The Reciprocal Influences of Perceived Risk for Alcoholism and Alcohol Use Over Time: Evidence for Aversive Transmission of Parental Alcoholism*

MOIRA M. HALLER, M.A.,[†] AND LAURIE CHASSIN, PH.D.

Department of Psychology, Arizona State University, 950 S. McAllister, P.O. Box 871104, Tempe, Arizona 85287-1104

ABSTRACT. Objective: This study examined how perceived risk for alcoholism and alcohol use influenced each other over time. We hypothesized an aversive transmission mechanism, by which some children of alcoholics may reduce their drinking because they perceive themselves to be at risk for future alcohol problems because of their parents' alcoholism. **Method:** Using participants ($N = 804$, 47% female) from an ongoing longitudinal study of children of alcoholics (e.g., Chassin et al., 1991), we examined the reciprocal prospective relations between perceived risk for alcoholism and drinking across three measurement occasions, and also tested whether perceived risk for alcoholism mediated the effect of perceived parental alcoholism on subsequent drinking. **Results:** Mediation analyses provided evidence for aversive transmission, in which the effect of perceived parental alcoholism on alcohol use during young adulthood was decreased to the extent that perceived

parental alcoholism predicted higher levels of perceived risk for alcoholism during emerging adulthood. Results indicated reciprocal effects between perceived risk for alcoholism and drinking over time, such that higher levels of perceived risk were associated with lower levels of drinking. Results were replicated using both self-report and collateral-report of alcohol use, and using both actual and perceived parental alcoholism. **Conclusions:** Young adults may avoid drinking when they perceive their parent(s) to be alcoholic, and consequently perceive themselves to be at elevated risk for alcoholism. Given that beliefs about risk for alcoholism are potentially modifiable, increasing self-perceived risk for alcoholism may be one feasible way to reduce the intergenerational transmission of alcohol disorders within families. (*J. Stud. Alcohol Drugs*, 71, 588-596, 2010)

BOTH POPULAR BELIEF AND a limited number of research studies suggest that some children of alcoholics (COAs) show "aversive transmission," in which they consciously limit their drinking or abstain from alcohol use altogether to avoid the negative outcomes that they perceive to be experienced by their alcoholic parent(s) (Harburg et al., 1982). Such an aversive transmission mechanism would suggest that COAs may perceive themselves to be at risk for developing alcohol problems themselves and that they may consequently limit their alcohol use. The present study tested whether young adults who perceive their parent(s) to be alcoholic(s) also perceive themselves to be at elevated risk for alcoholism, and whether this perceived risk of future alcoholism influences their later drinking behavior. Using a community sample, we examined the reciprocal prospective relations between perceived risk for alcoholism and alcohol use across three measurement occasions, and also tested the extent to which perceived risk for future alcoholism mediated the effect of perceived parental alcoholism on later alcohol use.

Individuals' beliefs about the possible consequences of drinking alcohol are often referred to as alcohol expectan-

cies, and they have been shown to prospectively predict drinking behavior (see Jones et al., 2001, for review). Alcohol expectancies are often categorized as either positive or negative, depending on whether the expected result is desirable or undesirable (Jones et al., 2001). Moreover, alcohol expectancies are also often described as being either proximal or distal (e.g., McMahon et al., 1994), depending on whether the anticipated effects of alcohol are immediate (e.g., feeling confident) or longer term/delayed (e.g., health effects). Because perceived risk for alcoholism reflects the extent that individuals believe that they will eventually become alcoholic (i.e., a delayed effect rather than immediate effect), perceived risk for alcoholism has been conceptualized as an alcohol expectancy that is both *negative* and *distal* (e.g., Noar et al., 2003).

Studies that have examined the association between negative expectancies and alcohol use have had mixed results (e.g., Jones et al., 2001; McMahon et al., 1994; Sharkansky and Finn, 1998). One reason for these conflicting findings may be that the direction of the relation between negative expectancies and alcohol use depends on whether the negative expectancy being studied is distal or proximal in nature, as well as the time lag between measurements. Research has suggested that negative expectancies that are relatively distal (e.g., becoming an alcoholic) rather than proximal (e.g., acting inappropriately) may be better predictors of changes in drinking behavior over longer time spans than they are predictors of immediate decisions about how much

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[†]Correspondence may be sent to Moira M. Haller at the above address or via email at: moira.haller@asu.edu.

to drink on a given occasion (McMahon et al., 1994; Noar et al., 2003). Overall, few studies outside of treatment settings have examined the long-term effects of distal negative expectancies on alcohol use (Watson and Sher, 1998), and even fewer studies have specifically examined the effect of perceived risk for alcoholism. Therefore, it is important to determine whether perceived risk for alcoholism may predict lower levels of alcohol consumption over time.

Because COAs may have witnessed their parents experience negative effects of alcohol use or may have heard discussion of these negative effects, it is plausible that they would have higher perceived risk for alcoholism than would non-COAs. Indeed, several studies have found that COAs tend to score higher than do non-COAs on measures of perceived risk for alcohol problems, drinking restraint (i.e., preoccupation with controlling alcohol use), and reasons for limiting drinking based on perceived or experienced negative consequences (Chassin and Barrera, 1993; Epler et al., 2009). Despite these differences, little evidence has indicated that these variables lead to decreased alcohol consumption for COAs compared with non-COAs either cross-sectionally or longitudinally (e.g., Chassin and Barrera, 1993; Epler et al., 2009). However, these studies tested parental alcoholism as a moderator of the relation between perceived risk for alcoholism and alcohol use, which may not fully capture the influence that parental alcoholism has on both of these variables. A more appropriate test of the proposed aversive transmission phenomenon would be a mediational model that tests whether parental alcoholism produces increased perceived risk for alcoholism, which, in turn, may predict reduced drinking. Thus, an aversive transmission mechanism suggests that there will be a pathway in which parental alcoholism reduces offspring drinking by conveying a perception of risk for alcoholism. Importantly, however, this pathway is not expected to fully explain parent alcoholism effects on offspring drinking. Rather, there are multiple pathways that work in the opposite direction. It is well-established that parental alcoholism has a strong effect on increasing levels of offspring drinking (e.g., Chassin et al., 1999; Jacob et al., 2003), but perhaps this effect can be lessened, at least to some extent, if parental alcoholism also results in higher perceived risk for alcoholism.

Although many studies have investigated alcohol expectancies, there are no studies to our knowledge that have examined whether parental alcoholism increases perceptions of risk for alcoholism among offspring, and whether higher perceived risk for alcoholism subsequently predicts reduced drinking. In fact, the studies conducted by Harburg and colleagues (1982, 1990) that originally described the aversive transmission pattern did not actually assess offspring beliefs about alcohol, and only 13% of parents reported using alcohol more than "sparingly." Moreover, offspring (ages 19–72) were assessed only one time that was 17 years after parents were assessed, thus limiting causal inferences about aversive

transmission across development. The present study, which follows participants across three assessments from emerging adulthood into adulthood, facilitates the examination of how perceived risk affects drinking over time. In addition, given that more stable patterns of drinking would be expected during adulthood, as opposed to the experimentation that typically occurs during adolescence, the design of the present study allows examination of how perceived risk influences the stabilization of drinking behavior and the "maturing out" of alcohol use (e.g., Bachman et al., 2002).

The current study hypothesized that perceived risk for alcoholism would partially mediate the effect of perceived parental alcoholism on subsequent alcohol use. That is, we hypothesized that those who believed that their parents were alcoholic would perceive themselves to be at elevated risk for alcoholism, which would, in turn, result in less drinking. However, it is also possible that COAs perceive themselves to be at high risk for future alcoholism because they consume alcohol at higher levels (i.e., their perceived risk is based on their past drinking behavior rather than their parents' alcoholism). Accordingly, we also examined whether alcohol use mediated the effect of parental alcoholism on subsequent perceived risk for alcoholism. Hypotheses were tested using a cross-lagged panel model with three waves of data, which allowed us to examine the reciprocal relations between drinking and perceived risk for alcoholism. Moreover, we examined participants' perceptions of their parents' alcoholism rather than parents' actual clinical diagnoses because individuals would not necessarily view themselves to be at risk for alcohol problems unless they believed that their parents were alcoholic. Thus, perceptions of parents' alcoholism should be more important for aversive transmission mechanisms than should parents' actual alcohol disorders.

Method

Participants

Participants ($N = 804$) for the present study were drawn from Wave 4 (emerging adulthood, $M_{\text{age}} = 21.2$ years, $SD = 2.3$), Wave 5 (young adulthood, $M_{\text{age}} = 26.5$ years, $SD = 2.5$), and Wave 6 (adulthood, $M_{\text{age}} = 32.8$ years, $SD = 2.7$) from a larger ongoing longitudinal study of familial alcoholism across three generations (e.g., Chassin et al., 1991). There were three annual waves of data collection and three additional follow-ups separated by 5 years. At Wave 1 (1988), the total sample consisted of 454 "target" adolescents and their parents; 246 of these adolescents had at least one biological alcoholic parent who was also a custodial parent (COAs), and the remaining 208 adolescents were demographically matched controls without an alcoholic parent. Sample retention was excellent at all follow-ups, with 90% ($n = 407$) of the original targets interviewed at Wave 4, 91% ($n = 411$) interviewed at Wave 5, and 89% ($n = 404$) interviewed at

Wave 6. Beginning at Wave 4, biological siblings within the same age range ("age-eligible siblings") were interviewed in addition to targets to increase sample size (total $n = 738$ at Wave 4; $n = 735$ at Wave 5; $n = 735$ at Wave 6). Through use of missing data techniques, the present study included any target and age-eligible siblings who were interviewed at least once at Waves 4, 5, or 6, resulting in a total sample of 804. Approximately 47% of participants were female ($n = 381$), 72% ($n = 580$) were non-Hispanic White, and 50% ($n = 401$) were COAs.

Recruitment

Alcoholic families were recruited using court records, health maintenance organization wellness questionnaires, and community telephone surveys. To qualify, parents had to live in Arizona, be of non-Hispanic White or Hispanic ethnicity, and be born between 1926 and 1960. Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III; American Psychiatric Association, 1980), diagnoses of lifetime parental alcoholism (abuse or dependence) were made during a face-to-face structured diagnostic interview using the DIS-III (Robins et al., 1981). Parents who refused to participate were diagnosed based on spousal report using the Family History-Research Diagnostic Criteria (Endicott et al., 1975). In all, 219 biological fathers and 59 biological mothers met DSM-III criteria for alcoholism. Matched non-alcoholic families (matched on child's age, family composition, ethnicity, and socioeconomic status) were recruited by using reverse directories to find families living in the same neighborhoods as the COA families.

Recruitment biases. The two primary sources of potential

recruitment biases for the longitudinal study were selective contact and refusal to participate. Potential participants who were and were not successfully contacted did not differ on alcoholism indicators, but those who were not contacted were more likely to be younger, from court sources, Hispanic, unmarried, and had a lower socioeconomic-status rating associated with their residence. Individuals who refused to participate were more likely than were participants to be Hispanic and married but did not differ from participants on age, sex, socioeconomic status, or alcoholism. See Chassin et al. (1992) for a complete description of sample recruitment and representativeness.

Procedure

Data were collected in person using computer-assisted interviews or, for families who located out of the geographic region, via telephone. To encourage self-disclosure, family members were interviewed simultaneously in separate rooms, and a Department of Health and Human Services Certificate of Confidentiality was used to emphasize confidentiality.

Measures

See Table 1 for descriptive statistics and correlations between all variables.

Demographic variables. Participants' age and gender (0 for men and 1 for women) were included as exogenous variables in the present study.

Perceived parental alcohol use disorder. Participants were asked at Wave 4 whether they thought their mother

TABLE 1. Descriptive statistics and zero-order correlations among study variables

	1	2	3	4	5	6	7	8	9	10	11	12
1. Wave 4 age	1											
2. Gender ^a	.046	1										
3. Perceived parental alscm.	.016	-.006	1									
4. W4 perceived risk for alscm.	-.107**	.085*	.115**	1								
5. W5 perceived risk for alscm.	-.010	.069	.096**	.513**	1							
6. W6 perceived risk for alscm.	.015	.067	.156**	.433**	.523**	1						
7. W4 alcohol use: Self-rep.	.120**	-.197**	.115**	-.278**	-.137**	-.169**	1					
8. W5 alcohol use: Self-rep.	-.099**	-.182**	.137**	-.266**	-.272**	-.292**	.513**	1				
9. W6 alcohol use: Self-rep.	-.084*	-.148**	.127**	-.221**	-.224**	-.304**	.397**	.614**	1			
10. W4 alcohol use: Coll.-rep.	.055	-.129*	.127**	-.236**	-.073	-.154**	.711**	.486**	.349**	1		
11. W5 alcohol use: Coll.-rep.	-.122**	-.201**	.146**	-.215**	-.223**	-.247**	.438**	.697**	.567**	.494**	1	
12. W6 alcohol use: Coll.-rep.	-.084*	-.141**	.125**	-.181**	-.155**	-.278**	.420**	.568**	.668**	.440**	.592**	1
<i>M</i>	21.15	381 (47%) female ^b	281 (35%) reported parental alscm. ^b	2.96	2.78	2.61	2.52	2.81	2.79	1.51	1.68	1.66
<i>SD</i>	2.34			0.73	0.81	0.83	1.89	2.04	2.18	1.19	1.28	1.34
<i>Range</i>	17.2-28.8			1.0-4.0	1.0-4.0	1.0-4.0	0.0-7.0	0.0-7.0	0.0-7.0	0.0-5.0	0.0-5.0	0.0-5.0

Notes: Alscm. = alcoholism; coll.-rep. = collateral-report. ^a0 = male, 1 = female; ^bpercentages rather than means are provided for dichotomous variables (gender and perceived parental alscm.).

* $p < .05$; ** $p < .01$.

or father is/was an alcoholic. This item is recommended by Crews and Sher (1992) to assess perceptions of parental alcoholism with an adapted version of the Short Michigan Alcoholism Screening Test. The variable was coded "1" if the participant believed either parent to be an alcoholic, and "0" if neither parent was believed to be an alcoholic. To include 95 participants who were not assessed at Wave 4, this study used participants' reports of parental alcoholism at Waves 5 or 6 for these cases. Although full-information maximum likelihood missing-data techniques were used to include participants who were missing data at Wave 4, data cannot be missing on exogenous variables. Analyses were also conducted after deleting these 95 cases, and the pattern of findings was unchanged.

There were 401 participants whose parents met actual lifetime DSM-III-R (American Psychiatric Association, 1987) criteria for an alcohol disorder at Wave 4. Of these 401 participants, 250 (62.3%) reported that they had an alcoholic parent, and 151 participants (37.7%) did not perceive any parental alcoholism. Only 7 the 151 COAs who did not perceive any parental alcoholism had a parent who was currently an alcoholic at Wave 4. Because most of these 151 participants were children of *recovered* alcoholics, they may have been unaware that their parent(s) had experienced problems with alcohol. Analyses were also conducted after deleting these 151 cases, but the pattern of findings was unchanged. Moreover, analyses modeling the effects of actual parental lifetime alcohol disorder and analyses modeling the effects of perceived parental alcoholism exhibited the same pattern of results.

Perceived risk for alcoholism. Participants' perceived risk for alcoholism was assessed with six items. Two items were phrased as expectancies ("Drinking alcohol will make me become an alcoholic," and "Drinking alcohol will cause me to develop a drinking habit I don't want"), and four items were phrased as reasons to limit drinking ("I don't want to develop a drinking habit," "If I didn't limit my drinking, I would develop a drinking habit that I couldn't break," "I'm afraid that I will become an alcoholic," and "I've seen the negative effects of someone else's drinking"; see Greenfield et al., 1989). High scores indicated high perceived risk for alcoholism (range: 1-4). Alpha reliability was .83 at Wave 4, .86 at Wave 5, and .85 at Wave 6.

Alcohol use. Participants self-reported their frequency of drinking beer, wine, and wine coolers during the past year at Waves 4, 5, and 6. Responses ranged from *never* (0) to *every day* (7). Between 21.3% and 26.1% of participants abstained from alcohol use at each wave, and participants who drank reported drinking on average more than "5 times in the past year" but less than "1-3 times a month" at each wave. Participants also selected a non-family member collateral informant who reported on the participant's frequency of drinking, with responses ranging from *never* (0) to *almost always or always* (5). Collaterals reported that between

23.5% and 26.5% of participants abstained at each wave and that participants who drank consumed on average more than "sometimes" but less than "often" in the past year at each wave. The current analyses used self-reported frequency and collateral-reported frequency as outcomes in separate models. Because of the zero inflation in alcohol use (i.e., the number of abstainers), additional zero-inflated Poisson regression models (see below) were conducted with the same pattern of findings. Correlations between self-reported and collateral-reported alcohol use were .711 at Wave 4, .697 at Wave 5, and .668 at Wave 6 (see Table 1; all $ps < .01$).

We present models with drinking frequency as the outcome to facilitate comparisons between self-report and collateral informant report models because the collateral surveys did not include a quantity of drinking report. However, models were also estimated with self-reported quantity (usual number of drinks) times frequency of alcohol use with identical results. Quantity responses ranged from *none* (0) to *nine or more drinks per occasion* (8). On average, participants who drank consumed between three and four drinks per occasion at each wave. Before analysis, the quantity times frequency variables were log-transformed and multiplied by 10 to range from 0 to 1.76 rather than 0 to 56 to reduce nonnormality and facilitate interpretation.

Model specification

The current models examined the reciprocal prospective relations between perceived risk for alcoholism and alcohol use over three time points. All autoregressive paths were estimated. Paths were also estimated from Wave 4 perceived risk for alcoholism to Wave 6 perceived risk for alcoholism, as well as from Wave 4 alcohol use to Wave 6 alcohol use. Perceived parental alcoholism was entered as an exogenous predictor, and age and gender were entered as exogenous covariates. Paths were estimated from all exogenous variables to alcohol use and perceived risk for alcoholism at each wave. Full-information maximum likelihood estimation was used to account for missing data, which provides unbiased parameter estimates when "missingness" at random is assumed (Schafer and Graham, 2002).

Results

Zero-order correlations are reported in Table 1. As predicted, participants who reported that their parents were alcoholic had higher perceived risk for alcoholism at all three waves ($r_{W4} = .115$, $r_{W5} = .096$, $r_{W6} = .156$; all $ps < .01$). The relations of most interest were between Wave 4 alcohol use and Wave 5 perceived risk, Wave 5 alcohol use and Wave 6 perceived risk, Wave 4 perceived risk and Wave 5 alcohol use, and Wave 5 perceived risk and Wave 6 alcohol use. Correlations indicated that these relations were all significant using both self-report and collateral-report for the

alcohol use variables (all $ps < .01$), such that higher levels of perceived risk for alcoholism were associated with lower levels of drinking.

The intraclass correlations (ICCs) for perceived risk for alcoholism and alcohol use were examined to assess the degree of similarity among siblings. The ICCs for perceived risk for alcoholism were .134 at Wave 4, .190 at Wave 5, and .232 at Wave 6. The ICCs for self-reported alcohol use were .270 at Wave 4, .424 at Wave 5, and .306 at Wave 6. The ICCs for collateral-reported alcohol use were .252 at Wave 4, .434 at Wave 5, and .347 at Wave 6. ICCs of this magnitude indicate substantial similarity among siblings and the need to take clustering within families into account when modeling the data.

Structural models

Models were tested using MPlus version 5.0 (Muthén and Muthén, 1998–2006). Because participants were nested

within families, the maximum-likelihood estimator with robust standard errors (MLR) was used, which computes parameter estimates for continuous outcomes with standard errors that are robust to nonnormality and nonindependence of observations. Model fit was estimated with the MLR chi-square statistic, comparative fit index (CFI), root mean square error of approximation (RMSEA), and the standardized root mean square residual (SRMR).

Before estimating the final models, we tested a multiple group model for men and women to examine whether structural paths differed across gender. The Satorra-Bentler chi-square difference test for nested models was used (Satorra, 2000). There was no significant improvement in model fit when paths were allowed to vary across gender in either the self-report, $\Delta\chi^2(25) = 29.95$, $p = .23$, or collateral-report, $\Delta\chi^2(25) = 30.05$, $p = .22$, of alcohol use model. Final models included gender as a covariate but did not test separate paths for men and women.

Both the self-report and the collateral-report alcohol use

FIGURE 1. Results of both the self-report and collateral-report of alcohol use models. Results of the collateral-report of alcohol use model are shown in parentheses. All path estimates are standardized. For ease of presentation, paths from the exogenous covariates gender and Wave 4 age to perceived risk for alcoholism and alcohol use at all three waves are not shown. Fit statistics for the self-report of alcohol use model: $\chi^2(2) = 0.806$, $p = .67$, comparative fit index (CFI) = 1.00, root mean square error of approximation (RMSEA) = .000, standardized root mean square residual (SRMR) = .003. Fit statistics for the collateral-report of alcohol use model: $\chi^2(2) = 1.59$, $p = .45$, CFI = 1.00, RMSEA = .000, SRMR = .005.

* $p < .05$; ** $p < .01$; *** $p < .001$.

TABLE 2. Effects of covariates on perceived risk for alcoholism and alcohol use

Variable	Perceived risk for alcoholism			Alcohol use		
	Wave 4	Wave 5	Wave 6	Wave 4	Wave 5	Wave 6
Self-report model						
Age	-.11**	.05	.03	.12**	-.18***	-.03
Gender	.09*	.01	.01	-.15***	-.04	-.04
Collateral-report model						
Age	-.11	.05	.03	.12*	-.18***	-.08*
Gender	.09*	.01	.01	-.09*	-.08*	-.02

Notes: All betas are standardized. Standard errors ranged from .03 to .04.

* $p < .05$; ** $p < .01$; *** $p < .001$.

models (see Figure 1) showed good fit to the data according to all four fit indices—self-report model: $\chi^2(2) = 0.806$, $p = .67$, CFI = 1.00, RMSEA = .000, SRMR = .003; collateral-report model: $\chi^2(2) = 1.59$, $p = .45$, CFI = 1.00, RMSEA = .000, SRMR = .005. For ease of presentation, Figure 1 does not show paths that were estimated from gender and age to perceived risk for alcoholism and alcohol use at each wave. These results are presented in Table 2. All autoregressive effects and correlations between cross-sectional covariances were significant (all $ps < .001$). The magnitude of the autoregressive paths indicated moderate stability of both perceived risk and drinking across waves.

Perceived parental alcoholism predicted greater perceived risk for alcoholism at Wave 4 and Wave 6 ($ps < .01$) but not at Wave 5 ($\beta = .03$, n.s.). In both the self-report and collateral-report alcohol use models, the cross-lagged paths indicated that higher levels of Wave 4 perceived risk for alcoholism prospectively predicted lower levels of Wave 5 alcohol use ($\beta_{\text{self}} = -.16$, $p < .001$; $\beta_{\text{collateral}} = -.15$, $p < .001$), which in turn predicted higher levels of Wave 6 perceived risk for alcoholism ($\beta_{\text{self}} = -.17$, $p < .001$; $\beta_{\text{collateral}} = -.17$, $p < .001$). Wave 5 perceived risk for alcoholism prospectively predicted lower levels of Wave 6 alcohol use in the model using self-report of alcohol use but not in the model using collateral-report ($\beta_{\text{self}} = -.09$, $p < .05$; $\beta_{\text{collateral}} = -.05$, n.s.). However, the nonsignificance of the path from Wave 5 perceived risk to Wave 6 alcohol use in the collateral-report model may have been due to more missing data in the collateral-report variables. Wave 4 alcohol use was not associated with Wave 5 perceived risk in either model.

To test the indirect effect of parental alcoholism on drinking via perceived risk for alcoholism, 95% bias-corrected asymmetric confidence limits were used (MacKinnon et al., 2004). As hypothesized, Wave 4 perceived risk for alcoholism significantly mediated the prospective effect of perceived parental alcoholism on Wave 5 alcohol use in both the self-report and collateral-report of alcohol use models (self-report model: 95% CI [-.022, -.004]); collateral-report model: 95% CI [-.017, -.003]). These findings indicate that the effect of parental alcoholism on Wave 5 alcohol use was decreased to the extent that parental alcoholism predicted higher levels of Wave 4 perceived risk for alcoholism. Al-

though Wave 5 perceived risk also predicted lower Wave 6 alcohol use in the self-report model, the path from parental alcoholism to Wave 5 perceived risk was nonsignificant ($\beta = .03$, n.s.). Thus, Wave 5 perceived risk did not mediate the effect of parental alcoholism on Wave 6 alcohol use in either model.

We next tested whether higher levels of alcohol use mediated the effect of parental alcoholism on subsequent perceived risk. In the collateral-report of alcohol use model, there was no support for an indirect effect of parental alcoholism on perceived risk through alcohol use. In the self-report of alcohol use model, there was a significant indirect effect of parental alcoholism on Wave 6 perceived risk by way of Wave 5 alcohol use (95% CI [-.055, -.008]). The effect of parental alcoholism on Wave 6 perceived risk was decreased to the extent that parent alcoholism predicted greater Wave 5 alcohol use. However, the indirect effect of parental alcoholism on Wave 5 perceived risk through Wave 4 alcohol use was nonsignificant (95% CI [-.021, .011]).

Additional zero-inflated Poisson analyses

Separate analyses modeled self-reported and collateral-reported frequency of alcohol use as a count variable using zero-inflated Poisson regression. Zero-inflated Poisson regression allows for prediction of being in the group coded 0 (i.e., abstaining, in the present study), as well as the count variable. Results were consistent with those using the frequency of the alcohol use variable. Higher perceived risk predicted lower levels of alcohol use for the “count” part of the model and also predicted greater likelihood of being in the zero group (i.e., abstaining). The predictors of abstinence and the extent of alcohol use did not differ from each other, and the zero-inflated Poisson regression model did not produce any different results compared with analyses using the continuous alcohol use variable. For parsimony, we report results using the continuous alcohol use variable.

Discussion

This study used three waves of assessment to examine how perceived risk for alcoholism and alcohol use may influ-

ence each other over time. It was hypothesized that perceived risk for alcoholism would predict lower levels of subsequent drinking and that the effect of parental alcoholism on drinking would be decreased when parental alcoholism also resulted in higher perceived risk for alcoholism. Results from mediation analyses provided evidence for an aversive transmission mechanism, in which the effect of perceived parental alcoholism on drinking during young adulthood (Wave 5) was decreased to the extent that perceived parental alcoholism predicted higher perceived risk for alcoholism during emerging adulthood (Wave 4). It appears that COAs may avoid drinking when they perceive their parent(s) to be alcoholic and thereby perceive themselves to be at elevated risk for alcoholism. Findings were replicated using collateral-report of drinking, ruling out the possibility of self-report bias.

More broadly, results demonstrated that greater levels of perceived risk for alcoholism during emerging adulthood predict subsequent reductions in alcohol use during young adulthood. Given that the mean age was 21.2 at Wave 4 and 26.5 at Wave 5, findings also suggest that perceived risk for alcoholism may influence the "maturing out" of alcohol problems that typically occurs during young adulthood. Numerous studies have shown that alcohol use tends to peak during the early 20s and then typically declines as individuals age (Bachman et al., 2002). Whereas past research has linked these normative declines in drinking to changes in personality (Littlefield et al., 2009), the adoption of a more conventional lifestyle (Bachman et al., 2002), and adult role transitions such as marriage or parenthood (O'Malley, 2004-2005), this is the first study to suggest that perceived risk for alcoholism may also predict these declines in drinking.

Results also provide clear evidence for an inverse prospective association between perceived risk for alcoholism and alcohol consumption, in which higher levels of perceived risk are associated with lower levels of drinking. Few studies have examined the relations between alcohol use and perceived risk for alcoholism—a type of distal negative expectancy. Studies on the relations between negative expectancies and alcohol use have had mixed results (Mann et al., 1987; McMahon et al., 1994; Sharkansky and Finn, 1998), perhaps because these relations are different in the long term than in the short term. Although we did not test the short-term effects of perceived risk for alcoholism, our findings with a 5-year time lag between assessments indicate inverse relations between perceived risk and drinking over time. These results are consistent with research suggesting the more distal and severe perceived consequences of drinking, such as becoming an alcoholic, may be more predictive of drinking reductions over long time spans rather than drinking on a given occasion close in time (Noar et al., 2003). Other studies have found that distal negative expectancies may even influence the decision to reduce or stop drinking among problem drinkers (Lee et al., 1999; McMahon et al., 1994).

Although our results suggest that parental alcoholism appears to exert an indirect, protective effect on drinking via increased perceived risk for alcoholism, results from the self-report of the alcohol use model also indicated that parental alcoholism exerts consistent, direct effects that increase drinking throughout adulthood. Indeed, numerous studies have demonstrated that parental alcoholism increases risk for offspring problems (e.g., Jacob et al., 2003). Yet, our findings suggest that COAs' risk for future drinking problems may be decreased to the extent that parental alcoholism also increases perceived risk for alcoholism. In other words, overall, parental alcoholism increases offspring drinking, but parental alcoholism may also have a small, protective effect on drinking if offspring perceive themselves to be at risk for future alcohol problems as a result of witnessing their parents' alcohol problems.

As for the reverse direction of effect, lower levels of Wave 5 alcohol use (both self-report and collateral-report) prospectively predicted higher levels of Wave 6 perceived risk for alcoholism. These results appear to counter-intuitively suggest that the less one drinks, the greater his or her perceived risk for alcoholism becomes (or, conversely, the more one drinks, the lower his or her perceived risk for alcoholism becomes). Given that Wave 4 perceived risk predicted lower levels of Wave 5 drinking, it is possible that the subset of individuals who reduce their drinking because of their perceived risk for alcoholism become increasingly convinced that they would be at risk if they were to drink (i.e., perceived risk at Wave 4 predicts lower drinking at Wave 5, which predicts even higher perceived risk at Wave 6).

Alternatively, given research that has suggested that COAs exhibit polarized patterns of alcohol consumption (e.g., Harburg et al., 1982), the effect of Wave 5 alcohol use on increased perceived risk at Wave 6 may represent the other extreme of drinking among COAs—those who are heavy drinkers and drink increasing amounts over time because they do not expect that drinking will cause problems for them. Young adults who consume high levels of alcohol may develop lower perceived risk for alcoholism because those who drink at these high levels may do so without believing that heavy alcohol use is problematic for them. Indeed, results showed that increased levels of Wave 5 alcohol use mediated the effect of parental alcoholism on Wave 6 perceived risk for alcoholism, such that the effect of parental alcoholism on perceived risk was decreased by way of increased alcohol use. Interestingly, studies have shown that COAs are often less sensitive to the negative (e.g., depressive effects) effects of alcohol (e.g., Schuckit and Smith, 1996), and more sensitive to the positive or stress-reducing effects of alcohol (e.g., Finn et al., 1990). Perhaps COAs who increase their drinking also experience fewer adverse effects and greater pleasurable effects from alcohol, and thus believe they are not at risk for alcoholism.

Our findings also shed light on the directionality of the

relations between perceived risk for alcoholism and alcohol use, indicating reciprocal prospective effects. Notably, prospective effects were obtained despite relatively high stability of both perceived risk for alcoholism and alcohol use over time. Given that the prospective effect of perceived risk on alcohol use was significant across two time lags using self-report of alcohol use, whereas the prospective effect of alcohol use on perceived risk was significant across only one time lag, results provide somewhat stronger evidence for the effect of perceived risk on drinking rather than the reverse.

In addition, although not the primary focus of this study, results also contribute to understanding of perceived risk for alcoholism within families. Previous studies have not evaluated the degree of similarity among siblings in their perceived risk for alcoholism. ICCs for perceived risk for alcoholism increased across waves, indicating that siblings become more similar in their perceived risk for alcoholism as they age. Siblings also became more similar in their alcohol use from Wave 4 to Wave 5 (but not from Wave 5 to Wave 6). As siblings age, they likely observe both their own consequences from drinking (or lack thereof), as well as those experienced by family members. Given that siblings share experiences on which to base their perceived risk for alcoholism, it would be expected that their perceived risk for alcoholism would become more similar as these experiences are acquired. Findings are also consistent with behavioral genetic research that has shown that heritable influences on substance use become stronger from adolescence into adulthood (Kendler et al., 2008).

Several limitations to the present study should be noted. First, the magnitudes of the reciprocal cross-lagged effects were relatively small. Given the small effects, future replication of our findings is necessary before firm conclusions can be made regarding perceived risk for alcoholism as a means of decreasing the effect of parental alcoholism on offspring drinking. Nonetheless, it should be noted that these effects were obtained using conservative statistical analyses, controlling for both gender and age, as well as both autoregressive effects and cross-sectional covariances, and were found for multiple measures of alcohol use, multiple reporters, and multiple definitions of parental alcoholism. Second, it is possible that third variables accounted for changes in both perceived risk and alcohol use, although the longitudinal design of the study provides evidence for prospective relations between perceived risk for alcoholism and alcohol use. Finally, given the 5-year time lag between assessments, this study did not address the possibility of more proximal changes in perceived risk for alcoholism or alcohol use.

In summary, to our knowledge this is the first study to longitudinally examine how alcohol use and perceived risk for alcoholism reciprocally influence each other over time during emerging adulthood through adulthood. The use of a community sample with large numbers of high-risk individuals facilitated the examination of this question. Although

results indicated that parental alcoholism increases both offspring drinking and perceived risk for alcoholism, our mediational analyses imply that: (a) the effect of parental alcoholism on alcohol use is decreased to the extent that COAs have higher levels of perceived risk for alcoholism, and (b) the effect of parental alcoholism on perceived risk for alcoholism is decreased to the extent that parental alcoholism predicts higher levels of alcohol use. Our findings thus provide evidence for aversive transmission, in which some COAs may reduce their drinking because they perceive themselves to be at risk for future alcoholism because of their parents' alcoholism. Replication of results using both self-report and collateral-report of alcohol use, and using both actual and perceived parental alcoholism, increases confidence in the findings.

Finally, our findings may have implications for interventions, given that beliefs about perceived risk for alcoholism are potentially modifiable (e.g., Darkes and Goldman, 1993). Prevention programs may want to include interventions that aim to increase personal awareness of risk for alcoholism, particularly risk related to problematic alcohol use among family members. Consistent with aversive transmission, increasing self-perceived risk for alcoholism among COAs may be a feasible way of reducing the intergenerational transmission of alcohol disorders. Future studies should examine what factors influence perception of risk for alcoholism, and how these factors differ for COAs versus non-COAs.

References

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.
- American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.
- Bachman, J. G., O'Malley, P. M., Schulenberg, J. E., Johnston, L. D., Bryant, A. L., & Merline, A. C. (2002). *The decline of substance use in young adulthood: Changes in social activities, roles, and beliefs*. Mahwah, NJ: Lawrence Erlbaum.
- Chassin, L., & Barrera, M. (1993). Substance use escalation and substance use restraint among adolescent children of alcoholics. *Psychology of Addictive Behaviors*, 7, 3-20.
- Chassin, L., Barrera, M., Bech, K., & Kossak-Fuller, J. (1992). Recruiting a community sample of adolescent children of alcoholics: A comparison of three subject sources. *Journal of Studies on Alcohol*, 53, 316-319.
- Chassin, L., Pitts, S. C., DeLucia, C., & Todd, M. (1999). A longitudinal study of children of alcoholics: Predicting young adult substance use disorders, anxiety, and depression. *Journal of Abnormal Psychology*, 108, 106-119.
- Chassin, L., Rogosch, F., & Barrera, M. (1991). Substance use and symptomatology among adolescent children of alcoholics. *Journal of Abnormal Psychology*, 100, 449-463.
- Crews, T. M., & Sher, K. J. (1992). Using adapted short MASTs for assessing parental alcoholism: Reliability and validity. *Alcoholism: Clinical and Experimental Research*, 16, 576-584.
- Darkes, J., & Goldman, M. S. (1993). Expectancy challenge and drinking reduction: Experimental evidence for a mediational process. *Journal of Consulting and Clinical Psychology*, 61, 344-353.

- Endicott, J., Anderson, N., & Spitzer, R. L. (1975). *Family history diagnostic criteria*. New York: Biometrics Research, New York Psychiatric Institute.
- Epler, A. J., Sher, K., & Piasecki, T. M. (2009). Reasons for abstaining or limiting drinking: A developmental perspective. *Psychology of Addictive Behaviors*, 23, 428-442.
- Finn, P. R., Zeitouni, N., & Pihl, R. O. (1990). Effects of alcohol on psychophysiological hyperreactivity to nonaversive and aversive stimuli in men at high risk for alcoholism. *Journal of Abnormal Psychology*, 99, 79-85.
- Greenfield, T. K., Guydish, J., & Temple, M. T. (1989). Reasons students give for limiting drinking: A factor analysis with implications for research and practice. *Journal of Studies on Alcohol*, 50, 108-115.
- Harburg, E., Davis, D., & Caplan, R. (1982). Parent and offspring alcohol use: Imitative and aversive transmission. *Journal of Studies on Alcohol*, 43, 497-516.
- Harburg, E., DiFranceisco, W., Webster, D. W., Gleiberman, L., & Schork, A. (1990). Familial transmission of alcohol use: II. Imitation of and aversion to parent drinking (1960) by adult offspring (1977)—Tecumseh, Michigan. *Journal of Studies on Alcohol*, 51, 245-256.
- Jacob, T., Waterman, B., Heath, A., True, W., Bucholz, K. K., Haber, R., ... Fu, Q. (2003). Genetic and environmental effects on offspring alcoholism: New insights using an offspring-of-twins design. *Archives of General Psychiatry*, 60, 1265-1272.
- Jones, B. T., Corbin, W., & Fromme, K. (2001). A review of expectancy theory and alcohol consumption. *Addiction*, 96, 57-72.
- Kendler, K. S., Schmitt, J. E., Aggen, S. H., & Prescott, C. A. (2008). Genetic and environmental influences on alcohol, caffeine, cannabis, and nicotine use from early adolescence to middle adulthood. *Archives of General Psychiatry*, 65, 674-682.
- Lee, N. K., Greely, J., & Oei, T. P. S. (1999). The relationship of positive and negative alcohol expectancies to patterns of consumption of alcohol in social drinkers. *Addictive Behaviors*, 24, 359-369.
- Littlefield, A. K., Sher, K., & Wood, P. K. (2009). Is "maturing out" of problematic alcohol involvement related to personality change? *Journal of Abnormal Psychology*, 118, 360-374.
- MacKinnon, D. P., Lockwood, C. M., & Williams, J. (2004). Confidence limits for the indirect effect: Distribution of the product and resampling methods. *Multivariate Behavior Research*, 39, 99-128.
- Mann, L. M., Chassin, L., & Sher, K. L. (1987). Alcohol expectancies and the risk for alcoholism. *Journal of Counseling and Clinical Psychology*, 55, 411-417.
- McMahon, J., Jones, B. T., & O'Donnell, P. (1994). Comparing positive and negative alcohol expectancies in male and female social drinkers. *Addiction Research*, 1, 349-365.
- Muthén, B. O., & Muthén, L. K. (1998-2006). *Mplus version 5.0, computer software and manual*. Los Angeles, CA: Author.
- Noar, S. M., LaForge, R. G., Maddock, J. E., & Wood, M. D. (2003). Rethinking positive and negative aspects of alcohol use: Suggestions from a comparison of alcohol expectancies and decisional balance. *Journal of Studies on Alcohol*, 64, 60-69.
- O'Malley, P. (2004-2005). Maturing out of problematic alcohol use. *Alcohol Research and Health*, 28, 202-204.
- Robins, L. N., Helzer, J. E., Croughan, J. L., & Ratcliff, K. S. (1981). National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics, and validity. *Archives of General Psychiatry*, 38, 381-389.
- Satorra, A. (2000). Scaled and adjusted restricted tests in multi-sample analysis of moment structures. In R. D. H. Heijmans, D. S. G. Pollock, & A. Satorra (Eds.), *Innovations in multivariate statistical analysis. A Festschrift for Heinz Neudecker* (pp. 233-247). New York: Kluwer Academic.
- Schafer, J. L., & Graham, J. W. (2002). Missing data: our view of the state of the art. *Psychological Methods*, 7, 147-177.
- Schuckit, M. A., & Smith, T. L. (1996). An 8-year follow-up of 450 sons of alcoholic and control subjects. *Archives of General Psychiatry*, 53, 202-210.
- Sharkansky, E. J., & Finn, P. P. (1998). Outcome expectancies and disinhibition on ad-lib alcohol consumption. *Journal of Studies on Alcohol*, 59, 198-206.
- Watson, A., & Sher, K. (1998). Resolution of alcohol problems without treatment: Methodological issues and future directions of natural recovery research. *Clinical Psychology: Science and Practice*, 5, 1-18.