Published in final edited form as:

Psychol Addict Behav. 2010 September ; 24(3): 386–396. doi:10.1037/a0019801.

Parental Alcohol Involvement and Adolescent Alcohol Expectancies Predict Alcohol Involvement in Male Adolescents

James A. Cranford,
University of Michigan

Robert A. Zucker,
University of Michigan

Jennifer M. Jester,
University of Michigan

Leon I. Puttler, and
University of Michigan

Hiram E. Fitzgerald
Michigan State University

Abstract

Current models of adolescent drinking behavior hypothesize that alcohol expectancies mediate the effects of other proximal and distal risk factors. This longitudinal study tested the hypothesis that the effects of parental alcohol involvement on their children's drinking behavior in mid-adolescence are mediated by the children's alcohol expectancies in early adolescence. A sample of 148 initially 9–11 year old boys and their parents from a high-risk population and a contrast group of community families completed measures of drinking behavior and alcohol expectancies over a 6-year interval. We analyzed data from middle childhood (M age = 10.4 years), early adolescence (M age = 13.5 years), and mid-adolescence (M age = 16.5 years). The sample was restricted only to adolescents who had begun to drink by mid-adolescence. Results from zero-inflated Poisson regression analyses showed that 1) maternal drinking during their children's middle childhood predicted number of drinking days in middle adolescence; 2) negative and positive alcohol expectancies in early adolescence predicted odds of any intoxication in middle adolescence; and 3) paternal alcoholism during their children's middle childhood and adolescents' alcohol expectancies in early adolescence predicted frequency of intoxication in middle adolescence. Contrary to predictions, child alcohol expectancies did not mediate the effects of parental alcohol involvement in this high-risk sample. Different aspects of parental alcohol involvement, along with early adolescent alcohol expectancies, independently predicted adolescent drinking behavior in middle adolescence. Alternative pathways for the influence of maternal and paternal alcohol involvement and implications for expectancy models of adolescent drinking behavior were discussed.

Correspondence concerning this article should be addressed to James A. Cranford, Ph.D., Addiction Research Center (UMARC), Rachel Upjohn Building, 4250 Plymouth Rd., Department of Psychiatry, University of Michigan, Ann Arbor, MI 48109-2700. jcranfor@med.umich.edu.

Publisher's Disclaimer: The following manuscript is the final accepted manuscript. It has not been subjected to the final copyediting, fact-checking, and proofreading required for formal publication. It is not the definitive, publisher-authenticated version. The American Psychological Association and its Council of Editors disclaim any responsibility or liabilities for errors or omissions of this manuscript version, any version derived from this manuscript by NIH, or other third parties. The published version is available at www.apa.org/pubs/journals/adb

Keywords

Adolescent alcohol expectancies; Adolescent drinking behavior; Parental drinking; Parental influence

Parental Alcohol Involvement and Adolescent Alcohol Expectancies Predict Alcohol Involvement in Male Adolescents

Does exposure to parental drinking influence the drinking behavior of their children once drinking onset has occurred? If so, are the effects mediated by childhood alcohol expectancies preceding the child's drinking? A probabilistic-developmental model of risk (Zucker, 1994; Zucker, Fitzgerald, & Moses, 1995; Zucker et al., 2008) emphasizes the cumulation of risk factors over time for the developmental course of problem drinking, and the specification of how various risk factors work together to produce negative outcomes has been identified as a priority topic (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). In this paper, we examine the longitudinal effects of parental alcohol involvement and adolescent alcohol expectancies (AEs) on subsequent alcohol involvement among adolescents who had begun drinking by mid-adolescence. Below, we review the literature on parental alcohol involvement and adolescent alcohol expectancies on later drinking behavior.

Effects of Parental Alcohol Involvement on Adolescent Drinking Behavior

An extensive body of evidence showed that parental substance use and family history of substance use are predictive of adolescent substance use (for reviews, see Ellis, Zucker, & Fitzgerald, 1997; Hawkins, Catalano, & Miller, 1992; Scheier, 2001; Sher, Grekin, & Williams, 2005; Wills & Yaeger, 2003; Windle, 1996). For example, Sher, Walitzer, Wood, and Brent (1991) examined the concurrent effects of parental alcoholism on adolescent alcohol involvement in a large sample of college freshmen (M age = 18.2 years). They found that children of alcoholics (COAs) reported higher levels of alcohol involvement (e.g., quantity-frequency of consumption, heavy drinking, and alcohol dependence symptoms) compared to non-COAs, although the extent to which alcoholic parents were actually drinking during the child's earlier years was not evaluated. Similar findings have been obtained with samples of younger adolescents. For example, working with a sample of alcoholic and nonalcoholic families from the community, Chassin, Rogosch, and Barrera (1991) examined the concurrent effects of parental alcoholism on the alcohol involvement of the adolescent offspring (M age = 12.7). Results showed that paternal but not maternal alcoholism predicted greater adolescent alcohol involvement, and this effect was stronger among the older adolescents.

Other evidence in addition to the Chassin et al. (1991) study indicated that the association between parental and offspring alcohol involvement differs depending on the gender of the parent and/or the child. However, the nature of the relationship has not been consistently replicated. Thus, Zhang, Welte, and Wieczorek (1999) found that paternal but not maternal drinking had a direct concurrent effect on adolescent boys' drinking (age range 16–19). By contrast, Ohannessian et al. (2004) found that maternal substance use consequences were more consistently related to concurrent adolescent psychopathology (including alcohol dependence, depression, and conduct disorder) than paternal substance use consequences. Furthermore, parental effects are not always observed; in another cross-sectional study, Cooper, Peirce, and Tidwell (1995) found no consistent associations between maternal or paternal drinking and adolescent substance use. Similarly, Yu (2003) showed that parental alcohol use was related to lifetime but not current alcohol use of their adolescent children (age range 15–18).

Although smaller in number, longitudinal studies have yielded similar results. White, Johnson, and Buyske (2000) followed adolescents across four waves from ages 15 to 28. Paternal and maternal drinking were predictive of a heavy drinking trajectory among sons and daughters. Wills, Sandy, Yaeger, and Shinar (2001) assessed 1,269 adolescents in 6th, 7th, and 8th grade and found that parental substance use (alcohol and tobacco use as reported by the child) predicted higher adolescent substance use (a latent variable comprised of alcohol, tobacco, and marijuana use) in grade 6, but did not predict increases in adolescent use over time. In one of the few longitudinal studies utilizing a high-risk sample, Chassin, Curran, Hussong, and Colder (1996) found that paternal and maternal alcoholism predicted initial levels of substance use (alcohol and illicit drug use) among girls and boys, but only paternal alcoholism and adolescent male gender predicted increases in substance use over a three year interval. Although the effects of paternal alcoholism were partially mediated by fathers' monitoring and adolescents' stress, negative affect, and associations with substance-using peers, these hypothesized mediators did not fully account for the paternal alcoholism effect.

Effects of Child and Adolescent Alcohol Expectancies on Adolescent Drinking Behavior

Alcohol expectancies are another potent risk factor for adolescent alcohol involvement. The available evidence indicates that alcohol expectancies are "among the strongest predictors of drinking, even after other variables are controlled" (Goldman, Del Boca, & Darkes, 1999, p. 219). Previous research using school-based samples showed that alcohol expectancies predicted drinking onset among adolescents (Bauman, Fisher, Bryan, & Chenoweth, 1985; Killen et al., 1996), and results from several longitudinal studies found that adolescent alcohol expectancies predicted increases in alcohol consumption over time (e.g., Aas, Leigh, Anderssen, & Jakobsen, 1998; Bauman et al., 1985; Christiansen, Smith, Roehling, & Goldman, 1989; Newcomb, Chou, Bentler, & Huba, 1988; Smith, Goldman, Greenbaum, & Christiansen, 1995). Similar findings have been obtained from longitudinal studies of college student samples (Darkes, Greenbaum, & Goldman, 2004; Goldman, Greenbaum, & Darkes, 1997; Kidorf, Sherman, Johnson, & Bigelow, 1995; Sher, Wood, Wood, & Raskin, 1996). In addition, there is longitudinal evidence that AEs predict the transition from nonproblem to problem drinking (Christiansen et al., 1989), and a recent study of high school students (*M* age = 16.2) found that tension-reduction AEs were concurrently associated with frequency of drunkenness (Catanzaro & Laurent, 2004).

Child and Adolescent Alcohol Expectancies as Mediators of the Effects of Parental Drinking on Adolescent Drinking

To this point, our review has indicated some linkages between a) parental and adolescent drinking, and b) adolescent AEs and adolescent drinking. Models of alcohol expectancies emphasize their role as mediators of the effects of more distal risk factors (Goldman et al., 1999; Petraitis, Flay, & Miller, 1995). Tests of social learning theory explanations of adolescent alcohol use have shown that exposure to parents who use alcohol has a direct relationship to alcohol expectancies (Zucker, Kincaid, Fitzgerald, & Bingham, 1995), which in turn predicts alcohol involvement (Petraitis et al.). The hypothesized mediational effects of AEs have been elaborated by a number of alcohol researchers (Goldman et al., 1999; Scheier & Botvin, 1997; Sher et al., 1991).

However, direct tests of the mediational role of alcohol expectancies, based on the assumption that alcohol expectancies are among the most proximal correlates of drinking behavior, have yielded conflicting findings. For example, the cross-sectional study by Sher et al. (1991) found that the effect of family history of paternal alcoholism on college students' alcohol involvement was mediated by the students' own level of behavioral undercontrol as well as by their (positive) alcohol expectancy level. Ouellette, Gerrard, Gibbons, and Reis-Bergan (1999) followed parents and their offspring over 4 years and showed that the effect of parental drinking (average

of maternal and paternal consumption) on adolescent alcohol consumption was mediated by adolescent alcohol expectancies. By contrast, a longitudinal study by Colder, Chassin, Stice, and Curran (1997) found that parental alcoholism had a direct effect on increases in adolescent heavy drinking that was *not* mediated by the adolescents' alcohol expectancies.

The Present Study

Taken together, the available evidence remains unclear with respect to the hypothesis that adolescent AEs mediate the effects of their parents' alcohol involvement on their own drinking during adolescence. Although evidence suggests linkages between a) maternal and paternal alcohol involvement and their adolescent children's drinking and b) the adolescent's alcohol expectancies on their own drinking, longitudinal studies have not produced consistent support for the mediational hypothesis. A major challenge to understanding these relationships is that both drinking and non-drinking adolescents have typically been included in the same analyses even though evidence has indicated that alcohol expectancies change substantially as a function of the transition from nondrinking to drinking (see Christiansen, Goldman, & Inn, 1982; Schell et al., 2005). Such changes have been linked to the concrete experience of drinking and the exposure to alcohol's pharmacodynamic effects (Aas et al., 1998). Thus, inclusion of both groups creates a confounded predictor (the expectancies). In this paper, we examine the possible mediational role of childhood alcohol expectancies in explaining the association between parental alcohol involvement and adolescent drinking behavior *once drinking has begun* (i.e., after the transition has occurred). Using longitudinal data, we tested the hypothesis that the association between parental drinking and adolescent drinking is mediated by adolescents' AEs.

Method

Participants

The present work is from the ongoing Michigan Longitudinal Study (MLS; Zucker et al., 2000), a prospective study that is following a community sample of initially intact families with high levels of substance use/abuse, along with a community contrast sample of families drawn from the same neighborhoods, but without the high substance abuse profile. The long term focus of the project is the emergence and development of substance abuse and problems in the children, and the patterns of stability and change in drug involvement among the parents.

A community-based but high alcohol-involved sample of initially intact families was recruited by identifying fathers on the basis of a drunk driving conviction with a high blood alcohol level (0.15 percent if a first conviction, 0.12 percent if not the first). Families were required to have at least one son in the 3–5 year old age range, and daughters in the 3–11 year old age range were recruited when present. Presence of fetal alcohol syndrome was ruled out by study exclusionary criteria. Both biological parents were required to be living with the child at the time of recruitment, and mothers' substance use status was free to vary. A contrast/control group of families who resided in the same neighborhoods as the drunk driver families but had no substance abuse history for either parent was also recruited. A second subset of families with a father who also had an alcohol use disorder was uncovered and recruited during the community canvass for controls (Zucker et al., 2000). After initial recruitment and assessment, individuals participated in multi-session assessments every 3 years. Data collection was completed by professional staff, graduate students, and carefully trained and supervised undergraduates.

Participants for the present study were biological fathers, mothers, and sons who completed relevant measures at child ages 9–11 (middle childhood), 12–14 (early adolescence), and 15–17 (mid-adolescence). For ease of presentation, we refer to these time points as baseline (T1),

T2, and T3 (although the MLS designation is T3, T4, and T5). A total of 259 MLS families completed the study protocol at T3. This total included a subset of girls potentially available for the study. However, the sample of girls available for this study ($n=21$) was not large enough to conduct meaningful analyses by gender, and because substantial sex differences are known to exist for many drinking indicators, we limited our analyses to boys. Also, because the present work is focused on individual differences in later adolescence, when drinking has to a large extent been initiated, we analyzed data only from families where the boys had begun drinking by T3 (when they were an average of 16.5 years old). Of the 259 boys who completed the T3 protocol, 57.1% ($N = 148$) had begun drinking. We analyzed data from these 148 boys and their parents at T1, T2, and T3. Based on responses to the question “How old were you the first time you ever took a drink? Do not count the times you were given a ‘sip’ by an adult,” the mean (SD) of drinking onset was 13.5 (2.5) years old (range = 5 to 17 years old). Although the sample of 148 boys included 5 male siblings of the MTCs, all participants were treated as independent based on an intraclass correlation of 0. At baseline, T2, and T3, average ages for children (with standard deviations in parentheses) were 10.4 (.9), 13.5 (.9), and 16.5 (1.0) years.

Parents at T1 were 148 mothers and fathers whose mean ages (SD) were 36.6 (4.0) and 39.2 (5.0) years, respectively. Couples had been married for an average of 11.4 years. Both couple members had completed about 2 years of education beyond high school, for mothers, M (SD) = 14.2 (1.9); for fathers, $M = 14.9$ (2.3), and median family income was \$40,000. All families were Caucasian because less than 4% of the population we sampled from was non-Caucasian. Given our sample size, this precluded effective analyses of race and ethnic differences.

Measures

Parental Lifetime Alcohol Use Disorder (AUD) and Alcohol Involvement at T1—

DSM-IV alcoholism diagnosis for both parents was assessed using several measures, including the Short Michigan Alcoholism Screening Test (SMAST; Selzer, Vinokur, & van Rooijen, 1975), the Drinking and Drug History Questionnaire (DDH; Zucker, 1991), and the Diagnostic Interview Schedule—Version IV (DIS-IV; Robins, Helzer, Croughan, & Ratcliff, 1981). The SMAST is a 13-item self-report screening inventory that assesses alcohol problems. The DDH contains a series of questions asking about alcohol and other drug use and alcohol-related consequences over the past 6 months. The DIS-IV is a structured diagnostic interview that collects extensive information about physical, alcohol- and drug-related symptoms, and other psychiatric symptoms. Trained clinicians used data from all three sources of data to create a best-estimate diagnosis (Leckman, Sholomskas, Thompson, Belanger, & Weisman, 1982) of a lifetime alcohol use disorder (abuse or dependence) for both parents. DIS data were used as the base supplemented by the DDH and SMAST data, guided by the principle that when a symptom was admitted, even from only one source, it probably was present. To evaluate the reliability of this pooled diagnosis, two raters independently diagnosed a series of 26 protocols. Agreement as evaluated by kappa was .81, indicating acceptable reliability. In this subsample of $N = 148$ drinking boys, 33.1% ($n=49$) of the mothers and 76.4% ($n=113$) of the fathers had a lifetime AUD. In terms of family risk status, 23.7% ($n=35$) of the families were control families; 18.9% ($n=28$) were families from the community in which the father had an AUD; and 57.4% ($n=85$) were families in which the father had a drunk driving conviction.

Less severe forms of parental alcohol involvement (e.g., frequency of alcohol consumption; Ary et al., 1993) have also shown longitudinal associations with adolescent drinking. Accordingly, we included a measure of average number of drinking days per month in the last 6 months from the DDH (Zucker, 1991). The average number of drinking days per month was lower for mothers ($M=4.1$, $SD=5.6$) than fathers ($M=8.6$, $SD=8.5$), paired t (135) = -6.2 , $p < .01$.

Alcohol Expectancies at T2—Alcohol expectancies (AEs) were assessed with the Beverage Opinion Questionnaire (BOQ; Fitzgerald, Zucker, & Noll, 1990) which was administered to participants starting when they were between the ages of 6 and 8 years and then again at T1, T2, and T3. This 25-item questionnaire assesses negative (5 items) and positive (20 items) expectancies for alcohol and, as a buffer, also includes 30 expectancy questions about soft drinks. The BOQ is based on the adolescent version of the Alcohol Expectancy Questionnaire (AEQ-A; Christiansen et al., 1982; Brown, Christiansen, & Goldman, 1987) and the adult version of the Alcohol Expectancy Questionnaire (AEQ; Brown, Goldman, Inn, & Anderson, 1980). The original version of the AEQ-A was developed for use with adolescents ages 12–19 (Christiansen et al., 1982) and consists of 90 items. To reduce participant burden we sought to reduce the number of items for inclusion in the BOQ. Further, because we began asking about alcohol expectancies when participants were between the ages of 6 and 8 years old, we selected those items that seemed likely to be most comprehensible when read to children by the interviewers. With these concerns in mind, we selected 23 items from the AEQ-A. In addition, we selected 2 items from the adult AEQ that focused on sleep. Each statement concerning alcohol is in the format “Drinking beer or wine would...”, followed by a phrase indicating an expectancy for alcohol, e.g., “Drinking beer or wine would *make me feel good*” (positive expectancy), “Drinking beer or wine would *make me feel angry*” (negative expectancy).¹ Adolescents were asked to respond to each item on a four-point scale (1=*agree completely*, 2=*somewhat agree*, 3=*somewhat disagree*, 4=*completely disagree*). Inspection of the item distributions showed that few participants selected the “somewhat agree” or “somewhat disagree” response options. Thus, we collapsed response options to create binary versions of each item, where 0=*disagree completely or somewhat disagree* and 1=*agree completely or somewhat agree*. Items were summed to create negative and positive expectancies scores for each participant. At T2, the positive AEs scale had an alpha of .88 ($M=2.4$, $SD=3.6$), the negative AEs scale had an alpha of .74 ($M=1.9$, $SD=1.4$), and the two scales were moderately correlated, $r = .44$, $p < .01$.

Adolescent Alcohol Involvement at T3—For the adolescent version of the DDH, participants were asked about average drinking days per month (drink days) over the past 6 months at T3 ($M=2.8$, $SD=3.8$)². In other words, adolescents reported drinking on approximately 18 days during the past 6 months. Adolescent participants at T3 were also asked to indicate how many times during the past 6 months they had gotten drunk or very high from drinking alcohol. Scores on this variable ranged from 0 (*not at all*) to 52 (*about twice a week*) with a mean (SD) of 9.8 (20.4) episodes of drunkenness during the past 6 months.

Analytic Plan

Correlation and regression analyses were used to test the study hypotheses. Our two dependent variables (number of drinking days and number of drunken episodes in the past 6 months) were count variables. Count variables can sometimes be modeled as Poisson variables, but the

¹Brown et al. (1987) noted that, compared to the adult version of the AEQ, for the AEQ-A “statements are worded more generally to accommodate adolescents who have had little or no experience with alcohol” (p. 485). Specifically, “the Adult AEQ involves statements regarding the effects of alcohol on the respondent, whereas the Adolescent AEQ focuses on the effect of alcohol on people in general” (p. 488). For example, the adult AEQ item “Drinking makes me feel good” was modified for the AEQ-A to “Drinking alcohol makes a person feel good and happy.” We agree with Brown et al. that such modifications likely make the AEQ-A items more applicable to the entire adolescent population, including “adolescents who have not yet had direct or personal experience with alcohol” (p. 489). Because the MLS by design recruited a sample of families that was likely to have extensive experience with alcohol, we decided to retain the original wording of the items such that they referred to the adolescent. Recognizing that even in a high-risk sample not all adolescents will have consumed alcohol, each item was framed as a pure expectancy, rather than as a putative effect of alcohol involvement. Returning to the earlier example, the AEQ item “Drinking makes me feel good” was modified for the AEQ-A to “Drinking alcohol makes a person feel good and happy,” and we in turn modified this item for the BOQ to “Drinking alcohol would make me feel good.”

²Three cases were identified as outliers (i.e., more than 3 standard deviations above the mean; Stevens, 1998) on this variable. All analyses were conducted with and without these three outlier cases. The results did not differ for the two sets of analyses. Because descriptive statistics were unduly influenced by these outlier cases, all descriptive statistics are reported with the outlier cases excluded.

Poisson distribution is restricted to a single parameter for the mean and the variance, and alcohol-related count variables often exceed this restriction by having larger variances than means (i.e., overdispersion; Horton, Kim, & Saitz, 2007). Although the present sample consisted only of those who had started drinking, a substantial percentage of participants reported that they did not drink or experience any drunken episodes in the past 6 months (29.1% and 34.5% of the sample, respectively). Count variables with large numbers of zeroes are referred to as “zero-inflated” (Karaszia & van Dulmen, 2008). Accordingly, zero-inflated Poisson (ZIP) regression analysis (Lambert, 1992) was used to examine predictors of drinking behaviors. For ZIP models, two regression equations are estimated simultaneously: 1) a logistic regression model is used to predict whether or not a given behavior occurs (i.e. membership in an “always zero” versus a “not always zero” latent group; Karaszia & van Dulmen, 2008), and 2) a Poisson regression model is used to predict the number of times a given behavior occurs (Atkins & Gallop, 2007; Muthen & Muthen, 2007). Estimating ZIP models thus allows for the possibility that different variables may predict whether or not someone drinks and how much or how often someone drinks (Atkins & Gallop, 2007). All ZIP models were estimated with the Mplus software package using maximum likelihood estimation with robust standard errors (Muthen & Muthen, 2007). For ease of interpretation of the logistic regression results, we report the reciprocal odds ratio for each predictor so that they represent the odds of being in the nonzero class, i.e. the odds of the occurrence of each drinking behavior.

Missing data—Because the pairwise sample sizes for the variables in our models ranged from $n=87$ to $n=148$, we used multiple imputation (MI; Rubin, 1987; Sinharay et al., 2001) to impute missing data. In MI, each missing value is replaced by $m > 1$ simulated values, resulting in m complete data sets (Schafer, 1997; Schafer & Graham, 2002). These m data sets are analyzed using standard analytic methods, and the results are combined to obtain parameter estimates and standard errors that take into account missing data uncertainty (Sinharay et al., 2001). MI is based on the assumption that the data are missing at random (MAR; Sinharay, Stern, & Russell, 2001). Since this assumption is not testable, we included several variables in the imputation model that could potentially be linked to the missingness of the imputed variables (Schafer, 1997; Sinharay et al., 2001).

The pattern of missing data appeared to be arbitrary, and so we used the Markov Chain Monte Carlo (MCMC) imputation method (Schafer, 1997). For the variables in the models we tested, the rate of missing information (λ) ranged from a low of .15 to a high of .48. Results from a simulation study (Graham, Olchowski, & Gilreath, 2007) showed with $m=10$ imputations: 1) the power to detect a small effect size when $\lambda=.50$ showed a decrease of only about 3% compared to simulations with $m=100$ imputations; and 2) the relative efficiency of a given parameter estimate when $\lambda=.50$ is .96 (compared to simulations with $m=100$ imputations). Accordingly, we used SAS PROC MI (SAS, 2004) to create $m=10$ imputed data sets. We then used PROC CORR to conduct correlational analyses on the $m=10$ imputed data sets, and PROC MIANALYZE to combine the results from analyses of the $m=10$ data sets. As noted earlier, for the ZIP models, we used the Mplus program with multiple imputation and maximum likelihood estimation with robust standard errors (Muthen & Muthen, 2007).

Attrition Analyses—For mothers and fathers, we compared T1 responders and nonresponders on the measure of drinking frequency at T3, and no significant differences between responders and nonresponders were observed. For adolescents, we compared T2 responders and nonresponders on the measures of positive and negative alcohol expectancies at T3, and no significant differences between responders and nonresponders were observed. These results indicate that any nonresponse bias was minimal.

Results

Correlations between all study variables are presented in Table 1. Maternal and paternal measures of AUD and drinking frequency were moderately correlated (r s ranged from .22 to .33). Interestingly, there was no significant association between paternal AUD and maternal drinking frequency, or between maternal AUD and paternal drinking frequency. No statistically significant correlations between parental alcohol involvement and adolescents' positive alcohol expectancies were observed, but there was a weak direct association between paternal AUD and adolescents' negative alcohol expectancies 3 years later at T2. Paternal AUD and maternal drinking frequency were significantly associated with both measures of adolescent alcohol involvement. Maternal AUD was also positively associated with adolescents' drinking frequency (but not frequency of drunkenness), and paternal drinking frequency was positively associated with adolescents' frequency of drunkenness (but not drinking frequency). Adolescents' positive AEs at T2 were positively related to frequency of drunkenness (but not drinking frequency) at T3. Adolescents' negative AEs at T2 were not significantly associated with either measure of adolescent alcohol involvement.

Baron and Kenny (1986) outline methods for testing mediational hypotheses. These steps include 1) establishing an association between the predictor and the outcome variable, 2) establishing an association between the predictor and the putative mediator variable, 3) establishing an association between mediator and the outcome variable when the predictor is statistically controlled, and 4) showing that the association between the predictor and the outcome is reduced in magnitude when the mediator variable is entered into the regression equation. Although demonstration of a relationship between the predictor and the outcome is not always required (Shrout & Bolger, 2002), an association between the predictor and the putative mediator variable is necessary to establish mediation. However, as seen in Table 1, only one of the measures of parental alcohol involvement was associated with adolescent AEs: paternal AUD showed a direct association with negative AEs. Further, negative AEs were not associated with either measure of adolescent alcohol involvement. These findings do not support the hypothesis that adolescent AEs mediate the effects of parental alcohol involvement on adolescent drinking.

We then examined the effects of T1 parental alcohol involvement and T2 adolescent AEs as independent predictors of adolescent drinking at T3. ZIP regression analyses were conducted using the Mplus statistical software program (Muthen & Muthen, 2007). Results are presented in Table 2. We first tested the effects of lifetime paternal and maternal AUD, T1 paternal and maternal alcohol involvement, and T2 adolescent AEs as predictors of average drinking days in the past 6 months at T3. Because there was variation in age within waves, we controlled for adolescents' age at T3 in all analyses. For the logistic regression of the binary part of the dependent variable, age was the only significant predictor, and every 1 unit increase in age resulted in a 2.10 increase in the odds of drinking on any days in the past 6 months. For the Poisson regression of the count part of the dependent variable, the only significant predictor was T1 maternal drinking. To gain perspective on the meaning of the Poisson coefficients, we used procedures outlined by Long (1997, p. 229). For each 1-unit increase in mother's average drinking days per month, adolescents' drinking days per month increased by a factor of 1.033, an increase of 3.3%, when all other predictors were statistically controlled. Although significant, this appears to be a relatively small effect. For example, at the average level of mother's average drinking days per month (4.3), the expected number of adolescents' drinking days is 3.2. At one standard deviation above the average level of mother's average drinking days per month (10.7), the expected number of adolescents' drinking days is 4.0.

Next, we tested the effects of lifetime paternal and maternal AUD, T1 paternal and maternal alcohol involvement, and T2 adolescent AEs as predictors of frequency of intoxication in the

past 6 months at T3. As seen in Table 2, for the logistic regression of the binary part of the dependent variable, age and negative and positive alcohol expectancies were significant predictors of any intoxication in the past 6 months. Increases in age and positive expectancies resulted in higher odds of any intoxication, and increases in negative expectancies resulted in lower odds of any intoxication. None of the maternal or paternal alcohol involvement variables were associated with any intoxication in the past 6 months. For the Poisson regression of the count part of the dependent variable, lifetime paternal AUD and T2 positive expectancies were significantly associated with the frequency of intoxication in the past 6 months.

We again used procedures outlined by Long (1997) to gain perspective on the meaning of the Poisson coefficients. For each 1-unit increase in positive alcohol expectancies at T2, adolescents' number of times intoxicated increased by a factor of $\exp(.069)=1.07$, an increase of 7.0%, when all other predictors were statistically controlled. Although significant, this appears to be a relatively small effect. For example, at the average level of positive alcohol expectancies at T2 (2.4), the expected number of times intoxicated is 8.3. At one unit above the average level of positive alcohol expectancies at T2 (3.4), the expected number of times intoxicated is 8.9. We also used procedures outlined by Long (1997) to calculate the additive change in number of times intoxicated for adolescents as a function of father's lifetime AUD. For adolescents with a non-AUD father, the expected number of times intoxicated in the past 6 months is 3.3. By contrast, for adolescents with an AUD father, the expected number of times intoxicated in the past 6 months is 11.0, holding all other variables constant. Thus, while lifetime paternal AUD and T2 positive expectancies were independently associated with increases in the number of times intoxicated in the past 6 months at T3, the effects of paternal lifetime AUD were particularly strong in magnitude.

Discussion

This study tested the hypothesis that alcohol expectancies mediate the effects of parental alcohol involvement on adolescent drinking behavior. In partial support of our hypotheses, we found that two aspects of parental alcohol involvement (i.e., paternal lifetime AUD and maternal average drinking days per month) during middle childhood (T1) predicted some dimensions of mid-adolescent drinking (T3). Contrary to our hypothesis, results showed that the effects of parental alcohol involvement were not mediated by adolescent AEs. Rather, parental drinking and positive and negative adolescent AEs had independent longitudinal associations with adolescent drinking behavior.

Parental Alcohol Involvement and Adolescents' Drinking Behaviors

Our findings with respect to the effects of parental alcohol involvement on adolescents' drinking behaviors are consistent with a long line of work indicating that parents have profound effects on the drinking behaviors of their children (e.g., Fitzgerald, Davies, & Zucker, 2002; Jacob & Johnson, 1997; Wills & Yaeger, 2003; Windle, 1996; Zucker et al., 2000, 2008). Our findings are unique, however, in showing that different aspects of paternal and maternal alcohol involvement are longitudinally associated with different aspects of their sons' alcohol involvement 6 years later. The finding that mothers' but not fathers' drinking behavior was predictive of subsequent alcohol involvement in their sons is consistent with the work of Brook and her colleagues (Brook, Whiteman, Gordon, & Cohen, 1986), who found that aspects of the mother-child relationship were stronger protective factors for adolescent drug use than were similar aspects of the father-child relationship. Brook et al. speculated that mothers have more influence on child-rearing practices than do fathers. Our findings indicate that this influence extends to the domain of alcohol involvement, at least in terms of adolescents' average drinking days per month (also see Christiansen & Goldman, 1983). Related to this point, the greater amount of time spent with mothers versus fathers may lead adolescents to more closely model

their own drinking behavior after that of their mothers. This may in part explain why maternal drinking behavior, but not maternal AUD, predicted their son's drinking behavior (Ohannessian & Hesselbrock, 2004).

By contrast, paternal alcoholism (but not paternal drinking behavior) was predictive of sons' alcohol involvement, and this effect was limited to frequency of intoxication. Paternal alcoholism is associated with a wide range of parenting variables, including less parental discipline (King & Chassin, 2004), lower levels of parental monitoring (Chassin et al., 1993; Chassin et al., 1996), and higher levels of child abuse and neglect (Richter & Richter, 2001); all of these variables are separately associated with earlier and heavier drinking among offspring. Our findings are thus consistent with recent work showing that COAs continue to show elevated levels of heavy drinking even when their fathers' alcoholism has remitted (DeLucia, Belz, & Chassin, 2001). In addition, parental alcoholism confers heightened genetic risk among some COAs (Zucker et al., 2008). The combination of socialization and genetic risk may explain the relatively large magnitude of the effect of paternal alcoholism on adolescents' intoxication.

With respect to the null findings for paternal drinking behavior, we note that paternal as compared to maternal drinking is more likely to occur on a sporadic basis for antisocial alcoholics (Jacob & Leonard, 1988), a group that has a substantial representation in the current sample of alcoholics. Thus, exposure to father drinking, especially during late preadolescence, would have been less available to the children than exposure to mother drinking. Last, to some degree the alcoholic fathers' drinking behavior was dampened during the earlier years of the study as a result of conviction for drunk driving. Such convictions sometimes required attendance at alcohol education classes and produced a dampening effect on fathers' consumption which would distort the relationship between their own undampened drinking and their children's alcohol involvement. This process was not in operation for the mothers.

The observation that paternal alcoholism – but not paternal drinking behavior – was predictive of sons' alcohol involvement is not readily attributable to a drinking-modeling explanation (see Sher et al., 2005). However, exposure to parental modeling was not directly measured in this study. Brown and colleagues (1999) showed that degree of exposure to an alcohol-abusing family member mediated the association between parental alcoholism and positive alcohol expectancies, and noted that variation in exposure to alcohol-abusing family members, even within families characterized by a biological history of alcoholism, might be considerable. In the absence of a direct measure of parental modeling, a drinking-modeling explanation for the present findings related to paternal AUD cannot be ruled out.

Adolescent Alcohol Expectancies and Drinking Behaviors

Our results also showed that adolescents' negative and positive AEs were longitudinally associated with higher odds of any intoxication 3 years later, and positive AEs further predicted frequency of drunkenness, independently of parental alcohol involvement. These findings replicate previous work in showing that AEs in early adolescence are longitudinally associated with drinking later in adolescence (Reese, Chassin, & Molina, 1994; Smith et al., 1995), and more generally with previous results showing that positive and negative expectancies are predictive of alcohol involvement (Goldman & Darkes, 2004). Further, the differential effects of negative and positive AEs on the occurrence and frequency of intoxication are consistent with evidence reported by Leigh and Stacy (2004), who suggested that "negative expectancy predicts abstinence while positive expectancy predicts amount of drinking among those who drink" (p. 224) (also see Chen, Grube, & Madden, 1994).

Results are also consistent with the hypothesis advanced by Sher and Gotham (1999) that AEs are *developmentally specific* risk factors for alcohol involvement. Alcohol schemas emerge as

early as age 3 (Zucker et al., 1995), and evidence indicated a shift in AEs from more negative to more positive during the period from middle childhood to early adolescence (grades 6 to 9, M ages 11.0 to 14.9; Dunn & Goldman, 1998, 2000; cf. Spiegler, 1983). Positive expectancies may better predict alcohol involvement than negative expectancies among younger participants, but the effects of negative AEs become stronger as a function of age (Leigh & Stacy, 2004). The current results add to this literature by showing that positive expectancies in middle adolescence have a stronger association to risky drinking than to overall frequency of drinking behavior.

Adolescent Alcohol Expectancies as Mediators of the Effects of Parental Alcohol Involvement

Our findings did not support the hypothesis that the effects of parental alcohol involvement are mediated by alcohol expectancies (Chassin et al., 1996). However, it is important to consider some aspects of the current study that limited our ability to draw firm conclusions about the mediation hypothesis. Our sample was by design limited to Caucasian males, many of whom were living in high-risk families, and this limits the generality of the findings. Also, our relatively small sample size was underpowered to detect mediation when the associations between a) the independent variable and the dependent variable, and b) the mediator and the dependent variable are in the small to moderate range (Fritz & MacKinnon, 2007).

Our own as well as other work suggests a complex relationship between parental drinking behavior and alcohol expectancies in children and adolescents. As noted earlier, several studies have found evidence for linkages between paternal alcohol involvement and their children's AEs (e.g., Brown et al., 1999). By contrast, Kraus, Smith, and Ratner (1994) found no cross-sectional associations between alcohol expectancies among 268 children in grades 2 through 4 and maternal and paternal drinking attitudes, parental problem drinking, and family history of AUD (also see Brown, Creamer, & Stetson, 1987; Henderson et al., 1994; Miller, Smith, & Goldman, 1990). Reasons for variability in the association between parental alcohol involvement and adolescent alcohol expectancies include sample heterogeneity and use of different measures of alcohol involvement and expectancies (e.g., Sher et al., 1991). Another important difference relates to the time lag between longitudinal assessments. Collins and colleagues (Collins & Graham, 2002) noted that the association between two variables can change dramatically across different measurement intervals and highlighted the importance of *temporal design* – defined as “the timing, frequency, and spacing of observations in a longitudinal study” (Collins, 2006, p.508) – for longitudinal studies of developmental processes (see Handley & Chassin, 2009). Greater attention to temporal design will clarify the status of mediational hypotheses about parental alcohol involvement and adolescent AEs (also see Sher et al., 1996).

Adolescent Alcohol Expectancies and Parental Alcohol Involvement as Independent Risk Factors for Adolescent Alcohol Involvement

The current findings are most consistent with the hypothesis that parental alcohol involvement and alcohol expectancies represent independent risk factors for subsequent alcohol involvement among adolescents (e.g., Mann, Chassin, & Sher, 1987). This pattern of results represents a conceptual replication of previous work showing unique longitudinal associations between parental alcohol involvement and alcohol expectancies and subsequent alcohol involvement in adolescents (e.g., Reese et al., 1994). However, to our knowledge, ours is the first study to find unique longitudinal effects of maternal drinking and paternal AUD (assessed in middle childhood) and positive and negative AEs (assessed in early adolescence) on different dimensions of alcohol involvement (assessed in middle adolescence). Thus, at least in this sample, the evidence suggests that a) cognitive factors may be of greater importance than parental factors in terms of *whether or not* an adolescent decides to engage in risk drinking;

and b) paternal AUD may be of greater importance than cognitive factors in terms of the frequency of engaging in risk drinking.

Limitations

The present study has several limitations. First, the sample was limited to adolescent boys, and there is some evidence that the effects of parental alcohol involvement and alcohol expectancies may be different for adolescent girls (see Pastor & Evans, 2003). Second, the sample was limited to Caucasians, and evidence showed that ethnicity moderates some associations between expectancies and alcohol involvement (Chartier, Hesselbrock, & Hesselbrock, 2009). Third, we relied on adolescents' self-reports of alcohol use. Although self-report measures of alcohol involvement seem to have adequate reliability and validity (Babor et al., 2000), there are numerous factors that influence the validity of self-reports, including age and forgetting (Brener, Billy, & Grady, 2003; Del Boca & Darkes, 2003), and these factors may have a stronger effect on self-report in younger adolescents. Fourth, the restricted range/class of families in the "low risk" group may have contributed to the nonsignificant correlations we observed between parental AUD and adolescent alcohol expectancies. In addition, our use of 3-year assessment intervals, combined with the relatively small sample size, might have reduced the likelihood of detecting some of the hypothesized mediational processes. Further research using designs that combine shorter (e.g., daily) and longer time lags will clarify the status of the expectancy mediation hypothesis.

Conclusions and Implications

Despite these limitations, the present study has several important strengths. Results build on our earlier work showing that alcohol schemas form as early as age 3 (Zucker et al., 1995), and our design allowed us to follow children and their parents from middle childhood to middle adolescence, which covers the critical period during which adolescents first begin experimenting with alcohol and other drugs (Zucker, 2006). Also, by tracking children who transitioned from non-drinkers in middle childhood to drinkers in middle adolescence, we were able to confirm that alcohol expectancies precede the development of risky drinking. Intervention studies have shown that alcohol expectancies are amenable to experimental manipulation, and challenges to expectancies predict reductions in alcohol consumption among males (e.g., Dunn, Lau, & Cruz, 2000). The present results highlight the potential utility of challenges targeting positive alcohol expectancies for reducing risky drinking behaviors.

Our findings are particularly important in light of recent evidence that family influences on adolescent substance use are more pervasive than peer and neighborhood influences (Ennett et al., 2008) and persist through late adolescence (Wood, Read, Mitchell, & Brand, 2004). An important avenue for further work is identification of how different risk and protective factors at different levels influence one another (Buu et al., 2009). Also, while expectancies are clearly important precursors of alcohol involvement, other cognitive constructs (e.g., substance use intentions; Anderson, Smith, & Fischer, 2003) and personality constructs (e.g., resilience; Lee & Cranford, 2008) should also be considered.

It is important to note that these results were obtained across a particularly crucial period in adolescent development. Parents and adolescents were assessed when offspring were in middle childhood (ages 9 – 11, *M* age = 10.6 years), early adolescence (ages 12 – 14, *M* age = 13.5 years), and mid-adolescence (ages 15 – 17, *M* age = 16.5 years). Evidence showed that positive AEs increase over this period (Dunn & Goldman, 1998), particularly among adolescents exposed to peer and parental drinking (Cumsille, Sayer, & Graham, 2000). Yet, in a recent review, Windle et al. (2008, p. S285) asserted that "Unfortunately, we do not yet have longitudinal data mapping the progression of expectancy endorsement and its prediction of subsequent drinking among children 10 to 12 years of age. This gap in the literature is an

important one that needs to be rectified.” Windle et al. noted that this period usually involves the transition to middle school and adolescence; as such, “this transition may become a turning point for some children, and their developmental trajectories may become characterized by maladaptive features.” The current study addresses this gap in the literature and demonstrates the unique longitudinal effects of maternal and paternal alcohol involvement and adolescent AEs for specific dimensions of underage drinking.

Acknowledgments

This work was supported by Grants T32 AA07477 and Grant R37 AA07065 from the National Institute on Alcohol Abuse and Alcoholism to Robert A. Zucker, Ph.D. We thank the families who participate in the Michigan Longitudinal Study and Susan K. Refior, whose sustained work with the families in this study has been a major contributor to the study’s continuation. We also thank the editor and the anonymous reviewers for helpful comments on previous drafts of this manuscript.

References

- Aas HN, Leigh BC, Anderssen N, Jakobsen R. Two-year longitudinal study of alcohol expectancies and drinking among Norwegian adolescents. *Addiction* 1998;93:373–384. [PubMed: 10328045]
- Anderson KG, Smith GT, Fischer SF. Women and acquired preparedness: Personality and learning implications for alcohol use. *Journal of Studies on Alcohol* 2003;64:384–392. [PubMed: 12817828]
- Ary DV, Tildesley E, Hops H, Andrews JA. The influence of parent, sibling, and peer modeling and attitudes on adolescent use of alcohol. *International Journal of the Addictions* 1993;28:853–880. [PubMed: 8359945]
- Atkins DC, Gallop RJ. Rethinking how family researchers model infrequent outcomes: A tutorial on count regression and zero-inflated models. *Journal of Family Psychology* 2007;21:726–735. [PubMed: 18179344]
- Babor TF, Steinberg K, Anton R, Del Boca F. Talk is cheap: Measuring drinking outcomes in clinical trials. *Journal of Studies on Alcohol* 2000;61:55–63. [PubMed: 10627097]
- Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology* 1986;51:1173–1182. [PubMed: 3806354]
- Bauman KE, Fisher LA, Bryan ES, Chenoweth RL. Relationship between subjective expected utility and behavior: A longitudinal study of adolescent drinking behavior. *Journal of Studies on Alcohol* 1985;46:32–38. [PubMed: 3974233]
- Brener ND, Billy JOG, Grady WR. Assessment of factors affecting the validity of self-reported health-risk behavior among adolescents: Evidence from the scientific literature. *Journal of Adolescent Health* 2003;33:436–457. [PubMed: 14642706]
- Brook JS, Whiteman M, Gordon AS, Cohen P. Some models and mechanisms for explaining the impact of maternal and adolescent characteristics on adolescent stage of drug use. *Developmental Psychology* 1986;22:460–467.
- Brown SA, Christiansen BA, Goldman MS. The Alcohol Expectancy Questionnaire: An instrument for the assessment of adolescent and adult alcohol expectancies. *Journal of Studies on Alcohol* 1987;48:483–491. [PubMed: 3669677]
- Brown SA, Creamer VA, Stetson BA. Adolescent alcohol expectancies in relation to personal and parental drinking patterns. *Journal of Abnormal Psychology* 1987;96:117–121. [PubMed: 3584659]
- Brown SA, Goldman MS, Inn A, Anderson LR. Expectations of reinforcement from alcohol: Their domain and relation to drinking patterns. *Journal of Consulting and Clinical Psychology* 1980;48:419–426. [PubMed: 7400427]
- Brown SA, Tate SR, Vik PW, Haas AL, Aarons GA. Modeling of alcohol use mediates the effect of family history of alcoholism on adolescent alcohol expectancies. *Experimental and Clinical Psychopharmacology* 1999;7:20–27. [PubMed: 10036606]
- Buu A, DiPiazza C, Wang J, Puttler LI, Fitzgerald HE, Zucker RA. Parent, family, and neighborhood effects on the development of child substance use and other psychopathology from preschool to the start of adulthood. *Journal of Studies on Alcohol and Drugs* 2009;70:489–498. [PubMed: 19515288]

- Catanzaro SJ, Laurent J. Perceived family support, negative mood regulation expectancies, coping, and adolescent alcohol use: Evidence of mediation and moderation effects. *Addictive Behaviors* 2004;29:1779–1797. [PubMed: 15530721]
- Chartier KG, Hesselbrock MN, Hesselbrock VM. Ethnicity and adolescent pathways to alcohol use. *Journal of Studies on Alcohol and Drugs* 2009;70(3):337–345. [PubMed: 19371484]
- Chassin L, Curran PJ, Hussong AM, Colder CR. The relation of parent alcoholism to adolescent substance use: A longitudinal follow-up study. *Journal of Abnormal Psychology* 1996;105:70–80. [PubMed: 8666713]
- Chassin L, Pillow DR, Curran PJ, Molina BSG, Barrera M. Relation of parental alcoholism to early adolescent substance use: A test of three mediating mechanisms. *Journal of Abnormal Psychology* 1993;102:3–19. [PubMed: 8436697]
- Chassin L, Rogosch F, Barrera M. Substance use and symptomatology among adolescent children of alcoholics. *Journal of Abnormal Psychology* 1991;100:449–463. [PubMed: 1757658]
- Chen MJ, Grube JW, Madden PA. Alcohol expectancies and adolescent drinking: Differential prediction of frequency, quantity, and intoxication. *Addictive Behaviors* 1994;19:521–529. [PubMed: 7832010]
- Christiansen BA, Goldman MS. Alcohol-related expectancies versus demographic/background variables in the prediction of adolescent drinking. *Journal of Consulting and Clinical Psychology* 1983;51:249–257. [PubMed: 6841769]
- Christiansen BA, Goldman MS, Inn A. Development of alcohol-related expectancies in adolescents: Separating pharmacological from social-learning influences. *Journal of Consulting and Clinical Psychology* 1982;50:336–344. [PubMed: 7096736]
- Christiansen BA, Smith GT, Roehling PV, Goldman MS. Using alcohol expectancies to predict adolescent drinking behavior after one year. *Journal of Consulting and Clinical Psychology* 1989;57:93–99. [PubMed: 2925979]
- Colder CR, Chassin L, Stice EM, Curran PJ. Alcohol expectancies as potential mediators of parent alcoholism effects on the development of adolescent heavy drinking. *Journal of Research on Adolescence* 1997;7:349–374.
- Collins LM. Analysis of longitudinal data: The integration of theoretical model, temporal design, and statistical model. *Annual Review of Psychology* 2006;57:505–528.
- Collins LM, Graham JW. The effect of the timing and spacing of observations in longitudinal studies of tobacco and other drug use: Temporal design considerations. *Drug and Alcohol Dependence* 2002;68 Supplement 1:S85–S96. [PubMed: 12324177]
- Cooper ML, Peirce RS, Tidwell MCO. Parental drinking problems and adolescent offspring substance use: Moderating effects of demographic and familial factors. *Psychology of Addictive Behaviors* 1995;9:36–52.
- Cumsille PE, Sayer AG, Graham JW. Perceived exposure to peer and adult drinking as predictors of growth in positive alcohol expectancies during adolescence. *Journal of Consulting and Clinical Psychology* 2000;68:531–536. [PubMed: 10883572]
- Darkes J, Greenbaum PE, Goldman MS. Alcohol expectancy mediation of biopsychosocial risk: Complex patterns of mediation. *Experimental and Clinical Psychopharmacology* 2004;12:27–38. [PubMed: 14769097]
- Del Boca F, Darkes J. The validity of self-reports of alcohol consumption: state of the science and challenges for research. *Addiction* 2003;98:1–12. [PubMed: 14984237]
- DeLucia C, Belz A, Chassin L. Do adolescent symptomatology and family environment vary over time with fluctuations in paternal alcohol impairment? *Developmental Psychology* 2001;37:207–216. [PubMed: 11269389]
- Dunn ME, Goldman MS. Age and drinking-related differences in the memory organization of alcohol expectancies in 3rd-, 6th-, 9th-, and 12th-grade children. *Journal of Consulting and Clinical Psychology* 1998;66:579–585. [PubMed: 9642899]
- Dunn ME, Lau C, Cruz IY. Changes in activation of alcohol expectancies in memory in relation to changes in alcohol use after participation in an expectancy challenge program. *Experimental and Clinical Psychopharmacology* 2000;8:566–575. [PubMed: 11127428]
- Ellis DA, Zucker RA, Fitzgerald HE. The role of family influences in development and risk. *Alcohol Health and Research World* 1997;21:218–226. [PubMed: 15706772]

- Ennett ST, Foshee VA, Bauman KE, Hussong A, Cai L, Luz H, Reyes M, Faris R, Hipp J, DuRant R. The social ecology of adolescent alcohol misuse. *Child Development* 2008;79(6):1777–1791. [PubMed: 19037949]
- Fitzgerald, HE.; Davies, WH.; Zucker, RA. Growing up in an alcoholic family: Structuring pathways for risk aggregation and theory-driven intervention. In: McMahon, RJ.; Peters, RD., editors. *The effects of parental dysfunction on children*. New York, NY: Kluwer; 2002. p. 127-146.
- Fitzgerald, HE.; Zucker, RA.; Noll, RB. *The Beverage Opinion Questionnaire*. East Lansing, MI: Unpublished manuscript, Michigan State University; 1990.
- Fritz MS, MacKinnon DP. Required sample size to detect the mediated effect. *Psychological Science* 2007;18(3):233–239. [PubMed: 17444920]
- Goldman MS, Darkes J. Alcohol expectancy multiaxial assessment: A memory network-based approach. *Psychological Assessment* 2004;16:4–15. [PubMed: 15023088]
- Goldman, MS.; Del Boca, FK.; Darkes, J. Alcohol expectancy theory: The application of cognitive neuroscience. In: Leonard, KE.; Blane, HT., editors. *Psychological theories of drinking and alcoholism*. 2nd ed.. New York: Guilford; 1999. p. 203-246.
- Goldman MS, Greenbaum PE, Darkes J. A confirmatory test of hierarchical expectancy structure and predictive power: Discriminant validation of the Alcohol Expectancy Questionnaire. *Psychological Assessment* 1997;9:145–157.
- Graham JW, Olchowski AE, Gilreath TD. How many imputations are really needed? Some practical clarifications of multiple imputation theory. *Prevention Science* 2007;8:206–213. [PubMed: 17549635]
- Handley ED, Chassin L. Intergenerational transmission of alcohol expectancies in a high-risk sample. *Journal of Studies on Alcohol and Drugs* 2009;70:675–682. [PubMed: 19737491]
- Hawkins JD, Catalano RF, Miller JY. Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance abuse prevention. *Psychological Bulletin* 1992;112:64–105. [PubMed: 1529040]
- Henderson MJ, Goldman MS, Covert MD, Carnevala N. Covariance structure models of expectancy. *Journal of Studies on Alcohol* 1994;55:315–326. [PubMed: 8022180]
- Horton NJ, Kim E, Saitz R. A cautionary note regarding count models of alcohol consumption in randomized clinical trials. *BMC Medical Research Methodology* 2007;7:9. [PubMed: 17302984]
- Jacob T, Johnson S. Parenting influences on the development of alcohol abuse and dependence. *Alcohol Health and Research World* 1997;21:204–209. [PubMed: 15706770]
- Jacob T, Leonard KE. Alcoholic-spouse interaction as a function of alcoholism subtype and alcohol consumption interaction. *Journal of Abnormal Psychology* 1988;97:231–237. [PubMed: 3385076]
- Karazsia BT, van Dulmen MHM. Regression models for count data: Illustrations using longitudinal predictors of childhood injury. *Journal of Pediatric Psychology* 2008;33:1076–1084. [PubMed: 18522994]
- Kidorf M, Sherman MF, Johnson JG, Bigelow GE. Alcohol expectancies and changes in beer consumption of first-year college students. *Addictive Behaviors* 1995;20:225–231. [PubMed: 7484316]
- Killen JD, Hayward C, Wilson DM, Haydel KF, Robinson TN, Taylor CB, et al. Predicting onset of drinking in a community sample of adolescents: The role of expectancy and temperament. *Addictive Behaviors* 1996;21:473–480. [PubMed: 8830905]
- King KM, Chassin L. Mediating and moderated effects of adolescent behavioral undercontrol and parenting in the prediction of drug use disorders in emerging adulthood. *Psychology of Addictive Behaviors* 2004;18:239–249. [PubMed: 15482079]
- Kraemer HC, Stice E, Kazdin AE, Offord DR, Kupfer DJ. How do risk factors work together? Mediators, moderators, and independent, overlapping, and proxy risk factors. *American Journal of Psychiatry* 2001;158:848–856. [PubMed: 11384888]
- Kraus D, Smith GT, Ratner HH. Modifying alcohol-related expectancies in grade-school children. *Journal of Studies on Alcohol* 1994;55:535–542. [PubMed: 7990463]
- Lambert D. Zero-inflated Poisson regression, with an application to defects in manufacturing. *Technometrics* 1992;34:1–14.

- Leckman JF, Sholomskas D, Thompson WD, Belanger A, Weissman MM. Best estimate of lifetime psychiatric diagnosis: A methodological study. *Archives of General Psychiatry* 1982;39:879–883. [PubMed: 7103676]
- Lee HH, Cranford JA. A study of Korean adolescents. Does resilience moderate the associations between parental problem drinking and adolescents' internalizing and externalizing behaviors? *Drug and Alcohol Dependence* 2008;96:213–221. [PubMed: 18440164]
- Leigh BC, Stacy AW. Alcohol expectancies and drinking in different age groups. *Addiction* 2004;99:215–227. [PubMed: 14756714]
- Long, JS. Regression models for categorical and limited dependent variables. Thousand Oaks, CA: Sage; 1997.
- Mann LM, Chassin L, Sher KJ. Alcohol expectancies and the risk for alcoholism. *Journal of Consulting and Clinical Psychology* 1987;55(3):411–417. [PubMed: 3597957]
- Miller PM, Smith GT, Goldman MS. Emergence of alcohol expectancies in childhood: A possible critical period. *Journal of Studies on Alcohol* 1990;51:343–349. [PubMed: 2359308]
- Muthen, LK.; Muthen, BO. Mplus user's guide. 5th ed.. Los Angeles, CA: Muthen & Muthen; 2007.
- Newcomb MD, Chou CP, Bentler PM, Huba GJ. Cognitive motivations for drug use among adolescents: Longitudinal tests of gender differences and predictors of change in drug use. *Journal of Counseling Psychology* 1988;35:426–438.
- Ohannessian CM, Hesselbrock VM. Do alcohol expectancies moderate the relationship between parental alcoholism and adult drinking behaviors? *Addictive Behaviors* 2004;29:901–909. [PubMed: 15219334]
- Ohannessian CM, Hesselbrock VM, Kramer J, Bucholz KK, Schuckit MA, Kuperman S, Nurnberger JJ Jr. Parental substance use consequences and adolescent psychopathology. *Journal of Studies on Alcohol* 2004;65:725–730. [PubMed: 15700509]
- Ouellette JA, Gerrard M, Gibbons FX, Reis-Bergan M. Parents, peers, and prototypes: Antecedents of adolescent alcohol expectancies, alcohol consumption, and alcohol-related life problems in rural youth. *Psychology of Addictive Behaviors* 1999;13:183–197.
- Pastor AD, Evans SM. Alcohol outcome expectancies and risk for alcohol use problems in women with and without a family history of alcoholism. *Drug and Alcohol Dependence* 2003;70:201–214. [PubMed: 12732414]
- Petratis J, Flay BR, Miller TQ. Reviewing theories of adolescent substance use: Organizing pieces in the puzzle. *Psychological Bulletin* 1995;117:67–86. [PubMed: 7870864]
- Reese FL, Chassin L, Molina BSG. Alcohol expectancies in early adolescents: Predicting drinking behavior from alcohol expectancies and parental alcoholism. *Journal of Studies on Alcohol* 1994;55:276–284. [PubMed: 8022174]
- Richter L, Richter DM. Exposure to parental tobacco and alcohol use: Effects on children's health and development. *American Journal of Orthopsychiatry* 2001;71:182–203. [PubMed: 11347360]
- Robins LN, Helzer JE, Croughan J, Ratcliff KS. National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics, and validity. *Archives of General Psychiatry* 1981;38:381–389. [PubMed: 6260053]
- Rubin, DB. Multiple imputation for nonresponse in surveys. New York, NY: John Wiley & Sons, Inc; 1987.
- SAS Institute Inc. SAS/STAT 9.1 user's guide. Cary, NC: SAS Institute Inc; 2004.
- Schaefer, JL. Analysis of incomplete multivariate data. New York, NY: Chapman & Hall; 1997.
- Schafer JL, Graham JW. Missing data: Our view of the state of the art. *Psychological Methods* 2002;7(2):147–177. [PubMed: 12090408]
- Scheier LM. Etiologic studies of adolescent drug use: A compendium of data resources and their implications for prevention. *Journal of Primary Prevention* 2001;22:125–168.
- Scheier LM, Botvin GJ. Expectancies as mediators of the effects of social influences and alcohol knowledge on adolescent alcohol use: A prospective analysis. *Psychology of Addictive Behaviors* 1997;11:48–64.
- Schell TL, Martino SC, Ellickson PL, Collins RL, McCaffrey D. Measuring developmental changes in alcohol expectancies. *Psychology of Addictive Behaviors* 2005;19:217–220. [PubMed: 16011394]

- Selzer ML, Vinokur A, van Rooijen L. A self-administered Short Michigan Alcoholism Screening Test (SMAST). *Journal of Studies on Alcohol* 1975;36:117–126. [PubMed: 238068]
- Sher KJ, Gotham HJ. Pathological alcohol involvement: A developmental disorder of young adulthood. *Development and Psychopathology* 1999;11:933–956. [PubMed: 10624733]
- Sher KJ, Grekin ER, Williams NA. The development of alcohol use disorders. *Annual Review of Clinical Psychology* 2005;1:493–523.
- Sher KJ, Walitzer KS, Wood PK, Brent EE. Characteristics of children of alcoholics: Putative risk factors, substance use and abuse, and psychopathology. *Journal of Abnormal Psychology* 1991;100:427–448. [PubMed: 1757657]
- Sher KJ, Wood MD, Wood PK, Raskin G. Alcohol outcome expectancies and alcohol use: A latent variable cross-lagged panel study. *Journal of Abnormal Psychology* 1996;105:561–574. [PubMed: 8952189]
- Shrout PE, Bolger N. Mediation in experimental and nonexperimental studies: New procedures and recommendations. *Psychological Methods* 2002;7:422–445. [PubMed: 12530702]
- Sinharay S, Stern HS, Russell D. The use of multiple imputation for the analysis of missing data. *Psychological Methods* 2001;6:317–329. [PubMed: 11778675]
- Smith GT, Goldman MS, Greenbaum PE, Christiansen BA. Expectancy for social facilitation from drinking: The divergent paths of high-expectancy and low-expectancy adolescents. *Journal of Abnormal Psychology* 1995;104:32–40. [PubMed: 7897051]
- Spiegler DL. Children's attitudes toward alcohol. *Journal of Studies on Alcohol* 1983;44:545–552. [PubMed: 6645535]
- Stevens, J. *Applied multivariate statistics for the social sciences*. 3rd ed.. Mahwah, New Jersey: Erlbaum; 1996.
- White HR, Johnson V, Buyske S. Parental modeling and parenting behavior effects on offspring alcohol and cigarette use: A growth curve analysis. *Journal of Substance Abuse* 2000;12:287–310. [PubMed: 11367605]
- Wills TA, Sandy JM, Yaeger A, Shinar O. Family risk factors and adolescent substance use: Moderation effects for temperament dimensions. *Developmental Psychology* 2001;37:283–297. [PubMed: 11370906]
- Wills TA, Yaeger AM. Family factors and adolescent substance use: Models and mechanisms. *Current Directions in Psychological Science* 2003;12:222–226.
- Windle M. Effect of parental drinking on adolescents. *Alcohol Health and Research World* 1996;20:181–184.
- Windle M, Spear LP, Fuligni AJ, Angold A, Brown JD, Pine D, Smith GT, Giedd, J, Dahl RE. Transitions into underage and problem drinking: Developmental processes and mechanisms between 10 and 15 years of age. *Pediatrics* 2008;121 Suppl4:S273–S289. [PubMed: 18381494]
- Wood MD, Read JP, Mitchell RE, Brand NH. Do parents still matter? Parent and peer influences on alcohol involvement among recent high school graduates. *Psychology of Addictive Behaviors* 2004;18:19–30. [PubMed: 15008682]
- Yu J. The association between parental alcohol-related behaviors and children's drinking. *Drug and Alcohol Dependence* 2003;69:253–262. [PubMed: 12633911]
- Zhang L, Welte JW, Wieczorek WF. The influence of parental drinking and closeness on adolescent drinking. *Journal of Studies on Alcohol* 1999;60:245–251. [PubMed: 10091963]
- Zucker RA. Scaling the developmental momentum of alcoholic process via the lifetime alcohol problems score. *Alcohol and Alcoholism* 1991;26 Supplement No. 1:505–510. [PubMed: 1804130]
- Zucker, RA. Pathways to alcohol problems and alcoholism: A developmental account of the evidence for multiple alcoholisms and for contextual contributions to risk. In: Zucker, RA.; Howard, J.; Boyd, GM., editors. *The development of alcohol problems: Exploring the biopsychosocial matrix of risk*. Rockville, MD: U.S. Department of Health and Human Services; 1994. p. 255-289.(NIAAA Research Monograph No. 26)
- Zucker, RA. Alcohol use and the alcohol use disorders: A developmental-biopsychosocial systems formulation covering the life course. In: Cicchetti, D.; Cohen, DJ., editors. *Developmental psychopathology, Vol 3: Risk, disorder, and adaptation*. 2nd ed.. Hoboken, NJ: John Wiley & Sons, Inc; 2006. p. 620-656.

- Zucker RA, Donovan JE, Masten AS, Mattson ME, Moss HB. Early developmental processes and the continuity of risk for underage drinking and problem drinking. *Pediatrics* 2008;121:S252–S272. [PubMed: 18381493]
- Zucker, RA.; Fitzgerald, HE.; Moses, HD. Emergence of alcohol problems and the several alcoholisms: A developmental perspective on etiologic theory and life course trajectory. In: Cicchetti, D.; Cohen, DJ., editors. *Developmental psychopathology: Risk, disorder, and adaptation*. Vol. Vol. 2. New York: John Wiley & Sons; 1995. p. 677-711.
- Zucker, RA.; Fitzgerald, HE.; Refior, SK.; Puttler, LI.; Pallas, DM.; Ellis, DA. The clinical and social ecology of childhood for children of alcoholics: Description of a study and implications for a differentiated social policy. In: Fitzgerald, HE.; Lester, BM.; Zuckerman, BS., editors. *Children of addiction: Research, health, and public policy issues*. New York: RoutledgeFalmer Press; 2000. p. 109-141.
- Zucker RA, Kincaid SB, Fitzgerald HE, Bingham CR. Alcohol schema acquisition in preschoolers: Differences between children of alcoholics and children of nonalcoholics. *Alcoholism: Clinical and Experimental Research* 1995;19:1011–1017.

Table 1

Zero-Order Correlations between Study Variables

Variable	1.	2.	3.	4.	5.	6.	7.	8.
1. Maternal Lifetime ALC DX	--							
2. Paternal Lifetime ALC DX	.22**	--						
3. Maternal Drinking Days Per Month in Last 6 Months T1	.32**	.04	--					
4. Paternal Drinking Days Per Month in Last 6 Months T1	.11	.33**	.27**	--				
5. Adolescent Positive AEs at T2	.13	.12	-.06	-.16	--			
6. Adolescent Negative AEs at T2	-.14	.20*	-.11	-.10	.44**	--		
7. Drinking Days Per Month in Last 6 Months at T3	.25**	.17*	.23*	.11	.14	-.02	--	
8. Frequency of Drunkenness in Last 6 Months at T3	.13	.22**	.27*	.26**	.22*	.09	.56**	--
<i>Mean</i>	.33	.76	4.1	8.6	2.4	1.9	2.8	9.8
<i>SD</i>	.47	.43	5.6	8.5	3.6	1.4	3.8	20.4

Note. N = 148 male adolescent drinkers and their parents. ALC DX = DSM-IV lifetime alcoholism diagnosis coded 0 = No ALC DX and 1 = ALC DX. AEs = Alcohol expectancies.

*
p < .05.

**
p < .01.

Table 2

Zero-Inflated Poisson Regression Analysis of Longitudinal Predictors of Adolescents' Average Number of Drinking Days Per Month and Number of Times Intoxicated in Past 6 Months

	Average Drinking Days in Past 6 Months at T3		Number of Times Intoxicated in Past 6 Months at T3	
	Any drinking days	Number of drinking days	Any intoxication	Number of times intoxicated
	$\exp(\gamma)^1$	$\exp(\beta)^2$	$\exp(\gamma)^1$	$\exp(\beta)^2$
Predictors				
Age	2.05*	1.19	2.11*	1.19
T2 Negative expectancies	0.63	0.99	0.61*	0.99
T2 Positive expectancies	1.19	1.01	1.39*	1.07*
T1 Maternal AUD	0.63	1.61	0.48	0.94
T1 Maternal drinking ³	1.01	1.03*	1.07	1.03
T1 Paternal AUD	1.38	1.61	2.43	3.35*
T1 Paternal drinking ³	1.05	0.99	1.04	1.03

¹ Exponentiated coefficient from logistic regression component of ZIP model.

² Exponentiated coefficient from Poisson regression component of ZIP model.

³ Average drinking days per month in the past 6 months.

* $p < .05$.

** $p < .01$.