

Alcohol Expectancies as Potential Mediators of Parent Alcoholism Effects on the Development of Adolescent Heavy Drinking

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This study used latent growth curve modeling to examine adolescent alcohol expectancies as potential mediators of the effects of parent alcoholism on escalation in adolescent heavy drinking. Data were drawn from a 3-year longitudinal study of a community sample of children of alcoholics (COAs) and demographically matched controls. Parent alcoholism had a direct effect on adolescent heavy drinking. Compared to non-COAs, COAs started out at higher initial levels of heavy drinking and increased their heavy drinking at a steeper rate over the three waves of measurement. However, expectancies concerning negative alcohol effects (negative expectancies) and expectancies concerning positive alcohol effects (positive expectancies) were unrelated to growth in adolescents' heavy drinking. Thus, alcohol expectancies did not mediate parent alcoholism effects on trajectories of adolescent drinking.

There was some cross-sectional evidence that adolescents' positive expectancies mediated the effects of parent alcoholism on initial levels of adolescent heavy drinking. Children of alcoholic fathers reported higher levels of positive expectancies than did non-COAs, and positive expectancies predicted high initial levels of heavy drinking. Although weak adolescent negative expectancies were associated with high initial levels of alcohol use, they were unrelated to parent alcoholism. Implications of these findings for COA risk and escalation of heavy drinking in the adolescent years are discussed.

Children of alcoholics (COAs) are at elevated risk for problem alcohol use in adolescence (Chassin, Rogosch, & Barrera, 1991; Pandina & Johnson, 1990), and for alcohol abuse and dependence in adulthood (Windle & Searles, 1990). However, little is known about the mechanisms underlying this risk, because few studies have examined potential mediators of COA risk in the adolescent years (cf. Rogosch, Chassin, & Sher, 1990). Consequently, little is known about the early development of COA risk for alcohol use or abuse. Adolescence is a crucial developmental stage in which to study COA risk, because drinking is initiated during this period (Jessor & Jessor, 1977; Johnston, O'Malley, & Bachman, 1988), and because early onset of drinking increases the risk for alcohol problems (Hawkins, Catalano, & Miller, 1992).

Alcohol expectancies (a cognitive variable that describes beliefs about alcohol effects) may mediate the effects of many factors that have been previously linked to adolescent drinking, such as parenting behavior, peer influences, temperament and personality, emotional distress, and parental drinking (see Sher, 1991). All of these factors may shape adolescents' beliefs about the effects of alcohol consumption, which in turn, are the proximal influences on drinking behavior (Goldman, Brown, & Christiansen, 1987). Alcohol expectancies may also mediate the relation between parent alcoholism and adolescent alcohol use, and therefore, they may be an important factor in the early sequelae of COA risk. Although there has been some disagreement regarding the measurement and structure of alcohol expectancies (Brown, Goldman, Inn, & Anderson, 1980; Leigh, 1989a; Leigh & Stacy, 1993), several studies have suggested that it is useful to consider both expectancies about the positive effects of alcohol (or positive expectancies) and expectancies about the negative effects of alcohol (or negative expectancies; Collins, Lapp, Emmons, & Isacc, 1990; Fromme, Stroot, & Kaplan, 1993; Leigh & Stacy, 1993). Positive and negative expectancies have been shown to be empirically independent and differentially predictive of alcohol consumption (Leigh & Stacy, 1993).

Alcohol expectancies may be expected to mediate the effects of parent alcoholism on adolescent alcohol use for a number of reasons. For example,

COAs may form positive expectancies because of socialization provided by their alcoholic parents. Alternatively, COAs may form positive expectancies based on their own direct experience with alcohol, because COAs are thought to derive greater pharmacological benefit from alcohol consumption (see Sher, 1991, for a review). Thus, diverse underlying mechanisms might result in COAs forming positive expectancies that serve as a common final pathway to explain elevated alcohol use. Alcohol expectancies also represent an important area for research efforts because of their relevance for intervention. Expectancies are potentially modifiable, and therefore, they are viable targets for alcohol prevention programs (Darkes & Goldman, 1993). Because of their theoretical interest and potential importance for preventive intervention, this study investigated whether alcohol expectancies mediated the relation between parental alcoholism and the development of adolescent heavy drinking.

Few previous studies of alcohol expectancies have directly tested this mediational model. However, past research has addressed several preconditions that are necessary to establish mediation. Baron and Kenny (1986) discussed these preconditions. First, the independent variable (i.e., parent alcoholism) must predict the dependent variable (i.e., adolescent drinking). Second, the independent variable must predict the mediator (i.e., parent alcoholism must predict adolescents' expectancies). Third, the mediator must predict the dependent variable while controlling for the effects of the independent variable (i.e., adolescents' expectancies must predict their drinking while controlling for the effects of parental alcoholism). Finally, mediation is demonstrated when the effect of the independent variable on the dependent variable is substantially reduced or eliminated when the mediator is introduced into the model. Although previous research has studied these preconditions in isolation, few studies have addressed all of the conditions, and no studies have tested this mediational model using longitudinal data. This study fulfills these goals.

The first criterion for mediation requires that parent alcoholism relate to adolescent drinking. Here there has been relatively consistent empirical support (Chassin et al., 1991; Hawkins et al., 1992; Sher, Walitzer, Wood, & Brent, 1991). Although one study found this relation only for older adolescents (Pandina & Johnson, 1990), that study included adolescents with second degree alcoholic relatives rather than only COAs. Thus, existing evidence suggests that adolescent COAs are at elevated risk for alcohol use compared to their non-COA peers.

The second criterion requires that parent alcoholism be related to adolescents' alcohol expectancies. There are several theoretical reasons to expect this relation. Some theories suggest that COAs have different physiological responses to alcohol than do non-COAs. For example, COAs have

been shown to experience greater stress response, dampening effects of alcohol (Sher & Levenson, 1982), and alcohol intoxication reduces muscle tension more for COAs than for non-COAs (Pihl, Peterson, & Finn, 1990). If COAs experience more reinforcing effects from alcohol, they would be expected to develop more positive expectancies than would non-COAs. In addition, adolescent COAs may develop more positive expectancies because of parental socialization about alcohol effects. Indeed, empirical evidence suggests that adolescent COAs have stronger positive expectancies than do non-COAs (Brown, Creamer, & Stetson, 1987; Mann, Chassin, & Sher, 1987; Sher et al., 1991).

Theoretically, COAs and non-COAs might also be expected to differ in their negative expectancies. Some findings suggest that COAs experience greater tolerance of alcohol effects (Pihl et al., 1990; Schuckit, 1994), and thus COAs may experience less behavioral impairment after alcohol consumption than do non-COAs. If so, COAs may develop less negative expectancies about alcohol effects. Although two studies have failed to find differences between COAs and non-COAs in their expectancies of cognitive and behavioral deterioration after alcohol use (Brown et al., 1987; Mann et al., 1987), we included negative expectancies as a potential mediator of COA risk to more fully represent the domain of expectancies.

The third criterion for mediation requires that alcohol expectancies predict drinking when drinking is regressed on both parent alcoholism and alcohol expectancies. This has rarely been directly tested. An exception is Reese, Chassin, and Molina (1994), who examined the moderating effect of parent alcoholism on the relation between adolescents' positive alcohol expectancies and adolescents' alcohol involvement using two panels of data. The moderating effect of parent alcoholism was not supported. However, positive expectancies were found to prospectively predict alcohol-related impairment above and beyond the effects of parental alcoholism. Other studies have assessed the relation between expectancies and drinking without considering the effects of parent alcoholism. Previous cross-sectional research has shown that adolescents with stronger positive expectancies have higher levels of alcohol use (Brown et al., 1980; Rosenhow, 1983; Smith & Goldman, 1994) and this relation has also been demonstrated prospectively (Bauman, Fisher, Bryan, & Chenoweth, 1985; Christiansen, Smith, Roehling, & Goldman, 1989; Smith, Goldman, Greenbaum, & Christiansen, 1995; Stacy, Widaman, & Marlatt, 1990). Weak negative expectancies also predict high levels of alcohol use cross-sectionally (Christiansen et al., 1989; Leigh & Stacy, 1993; Rosenhow, 1983; Stacy et al., 1990), but not prospectively (Christiansen et al., 1989; Stacy et al., 1990).

The final criterion for mediation requires that the effect of parental alcoholism on adolescent drinking be substantially reduced (or eliminated)

when adolescent expectancies are entered into the model. This has rarely been tested. In a cross-sectional study of college students, Sher et al. (1991) found that parent alcoholism was associated with behavioral undercontrol (an aggregate of impulsivity, extraversion, antisociality, and sensation seeking), which predicted positive alcohol expectancies. Consistent with mediation, these expectancies in turn predicted heavy alcohol involvement. Another cross-sectional study of college students did not support alcohol expectancies as mediators of family history of alcohol abuse (Henderson, Goldman, Coover, & Carnevalla, 1994). However, in that study family history of alcohol abuse was operationalized as a continuous variable. This strategy may have obscured important effects of parent alcoholism because subclinical levels of problem drinking and clinically diagnosable alcohol abuse and dependence could not be disaggregated.

Not only have there been few tests of expectancies mediating the effects of parental alcoholism on adolescent drinking, but studies that have done so are limited by methodological problems. Studies typically fail to directly ascertain either parental alcohol abuse or dependence diagnoses or both, relying instead on offspring report. Moreover, existing mediational models have used cross-sectional data. Thus, it is difficult to discern the direction of effects between COAs' expectancies and their drinking behavior. This study addresses these limitations by directly ascertaining parental alcohol abuse and dependence diagnoses and by using a longitudinal design.

Finally, most longitudinal studies examining the effect of expectancies on adolescent drinking have typically used two points of measurement (e.g., Reese et al., 1994). Although this strategy may be useful in identifying the temporal order of expectancies and alcohol consumption, and the amount of change in drinking between two time points, it does not assess the developmental process of growth in drinking (Rogosa & Willett, 1985). Growth trajectories of drinking are important outcomes to study in the adolescent years, because alcohol consumption is expected to escalate over time during this developmental period (Johnston et al., 1988). Smith et al. (1995) examined growth trajectories of adolescent drinking and found that high levels of positive expectancies were related to steep rates of growth in drinking. The mediating role of expectancies on COA risk was not examined. Previously, we showed that parent alcoholism predicts rapid escalation in adolescent alcohol and drug use (Chassin & Barrera, 1993; Chassin, Curran, Hussong, & Colder, 1996; Curran, Stice, & Chassin, 1997). However, this effect of parent alcoholism remained substantial after considering several hypothesized mediators (e.g., parenting, environmental stress and negative affect, and affiliations with drug using peers). This suggests that other mediators are necessary to explain COA risk, and expectancies, because they are thought to be proximal influences, are potentially impor-

tant mediators to investigate. This study used latent growth curve analysis (LGC) to examine whether adolescents' alcohol expectancies mediated the effects of parent alcoholism on both initial level of alcohol use, and growth trajectories of alcohol use over three waves of measurement.

In summary, this study used a longitudinal design to assess the effects of parent alcoholism and alcohol expectancies on adolescents' growth trajectories of heavy alcohol use. We hypothesized that COAs would show higher initial levels of heavy drinking and greater escalation in heavy drinking over time than would non-COAs, and that alcoholic parents would transmit strong positive expectancies and weak negative expectancies to their adolescents. We predicted that these alcohol expectancies, in turn, would predict the initial level and the slope of the growth curve of adolescent heavy drinking.

METHODS

Participants

The total sample consisted of 454 adolescents and their parents. Of these adolescents, 246 had at least one biological and custodial alcoholic parent (COAs), and 208 were demographically matched controls with no biological or custodial alcoholic parents (non-COA controls). The sample consisted of 428 adolescents (197 female adolescents and 231 male adolescents) who had complete data concerning alcohol use and alcohol expectancies for all three waves of measurement. The adolescents ranged in age from 10.5 to 15.5 (mean age = 12.7) at Wave 1. Adolescents were categorized as either Hispanic (23.6%) or non-Hispanic Caucasian (76.4%), according to self-reports. Parents' level of education ranged from grade school to graduate school with a modal level of some college attendance. At Wave 1, 82.5% of the adolescents lived with both biological parents, 9.9% lived in a blended family, and 7.7% lived in single-parent families.

Recruitment Procedures

COA families were recruited using community telephone surveys ($N = 120$), questionnaires from a health maintenance organization ($N = 22$), and court Drinking Under the Influence (DUI) records ($N = 103$). One family was referred by a local Veteran's Administration hospital. Initial screening was performed by project staff, or by agency staff when records were confiden-

tial. COAs had to meet the following criteria: non-Hispanic Caucasian or Hispanic ethnicity, Arizona residency, ages 10.5 to 15.5, English speaking, and having no cognitive limitations that would preclude interview. Participants recruited from telephone surveys were interviewed on the phone to screen for demographic eligibility and possible alcoholism indicators (i.e., reports that either parent had been alcoholic, received help for drinking, attended an AA meeting, or been hospitalized for a drinking problem). In addition to telephone surveys, wellness questionnaires returned between 1986 and 1988 by newly enrolling members of a local HMO were screened for demographic eligibility and possible alcoholism indicators (i.e., more than 25 drinks per week, more than two alcohol-related negative consequences, or self-labeling as an alcoholic). Finally, records of convicted DUI offenders were screened for demographic eligibility and indicators of possible alcohol problems, including Blood Alcohol Contents (BACs) more than .14 or Michigan Alcohol Screening Test (MAST) scores more than 6 (Selzer, 1971). For COA families, direct interview data had to confirm that a biological and custodial parent met Diagnostic and Statistical Manual of Mental Disorders, 3rd Edition (DSM-III) criteria for lifetime diagnosis of alcohol abuse or dependence by the Diagnostic Interview Schedule (DIS, Version III; Robins, Helzer, Croughan, & Ratcliff, 1981) or by spouse report using Family History Research Diagnostic Criteria (FH-RDC; Andreasen, Endicott, Spitzer, & Winokur, 1977) if a parent could not be interviewed.

Demographically matched control families were recruited through telephone interviews. When a COA family was interviewed, reverse directories were used to locate families in the same neighborhood. Control families were matched to COA families on child's age within 1 year, family structure, and ethnicity of the alcoholic parent. Families were also matched on socioeconomic status using property value codes in reverse directories or parent's report of income if no property code was available. Direct interview verified that neither parent met DSM-III criteria for alcohol abuse or dependence. The recruitment procedures are presented in greater detail elsewhere (Chassin, Barrera, Bech, & Kossak-Fuller, 1992).

Recruitment biases due to selective contact with participants and participant refusals have been discussed elsewhere (Chassin et al., 1991, 1992). Analyses that examined participation bias revealed that the sample was unbiased with respect to alcoholism indicators that were available in archival records (e.g., blood-alcohol level at time of arrest, MAST scores). However, individuals who refused to participate were more likely to be Hispanic and to be married at the time of arrest. Although the magnitude of the bias was small and unrelated to archival indicators of alcoholism, some caution should be used in generalization.

Procedure

Data were collected in three annual computer-assisted interviews with the adolescents and their parents. Confidentiality was assured with a Department of Health and Human Services Certificate of Confidentiality. In support of the validity of the self-reports, 95.2% of the court participants reported their DUI arrests in the interviews. Interviewers were blind to group membership (COA vs. non-COA), as well as to the hypotheses of the study. Interviews lasted 1 to 2 hr and families were paid \$50 for their participation.

Measures

Demographics. Adolescents' age, gender (0 = female adolescents, 1 = male adolescents), ethnicity (0 = non-Hispanic Caucasian, 1 = Hispanic), and level of parent education were used as demographic variables. Mothers and fathers reported their level of education using a 7-point scale ranging from 1 (*grade school*) to 7 (*graduate school*), and these variables were averaged to form a parental education variable.

Parental alcoholism. Maternal and paternal DSM-III lifetime diagnosis of alcohol abuse or dependence was assessed using a computerized version of the DIS, Version III (Robins et al., 1981) or using the FH-RDC (Andreasen et al., 1977). Of the parents in this analysis, 44 of the biological mothers and 206 of the biological fathers met diagnostic criteria. Parent alcoholism was considered a dichotomous variable (0 = non-alcoholic and 1 = alcoholic).

Alcohol expectancies. Alcohol expectancies were measured using items from Mann et al.'s (1987) adaptation of the Alcohol Expectancies Questionnaire (Adolescent form, Christiansen, Goldman, & Inn, 1982). The response scale for all items was expanded to a 5-point scale ranging from 1 (*strongly disagree*) to 5 (*strongly agree*). Positive expectancies included: social enhancement effects (five items, e.g., "Drinking alcohol helps me celebrate social occasions"), personal power expectancies (seven items, e.g., "Drinking alcohol makes me feel more powerful than others"), tension reduction expectancies (seven items, e.g., "Drinking alcohol helps me relax"), and enhanced cognitive-motor functioning expectancies (five items, e.g., "I can do things better after drinking alcohol"). One social enhancement expectancy item was dropped from this analysis because of a low item total correlation for the subscale ($r = .12$). Cronbach's alphas for the subscales ranged from .75 to .91 across the three waves. Expectancies about the

negative effects of alcohol were measured using four items from Mann et al. (1987) reflecting impaired cognitive and behavioral functioning (e.g., "I do stupid, strange, or silly things after I have been drinking"). Cronbach's alpha for this scale ranged from .74 to .75 across the three waves.

Adolescent heavy alcohol use. Because expectancies have shown a particularly strong relation to quantity-type measures of alcohol use (cf. Leigh, 1989b), this study investigated adolescent heavy drinking as the outcome measure. Previous research has operationalized adolescent heavy drinking using survey questions about the quantity of consumption of alcohol (five or more drinks in a row) and questions about the frequency of intoxication (e.g., Jessor & Jessor, 1977; Johnston et al., 1988). Consistent with this research, we assessed heavy drinking using adolescents' self-report of the number of times in the last year they consumed five or more drinks (of beer, wine, wine cooler, or hard liquor) and the number of times they had been drunk. These two items were averaged for analysis. Cronbach's alpha for this measure was .92 for Wave 1, .92 for Wave 2, and .88 for Wave 3. The percentage of adolescents who reported heavy drinking at each wave stratified by age and gender is presented in Table 1. Consistent with epidemiological data (Johnston et al., 1988), heavy drinking was more prevalent for middle adolescents than for early adolescents, and prevalence rates increased from Wave 1 to Wave 3. For early male adolescents, the prevalence of heavy drinking decreased from Wave 1 to Wave 2. The low prevalence rates for early adolescents and the small subsample size suggest that this small decrease may be attributed to sporadic experimentation among a few boys.¹

RESULTS

Our data analytic strategy followed several steps. First, the factor structure of alcohol expectancies was examined. Second, change over time in expectancies and heavy drinking was tested to establish growth patterns in these variables. Finally, our mediational hypothesis was examined by testing the direct effects of parent alcoholism on growth in adolescent drinking,

¹The threshold for heavy drinking may differ with age. We reestimated all of the LGC models using adolescents' self-reports of their typical quantity of consumption of alcohol (number of beers, glasses of wine, wine coolers, drinks of hard liquor). There were no substantive differences in the models when heavy drinking and quantity of consumption outcomes were examined, and therefore, age differences in the threshold for "heavy" drinking did not seem to affect the findings.

TABLE 1
Percentage of Heavy Drinkers Stratified by Age and Gender

	Females (%)	Males (%)
Early adolescents ^a		
Wave 1	2.5	4.9
Wave 2	6.2	2.9
Wave 3	18.7	10.8
Middle adolescents ^b		
Wave 1	24.8	23.3
Wave 2	29.9	37.2
Wave 3	33.3	45.0

Note. Adolescents were considered heavy drinkers if, on at least one occasion in the past year, they drank five or more drinks *or* got drunk.

^a10.5 to 12.5 years old at Wave 1; females: *N* = 80, males: *N* = 102. ^b12.6 to 15.5 years old at Wave 1; females: *N* = 117, males: *N* = 129.

and then testing whether these effects were mediated by adolescent alcohol expectancies.

Confirmatory Factor Analyses of Expectancies

We first tested whether a 2-factor model of alcohol expectancies fit the data at Wave 1. This model was estimated using LISREL VII (Joreskog & Sorbom, 1989) based on the variance/covariance matrix. The negative and positive expectancies factors were indicated by four items and four subscale scores, respectively. Each indicator was constrained to load on only one factor, and the latent factors were freely correlated. Fit indices suggested that this factor model adequately fit the data, Comparative Fit Index (CFI, Bentler, 1990) = .97, and Tucker-Lewis (TLI, Tucker & Lewis, 1973) = .96, but the model chi-square, $\chi^2(19, N = 428) = 58.19, p < .001$, suggested that the ratio of model chi-square to model degrees of freedom was not ideal (3:1). One correlated error was freed to ensure proper specification of the factor model, and this adjusted model produced an adequate fit of the data, $\chi^2(18, N = 428) = 41.17, p = .001$, CFI = .98, and TLI = .97. All indicators loaded significantly ($p < .05$) on their respective factors, and the factors were modestly correlated ($r = -.15, p < .10$).

After establishing the factor structure for Wave 1, it was important to demonstrate that this factor structure also fit the data from Waves 2 and 3. This was tested by simultaneously specifying the factor structure developed from Wave 1 for Wave 1, 2, and 3 data. We specified a six-factor measurement model that included a positive and negative expectancies factor from each wave, and hierarchically constrained parameters to be

invariant over time (see Bollen, 1989). Measurement error variances were auto-correlated and the latent factors were freed to correlate. Hierarchical model comparisons (chi-square change) showed that the factor loadings and factor correlation were equivalent for Waves 1, 2, and 3 of data.² The final model with equivalent factor loadings and factor correlation fit the data well, $\chi^2(228, N = 428) = 398.34, p < .001, CFI = .96, TLI = .97$. These findings suggest that the underlying factor structure was similar for each wave of data collection. Accordingly, means were calculated to form positive and negative expectancies scale scores for each wave of measurement to be used in subsequent analyses.

Descriptive statistics and correlations for expectancies, heavy drinking, demographic, and parent alcoholism variables are presented in Table 2. The correlations suggested that expectancies and heavy drinking were moderately stable across time, and that the correlations between heavy drinking and expectancies were similar at each wave of measurement. Skewness and kurtosis suggested that positive and negative expectancies were approximately normally distributed, and that the heavy drinking variables were nonnormally distributed. Later we address the nonnormal distribution of heavy drinking variables (see footnote 7).

Testing for Change in Heavy Drinking and Alcohol Expectancies

Latent growth curve analysis. Our first step in LGC model building was to test the shape of the growth curves (e.g., no growth, linear growth, nonlinear growth) for expectancies and heavy drinking. Specifically, a one-factor, three-indicator FOCUS model (Factor of Curves; McArdle, 1988) was used for each construct of interest using EQS (version 3.0, Bentler, 1989) based upon both the sample covariance matrix and sample means.

Three separate FOCUS models were estimated, one for positive expectancies, one for negative expectancies, and one for adolescent heavy drinking. First, a "no growth" model was estimated by constraining all three factor loadings of the repeated measures equal to 1 while freely estimating the mean and variance of the latent factor. All model chi-square test statistics were significant (all p values $< .05$), with chi-square values ranging from 18.52 to 143.74, all with $df = 4$ and $N = 428$. Thus, the no growth model fit the data poorly, indicating that the three constructs significantly changed over the three waves of measurement.

²Measurement error variances/covariances significantly varied across time. However, the lack of invariance of the error structure is of little practical significance because it does not substantially influence the interpretation of the factors (Bollen, 1989).

TABLE 2
Descriptive Statistics and Zero-Order Correlations for All Predictor and Criterion Variables

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Wave 1 heavy drinking	1.00														
2. Wave 2 heavy drinking	.71	1.00													
3. Wave 3 heavy drinking	.51	.70	1.00												
4. Wave 1 positive expectancies	.33	.27	.20	1.00											
5. Wave 2 positive expectancies	.30	.36	.28	.51	1.00										
6. Wave 3 positive expectancies	.20	.28	.33	.20	.28	1.00									
7. Wave 1 negative expectancies	-.13	-.10	-.12	-.01	-.10	-.05	1.00								
8. Wave 2 negative expectancies	-.15	-.17	-.17	-.11	.04	-.01	.27	1.00							
9. Wave 3 negative expectancies	-.08	-.08	-.11	-.04	.02	.12	.24	.35	1.00						
10. Age	.33	.33	.27	.25	.20	.23	.02	-.16	-.12	1.00					
11. Gender	.03	.04	.08	-.05	-.01	.03	.04	-.02	.08	-.07	1.00				
12. Parent education	-.02	-.03	-.04	.03	-.02	-.01	.05	.11	.09	-.02	.06	1.00			
13. Ethnicity	.06	.02	-.01	.10	.11	.07	.01	-.09	-.10	.06	-.04	-.32	1.00		
14. Maternal alcoholism	.09	.13	.13	.06	.04	.03	-.04	-.02	-.04	.01	-.09	-.04	-.10	1.00	
15. Paternal alcoholism	.15	.18	.20	.09	.13	.08	-.04	-.08	-.08	-.06	.02	-.21	.13	.03	1.00
M	.24	.37	.55	1.82	1.88	2.05	3.83	3.76	3.61	12.71	.54	4.32	.24	.11	.46
SD	.76	.98	1.18	.72	.76	.74	1.01	.98	.95	1.47	.50	1.38	.43	.32	.50
Skewness	4.05	3.36	2.64	.66	.75	.22	-.88	-.75	-.59	-.11	-.16	-.16	1.25	2.43	.14

Note. Absolute values greater than .10 are significant at $p < .05$. Absolute values greater than .15 are significant at $p < .01$. Gender, ethnicity, and maternal and paternal alcoholism were dichotomous variables (coded 0 or 1). Their means represent the percentage of male adolescents, of Hispanic adolescents, of adolescents with an alcoholic mother, and of adolescents with an alcoholic father, respectively.

A series of nested models was then estimated freeing various combinations of factor loadings to determine the shape of the growth curves. The no growth model served as a comparison model. Results suggested that adolescent heavy drinking increased linearly over the three waves of measurement. Positive expectancies showed no growth from Wave 1 to Wave 2, but a significant increase from Wave 2 to Wave 3. Negative expectancies showed no growth from Wave 1 to Wave 2, but a significant decrease from Wave 2 to Wave 3. Because positive and negative expectancies only changed from Wave 2 to Wave 3, individual growth curves and individual differences in growth could not be modeled (see Rogosa, 1988). Accordingly, we used the Wave 1 measures of positive and negative expectancies in subsequent models, because these measures best characterized alcohol expectancies over the three waves of data, and because these measures allowed for prospective prediction of adolescent heavy drinking.^{3,4}

The Effects of Parent Alcoholism on Adolescent Heavy Drinking

Before examining the mediational effect of alcohol expectancies on COA risk, it was necessary to show that parent alcoholism had a direct effect on adolescent heavy drinking. An LGC model was estimated to assess the direct effects of parent alcoholism on both initial levels of heavy drinking (the intercept of the growth curve) and the slope of the growth curve over time (see Figure 1). A latent factor representing the intercept of the growth curve was defined by setting all three of the factor loadings from Wave 1,

³The observed pattern of change in alcohol expectancies may suggest that expectancies did not influence heavy drinking until Waves 2 and 3. Thus, by examining only Wave 1 expectancies in our LGC models, prospective relations between heavy drinking and expectancies may have been obscured. However, a prospective regression model showed that Wave 2 positive and negative alcohol expectancies did not predict Wave 3 heavy drinking. Thus, examining Wave 1 expectancies in our LGC models did not seem to obscure important longitudinal relations.

⁴Although the group growth curve for heavy drinking was positive and linear, the shape of the growth trajectories of certain subpopulations may have deviated significantly from this pattern, and thus, inappropriately influenced the group level parameters. Visual inspection of individual growth curves showed no apparent deviation for growth patterns of positive and negative expectancies. However, 27 cases were determined to be outliers with respect to growth in heavy drinking. Our FOCUS models, as well as our subsequent LGC models were estimated with ($N = 428$) and without the divergent cases ($N = 401$) to determine the influence of these aberrant growth patterns. No substantive differences were found, indicating that our results were robust to heterogeneity of growth patterns. Nonetheless, because heterogeneity of growth is thought to inappropriately influence parameter estimates, results without the outlying cases ($N = 401$) are presented.

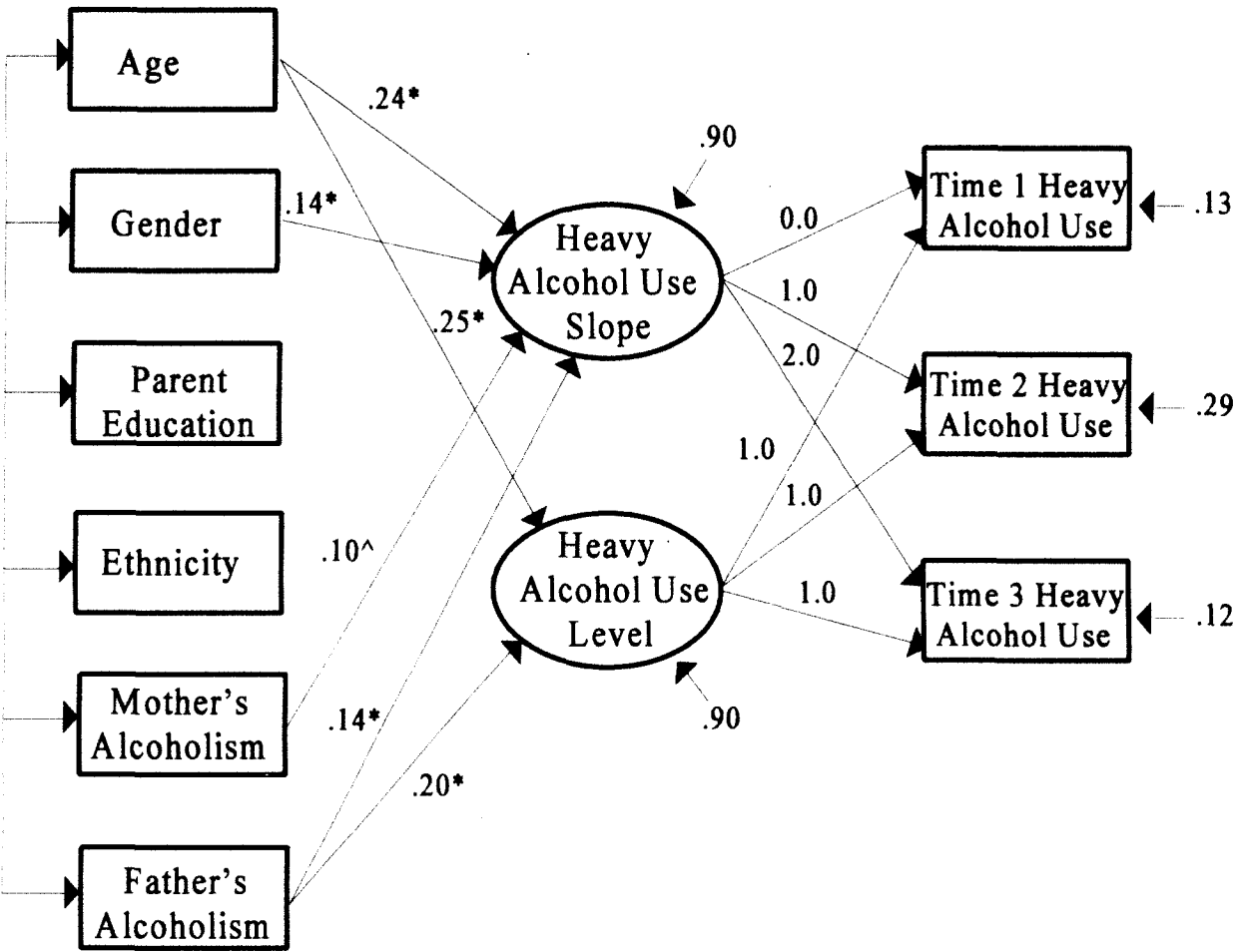


FIGURE 1 Direct effects model of heavy alcohol use. Standardized parameters are presented. Only significant paths are shown. ^ $p < .10$ and * $p < .05$.

2, and 3 measures of heavy drinking to 1.0. A second latent factor representing the shape growth curve (linear growth) was defined by setting the first factor loading to 0 (thus, not allowing the Wave 1 measure to load on this factor), fixing the second loading to 1.0, and fixing the third loading to 2.0. We regressed the heavy drinking slope and intercept factors on demographic variables (age, gender, ethnicity, and parent education) and parent alcoholism diagnoses (see Figure 1). The structural paths from maternal and paternal alcoholism diagnoses to the latent intercept and slope factors were freely estimated. To control for the effects of demographics on heavy drinking, structural paths from the demographic variables to the heavy drinking intercept and slope latent factors were iteratively freed if the path significantly reduced the model chi-square (as indicated by a LaGrange multiplier ≥ 6.6 , $p < .01$). Using this procedure, three paths were freed.⁵ The final model

⁵There were no substantive changes in the effects of parent alcoholism after freeing paths from demographic variables.

fit the data well, $\chi^2(13, N = 401) = 13.84, p = .385, CFI = .99, TLI = .99$, and is presented in Figure 1.

As shown in Figure 1, age was positively related to the heavy drinking intercept and slope factors, indicating that older adolescents reported higher initial levels of heavy drinking and increased their heavy drinking at a significantly steeper rate than did younger adolescents. A significant relation was also found between gender and the heavy drinking slope factor, suggesting that boys increased their heavy drinking at a significantly steeper rate than did girls. Both maternal and paternal alcoholism positively predicted the heavy drinking slope factor, and paternal alcoholism positively predicted the heavy drinking intercept factor. This suggests that offspring of male alcoholics reported higher initial levels of heavy drinking at Wave 1 than did non-COAs, and that children of maternal and paternal alcoholics increased their heavy drinking at a significantly steeper rate of growth compared to non-COAs. In sum, these findings suggest a direct positive effect of parent alcoholism diagnosis on initial levels of heavy drinking and on the rate of increase of heavy drinking over time.

The Mediational Effect of Alcohol Expectancies on COA Risk

After establishing a direct effect of parent alcoholism on adolescent drinking, we tested the relation between parent alcoholism and adolescent alcohol expectancies, and the relation between adolescent alcohol expectancies and adolescent heavy drinking. An LGC model was specified by adding Wave 1 positive and negative expectancies variables to the model (see Figure 2). All structural paths from maternal and paternal alcoholism diagnoses to expectancies and to the latent heavy drinking slope and intercept factors were freed. Then, to control for the effects of demographic variables on heavy drinking and expectancies, paths from the demographic variables to expectancies and to the latent heavy drinking slope and intercept factors were iteratively freed as previously described. Using this iterative procedure five paths from demographic variables were freed.⁶ The final model fit the data well, $\chi^2(22, N = 401) = 21.98, p = .461, CFI = .99, TLI = .99$, and is presented in Figure 2.

⁶There were no substantive changes in the effects of parent alcoholism on adolescent alcohol expectancies and heavy drinking and the effects of alcohol expectancies on heavy drinking after freeing paths from demographic variables.

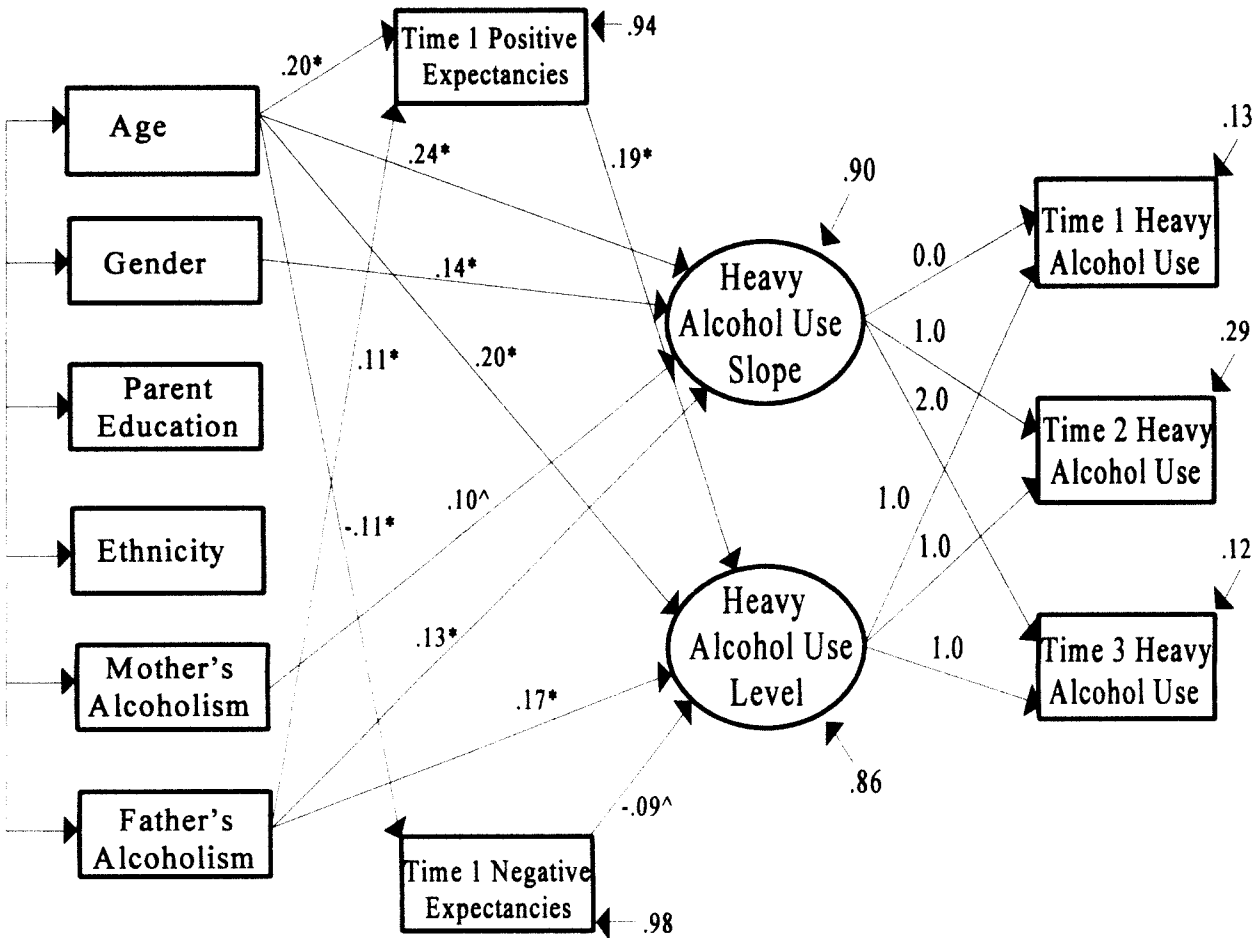


FIGURE 2 Mediation model of heavy alcohol use with Wave 1 expectancies. Standardized parameters are presented and only significant paths are shown. ^ $p < .10$ and * $p < .05$.

As shown in Figure 2, significant direct paths from demographic variables and parental alcoholism to the slope and intercept alcohol use factors remained as described in our direct effects model discussed previously (and shown in Figure 1). Regarding expectancies, age was positively associated with positive expectancies and negatively associated with negative expectancies, suggesting that older adolescents reported higher levels of positive expectancies and lower levels of negative expectancies at Wave 1 than did younger adolescents.

There was little support for the mediational effect of expectancies on COA risk for heavy drinking. As predicted, father's alcoholism was significantly associated with heightened positive expectancies that were associated with higher initial levels of heavy drinking. However, there was no significant relation between maternal alcoholism and positive expectancies or between either maternal or paternal alcoholism and negative expectancies. Moreover, although high levels of positive expectancies and low levels of negative expectancies

were significantly associated with higher initial levels of heavy drinking, initial expectancies did not predict growth over time in adolescent heavy drinking.⁷

These findings suggest that only positive expectancies met the first three criteria for mediation as discussed by Baron and Kenny (1988), and then only for the cross-sectional prediction of heavy drinking. The specific indirect effect from paternal alcoholism to initial levels of adolescent heavy drinking as mediated through positive expectancies was significant, $t(400) = 1.90, p = .056$, using Sobell's method (Sobell, 1989). However, with regard to Baron and Kenny's (1986) fourth criteria for mediation, there was a small change in the direct path from paternal alcoholism to initial levels of drinking when expectancies were included in the model (change in standardized path coefficient = .03), and the effect of father's alcoholism was not eliminated when positive expectancies were introduced into the model. Taken together, these findings suggest that positive expectancies can be considered a partial mediator of the effects of paternal alcoholism on initial levels of adolescent heavy drinking.

Gender Differences in the Mediation Model

Because some studies have found gender differences in associations between alcohol expectancies and drinking (Newcomb, Chou, Bentler, & Huba, 1988; Windle & Barnes, 1988), we tested for differences in the mediational model across gender. The sample was split by gender and the mediational model was respecified.⁸ All parameters were initially constrained to be invariant. LaGrange multipliers were used to identify parameters that significantly varied across gender ($p < .05$), and these parameters were iteratively freed. Three parameters were found to significantly vary across gender. More variation in escalation and initial levels of heavy drinking was found for boys than for girls, and the path from age to negative expectancies was significant for girls, $t(180) = -3.20, p < .01$, but not for boys,

⁷The heavy drinking variables in this study were significantly skewed (see Table 1). To examine the impact of nonnormality on these findings, our mediational model was reestimated using the Satorra-Bentler method of estimation, an estimation method that is more robust to nonnormality than maximum likelihood (Satorra & Bentler, 1988). The latent intercept and slope factors were replaced with manifest variables that were calculated using the regression method (Loehlin, 1992). There were no substantive differences between this model and our original LGC model, suggesting that the nonnormal distribution of our heavy drinking variables did not bias our findings.

⁸Because of sample size constraints, a full series of cross-group comparisons was not feasible with our LGC models. To compare our findings across gender, the latent intercept and slope factors were replaced with the manifest variables in the mediational model (see footnote 7).

$t(219) = -.21, p < .60$. Older girls reported lower levels of negative expectancies than did younger girls. There were no differences in links between parent alcoholism and expectancies, and between expectancies and the level and slope factors. Thus, there was no evidence of gender differences in our mediational findings.

Post Hoc Analyses

The effects of recent parent alcohol-related impairment. The weak relations between parent alcoholism and expectancies might be due to our use of lifetime diagnosis that collapses recent and "recovered" alcoholism. Stronger effects on expectancies might be revealed by considering parents' recent alcohol-related impairment, and the potential interactive effects between alcoholism diagnosis and recent alcohol-related impairment. To test this possibility we performed a series of 2 (parent alcoholism diagnosis) \times 2 (recent parent alcohol-related impairment) \times 2 (adolescents' gender) analyses of covariance (ANCOVAs) with age as a covariate and heavy drinking as the dependent variable. Parents were considered to show recent impairment if they reported experiencing at least one social consequence or dependency symptom at Wave 1 (from a list of 26 items) within the last year. For noninterviewed parents ($N = 107$), spouse report at Wave 2 was used to determine impairment. ANCOVAs were performed separately for mothers and fathers and separately for positive and negative expectancies as the dependent variables. Small cell sizes precluded a test of the three-way interactions, but all possible two-way interactions were included in the models. For negative expectancies, no effects of parent alcohol-related impairment or alcoholism diagnosis were found.

For positive expectancies, mothers' alcohol-related impairment had a marginal effect, $F(1, 393) = 2.65, p = .10$, such that stronger positive expectancies were found for adolescents whose mothers reported recent alcohol-related impairment. For fathers, there was a significant gender by impairment interaction, $F(1, 393) = 5.44, p = .02$. For boys, fathers' recent impairment was associated with high positive expectancies (adjusted M s = 1.73 and 1.94, and SD s = .66 and .79, respectively), $t(218) = 1.86, p < .06$. For girls, fathers' recent impairment was associated with less positive expectancies (adjusted M s = 1.90 and 1.66, and SD s = .73 and .71, respectively), $t(179) = 1.83, p < .07$. Also for fathers, there was a significant alcoholism diagnosis by gender interaction, $F(1, 393) = 3.52, p = .06$. For both boys and girls, paternal alcoholism was associated with higher positive expectancies, but this effect was stronger for girls than for boys (for girls adjusted M s = 1.85 and 1.61, and SD s = .66 and .73, respectively; for boys adjusted M s = 1.94 and 1.82, and SD s = .67 and .75, respectively).

In summary, considering recent alcohol-related impairment did not reveal evidence of links between parent alcoholism and negative expectancies. However, unlike diagnoses in our LGC model, mothers' alcohol-related impairment was linked to positive expectancies and fathers' alcohol-related impairment was differentially linked to positive expectancies for boys and girls.

The effects of alcohol expectancies on heavy drinking onset. The lack of effects of alcohol expectancies on growth trajectories of heavy drinking were surprising, because previous studies have supported prospective relations between alcohol expectancies and adolescent alcohol use using samples of similar ages (Bauman et al., 1985; Smith et al., 1995). However, these previous studies examined alcohol use onset.⁹ To examine the prospective effect of alcohol expectancies on the onset of heavy drinking, we performed a logistic regression. For abstainers at Wave 1, we calculated a dichotomous outcome (0 = abstained from heavy drinking at Wave 2 or 1 = initiated heavy drinking at Wave 2), and regressed this outcome on Wave 1 positive and negative expectancies. Negative expectancies were unrelated to onset of heavy drinking. However, positive expectancies marginally predicted onset, $\beta = .38$, Wald $\chi^2(1, N = 348) = 3.03$, $p < .08$, such that high positive expectancies increased the likelihood of transitioning into heavy drinking. Taken together, our LGC models and logistic regressions suggest that positive expectancies are prospectively related to onset of adolescent heavy drinking, but unrelated to growth trajectories of heavy drinking. Negative expectancies seem to be unrelated to both onset of heavy drinking and growth in heavy drinking.

DISCUSSION

The main goal of this study was to examine the role of adolescent alcohol expectancies as a potential mediator of the relation between parent alcoholism and adolescent heavy drinking using a longitudinal design. Our longitudinal data and LGC analyses permitted us to examine growth in adoles-

⁹Expectancies for social facilitation have prospectively predicted alcohol use in some studies (Christiansen et al., 1989; Smith et al., 1995), suggesting that the current aggregate positive expectancies variable may have obscured prospective relations. In our mediational model, we replaced positive expectancies with a social facilitation variable similar to that used in previous studies. Our findings did not change. Expectancies for social facilitation predicted initial levels of drinking, but did not predict the slope of the growth curve. Thus, lack of prospective effects in this study is not attributable to using an aggregate positive expectancies variable.

cent heavy drinking and the influence of initial levels of positive and negative expectancies on this developmental process.

Relations Between Parent Alcoholism and Adolescent Heavy Drinking

As expected developmentally, adolescents showed significant growth over time in their heavy drinking. This is consistent with epidemiological studies that have shown adolescence to be a period in which alcohol use escalates (Johnston et al., 1988). Critical developmental tasks of adolescence include establishing autonomy, assuming adult roles, and identity formation, and these tasks are often achieved through exploring a wide variety of behaviors (Steinberg, 1989). As suggested by Baumrind (1991), growth in alcohol use may represent such exploration.

Our findings also suggested that both maternal and paternal alcoholism predicted steeper growth rates. Adolescent COAs escalated in their heavy drinking more rapidly than did non-COAs. Although previous research has shown increased risk for alcohol use and abuse among COAs, few studies have examined the early development of COA risk over time. Rapid escalation of heavy drinking among COAs may reflect an early manifestation of problem drinking and alcohol abuse. If so, then it is important for future research to distinguish between factors that influence normative growth trajectories of alcohol use and those related to rapid escalation. Other analyses from this longitudinal project have shown that COAs also rapidly escalate in their substance use (an aggregate of alcohol and drug use, Chassin et al., 1996), suggesting that COA risk is associated with rapid escalation in alcohol and drug use, as well as heavy drinking.

The Mediational Role of Adolescent Alcohol Expectancies

Our findings did not support a relation between alcohol expectancies at Wave 1 and growth in heavy drinking over time. Thus, alcohol expectancies were not prospective predictors of heavy drinking, and did not mediate parent alcoholism effects on adolescent growth trajectories of heavy drinking. The lack of prospective effects of alcohol expectancies was surprising because previous research has supported such effects using samples of similar ages (Bauman et al., 1985; Christiansen et al., 1989; Smith et al., 1995). However, previous studies either had methodological problems (e.g., did not control for initial levels of drinking when examining prospective effects of positive expectancies on alcohol use at one follow-up point (e.g., Christiansen et al., 1989), or they examined alcohol use onset (e.g., Smith et al., 1995). Indeed, our post hoc analysis suggested that strong positive expect-

tancies prospectively predicted heavy drinking onset, whereas negative expectancies were unrelated to onset. Thus, positive expectancies may be prospectively related to onset of adolescent heavy drinking, but unrelated to growth trajectories of heavy drinking. Negative expectancies seem to be unrelated to both outcomes.

It is possible that after drinking has been initiated, the true relation between alcohol expectancies and heavy drinking is a reciprocal one (Bauman et al., 1985; Goldman et al., 1987; Leigh, 1989b; Smith et al., 1995). If so, it would have been important to model growth in alcohol expectancies, and examine how this growth relates to adolescent drinking. For example, growth in expectancies might be significantly correlated with growth in heavy drinking. Although a reciprocal model is theoretically appealing, the pattern of change shown for both alcohol expectancies and drinking in the these data is not consistent with such a model, because growth in expectancies seems to have lagged behind that of heavy drinking.

Social psychological research on attitude-behavior relations suggests another explanation for the lack of prediction of growth in heavy drinking from alcohol expectancies. Self-monitoring represents individual differences in how much a person uses social comparison information to guide their behavior in a social situation (Snyder, 1979). Individuals high in self-monitoring are thought to be concerned with socially appropriate self-presentation and rely on social comparison information to guide their behavior, whereas individuals low in self-monitoring are thought to rely on internal cues to guide their behavior and to devalue the impression they make on others (Snyder, 1974). Previous research has indicated that the attitude-behavior relation is stronger for individuals low in self-monitoring than for individuals high in self-monitoring (Kardes, Sanbonmatsu, Voss, & Fazio, 1986). It has been argued that self-monitoring changes developmentally with a peak in adolescence (Lapsley, Jackson, Rice, & Shadid, 1988). If early adolescents are high in self-monitoring, they may rely on situational cues, rather than their own attitudes, to guide drinking behavior. Thus, developmental differences in self-monitoring may explain the relatively weak relations between alcohol expectancies (which can be considered attitudes about drinking) and growth in adolescent heavy drinking.

Regarding links between parent alcoholism and adolescent alcohol expectancies, our findings showed that adolescents with alcoholic fathers expected more positive benefits from alcohol consumption. Moreover, strong expectancies for the positive benefits of alcohol use were associated with high levels of Wave 1 heavy drinking. Thus, there was cross-sectional support for positive expectancies mediating the relation between parent alcoholism and adolescent heavy drinking. Previous research has supported a similar mediational pathway (Sher et al., 1991), and other studies

have shown that male alcoholics transmit strong positive expectancies to their offspring (Brown et al., 1987; Mann et al., 1987). This transmission may occur through multiple mechanisms. For example, COAs have been reported to derive greater stress response dampening effects of alcohol than do non-COAs (Sher & Levenson, 1982), and this enhanced reinforcement may be reflected in more positive alcohol expectancies. Environmental mechanisms such as parent modeling of positive alcohol effects could also account for these findings.

Post hoc analyses suggested that mothers' alcohol-related impairment was linked to positive expectancies and fathers' alcohol-related impairment was differentially linked to positive expectancies for boys and girls. However, these findings were based on a small number of maternal alcoholics, and the gender interactions were not evident in our multivariate model. Thus, substantive conclusions regarding the effects of recent parent alcohol-related impairment await future replication.

Neither parent alcoholism diagnosis nor recent parental alcohol-related impairment were related to adolescents' negative expectancies. In considering negative expectancies, multiple mechanisms of transmission may work in opposite directions to offset each other. For example, biologically based mechanisms may lead COAs to experience less negative alcohol effects, which in turn should weaken negative expectancies. However, social environmental modeling mechanisms may work in the opposite direction. That is, COAs may observe their parents experiencing negative consequences of drinking, and these observations may increase negative expectancies. These mechanisms, working in opposite directions, may offset each other, resulting in the lack of a relation between negative expectancies and parent alcoholism.

Limitations and Conclusions

Although this study addressed some important methodological and theoretical issues in COA research, it is important to recognize some of the limitations. First, the inclusion of only three time points of measurement limited our ability to model patterns of growth for positive and negative expectancies. This precluded us from examining potential links between growth in drinking and growth in alcohol expectancies. Future studies examining the development of adolescent drinking over time with more than three time points over a longer span of adolescence are necessary to examine potential reciprocal relations between expectancies and drinking. Second, expectancies and adolescent heavy drinking were assessed using adolescent self-report. Thus, relations between these measures may be inflated due to common variance associated with the reporter. Future

research should use multiple sources of information to minimize this problem. Third, alcoholism has been treated as a unitary disorder, and no attempt has been made to subtype particular forms of alcoholism. A direction for future research is to examine the effects of subtypes of alcoholism and gender effects with larger sample sizes.

Finally, the mechanisms that account for how alcoholic parents influence their offsprings' growth trajectories of heavy drinking remain unclear. Our findings suggest that adolescent alcohol expectancies cannot explain the relation between parent alcoholism and growth in adolescents' heavy drinking. Other mechanisms are suggested by several theories of adolescent alcohol use and include family socialization of unconventional behavior (Kandel, 1980), social learning mechanisms such as modeling (Bandura, 1977), negative affect regulation (Kaplan, Martin, & Robbins, 1984), or the transmission of difficult temperament (Tarter, Alterman, & Edwards, 1985). Indeed, Chassin et al. (1996) found that parent monitoring and environmental stress partially mediated the effects of parent alcoholism on growth trajectories of adolescent substance use. This supports family socialization and negative affect regulation mechanisms of COA risk, and it is important for future research to test other potential mechanisms.

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