									
0	695	15.4 (2.7)	16.4 (3.0)	864	15.9 (2.6)	16.3 (3.7)	1559	15.7 (2.7)	16.3 (3.4)
1	762	15.2 (2.7)	16.2 (2.9)	1029	16.2 (2.5)	16.4 (3.6)	1791	15.8 (2.6)	16.3 (3.3)
2	379	15.6 (2.4)	16.7 (2.6)	446	16.3 (2.4)	16.5 (3.8)	825	16.0 (2.4)	16.6 (3.3)
3 or more	215	15.2 (2.5)	16.3 (2.6)	270	16.3 (2.7)	16.6 (4.1)	485	15.8 (2.6)	16.4 (3.5)
<i>Full Sisters</i>									
0	741	15.4 (2.7)	16.3 (3.0)	1016	16.0 (2.5)	16.5 (3.6)	1757	15.7 (2.6)	16.4 (3.4)
1	795	15.3 (2.6)	16.3 (2.8)	883	16.2 (2.5)	16.4 (3.8)	1678	15.8 (2.6)	16.4 (3.4)
2	318	15.5 (2.6)	16.5 (2.7)	419	16.1 (2.5)	16.2 (3.8)	737	15.8 (2.6)	16.3 (3.4)
3 or more	197	15.4 (2.5)	16.4 (2.7)	291	16.2 (2.8)	16.3 (4.0)	488	15.9 (2.7)	16.3 (3.5)
<i>Full Older Brothers</i>									
0	984	15.4 (2.7)	16.3 (3.0)	1358	16.0 (2.5)	16.4 (3.7)	2342	15.8 (2.6)	16.3 (2.4)
1	651	15.3 (2.6)	16.3 (2.8)	776	16.2 (2.6)	16.3 (3.7)	1427	15.7 (2.6)	16.3 (3.3)
2	276	15.5 (2.4)	16.4 (2.8)	304	16.3 (2.4)	16.5 (3.7)	580	15.9 (2.4)	16.5 (3.2)
3 or more	139	15.2 (2.6)	16.4 (2.6)	171	16.5 (2.5)	17.0 (4.1)	310	15.9 (2.6)	16.7 (3.5)
<i>Full Older Sisters</i>									
0	1110	15.3 (2.7)	16.3 (3.0)	1442	16.1 (2.5)	16.6 (3.6)	2552	15.7 (2.6)	16.4 (3.4)
1	580	15.4 (2.6)	16.5 (2.7)	687	16.2 (2.6)	16.2 (3.9)	1267	15.8 (2.6)	16.3 (3.4)
2	215	15.3 (2.6)	16.2 (2.9)	294	16.0 (2.4)	16.1 (3.8)	509	15.7 (2.5)	16.2 (3.4)
3 or more	146	15.5 (2.6)	16.4 (2.7)	186	16.3 (2.6)	16.4 (3.8)	332	16.0 (2.6)	16.4 (3.4)

Table 1.b continued

Measure	Males			Females			Total		
	N	Mean AFD	Mean AFI	N	Mean AFD	Mean AFI	N	Mean AFD	Mean AFI
<i>CIAO Brothers (< 3 Yrs. Older)</i>									
0	1156	15.2 (2.8)	16.2 (2.9)	1570	15.9 (2.7)	16.3 (3.7)	2726	15.6 (2.7)	16.3 (3.4)
1 or 2	229	14.7 (3.0)	15.8 (3.1)	326	15.8 (2.8)	16.3 (3.7)	555	15.3 (2.9)	16.1 (3.4)
<i>CIAO Sisters (< 3 Yrs. Older)</i>									
0	1182	15.1 (2.8)	16.1 (2.9)	1574	15.9 (2.7)	16.4 (3.8)	2756	15.5 (2.8)	16.3 (3.4)
1 or 2	203	15.1 (2.8)	16.0 (3.2)	322	15.9 (2.4)	16.0 (3.6)	525	15.6 (2.6)	16.0 (3.4)
<i>CIAO Brothers (< 3 Yrs. Older)</i>									
0	1236	15.2 (2.8)	16.1 (3.0)	1721	15.9 (2.7)	16.3 (3.7)	2957	15.6 (2.8)	16.2 (3.4)
1 or 2	149	14.7 (3.0)	16.0 (2.8)	175	15.8 (2.7)	16.3 (3.6)	324	15.3 (2.9)	16.1 (3.2)
<i>CIAO Brothers (\leq 2 Yrs. Older)</i>									
0	1574	15.9 (2.7)	16.4 (3.8)	1182	15.1 (2.8)	16.1 (2.9)	2756	15.5 (2.8)	16.3 (3.4)
1 or 2	322	15.9 (2.4)	16.0 (3.6)	203	15.1 (2.8)	16.0 (3.2)	525	15.6 (2.6)	16.0 (3.4)

Note: AFD = Age at First Drink; AFI = Age at First Intoxication; CIAO = Close in Age Older. Standard deviations given in parentheses.

Table 2

F-Statistics and p-Values for Analyses Assessing the Influence of Sibship Factors on Conduct Disorder: Raw and Rank-Transformed Data

Measure	Raw Data		Rank Data	
	<i>F</i>	<i>p</i> -value	<i>F</i>	<i>p</i> -value
Sibship Size				
<i>Full Siblings</i>	3.50	.01	3.50	.02
Sex	282.83	< .0001	355.90	< .0001
Full Siblings*Sex	4.12	.006	2.01	.11
<i>Full Older Siblings</i>	3.92	.008	3.27	.02
Sex	324.71	< .0001	402.38	< .0001
Full Older Siblings*Sex	3.32	.02	1.44	.23
<i>CIAO Siblings (< 3 Yrs. Older)</i>	.01	.91	.36	.55
Sex	189.26	< .0001	239.53	< .0001
CIAO Siblings*Sex	.29	.59	.01	.93
<i>CIAO Siblings (< 2 Yrs. Older)</i>	.99	.32	1.60	.21
Sex	148.48	< .0001	189.14	< .0001
CIAO Siblings*Sex	.18	.67	.99	.32
Sibship Composition				
<i>Full Brothers</i>	.99	.40	.78	.51
Sex	284.48	< .0001	343.46	< .0001
Full Brothers*Sex	.98	.40	.51	.68
<i>Full Sisters</i>	3.41	.02	4.62	.003
Sex	279.52	< .0001	342.04	< .0001
Full Sisters*Sex	1.91	.13	.91	.43
<i>Full Older Brothers</i>	1.26	.29	.65	.58
Sex	218.91	< .0001	257.21	< .0001
Full Older Brothers*Sex	.93	.42	.40	.75
<i>CIAO Brothers (< 3 Yrs. Older)</i>	.00	.95	.27	.60
Sex	121.46	< .0001	161.22	< .0001
CIAO Brothers*Sex	.40	.53	.02	.88
<i>CIAO Sisters (< 3 Yrs. Older)</i>	.17	.68	.01	.94
Sex	119.04	< .0001	145.05	< .0001
CIAO Sisters*Sex	.14	.70	.15	.70

Table 2 continued

Measure	Raw Data		Rank Data	
	<i>F</i>	<i>p</i> -value	<i>F</i>	<i>p</i> -value
<i>CIAO Brothers (≤ 2 Yrs. Older)</i>	.91	.34	1.80	.18
Sex	82.23	< .0001	114.13	< .0001
CIAO Brothers*Sex	.06	.81	.41	.52
<i>CIAO Sisters (≤ 2 Yrs. Older)</i>	.25	.62	.12	.73
Sex	85.92	< .0001	98.75	< .0001
<i>CIAO Brothers*Sex</i>	.64	.42	.36	.55

Note: CIAO = Close in Age Older.

Table 3.a

Sibship Size: Effect of Having at Least One Sibling

Measure	AFD		AFI	
	<i>F</i>	<i>p</i> -value	<i>F</i>	<i>p</i> -value
<i>Full Siblings</i>	2.15	.14	.13	.72
<i>Full Older Siblings</i>	.41	.52	1.26	.26
<i>CIAO Siblings (< 3 Yrs. Older)</i>	.84	.36	1.22	.27
<i>CIAO Siblings (≤ 2 Yrs. Older)</i>	.13	.71	1.22	.27

Note: AFD = Age at First Drink; AFI = Age at First Intoxication; CIAO = Close in Age Older.

Table 3.b

Sibship Composition: Effect of Having at Least One Sibling

Measure	AFD		AFI	
	<i>F</i>	<i>p</i> -value	<i>F</i>	<i>p</i> -value
<i>Full Brothers</i>	4.78	.03	.10	.76
<i>Full Sisters</i>	.94	.33	.33	.56
<i>Full Older Brothers</i>	.90	.34	.09	.76
<i>Full Older Sisters</i>	.76	.39	1.14	.28
<i>CIAO Brothers (< 3 Yrs. Older)</i>	1.01	.32	.12	.73
<i>CIAO Sisters (< 3 Yrs. Older)</i>	0.00	.99	1.45	.23
<i>CIAO Brothers (\leq 2 Yrs. Older)</i>	.77	.38	1.27	.26
<i>CIAO Sisters (\leq 2 Yrs. Older)</i>	.16	.69	.12	.73

Note: AFD = Age at First Drink; AFI = Age at First Intoxication; CIAO = Close in Age Older.

Table 4.a

Sibship Size: Effects of Different Numbers of Siblings

Measure	AFD		AFI	
	<i>F</i>	<i>p</i> -value	<i>F</i>	<i>p</i> -value
<i>Full Siblings</i>	2.70	.04	.47	.70
Sex	53.99	< .0001	62.91	< .0001
Full Siblings*Sex	.79	.50	.71	.54
<i>Full Older Siblings</i>	.77	.51	.31	.82
Sex	74.96	< .0001	86.18	< .0001
Full Older Siblings*Sex	1.54	.20	1.07	.36
<i>CIAO Siblings (< 3 Yrs. Older)</i>	1.90	.17	1.82	.18
Sex	44.70	< .0001	52.25	< .0001
CIAO Siblings*Sex	2.50	.11	0.00	.97
<i>CIAO Siblings (≤ 2 Yrs. Older)</i>	.63	.43	1.57	.21
Sex	37.03	< .0001	36.44	< .0001
CIAO Siblings*Sex	2.21	.14	.06	.81

Note: AFD = Age at First Drink; AFI = Age at First Intoxication; CIAO = Close in Age Older.

Table 4.b

Sibship composition: Effects of different numbers of brothers/sisters.

Measure	AFD		AFI	
	<i>F</i>	<i>p</i> -value	<i>F</i>	<i>p</i> -value
<i>Full Brothers</i>	1.40	.24	.	.
Sex	64.65	< .0001	.	.
Full Brothers*Sex	1.53	.20	.	.
<i>Full Sisters</i>	.39	.76	.10	.96
Sex	57.10	< .0001	65.96	< .0001
Full Sisters*Sex	1.03	.38	.87	.46
<i>Full Older Brothers</i>	.55	.65	.	.
Sex	59.55	< .0001	.	.
Full Older Brothers*Sex	2.48	.06	.	.
<i>Full Older Sisters</i>	1.55	.20	.50	.68
Sex	47.54	< .0001	55.52	< .0001
Full Older Sisters*Sex	.96	.41	.21	.89
<i>CIAO Brothers (< 3 Yrs. Older)</i>	1.51	.22	.25	.62
Sex	32.92	< .0001	39.11	< .0001
CIAO Brothers*Sex	1.41	.23	.27	.60
<i>CIAO Sisters (< 3 Yrs. Older)</i>	.16	.69	1.57	.21
Sex	30.42	< .0001	26.74	< .0001
CIAO Sisters*Sex	.98	.32	.83	.36
<i>CIAO Brothers (≤ 2 Yrs. Older)</i>	.62	.43	.90	.34
Sex	19.58	< .0001	22.97	< .0001
CIAO Brothers*Sex	.29	.59	0.00	.99
<i>CIAO Sisters (≤ 2 Yrs. Older)</i>	.16	.69	.46	.50
Sex	23.94	< .0001	17.42	< .0001
CIAO Sisters*Sex	2.06	.15	.10	.74

Note: AFD = Age at First Drink; AFI = Age at First Intoxication; CIAO = Close in Age Older.