

## Pathways of Risk for Accelerated Heavy Alcohol Use Among Adolescent Children of Alcoholic Parents

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The current study examined two questions. First, do internalizing symptoms and externalizing behavior each mediate the relations between parent psychopathology (alcoholism, antisocial personality disorder, and affective disorder) and growth in adolescent heavy alcohol use? Second, are there gender differences in these mediated pathways? Using latent curve analyses, we examined these questions in a high-risk sample of 439 families (53% children of alcoholic parents; 47% female). Collapsing across gender, adolescent-reported externalizing behavior mediated both the relation between parent alcoholism and growth in heavy alcohol use and the relation between parent antisociality and growth in heavy alcohol use. Parent-reported externalizing behavior only mediated the relation between parent antisociality and growth in heavy alcohol use in males. No support was found for internalizing symptoms as a mediator of these relations. Avenues are suggested for further exploring and integrating information about different mediating processes accounting for children of alcoholics' risk for heavy alcohol use.

**KEY WORDS:** Parent alcoholism; adolescent alcohol use; externalizing behavior; internalizing symptoms; gender.

Children of alcoholic parents (COAs) have a greater risk of early and accelerated alcohol use, and other behavior problems, than do children whose parents are not alcoholic (Chassin, Curran, Hussong, & Colder, 1996; Sher, 1991; West & Prinz, 1987). COAs' risk for developmental difficulties begins early (ages 3 to 5) and includes elevated motoric activity, attention deficits, reactivity, impulsivity, and anxiety (Fitzgerald et al., 1993; Jansen, Fitzgerald, Ham, & Zucker, 1995; Johnson & Jacob, 1995; Roosa, Beals, Sandler, & Pillow, 1990; West & Prinz, 1987). To better understand potential relations among these developmental outcomes, the current study assessed (1) whether internalizing symptoms and externalizing be-

haviors each form pathways of risk for COAs leading to heavy alcohol use during adolescence, and (2) whether male and female COAs follow different pathways of risk for heavy alcohol use.

### *Relations Between Adolescent Symptomatology and Heavy Alcohol Use*

The distinction between externalizing behavior and internalizing symptoms is common in adolescent symptomatology (Achenbach, 1982) and in theories of pathways leading to adolescent problem behaviors and substance use (Windle, 1994; Zucker, 1986). According to Zucker, these internalizing and externalizing pathways are distinct in course, predisposing factors, and etiology. The externalizing path, expected to be more common in males, involves an early onset of substance use following elevations in childhood attentional deficits, activity levels, and conduct problems. These externalizing behaviors may increase the likelihood of future alcohol involvement

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directly (Windle, 1994); or, externalizing behaviors and alcohol problems may both be manifestations of a single underlying trait that is present throughout development (Loeber, 1988). Regardless, multiple theorists suggest that the relation between externalizing behavior and substance use is at least partially due to temperamental or predisposing traits, with peer contexts and parenting styles contributing to the expression of disruptive behavior over time (Loeber, 1988; Windle, 1994).

Substantial research consistently supports the association between adolescent alcohol use and delinquent or externalizing behavior, involving either comorbid diagnoses (e.g., conduct and oppositional defiant disorder; Rhode, Lewinsohn, & Seeley, 1996) or subclinical acting out behaviors (e.g., Loeber, 1988; Van Kammen, Loeber, & Stouthamer-Loeber, 1991). For example, Windle (1990) found that delinquency in early adolescence predicted subsequent substance use even after controlling for substance use in early adolescence. Similar relations have been reported between aggression in first grade and substance use 10 years later in males, but not in females (Ensminger, Brown, & Kellam, 1982), and between disruptive behavior disorder and later onset of alcohol abuse/dependence for both females and males (Rhode et al., 1996).

More controversial is the relation between internalizing symptoms and adolescent alcohol and drug use. The internalizing pathway focuses on the relation between negative emotions and substance use and is expected to be more common in females. Windle (1994) suggested that such internalizing symptoms as anxiety, depression, and avoidance increase risk for subsequent substance use as children seek to cope with escalating withdrawal, isolation, and poor social skills over time. Sometimes referred to as a self-medication model (Sher, 1991), adolescent substance use is seen as a purposeful effort to dampen experienced anxiety and depression.

Examining such internalizing models, Swaim, Oetting, Edwards, & Beauvais (1989) found that adolescents with greater negative affect were more likely to associate with drug-using peers, and, in turn, to use drugs themselves. Moreover, adolescent substance use was more strongly associated with peers' drug use than with the adolescent's internalizing symptoms, leading these authors to conclude that peer behavior, and not negative affect, is an important predictor of adolescent substance use. Consistent with this conclusion, theories of adult alcoholism describe internalizing symptoms as precursors of late

onset alcohol abuse (Sher, 1991; Zucker, 1986), suggesting that alcohol use motivated by negative affect may not occur as a consistent problem until after adolescence. In contrast, Kaplan (1980) described internalizing symptoms (or self-derogation) as central in leading adolescents to seek out the deviant peer associations in which they eventually begin to use substances. In short, contradictory perspectives provide an unclear understanding of the proposed relation between internalizing symptoms and later substance use among adolescents.

Similar mixed findings appear within the empirical literature, with some studies failing to find a relation between internalizing symptoms and adolescent substance use (Windle & Barnes, 1988) and others supporting this relation. Previous research indicates concurrent associations between depression and adolescent heavy alcohol use (Hussong & Chassin, 1994), and also that emotional distress assessed at age 7 distinguishes between frequent and experimental substance use among 18-year-olds (Shedler & Block, 1990). Moreover, Henry et al. (1993), found that, depressive symptoms uniquely predicted subsequent polydrug use among males (although not females) in their New Zealand sample. Finally, in their study of more severe disturbance, Rhode et al. (1996) found that among adolescents with comorbid anxiety and alcohol abuse/dependence disorders, the onset of anxiety disorders preceded that of alcohol abuse/dependence.

In sum, theory and research consistently support the externalizing path leading to adolescent substance use, especially among males, although the literature is much less clear regarding the internalizing path. Due to gender differences in the prevalence of internalizing symptoms and externalizing behavior and to previous support for gender differences in the relation between symptomatology and adolescent alcohol use, theoretical speculation about gender differences in these two pathways to heavy alcohol use appears warranted, but not well examined. The current study extended previous research by examining these two pathways to adolescent substance use within a prospective study, utilizing multiple reporters of adolescent symptomatology, focusing on the prediction of intraindividual change in heavy alcohol use over time, and examining gender differences in the relation between each form of symptomatology and growth in heavy alcohol use over time. Moreover, internalizing symptoms and externalizing behavior were examined as potential mediators of

COAs' risk for accelerated heavy alcohol use over time.

### *Specificity of COAs' Risk for Negative Outcomes*

Does parent alcoholism actually account for COAs' greater developmental risks? Epidemiological Catchment Area studies have found that alcoholic women and men are 2.7 and 1.7 times more likely to report depressive and dysthymic disorders and 12.3 and 3.8 times more likely to report antisocial personality disorder than are their nonalcoholic counterparts (Helzer & Pryzbeck, 1988). Given such rates of comorbid disorder, COAs may face developmental risks associated not only with parent alcoholism but also with co-occurring parent psychopathology. Like COAs, children of depressed parents show greater internalizing symptoms, externalizing behaviors, functional impairment, social and academic deficits and lack of competence compared to children of nondepressed parents (Downey & Coyne, 1990). Moreover, Johnson and Jacob (1995) found that children of depressed and alcoholic fathers showed similar elevations in internalizing and externalizing behaviors as compared with children of non-disturbed fathers. Similarly, children of antisocial parents show elevated levels of attention deficit, oppositional defiant, conduct, and anxiety disorders than do children of nonantisocial parents (Earls, Reich, Jung, & Cloninger, 1988; Frick et al., 1992). These developmental risks raise the possibility that the negative outcomes seen among COAs are actually associated with parent disorders that are often comorbid with parent alcoholism rather than with parent alcoholism per se.

Comparing developmental outcomes among children of alcoholic and antisocial parents, Frick et al. (1992) found that parent antisociality, and not parent alcoholism, was a unique predictor of child oppositional and conduct problems. Father's antisociality, rather than alcoholism, also uniquely predicted adolescent aggression in a study of adolescent male adoptees conducted by Cadoret, Yates, Troughton, Woodworth, and Stewart (1995). Although providing preliminary support for the importance of comorbid parent disorder in understanding the unique developmental risks associated with parent alcoholism, these studies were limited by their indirect methods of ascertaining parental psychopathology (e.g., archival records, child reports) and reliance on

nonrepresentative samples (e.g., treatment or twin/adoption samples).

Overcoming these limits through parent assessment and community sampling, Chassin, Rogosch, and Barrera (1991) found unique associations between parent psychopathology and adolescent heavy alcohol use such that parent alcoholism was uniquely related to adolescent alcohol involvement and internalizing symptoms; parent affective disorder uniquely predicted adolescent internalizing symptoms; and parent antisocial personality disorder uniquely predicted adolescent externalizing behaviors and drug use. Moreover, Chassin et al. (1996) found that father's alcoholism diagnosis was the only one of these parent diagnoses to *uniquely* predict changes in adolescent substance use over time, such that COAs reported greater substance use at Time 1 and faster escalation of substance use involvement throughout adolescence.

Just as parent disorders seem to show different unique associations with adolescent outcomes, the multiple pathways through which adolescents come to manifest elevated substance use may be related to different types of parent disorder. In other words, although children of parents with alcohol, antisocial and affective disorders all show greater heavy alcohol use during adolescence (Cadoret et al., 1995; Chassin et al., 1996; Downey & Coyne, 1990), the pathways or mediators leading to heavy alcohol use may differ across each form of parent psychopathology.

Comparing pathways of risk associated with parental alcoholism and antisocial personality disorders, Cadoret et al. (1995) found that biological paternal alcoholism directly predicted adoptee drug abuse, whereas biological paternal, antisocial personality disorder indirectly influenced both adoptee antisocial personality disorder and drug abuse via increased offspring aggression. These findings suggest that the externalizing pathway leading to adolescent substance use may be associated with parent antisociality, rather than with parent alcoholism, whereas other pathways may be needed to explain the association between parent alcoholism and elevated adolescent substance use. A number of limitations of Cadoret's adoptee study, however, qualify this conclusion including the omission of females, indirect assessment of parent psychopathology (via archival records) and cross-sectional assessment of the relation between adoptee aggression and substance use.

In sum, the current study was designed to address these limitations and to further examine differences in the pathways from various types of parent

psychopathology to heavy alcohol use in adolescence. Specifically, the current study examined the hypotheses that (1) internalizing symptoms and externalizing behavior each mediate the unique relation (when controlling for other parent psychopathology) between parent alcoholism and early and accelerated adolescent heavy alcohol use, and that (2) females are more likely to follow the internalizing path and males more likely to follow the externalizing path to heavy alcohol use. A strength of the current study was the use of longitudinal and multiple-reporter data gathered within a community-based, high-risk sample. Latent curve modeling was also used in the current study, permitting a powerful examination of these two hypotheses.

## METHOD

### *Participants*

At Time 1, 454 adolescents (aged 10.5 to 15.5 years,  $M = 12.7$ ,  $SD = 1.4$ ) and their parents participated in the study. COAs ( $n = 246$ ) had at least one biological alcoholic parent who was also a custodial parent. Controls ( $n = 208$ ) had no biological nor custodial alcoholic parents. Sample retention over the 3-year study was high, and only 10 families did not provide complete data at all three time points. Five additional participants were identified as influential outliers (see Results section) and excluded from this study, leaving a final sample of 439 families.

*T*-tests and chi-square comparisons showed that the 15 participants who were dropped from analyses did not significantly differ at Time 1 in age, gender, parent education, or adolescent externalizing behaviors from the 439 who were retained. However, those dropped from the analyses reported higher rates of adolescent heavy alcohol use, maternal and paternal alcoholism, parent affective disorder, and parent antisocial personality disorder ( $p < .01$ ). Of the 439 retained adolescents, 53% were COAs, 47% were female, 23% were Hispanic, and 92% lived in two-parent homes at Time 1. COAs and controls did not significantly differ in these characteristics. However, COAs reported lower parent education as well as greater heavy alcohol use and adolescent externalizing behavior (all  $ps < .05$ ) as compared with controls at Time 1.

### *Recruitment*

COA families were recruited using court records ( $n = 103$ ), wellness questionnaires from a health maintenance organization ( $n = 22$ ), and community telephone surveys ( $n = 120$ ; see Chassin, Barrera, Bech, & Kossak-Fuller, 1992). COAs had to be non-Hispanic Caucasian or Hispanic, Arizona residents, aged 10.5 to 15.5 years, and English speaking. Moreover, a biological and custodial parent had to meet the criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.) (DSM-III; American Psychological Association, 1980) for alcohol abuse or dependence or Family History-Research Diagnostic Criteria (FH-RDC; Andreasen, Endicott, Spitzer, & Winokur, 1977), based on spouse reports (if the alcoholic parent was not interviewed). Demographically matched controls were recruited using telephone interviews. Controls were screened to match the COA participant in ethnicity, family composition, age, and socioeconomic status. Neither biological nor custodial parents could meet DSM-III criteria (or FH-RDC criteria) for alcohol abuse or dependence.

By requiring alcoholic parents to be custodial as well as biological (to allow both environmental and biological risk exposure), our resulting sample overrepresented two-parent families. Also, those who refused participation were more likely to be Hispanic. However, the sample was unbiased with respect to alcoholism indicators that were available in archival records. In support of the representativeness of the alcoholic sample, the parents' comorbidities were similar to those reported in the Epidemiological Catchment Area Study (Helzer & Pryzbeck, 1988). However, the underrepresentation of single-parent families and the higher refusal rate for Hispanics suggest caution in generalization.

### *Procedure*

Trained staff conducted computer-assisted, in-person interviews with adolescents and their parents at either their residence or the university campus. All measures were close ended and preprogrammed into the computerized batteries using automated skip patterns. Interviewers read items aloud to subjects who had the option of responding verbally or through direct keyboard entry. To minimize contamination effects, family members were interviewed simultaneously and in separate rooms (in most cases). Although in-

Table I. Gender Differences in Adolescent Symptomatology and Heavy Alcohol Use Scores

Composite scores for symptomatology & heavy alcohol use	Girls		Boys		T-test	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>t</i>	<i>p</i>
Heavy alcohol use						
Time 1	.41	1.24	.47	1.55	-0.48	.63
Time 2	.55	1.41	.85	2.19	-1.69	.09
Time 3	.92	2.00	1.30	2.62	-1.68	.09
Time 1 internalizing symptoms						
Self-report	2.32	.77	2.11	.68	3.0	.00
Parent-report	.36	.27	.38	.28	-.44	.66
Time 1 externalizing behaviors						
Self-report	1.64	.49	1.71	.52	-1.26	.21
Parent-report	0.26	.22	.35	.28	-3.73	.00

initially blind to group membership, interviewers were aware of family alcohol and drug use through interview responses. To encourage honesty and ensure privacy, we informed families of our Department of Health and Human Services Certificate of Confidentiality. Families participated in three such 1 to 2-hour interviews on an annual basis beginning in 1988.

### Measures

*Parent Alcoholism and Associated Psychopathology.* Lifetime DSM-III (APA, 1980) diagnoses of alcohol abuse or dependence, affective disorder (major depression or dysthymia), and antisocial personality disorder were obtained using a computerized version of the Diagnostic Interview Schedule (DIS, Version III; Robins, Helzer, Croughan, & Ratcliff, 1981). Bipolar disorder was excluded due to lower base rates and need to constrain interview length. If only one

parent was interviewed, alcoholism diagnosis for the other parent was made using spouse reports according to FH-RDC. For the current analyses, alcoholism diagnoses of the biological father and mother were considered (separately) as dichotomous variables. Among the 439 families in the current analyses, 53 mothers and 206 fathers met these criteria.

Parents' (lifetime) affective disorder and antisocial personality disorder were assessed using the DIS, and were considered as separate dichotomous variables, either present (in one or both parents) or absent.<sup>5</sup> If only one parent was interviewed, then family level diagnoses were based on this individual's diagnostic status.<sup>6</sup> This occurred for 113 (25.7%) families (22 mothers and 91 fathers) in assessing antisocial personality disorder and for 110 (25.1%) families (23 mothers and 87 fathers) in assessing affective disorder. Of the 439 families in the current analyses, 62 reported parent affective disorder and 35 reported parent antisocial personality disorder.

*Adolescent Heavy Alcohol Use.* Adolescents reported how often they had been drunk and how often they had consumed 5 or more drinks over the past year. The 8-point response scale ranged from *never* (0) to *every day* (7). The mean of the two items constituted the alcohol involvement score. Interitem correlations ranged from .79 to .83 over measurement waves and no gender differences in heavy alcohol use were found across the three time points (see Table I).

Rates of drinking at Time 1, as assessed by a single item regarding lifetime alcohol use, reflect the young age of the current sample and the expected increase in the number of adolescents engaging in alcohol use over time. At Time 1, 42% of the current sample reported having ever tried alcohol whereas 13% reported having ever had five or more drinks

<sup>5</sup>Because prevalence rates of these disorders were lower than those for alcoholism (due to our sample design), we were unable to create separate variables for mother's versus father's diagnostic status on these disorders.

<sup>6</sup>The assumption underlying this measurement was that missing parent reports would not indicate parent psychopathology in the absence of psychopathology in the reporting parent. Although this assumption may have resulted in differential reliability among the measures of parental psychopathology, this possibility cannot account for the current pattern of findings. If differential reliability were truly responsible for the pattern of unique relations between parent psychopathology and child symptoms, then parent alcoholism (the diagnosis not impacted by this assumption) should be most strongly and consistently associated with child adjustment across outcomes. However, as described later, unique relations between parent psychopathology and child symptoms varied across the outcomes of externalizing behavior, internalizing symptoms and heavy alcohol use.

in a row in the past year and 11% reported having ever been drunk in the past year. By Time 3, 57% of the sample reported lifetime alcohol use whereas 25% reported having had five or more drinks in a row in the past year and 24% reported having been drunk in the past year.

**Symptomatology.** Both parents and adolescents reported on adolescent internalizing symptomatology and externalizing behavior occurring in the 3 months prior to Time 1. To shorten the larger assessment battery and to create parallel self and parent report instruments, we chose items from the Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1981) including seven items assessing internalizing symptoms and 21 items assessing externalizing behavior (see Table 1).<sup>7</sup> Adolescents reported their internalizing symptoms ( $M = 2.2$ ,  $SD = 0.73$ ) and externalizing behavior ( $M = 1.7$ ,  $SD = 0.51$ ) using a 5-point response scale. Mothers and fathers reported adolescent internalizing symptoms ( $M = 0.41$ ,  $SD = 0.34$ ;  $M = 0.31$ ,  $SD = 0.28$ , respectively) and externalizing behaviors ( $M = 0.33$ ,  $SD = 0.26$ ;  $M = 0.30$ ,  $SD = 0.25$ , respectively) using a response scale ranging from 0 to 2. (See Table I for gender differences in adolescent symptomatology composites.) To assess internal reliability of these abbreviated scales, we examined Cronbach's alpha and found them acceptable across reporters ( $\alpha = .62$  to  $.76$  for internalizing symptoms;  $\alpha = .86$  to  $.88$  for externalizing behavior). When two parents were interviewed, a mean of mother and father reports formed a single score for parent-reported adolescent symptomatology. When only one parent was interviewed, the participating parent's report formed the parent reported score on adolescent symptomatology ( $n = 122$ ). Correlations

across reporters were as follows for internalizing and externalizing, respectively: mother-adolescent ( $r = .21$ ;  $.40$ ), father-adolescent ( $r = .19$ ;  $.30$ ), and mother-father ( $r = .29$ ;  $.63$ ).

## RESULTS

### *Analytic Strategy*

We used latent curve (LC) modeling to analyze the two hypotheses of the current study. Both technical discussions (McArdle & Epstein, 1987; Meredith & Tisak, 1990) and longitudinal applications (Curran, Stice, & Chassin, 1997; Stoolmiller, 1994) of this technique are available in the literature. In the current study, all models were estimated using EQS (Version 3.0; Bentler, 1989) based on the sample covariance matrix and a column vector of means (see Table II). Model fit was assessed using the chi-square test statistic, the Tucker-Lewis Index (TLI; Tucker & Lewis, 1973), the Comparative Fit Index (CFI; Bentler, 1990), and the root mean squared error of approximation (RMSEA; Browne & Cudeck, 1993; Steiger & Lind, 1980). Fit indices greater than .90 and an RMSEA less than .05 were taken to indicate acceptable fit.

Before interpretation of final parameter estimates and model fit, outlier analyses were conducted to determine whether those adolescents whose individual trajectories of heavy alcohol use most deviated from the group trajectory (i.e., who showed a greater than 2 standard deviation decrease in heavy alcohol use over any interval) unduly influenced model interpretation. Five individuals were found to be influential outliers in this manner and were dropped from further analyses, creating the final sample of 439.

### *Modeling Adolescent Heavy Alcohol Use*

The first step was to examine both *group* and *individual* characteristics of growth in heavy alcohol use over time. LC analysis is a random effects model that allows for the separate estimation of the initial status or *intercept* of the growth trajectory from the rate of change or *slope* of this growth trajectory. This two part growth trajectory is defined analytically by a two factor latent variable or unconditional growth model. In the current study, three measures (Times 1, 2, and 3 heavy alcohol use) served as indicators of these two latent growth factors. The mean of the

<sup>7</sup>Specific items from the CBCL assessing internalizing included: "I felt lonely;" "I felt that I had to be perfect;" "I was too fearful or anxious;" "I felt too guilty; I was unhappy, sad, or depressed;" "I felt overtired or lacked energy;" "and" "I felt worried." We selected items for the externalizing scale from the Youth Self-Report Survey (loading on the externalizing and aggression subscales for both boys and girls), and added items concerning nonaggressivity and conduct disorder. Specific items assessing externalizing behavior included: "I argued a lot;" "I skipped or ditched school;" "I stole things outside of home;" "I was mean or cruel to others;" "I tried to get a lot of attention;" "I destroyed my own things;" "I stole things at home;" "I disobeyed at home;" "I disobeyed at school;" "I started fights;" "I screamed a lot;" "I lied or cheated;" "I physically attacked people;" "I destroyed things belonging to others;" "I showed off or clowned;" "I threatened to hurt people;" "I couldn't concentrate;" "I was quick tempered;" "I swore or used obscene language;" "I hung around with kids who got in trouble;" "I acted without stopping to think."

Table II. Variance-Covariance Matrix and Vector of Means for Current Study

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
(1) Parent affective disorder	.122												
(2) Parent antisocial personality disorder	.018	.074											
(3) Biological mother's alcoholism diagnosis	.017	.018	.106										
(4) Biological father's alcoholism diagnosis	.032	.026	.005	.250									
(5) Child's age	-.012	-.010	.002	-.047	2.134								
(6) Child's gender	-.002	-.001	-.016	-.001	-.042	.250							
(7) Adolescent report of externalizing behaviors	.019	.022	.011	.035	.172	.015	.258						
(8) Parent report of externalizing behaviors	.018	.019	.010	.024	-.042	.023	.047	.066					
(9) Adolescent report of internalizing behaviors	.027	.014	.008	.029	.177	-.052	.210	.029	.531				
(10) Parent report of internalizing behaviors	.024	.012	.013	.021	-.019	.003	.024	.035	.047	.076			
(11) Time 1 heavy alcohol use	.013	.035	.024	.098	.668	.016	.231	.057	.120	.038	2.001		
(12) Time 2 heavy alcohol use	.041	.091	.069	.176	.874	.075	.350	.112	.161	.069	1.874	3.493	
(13) Time 3 heavy alcohol use	.044	.091	.090	.239	.930	.094	.428	.118	.171	.021	1.719	3.152	5.569
(14) Vector of means	.141	.080	.121	.469	12.697	.531	1.677	.311	2.210	.371	.442	.713	1.123

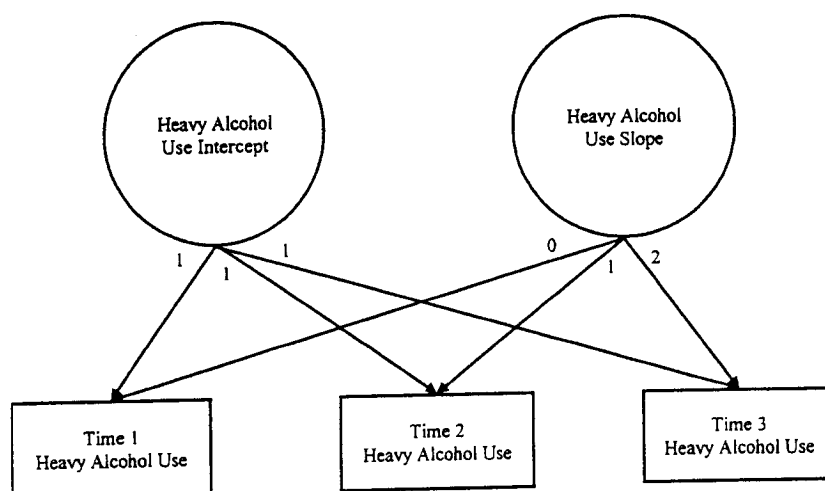


Fig. 1. Heavy alcohol use latent growth model. Model fit: ( $df = 2$ ,  $N = 439$ ) = 3.6,  $p = .16$ ,  $TLI = .99$ ,  $CFI = .99$ ,  $RMSEA = .04$ .

intercept factor represented the group mean on heavy alcohol use at Time 1 (defined by setting the factor loadings on the intercept to 1.0), and the variance of the intercept factor represented individual variability around this group mean. The slope factor was set to reflect linear growth (defined by setting factor loadings to 0, 1, 2 across time bound indicators), which best described the shape of the group's underlying growth trajectory as judged by parsimony and model fit [ $\chi^2$  (2,  $N = 439$ ) = 3.6,  $p = .16$ ;  $TLI = .99$ ,  $CFI = .99$ ,  $RMSEA = .04$ ; Fig. 1]. Significant individual variability was found in both the intercept factor ( $\psi = 1.9$ ,  $p < .001$ ) and, most importantly, in the growth factor ( $\psi = 0.7$ ,  $p < .001$ ). Thus the group as a whole reported significant linear increases

in heavy alcohol use over time as well as significant individual variability in rates of growth over time.

#### Testing Mediators of COA Risk for Growth in Adolescent Heavy Alcohol Use

To test the first hypothesis, we used mediational, LC analyses to examine four models, one for each of the potential mediators of: adolescent-reported externalizing behaviors, parent-reported externalizing behaviors, adolescent-reported internalizing symptoms, and parent-reported internalizing symptoms. Each model contained six exogenous variables (parent affective disorder, parent antisocial personality disorder, biological mother's alcoholism diagnosis, biological father's alcoholism diagnosis, the

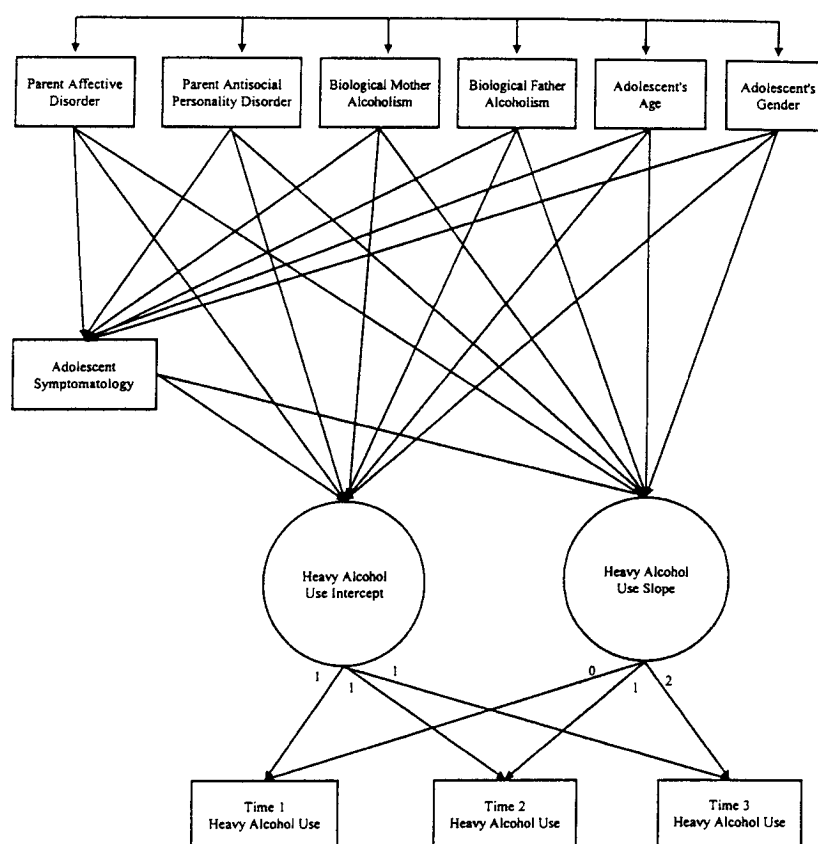


Fig. 2. Estimated model.

adolescent's age and the adolescent's gender), the mediator of interest and the two growth factor model of heavy alcohol use (Fig. 2).

All exogenous variables were freely intercorrelated. Structural parameters tested whether the six exogenous variables significantly predicted (1) each mediator (assessed at time one), (2) the intercept of the growth trajectory, and (3) the slope of the growth trajectory. Pathways between adolescent symptomatology and the intercept and slope factors were also estimated. Because the intercept factor estimates the beginning of the growth trajectories (at time 1), predictions of this factor are essentially concurrent associations. In contrast, the slope factor for heavy alcohol use indexes change over time, and predictions of the slope factor are truly prospective and emphasized in the current report. These LC models permitted comparisons of (1) the unique effects of different forms of parent psychopathology on adolescent symptomatology and on growth in heavy alcohol use, and (2) the unique effects of each mediational pathway from the different forms of parent psycho-

pathology through internalizing symptomatology and externalizing behaviors, predicting growth in adolescent heavy alcohol use.

**Adolescent-Reported Externalizing Behavior.** The estimated model fit the data well in the analyses of adolescent-reported externalizing behavior as a mediator of the relation between parent psychopathology and growth in heavy alcohol use [ $\chi^2(9, N = 439) = 13.5, p = .14$ ; TLI = .97, CFI = .99, RMSEA = .03]. Elevated adolescent externalizing behaviors were associated with having an antisocial parent ( $\beta = .12, p < .05$ ), having an alcoholic father ( $\beta = .12, p < .05$ ) and being older ( $\beta = .25, p < .001$ ). Moreover, adolescents with an alcoholic father ( $\beta = .13, p < .01$ ), with greater externalizing behaviors ( $\beta = .25, p < .001$ ) or who were older ( $\beta = .31, p < .001$ ) reported greater heavy alcohol use at the start of their growth trajectories than did those with nonalcoholic fathers, those with fewer externalizing behaviors, or those who were younger. Unique risk factors associated with change in heavy alcohol use over time (i.e., predictors of the slope factor) included



maternal ( $\beta = .12, p < .05$ ) and paternal ( $\beta = .15, p < .05$ ) alcoholism, externalizing behaviors ( $\beta = .19, p < .01$ ) and male gender ( $\beta = .12, p < .05$ ). These findings suggest that those adolescents showing greater acceleration in heavy alcohol use over time were more likely to have an alcoholic parent, to show greater externalizing behavior, and to be male.

To examine whether these associations formed pathways of risk leading from parent psychopathology to growth in heavy alcohol use, indirect effects were formally tested in EQS (Bentler, 1989; Sobel, 1982). Significant indirect effects were found for the pathways associated with parent antisocial personality disorder ( $\beta = .02, p < .05$ ) and father's alcoholism diagnosis ( $\beta = .02, p < .05$ ). These unique effects suggest that children of antisocial parents as well as alcoholic fathers show greater externalizing behavior in early adolescence that, in turn, increases their risk for accelerated heavy alcohol use during adolescence. However, the remaining unique, main effect of parent alcoholism on growth in adolescent heavy alcohol use suggested that additional pathways are needed to completely explain COAs' risk for heavy alcohol use.

*Parent-Reported Externalizing Behavior.* The estimated model also fit the data well in analyses of parent report of adolescents' externalizing behaviors [ $\chi^2(9, N = 439) = 13.2, p = .15$ ; TLI = .97, CFI = .99, RMSEA = .03]. Parents reported greater externalizing behaviors by their adolescents in families having alcoholic fathers ( $\beta = .12, p < .01$ ) as well as a parent with affective ( $\beta = .12, p < .01$ ) or antisocial disorders ( $\beta = .22, p < .001$ ). Parents also reported more externalizing behaviors in males than in females ( $\beta = .18, p < .001$ ). Paternal alcoholism ( $\beta = .14, p < .01$ ) and age ( $\beta = .38, p < .001$ ) were associated with higher initial heavy alcohol use as in the adolescent report model. In addition, adolescents for whom parents reported more externalizing behavior ( $\beta = .18, p < .001$ ) also reported higher initial heavy alcohol use. As in the adolescent report model, change in heavy alcohol use over time was uniquely associated with both maternal ( $\beta = .12, p = .05$ ) and paternal ( $\beta = .16, p < .01$ ) alcoholism and age ( $\beta = .16, p < .01$ ). Greater parent-reported externalizing behavior ( $\beta = .11, p < .10$ ) and male gender ( $\beta = .11, p < .10$ ) were only marginally associated with steeper growth in heavy alcohol use. Analyses of indirect effects involving parent-reported externalizing behaviors on growth in heavy alcohol use found marginal support for a pathway from parent antisocial personality disorder through

externalizing behavior to growth in heavy alcohol use ( $\beta = .02, p < .10$ ).

*Adolescent-Reported Internalizing Symptoms.* Analyses of adolescent-reported internalizing symptoms as a mediator of the relations between parent psychopathology and growth in heavy alcohol use resulted in a good fit of the estimated model to the data [ $\chi^2(9, N = 439) = 11.51, p = .24$ , TLI = .98, CFI = .99, RMSEA = .025]. Older adolescents and females reported greater internalizing symptomatology than did their younger ( $\beta = .17, p < .001$ ) or male peers ( $\beta = -.13, p < .01$ ). Parent affective disorder was also marginally associated with adolescent internalizing symptoms ( $\beta = .09, p < .10$ ). As in previous models, paternal alcoholism ( $\beta = .16, p < .01$ ) and age ( $\beta = .36, p < .001$ ) were associated with higher initial heavy alcohol use, but internalizing symptoms were not associated with initial heavy alcohol use. Paternal ( $\beta = .17, p < .01$ ) and maternal ( $\beta = .13, p < .05$ ) alcoholism and age ( $\beta = .15, p < .05$ ) were again associated with steeper escalations in heavy alcohol use. Moreover, males ( $\beta = .14, p < .05$ ), and to a lesser extent children of antisocial parents ( $\beta = .10, p < .10$ ), reported greater acceleration in heavy alcohol use than did females or adolescents of nonantisocial parents. The lack of association between parent report of adolescents' internalizing symptoms and growth in heavy alcohol use precluded the possibility of significant mediational pathways in this model.

*Parent-Reported Internalizing Symptoms.* The estimated model fit the data well in analyses of parent reports of adolescents' internalizing symptoms as a mediator of the relation between parent psychopathology and growth in heavy alcohol use [ $\chi^2(9, N = 439) = 16.7, p = .05$ , TLI = .95, CFI = .99, RMSEA = .044]. Parents reported greater internalizing symptoms in adolescents from families having a parent with affective disorder ( $\beta = .20, p < .001$ ), alcoholic mothers ( $\beta = .09, p < .05$ ), or alcoholic fathers ( $\beta = .09, p < .05$ ). Paternal alcoholism ( $\beta = .15, p < .01$ ) and age ( $\beta = .37, p < .001$ ) were again associated with higher initial heavy alcohol use. In addition, adolescents with greater internalizing symptoms as reported by parents showed higher initial heavy alcohol use ( $\beta = .10, p < .05$ ). As in the adolescent report model, steeper escalations in heavy alcohol use were associated with maternal ( $\beta = .13, p < .05$ ) and paternal ( $\beta = .18, p < .01$ ) alcoholism, age ( $\beta = .15, p < .05$ ), male gender ( $\beta = .14, p < .05$ ), and, marginally, parent antisocial personality disorder ( $\beta = .11, p < .10$ ), but not with parent reported internalizing symptoms.

### *Gender Differences in Mediation Pathways*

We also conducted multiple-group, LC analyses (McArdle, 1989) to provide a formal test of the interaction between the mediating mechanisms and adolescent gender, testing Hypothesis 2. First we examined the unconditional growth model for heavy alcohol use. We estimated baseline models within gender to determine whether gender specific modifications were needed (using LaGrange multiplier indices, Bentler, 1989,  $\chi^2(1) > 6.6$ ,  $p < .01$ , and following Bollen, 1989; Byrne, 1989; Byrne, Shavelson, & Muthen, 1989). Based on these indices, an error covariance between the time one and two measures of heavy alcohol use was added to each baseline model, leading to an adequate fit to the data for both males [ $\chi^2(1, N = 233) = .18$ ,  $p = .67$ ; TLI = 1.0, CFI = 1.0, RMSEA = .00] and females [ $\chi^2(1, N = 206) = 2.4$ ,  $p = .12$ ; TLI = .98, CFI = .99, RMSEA = .01]. In a multiple-group framework, we then imposed nested equality constraints on estimated parameters (factor variances and covariance followed by factor means) in these models across gender. The failure of such constraints to hold suggested that males, as compared with females, showed greater variability in heavy alcohol use at each time point and in the intercept and the slope factors for heavy alcohol use. Moreover, gender differences in the mean of the slope factor indicated that males escalated faster in their heavy alcohol use over time than did females. No gender differences were found in the mean of the intercept factor. The final partially constrained, multiple-group model fit the data well [ $\chi^2(4, N = 439) = 4.2$ ,  $p = .38$ ; TLI = .99, CFI = 1.0, RMSEA = .03], suggesting no gender differences in the shape of growth in heavy alcohol use over time.

We next examined gender differences in the mediational pathways by examining, within a multiple-group framework, the same four mediational, LC models presented earlier. Equality constraints were first imposed on the two growth factors in the full mediational model as indicated by the results of the unconditional model. We then imposed equality constraints on each parameter in a serial fashion to determine whether males and females differed in the structural parameter estimates of the full mediational model (e.g., Bollen, 1989). Models of partial invariance were constructed for each of the four mediational models.

Gender differences impacting mediational pathways were only found in the model of parent report of adolescents' externalizing behaviors. Specifically,

(parent-reported) externalizing behaviors predicted steeper escalations in heavy alcohol use over time in boys ( $\beta = .21$ ,  $p < .01$ ) but were unrelated to change in heavy alcohol use in girls ( $\beta = -.16$ ,  $p > .10$ ). Examination of indirect effects within gender revealed a marginally significant indirect effect from parent antisocial personality disorder to growth in heavy alcohol use in boys through externalizing behaviors ( $\beta = .04$ ,  $p < .10$ ) but no indirect effect in girls ( $\beta = -.03$ ,  $p > .10$ ). These findings suggest that boys, but not girls, with antisocial parents display greater externalizing behaviors in early adolescence that increases risk for escalation in heavy alcohol use over time.

### DISCUSSION

In the current study, we examined two hypotheses. First, we tested whether internalizing symptoms and externalizing behavior each mediated the unique relation between parent alcoholism and early, accelerated adolescent heavy alcohol use, when controlling for comorbid parent psychopathology. Second, we tested whether females were more likely to follow the internalizing path and males more likely to follow the externalizing path to heavy alcohol use. Three primary findings provide mixed support for these hypotheses. First, greater externalizing behavior among children of alcoholic and antisocial parents at least partly explained why these children show accelerated heavy alcohol use through adolescence when compared with their peers. Second, adolescents with greater internalizing symptoms were not at risk for accelerated heavy alcohol use over time, indicating that the internalizing pathway does not explain elevated risk for heavy alcohol use among COAs or their peers. Finally, COAs continued to show accelerated heavy alcohol use over time, even after controlling for other forms of parent disorder and early adolescent symptomatology. In other words, parent alcoholism was a powerful and unique predictor of change in adolescent heavy alcohol use over time that could not be fully explained by adolescent developmental risk associated with comorbid parent disorder or early adolescent symptomatology.

#### *The Externalizing Path*

Externalizing behavior predicted not only concurrent heavy alcohol use among adolescents, repli-

cating previous research (Ensminger et al., 1982; Loeber, 1988; Van Kammen et al., 1991; Windle, 1990), but also prospectively predicted individual differences in change in heavy alcohol use over time. Moreover, externalizing behavior was a unique mediator of the relation between parent psychopathology and accelerated heavy drinking, though precise findings varied across parent versus adolescent report of externalizing behavior as well as across adolescent gender. Consistent with Cadoret et al. (1995), *parent reports* of adolescents' externalizing behaviors uniquely mediated the relation between *parent antisociality* (but not parent alcoholism) and heavy alcohol use *among males*. However, *adolescent reports* of externalizing behavior uniquely mediated both the relation between *parent alcoholism* and growth in heavy alcohol use and the relation between *parent antisociality* and growth in heavy alcohol use *across gender*.

These findings support the unique risk for children of antisocial parents associated with externalizing behavior leading to adolescent heavy alcohol use. However, they provide inconsistent support for a similar externalizing pathway uniquely associated with parent alcoholism. To better understand these findings for COAs, future research on the externalizing pathway to substance use may need to consider gender differences in adolescent styles of deviance or externalizing behavior and in parental awareness of these different types of externalizing behavior. Specifically, studies of social development, dominance and delinquency find that females show more relational or covert aggression (i.e., harming others' through interpersonal relationships) whereas males show more overt or physical aggression (Crick & Grotpeter, 1995; Savin-Williams, 1979). Given these differences, parents may be less aware of their daughters' than of their sons' externalizing behaviors, resulting in a greater discrepancy between parent and daughter, rather than parent and son, reports of externalizing behaviors.<sup>8</sup>

Future research on the externalizing path to substance use should examine whether gender differences in the association between externalizing behaviors and growth in heavy alcohol use exist across different types (overt and covert) of externalizing be-

havior and disorder (e.g., oppositional defiant, conduct, and attention deficit-hyperactivity disorder).

### *The Internalizing Path*

The lack of support for internalizing symptoms as a predictor of future adolescent heavy alcohol use is consistent with views of negative affect based alcoholism as having an adult onset (Cloninger, 1987), a deemphasis on the theoretical role of negative emotions in adolescent substance use (Swaim et al., 1989), and some of the previous research regarding adolescent substance use (Windle & Barnes, 1988). However, these views and the current findings must be reconciled with previous studies that support the relation between internalizing symptoms and alcohol use among adolescents (Henry et al., 1993; Hussong & Chassin, 1994; Rhode et al., 1996; Shedler & Block, 1990).

One possibility, supported by research on adult alcoholism, is that internalizing symptoms are a result and not a precursor of alcohol involvement (Aneshensel & Huba, 1983). Consistent with this interpretation, in the current study, parent reports of adolescents' internalizing symptoms were related to higher initial heavy drinking but not to future changes in heavy alcohol use. Alternatively, some types of internalizing symptoms may serve as precursors and others as consequences of alcohol involvement. If so, we should examine the internalizing path with respect to the relations between alcohol use and different types of internalizing symptoms. This hypothesis has received some support in studies of adult alcoholics, where retrospective reports suggest that the onset of anxiety disorders precedes and that of depression follows the onset of alcohol abuse and dependence (Chambless, Cherney, Caputo, Rheinstein, 1987; Merikangas, Risch, Weissman, 1994).

Still a third possibility is that the internalizing path creates developmental risk for only a subgroup of individuals. To evaluate this hypothesis, important moderators of the relation between internalizing symptoms and heavy alcohol use must be identified. For example, impulsivity may raise risk for affect-related substance use by reducing the likelihood that adolescents will consider other coping mechanisms or the potential consequences of their substance use. In support of this hypothesis, Hussong and Chassin (1994) found that depression was most strongly related to heavy alcohol use among more impulsive adolescents. Additional potential moderators identi-

<sup>8</sup>This pattern of findings existed in the current data, though a Fisher's z-score transformation test of the differences between parent-son versus parent-daughter correlations of reported externalizing behavior produced nonsignificant results.

fied by Cooper, Frone, Russell, and Mudar (1995) include adolescent coping styles and alcohol expectancies. More specifically, impoverished coping resources may also raise risk for affect-related substance use among adolescents who rely heavily on their peer groups for support and guidance. Deviant peer groups may provide less support for adolescents and raise risk for responding to internalizing symptoms by engaging in the drug-using behavior of the peer group as a coping mechanism. Unlike previous authors who have discussed deviant peers and negative affect as competing risk factors for adolescent heavy alcohol use (e.g., Swaim et al., 1989), it may be that these influences are actually synergistic and describe a larger context placing adolescents at risk for problematic alcohol and drug use.

In short, the results of the current study suggest that internalizing symptoms do not form a general pathway of risk for COAs' accelerated heavy alcohol use. However, internalizing symptoms may still play an important role in adolescent heavy alcohol use either as a consequence of alcohol and drug involvement or as a precursor for particular subgroups demarcated by greater impulsivity, poorer social resources, or parent alcoholism. Because internalizing pathways of risk have received far less attention and only equivocal support, these potential avenues for understanding the relation between internalizing symptoms and alcohol use in adolescence deserve further investigation.

### Conclusions

One potential limitation of the current study, and many like this, is the effect of differential reliability when comparing externalizing behaviors versus internalizing symptoms as predictors of growth in heavy alcohol use. Although the unique effects of each potential mediator were not tested in the current study, externalizing behavior was a more consistent and robust predictor of adolescent alcohol use than were internalizing symptoms. This conclusion should be tempered by the differential reliability of the symptomatology measures (a common problem in adolescent psychopathology research; Achenbach & Edelbrock, 1981), which may have resulted in greater power to detect a relation between externalizing behaviors and alcohol use than between internalizing symptoms and alcohol use. Despite this potential limitation, the current findings are consistent with previous research in which conduct problems, but not depressive symp-

toms, uniquely predicted later adolescent alcohol use (Henry et al., 1993).

Despite potential limitations, the current study offers a strong test of the role of early adolescent symptomatology in the development of heavy alcohol use among high-risk adolescents. The current results suggest that early adolescent symptomatology may serve to at least partially explain the unique risk for accelerated heavy alcohol use among adolescents of antisocial or alcoholic parents. More specifically, early externalizing behaviors, but not internalizing symptoms, indicate risk for more rapid growth in heavy alcohol use over the adolescent period of development. These findings must be considered within the context of previous research in which COAs' elevated risk for adolescent substance use has also been associated with deficits in parenting, peer influences, temperament, alcohol expectancies, differential pharmacological sensitivity, and cognitive dysfunction (Chassin et al., 1996; Sher, 1991). Such multiple mediators of COAs' risk for substance use must be considered in concert to attain a more integrated understanding of the individual processes of risk leading to adolescent alcohol involvement. To better understand these processes of risk, future research should examine what forms of externalizing behavior may be particularly salient in predicting growth in heavy alcohol use during adolescence. Moreover, future research is needed to determine whether internalizing symptoms may help explain why particular subgroups of adolescents (e.g., COAs, impulsive adolescents, or those with fewer supportive resources) may be particularly vulnerable to heavy alcohol use. Through a more careful examination of the relations among early adolescent symptomatology and later alcohol behavior, we may begin to understand the processes underlying the developmental risks of COAs and to identify different pathways leading to problematic alcohol use among adolescents.

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