

## Relation of Parental Alcoholism to Early Adolescent Substance Use: A Test of Three Mediating Mechanisms

Laurie Chassin, David R. Pillow, Patrick J. Curran, Brooke S. G. Molina, and Manuel Barrera, Jr.

The current study assessed 3 hypothesized mediating mechanisms underlying the relation between parental alcoholism and adolescent substance use. Using structural equation modeling, we analyzed data obtained from a large community sample of adolescent children of alcoholics and a demographically matched comparison group. Results suggested that parental alcoholism influenced adolescent substance use through stress and negative affect pathways, through decreased parental monitoring, and through increased temperamental emotionality (which was associated with heightened negative affect). Both negative affect and impaired parental monitoring were associated with adolescents' membership in a peer network that supported drug use behavior. The data did not support a link between parental alcoholism and temperamental sociability.

Because children of alcoholics (COAs) are at increased risk for adult alcoholism, they have been a focus of research attention (see Sher, 1991; West & Prinz, 1987, for reviews). However, there have been conflicting findings concerning whether COAs are at elevated risk for substance use during adolescence, with some studies finding elevated risk (e.g., Herjanic, Herjanic, Penick, Tomelleri, & Armbruster, 1977; Merikangas, Weissman, Prusoff, Pauls, & Leckman, 1985) and others finding no elevated risk for adolescent alcohol use (Johnson, Leonard, & Jacob, 1989; Pandina & Johnson, 1989) or drug use (Knop, Teasdale, Schulsinger, & Goodwin, 1985).

The reasons for the conflicting findings appear to be primarily methodological, including variations in sampling strategies, definitions of parental alcoholism, and whether parental alcoholism is assessed directly or by adolescents' reports (cf. Sher, 1991). Moreover, studies have not often considered the effects of co-occurring factors that influence the magnitude of COA risk for adolescent substance use (e.g., parental psychiatric disorders, environmental stress). To the extent that samples vary along these dimensions, the magnitude of the effect of parental alcoholism on adolescent substance use will also vary. Finally, COAs are heterogeneous with regard to risk; only a minority of COAs will actually develop negative outcomes. This heterogeneity reduces the magnitude of differences between COA and non-COA groups, so that studies with small sample sizes may lack the power to detect parental alcoholism effects (Sher,

1983). The net result of these methodological problems has been inconsistent demonstrations of the effect of parental alcoholism on adolescent substance use. However, at least two recent studies with large samples have demonstrated both statistically reliable and clinically meaningful elevations in alcohol and drug use among COAs in early adolescence (Chassin, Rogosch, & Barrera, 1991) and late adolescence (Sher, Walitzer, Wood, & Brent, 1991).

Given the uncertainty about the magnitude of COA risk for adolescent substance use and the methodological and practical difficulties of conducting these studies, it is not surprising that we know little about potential mediating mechanisms by which parental alcoholism may result in elevations in adolescent substance use. Some hypotheses have been explored with respect to the intergenerational transmission of adult alcoholism. These mechanisms have included parent modeling of alcohol abuse and exposure to alcohol, differential sensitivity to the positive or negative effects of alcohol, and a greater tendency to develop tolerance or negative alcohol effects (see Sher, 1991, for a review). Critical supportive data have been lacking because there have been few appropriate analyses that simultaneously test all the requisite links between parental alcoholism, a potential mediator, and offspring drinking behavior, particularly for young adolescents (cf. Rogosch, Chassin, & Sher, 1990). Nevertheless, models of substance use for the general adolescent population can be used to generate theory-based hypotheses.

---

Laurie Chassin, David R. Pillow, Patrick J. Curran, Brooke S. G. Molina, and Manuel Barrera, Jr., Department of Psychology, Arizona State University.

This study was supported by Grant DA05227 from the National Institute on Drug Abuse to Laurie Chassin and Manuel Barrera.

We thank Kirsten Beck, Julie Kossak-Fuller, and Polly Lewitt for coordinating the interviewing and data collection activities. We also thank Leona Aiken, Craig Colder, David Mackinnon, Clark C. Presson, Mark Reiser, Kenneth J. Sher, and Steven West for statistical consultation and comments on this manuscript.

Correspondence concerning this article should be addressed to Laurie Chassin, Department of Psychology, Arizona State University, Tempe, Arizona 85287-1104.

### Models of Adolescent Substance Use

#### *Parenting and Socialization Models*

It has been repeatedly demonstrated that parent modeling of substance use and ineffective parental control practices are related to adolescent substance use (e.g., Brook, Whiteman, & Gordon, 1983; Jessor & Jessor, 1977). For the present article, the most closely relevant application of a parental socialization framework is the social interactional theory of Patterson and his colleagues (Dishion, Patterson, & Reid, 1988; Patterson & Reid, 1984). Dishion et al. (1988) demonstrated that parental drug use was related to early adolescent drug sampling. More

important, the effect was both direct (interpreted as the result of modeling and opportunities for substance use) and indirect (through impaired parental control). Parental drug use impaired parent monitoring of the child's behavior and activities. Impaired monitoring, in turn, was related to the child's embeddedness in a peer subculture that included peer drug use and delinquency. This peer network provided the proximal pathway into early drug use behaviors. A parallel argument could be made for the impact of parental alcoholism on adolescent substance use. Alcoholic parents may not only provide models for drinking and access to alcoholic beverages, but they also may be impaired in their ability to monitor their children's behavior. In such cases, adolescents might not be restrained from deviant peer associations (including associations with drug-using peers). This socialization mechanism (linking parental alcoholism to impaired parent monitoring to associations with drug-using peers and drug use) was tested in the current study.

### *Stress and Negative Affect Regulation Models*

An alternative (although not mutually exclusive) perspective focuses on environmental stress and the negative affect regulation functions of substance use. According to this model, substance use serves as a coping strategy to regulate the negative affect that is associated with life stress. Stress and negative affect regulation models have been more commonly associated with later stage substance abuse than with adolescent substance use initiation (Swaim, Oetting, Edwards, & Beauvais, 1989). Nevertheless, some studies have supported links between stress and negative affect and substance use in adolescence. For example, Paton, Kessler, and Kandel (1977) found that depressed mood prospectively predicted both the initiation of marijuana use and other illegal drug use in adolescence. Wills (1986) found that life stress events were prospective predictors of substance use among junior high school students. Perhaps most relevant to the current study, Newcomb and Harlow (1986) found evidence for a mediational model in which uncontrollable negative life events were related to perceived loss of control and meaninglessness which, in turn, were linked to adolescent substance use. Thus, despite being more commonly viewed as explaining substance abuse, stress and negative affect regulation have also been linked to adolescent substance use.

A stress and negative affect regulation model provides a plausible theoretical explanation for increased risk for substance use among adolescent COAs. Laboratory data suggest that young adult COAs derive greater stress response dampening benefits of alcohol use than do their non-COA peers (Levenson, Oyama, & Meek, 1987). Moreover, parental alcoholism is associated with elevated levels of environmental stress among adolescent offspring (Roosa, Sandler, Gehring, Beals, & Cappo, 1988). If COAs experience elevated stress levels, and if they experience greater stress response dampening effects of alcohol, then a stress and negative affect reduction mechanism may underlie COA risk for adolescent substance use.

A potentially important distinction between negative affect regulation models of adolescent substance use compared with substance abuse involves the role of the peer environment. In studying substance abuse, these models have posited self-medication functions of alcohol and drugs. However, in considering

adolescent substance use, there are data to support a somewhat different mechanism involving additional mediation through a drug-use-promoting peer environment. Swaim et al. (1989) found that adolescents who experienced high levels of negative affect were more likely to associate with drug-using peers, who were the proximal influence agents for drug use. Similarly, Kaplan (1980) suggested that adolescents who suffered negative self-evaluations were more likely to seek out deviant peer networks as a way of repairing damaged self-esteem. Thus, stress and negative affect may raise risk for early adolescent drug use not through self-medication motives but rather by increasing the likelihood of associations with a drug-use-promoting peer group. This peer group affiliation might help explain how stress and negative affect could be related to adolescent substance use initiation (in addition to the more typically conceptualized self-medication effects on later stage substance abuse). Because of these two different interpretations, we tested both direct relations between negative affect and substance use and indirect effects (mediated through associations with drug-using peers).

### *Personality and Temperament Models*

A final set of predictors of adolescent drug use are intrapersonal characteristics that have been referred to as "personality" (e.g., Brook, Whiteman, Gordon, & Cohen, 1986; Jessor & Jessor, 1977) or "temperament" (Windle, 1990). In this domain, many characteristics have been associated with adolescent substance use. Adolescents who are rebellious, nonconventional, impulsive, sensation-seeking, and extraverted have been shown to be at risk for substance use (Brook et al., 1986; Jessor & Jessor, 1977; Kandel, 1978).

Some researchers have suggested that temperamental characteristics might mediate the effects of parental alcoholism (see Windle, 1990, for a review). For example, Tarter, Alterman, and Edwards (1985) argued that COAs are more likely to be temperamentally high in activity, low in persistence, slow to soothe after stress, and emotionally labile and disinhibited, and that these characteristics are also associated with early onset alcohol abuse among men. Other investigators have suggested that COAs display a broad pattern of behavioral undercontrol perhaps coupled with a propensity to experience negative affective states (Sher, 1991). If these dimensions are both heritable and related to risk for alcohol use, then they are potential candidates for mediating the effects of parental alcoholism on adolescent substance use.

Despite these speculations, however, supportive data concerning temperamental characteristics as mediators of parental alcoholism effects are still lacking. One attempt to test behavioral undercontrol as a mediator of alcohol use among adolescent COAs failed to find supportive data (Rogosch et al., 1990), although a recent study with college students and more comprehensive assessment did support this model (Sher et al., 1991). Thus, little is known about temperamental characteristics as mediators of parental alcoholism effects.

The current study focused on emotionality and sociability as two potential temperamental mediators of parental alcoholism effects. There were several reasons for this choice. First, most theoretical models of temperament recognize emotionality and

sociability as components (Goldsmith et al., 1987). Second, emotionality and sociability map readily onto the widely investigated adult personality dimensions of neuroticism and extraversion (Eysenck & Eysenck, 1969), providing relatively clear developmental links to adult personality. Third, emotionality and sociability relate clearly to the major explanatory variables that have been implicated in adolescent substance use. Sociability should directly influence an adolescent's risk of exposure to peer influences, and emotionality should directly influence affect regulation motives for substance use. Thus, although other temperamental mediators may also exist (most notably some sort of "behavioral undercontrol"), emotionality and sociability are of clear theoretical importance.<sup>1</sup>

In sum, the current study investigated parent monitoring, stress and negative affect, and adolescent temperament as mediating mechanisms underlying the effects of parental alcoholism on adolescent substance use. Because of theoretical interrelations among the mechanisms and to avoid overestimating the importance of any one pathway, we investigated the unique effects of each mediational pathway in the context of an integrative model. Methodologically, we attempted to address several limitations of previous COA studies. We used a large, actively recruited, community sample and directly verified parental alcoholism rather than relying on offspring report. We considered both lifetime diagnoses of parental alcoholism as well as current parental alcohol consumption. We disaggregated maternal and paternal alcoholism, and we controlled for the effects of co-occurring parent antisocial personality and depression.

### *Operationalizing the Three Mediating Mechanisms*

The integrative model that we tested is depicted in Figure 1. To operationalize the parenting model, we hypothesized that both lifetime parental alcoholism diagnoses and current parental alcohol consumption were associated with decreased parent monitoring of the adolescent's behavior. Decreased monitoring was thought to result in associations with drug-using peers and ultimately in adolescent substance use. Parental alcoholism and current parental alcohol consumption were hypothesized to directly affect adolescent substance use (e.g., through modeling and increased access to alcohol).

To operationalize the stress and negative affect regulation model, we hypothesized that parental alcoholism diagnoses and current parental alcohol consumption would be associated with elevations in environmental stress. Stress, in turn, was thought to result in a negative affective state that would either directly influence substance use or result in associations with drug-using peers and adolescent substance use.

To operationalize the temperament model, we hypothesized that lifetime parental alcoholism diagnoses (but not current parental alcohol consumption) were related to elevations in adolescent emotionality and sociability. Emotionality was then thought to increase the probability of negative affective states, which would lead to associations with drug-using peers and adolescent substance use. Sociability was thought to affect drug use by increasing exposure to drug-using peers.

Finally, because demographic factors and co-occurring parent psychopathology could affect adolescent substance use, we

controlled for the effects of child age, child gender, parent educational attainment, parent affective disorders, and parent antisocial personality disorders.

## **Method**

### *Subjects*

The total sample consisted of 454 adolescents ages 10.5 to 15.5 years (average age = 12.7 years) and their parents. Of these adolescents, 246 had at least one biological alcoholic parent who was also a custodial parent (COAs), and the remaining subjects were 208 demographically matched adolescents with no biological or custodial alcoholic parents (controls). Because the current study used reports from the child, mother, and father, 38 single-parent families (8.4% of the total sample) were excluded.<sup>2</sup> This resulted in a possible sample of 416 families.

<sup>1</sup> The major alternative to our choice of emotionality and sociability as temperamental mediators is behavioral undercontrol. Many different characteristics have been considered under this rubric (e.g., impulsivity, aggression, sensation-seeking, and overactivity). However, we did not focus on this construct because a clear theoretical specification of its components has not been provided, and it is unclear if it can be accurately described as a single construct (Windle, 1990). Moreover, there is no clear consensus among different theoretical models concerning which dimensions of behavioral undercontrol should properly be considered as part of temperament. Finally, many attempts to measure behavioral undercontrol rely on indicators of antisocial behavior. As Windle (1990) and Nathan (1988) have noted, under these circumstances, behavioral undercontrol may be simply an indicator of other deviant behaviors that are associated with substance use, rather than being a temperamental characteristic that predisposes an individual to substance use. For these reasons we did not test behavioral undercontrol as a temperamental mediator. However, future examinations of this construct are warranted if clear operational definitions can be derived.

<sup>2</sup> To assess whether the exclusion of single parent families (33 single mothers and 5 single fathers) introduced bias into the sample, comparisons were performed between two-parent and single-parent families (*t* tests and chi-squares). The groups were compared on all variables in Table 1 except for family composition and the percentages of interviewed mothers and fathers. Comparisons involving paternal factors were based either on maternal report (e.g., father's alcoholism) or on the small number of single fathers who participated. There were no significant differences between the single-parent and two-parent families for 18 of the 29 comparisons. The groups did not significantly differ in father's education, father's ethnicity, child gender, child age, father's affective disorder or antisocial personality, mother's affective disorder, father's frequency of alcohol or drug use in the past 3 months, mother's drug use in the past 3 months, father's monitoring of the adolescent's behavior, the adolescent's life stress in the past 3 months, the adolescent's sociability, and the adolescent's association with drug-using peers. Most important, the groups did not significantly differ on any of the adolescent's substance use outcomes. However, single-parent families had mothers with higher educational attainment, fewer Hispanic mothers, more maternal alcoholism and antisocial personality diagnoses, less maternal monitoring of the adolescent, greater maternal quantity and frequency of alcohol use in the past 3 months, less paternal alcoholism, higher quantity of paternal alcohol use in the past 3 months, higher levels of adolescent emotionality, and higher levels of adolescent negative affect (all *ps* < .10). The directions of the differences were not consistent in terms of putting one or the other group at higher risk. Moreover, the differences between the two groups were generally minor, and there were no significant differences in adolescent substance use outcomes.

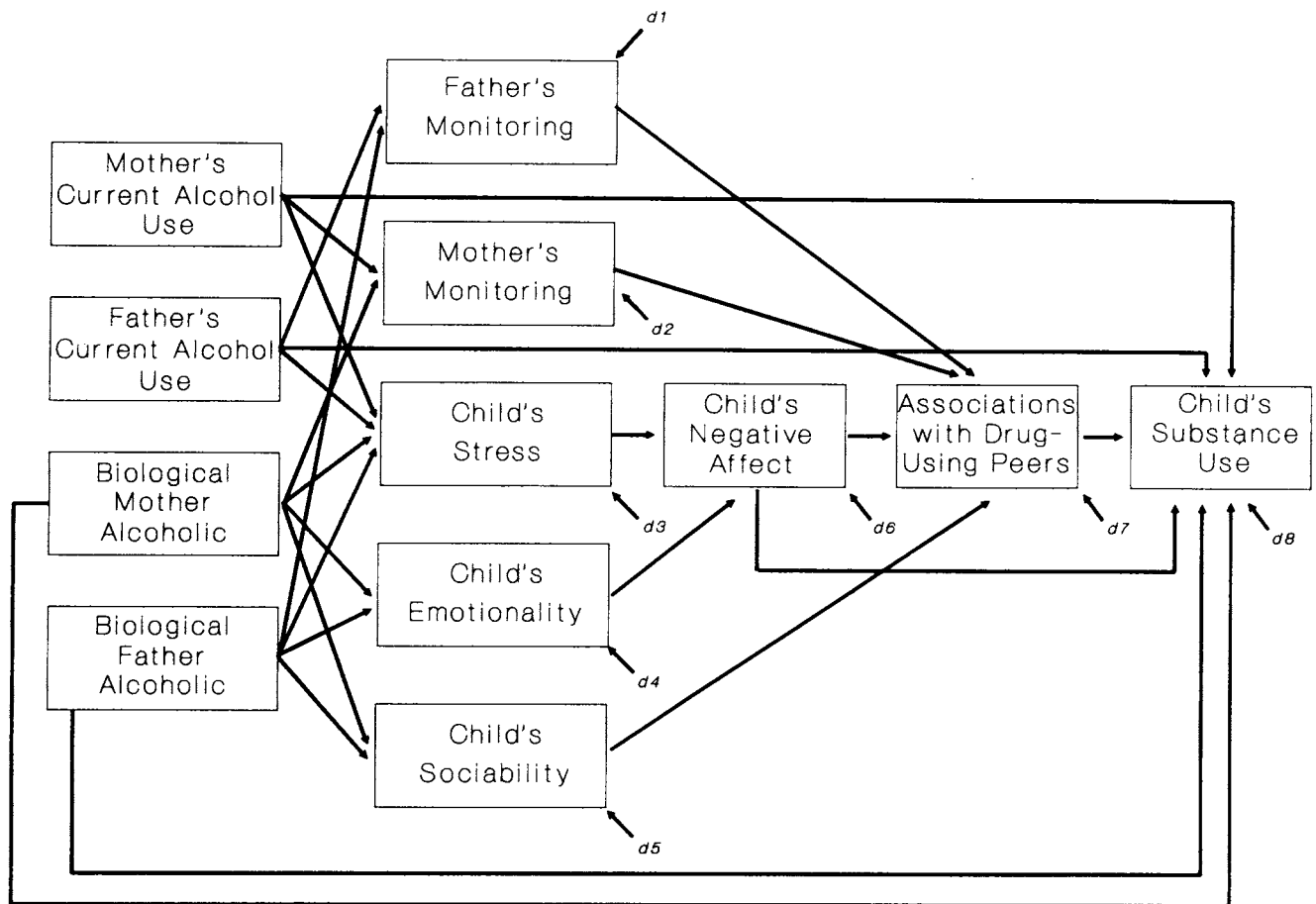


Figure 1. The hypothesized effects of parent alcoholism on child substance use through mediational pathways involving parental monitoring, stress–negative affect, and child temperament. (d1–d8 = structural disturbances.)

Sixty-six fathers and 21 mothers were not interviewed, and 2 families had incomplete data. Thus, there was a final sample of 327 families with complete data provided by two parents and the adolescent. Because of the subject loss, we estimated the structural models both with the smaller sample ( $n = 327$ ) and with the larger sample (using data imputation methods,  $n = 416$ ).

### Recruitment Procedures

The recruitment procedures are presented in detail elsewhere (Chassin et al., 1991; Chassin, Barrera, Bech, & Kossak-Fuller, 1992). COA families were recruited using court records ( $n = 103$ ), wellness questionnaires from a health maintenance organization ( $n = 22$ ), and community telephone surveys ( $n = 120$ ). One family was referred by a local Veteran's Administration hospital. Screening and recruitment were done by research team members (or by participating agencies when required because of confidentiality concerns).

COAs had to meet the following criteria: Anglo or Hispanic ethnicity, Arizona residency, age 10.5–15.5 years, English-speaking, and have no cognitive limitations that would preclude interview (e.g., severe mental retardation or psychosis). Finally, direct interview data had to confirm that a biological and custodial parent met *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.; *DSM-III*; American

Psychiatric Association, 1980) criteria for alcohol abuse or dependence (lifetime diagnoses using the Diagnostic Interview Schedule) or Family History–Research Diagnostic Criteria (FH–RDC), on the basis of reports by the other parent (if the alcoholic parent was not interviewed).

Demographically matched control families were recruited using telephone interviews. When a COA subject was recruited, reverse directories were used to locate families living in the same neighborhood. Families were screened to match the COA subject in ethnicity, family composition, target child's age (within 1 year), and socioeconomic status (using the property value code from the reverse directory). Direct interview data were used to confirm that neither biological nor custodial parents met *DSM-III* criteria (or FH–RDC criteria) for lifetime diagnoses of alcohol abuse or dependence.<sup>3</sup>

<sup>3</sup> Families who (on interview) reported indications of alcohol problems including either pathological patterns of use or associated impairment were eliminated from the control group, even though the symptoms were not severe enough to obtain a *DSM-III* alcoholism diagnosis. These consequences might indicate a subclinical alcohol problem or the beginning stages of an alcohol problem. Given that parents in these age ranges have not yet passed through the period of risk for late onset alcohol problems, some of these parents might be diagnosed as alcoholic at some future time (Pandina & Johnson, 1989).

Recruitment biases because of selective contact with subjects or subject refusals are discussed in detail elsewhere (see Chassin et al., 1991; Chassin et al., 1992). Although contact rates were low (38.3% from archival records and 44.2% from reverse directories), participation rates were high (72.8% of eligible COA families and 77.3% of eligible control families participated). Analyses to assess participation bias found that the sample was unbiased with respect to alcoholism indicators that were available in archival records (e.g., blood-alcohol level at the time of the arrest, Michigan Alcoholism Screening Test results). However, subjects who refused participation were more likely to be Hispanic and, if there was an arrest record, more likely to be married at the time of the arrest. Although the magnitude of the bias was small and unrelated to archival indicators of alcoholism, some caution is warranted in generalization.

### *Characteristics of the Alcoholic Sample*

The total alcoholic sample was typically characterized by an early onset of drinking problems, with 74.6% of the alcoholic fathers and 58.5% of the alcoholic mothers reporting drinking problems at or before age 25. Only a minority of the biological alcoholic parents reported receiving alcoholism treatment (31% of the biological alcoholic fathers and 33% of the biological alcoholic mothers). Some confidence in the representativeness of our alcoholic sample can be drawn by comparing our rates of comorbidity with recently reported epidemiological data (*Epidemiological Catchment Area Study* [ECA] data, Helzer & Pryzbeck, 1988). Our interviewed alcoholic men showed a prevalence of 5.5% for major depression (compared with 5% in the ECA data) and 16.4% for antisocial personality (compared with 15% in the ECA data). Our interviewed alcoholic women showed somewhat lower rates of major depression (12.1% in the current sample vs. 19% in the ECA data) and somewhat higher rates of antisocial personality (15.5% in the current sample vs. 10% in the ECA data).

### *Differences Between Alcoholic and Control Families*

The characteristics of the COA and control groups (for the two-parent families in the sample) are shown in Table 1. The samples did not differ in child gender, age, family composition, the percentage of custodial or biological fathers who were interviewed, the percentage of custodial mothers who were interviewed, maternal monitoring of adolescent behavior, adolescent sociability, or maternal frequency of current alcohol and drug use. However, the COA sample had lower levels of parent educational attainment, marginally higher proportions of Hispanics, a lower proportion of biological mothers interviewed, higher prevalence of parental psychopathology and current paternal alcohol and drug use, less paternal monitoring of adolescent behavior, more recent life stress events, higher adolescent emotionality and negative affect, and more associations with drug-using peers. Most important, adolescent COAs reported more alcohol and drug use in the past 3 months and more social consequences and dependency symptoms of alcohol and drug use in the past year than did controls (chi-square and *t*-test comparisons, see Table 1).

### *Extent of Substance Use in the Adolescent Sample*

Because of the young age of the sample, the prevalence of substance use was generally low (see Table 1). Accordingly, the current study is best viewed as examining substance use initiation (rather than substance abuse). However, these low prevalences should not be taken to

minimize the significance of substance use in this sample. Indeed, some researchers have suggested that any substance use occurring in this age group is of prognostic significance in terms of risk for later substance abuse (Robins & Pryzbeck, 1985). Moreover, use rates in our COA sample exceeded national data for older adolescents. For example, the National Household Survey (National Institute on Drug Abuse, 1989) found current (past month) use of alcohol for 25.2% of adolescents aged 12–17, whereas 34.1% of our COAs aged 12–15 used alcohol in the past 3 months. Comparable data for current marijuana use showed 6.5% of our COAs compared with 6.4% in the (older) national sample.

### *Procedure*

The procedures are described in detail elsewhere (Chassin et al., 1991). Data were collected using computer-assisted interviews with the adolescents and their parents. Interviewers were adults from the community with at least a bachelor's-level education who were recruited through local newspaper ads. Interviewers received a six-session training program including general elements of interviewing, specific training in the computer-assisted interview, and procedures for maintaining a standardized administration. Because all responses were closed-ended and all skip patterns and branching patterns were preprogrammed, interviewers did not make any diagnostic decisions. Interviewers were blind to the group membership of the family (although the interview itself revealed the extent of alcohol and drug use in the family).

To minimize contamination, family members were interviewed individually on one occasion by different interviewers (in all but 11 cases), and privacy was emphasized. Confidentiality was assured and reinforced with a Department of Health and Human Services (DHHS) Certificate of Confidentiality. One check on the validity of self-reports was obtained by comparing subjects' self-reported drunk driving arrests with court records. In support of the validity of the self-reports, 95.2% of the court subjects reported their driving while intoxicated arrests in the interview.

### *Measures*

The measures of interest were part of a larger interview battery and are summarized in Table 2. Child age, gender, and average level of parent educational attainment were used as control variables in the model.

*Parental alcoholism and associated psychopathology:* Lifetime *DSM-III* diagnoses of alcoholism (abuse or dependence), affective disorder (major depression or dysthymia), and antisocial personality (excluding items on deviant sexuality and child abuse and neglect) were obtained using a computerized version of the Diagnostic Interview Schedule (DIS, Version III; Robins, Helzer, Croughan, & Ratcliff, 1981). The DIS was designed to permit administration by lay interviewers in large epidemiological studies. Initial evaluation studies compared diagnoses obtained by lay interviewers with the DIS with those diagnoses obtained by psychiatrists who could also ask supplemental questions (Robins et al., 1981; Robins, Helzer, Ratcliff, & Seyfried, 1982). Alcohol abuse and dependency had one of the highest levels of agreement ( $kappa = .86$ ) and had excellent sensitivity ( $kappa = .86$ ) and specificity ( $kappa = .98$ ). Levels of agreement, sensitivity, and specificity were also adequate for antisocial personality ( $kappas = .63, .75, \text{ and } .96$ ) and major depression ( $kappas = .63, .80, \text{ and } .84$ ). Dysthymia was not evaluated. In this study, diagnoses were not excluded if other disorders co-occurred or preceded them (cf. Burnam et al., 1987).

In cases in which only one parent was interviewed, lifetime alcoholism diagnoses for the noninterviewed parent were made using reports by the other parent according to FH-RDC. For noninterviewed par-

---

By eliminating families with indications of drinking problems from the control group, this possibility becomes less likely.

Table 1

*Characteristics of Children of Alcoholics (COA) and Control Two-Parent Families*

Measure	COA	Control	Measure	COA	Control
<b>Demographics</b>			<b>Parent current alcohol and drug use</b>		
Father's education			Father's frequency of alcohol use in past 3 months <sup>b</sup>	2.85	2.03***
% High school or less	41.7	24.7****	Father's quantity of alcohol use in past 3 months <sup>c</sup>	3.37	1.55****
% College graduate	19.8	31.0	Father's drug use in past 3 months <sup>d</sup>	.09	.02***
Mother's education			Mother's frequency of alcohol use in past 3 months <sup>b</sup>	1.58	1.31
% High school or less	47.4	36.3**	Mother's quantity of alcohol use in past 3 months <sup>c</sup>	1.64	1.09****
% College graduate	17.4	23.6	Mother's drug use in past 3 months <sup>d</sup>	.04	.01
Father's ethnicity (% Hispanic)	28.2	20.6*	<b>Hypothesized mediating variables<sup>e</sup></b>		
Mother's ethnicity (% Hispanic)	25.7	18.5*	Father's monitoring of child's activities <sup>f</sup>	3.92	4.15***
Child gender (% female)	47.6	47.6	Mother's monitoring of child's activities <sup>f</sup>	4.45	4.54
Child age	12.6	12.8	Child's life stress in past 3 months (number of events) <sup>g</sup>	3.94	2.49****
<b>Family composition</b>			Child's emotionality <sup>h</sup>	2.50	2.36**
% Step families	13.2	10.6	Child's sociability <sup>h</sup>	3.95	3.90
% With 2 biological parents	86.8	89.4	Child's negative affect (past 3 months) <sup>i</sup>	2.15	2.03*
% Custodial fathers interviewed	84.6	83.6	Peer drug use and tolerance of drug use <sup>j</sup>	.14	-.16***
% Custodial mothers interviewed	93.8	96.3	<b>Child's current substance use</b>		
% Biological fathers interviewed	80.2	78.3	Alcohol and substance use in past 3 months <sup>k</sup>	.27	.11***
% Biological mothers interviewed	87.2	93.7**	% Used alcohol in past 3 months	28.6	13.2****
<b>Parent psychopathology (lifetime DSM-III diagnosis)<sup>a</sup></b>			% Used drugs in past 3 months	8.4	2.1***
% Fathers with affective disorders	10.9	2.5***	% Reporting consequences or dependency symptoms in past year	19.4	7.4****
% Fathers with antisocial personality	15.1	0.6****			
% Mothers with affective disorders	13.6	4.4***			
% Mothers with antisocial personality	2.3	0.0**			

Note. Sample sizes vary across constructs because of missing data.  $N = 416$  two-parent families.

<sup>a</sup> DSM-III = *Diagnostic and Statistical Manual of Mental Disorders, Third Edition*. Diagnoses only for interviewed custodial parents. Affective disorders include Major Depression or Dysthymia.

<sup>b</sup> Frequency of alcohol use response scale: (0) *not at all*, (1) *once or twice*, (2) *1 day a month*, (3) *2-3 days a month*, (4) *1 day a week*, (5) *2 or 3 days a week*, (6) *4 or 5 days a week*, (7) *every day*.

<sup>c</sup> Quantity of alcohol use response scale: (0) *none*, (1) *1 to (7) 7-8*, (8) *9 or more drinks per occasion*.

<sup>d</sup> Current (3 month) drug use response scale: (0) *never* to (8) *every day*.

<sup>e</sup> Unlike the constructs used in the model, scores here are untransformed to aid in interpretation. Also for ease of interpretation (and unlike variables in the model), multiple reporter constructs are averaged across reporters rather than based on factor score regression weights. Note also that in the model, parent alcoholism is disaggregated into maternal and paternal alcoholism.

<sup>f</sup> Scores are on a 5-point scale with higher values reflecting greater monitoring.

<sup>g</sup> Scores are averaged across mother, father, and child reports.

<sup>h</sup> Scores are averaged across mother, father, and child reports. Scores are on a 5-point scale with higher values indicating higher levels of emotionality and sociability.

<sup>i</sup> Scores are an average of child's self-reported internalizing symptomatology, self-esteem, and perceived control. Scores are on a 5-point scale with higher values reflecting higher levels of negative affect.

<sup>j</sup> Higher values indicate greater peer use and tolerance for use.

<sup>k</sup> Average frequency of alcohol use, heavy alcohol use, and other drug use response scale: *no use* (0) to *daily use* (7).

\*  $p < .10$ . \*\*  $p < .05$ . \*\*\*  $p < .01$ . \*\*\*\*  $p < .001$ .

ents, no information was available concerning affective disorders or antisocial personality. For the current analyses, the lifetime alcoholism diagnoses of the biological father and the biological mother were each considered (separately) as dichotomous variables. Among the two-parent families, there were 44 biological mothers and 206 biological fathers who met these alcoholism criteria.

Parent affective disorders and antisocial personality disorders were treated (separately) as control variables in the model. For each family, diagnoses of affective disorders and antisocial personality were considered as dichotomous variables, either present (in one or both parents) or absent.

**Current parental alcohol and drug use.** Because the alcoholism diagnoses were lifetime measures, it was also necessary to assess the extent to which parental alcohol and other drug use persisted in the

child's current environment. This was assessed using parents' self-reported frequency and quantity of alcohol use and frequency of use of eight illicit drugs in the past 3 months. Frequency of drinking responses ranged from (0) *not at all* to (7) *every day*; quantity responses ranged from (0) *none* to (8) *nine or more drinks per occasion*. In the structural model, parents' current drinking was represented by quantity and frequency products that were calculated separately for each parent. A natural log power transformation was used to reduce skewness (after transforming, skewness was .23 for father's alcohol use and .83 for mother's alcohol use).

The average frequency of illicit drug use in the past 3 months (on a 9-point scale from 0 [*no use*] to 8 [*daily use*]) was calculated for each parent and is presented in Table 1. However, because of extremely low rates of current maternal drug use (97% reporting no use in the past 3

months) and the lack of relation between current paternal drug use and the outcome measure ( $r = .06$ , n.s.), current parental drug use was not considered further.

*Mother and father monitoring of child behavior.* The extent to which parents monitored their child's behavior in the past 3 months was assessed by mother and father self-report (three items, e.g., "I had a pretty good idea of [the child's] plans for the day"). Internal consistency (coefficient  $\alpha$ ) was .75 for father's monitoring and .79 for mother's monitoring. A single score was computed for each parent using the mean of the three self-report items.

*Child's current life stress.* Parents and children reported whether each of 22 life events had occurred to the child within the past 3 months. Children also reported on four additional peer-related events. Although life events measures represent only one approach to assessing life stress, they had several advantages for the present study. First, we could use events that had been previously rated as uncontrollable by the child. Because these events are not likely to have been "caused" by the child's personal characteristics, this aids in the interpretative clarity of the stress mediator. Second, a life events approach was appropriate to our focus on gathering multiple-reporter data when possible. Relatively discrete, significant negative life events could be observed and reported by both parents and child.

Events were taken from the Children of Alcoholics Life Events Schedule (COALES; Roosa et al., 1988) and the General Life Events Schedule for Children (GLESC; Sandler, Ramirez, & Reynolds, 1986), supplemented with several items from other child life events schedules. The GLESC and COALES items had previously been rated by experts as negative and uncontrollable by the child (Roosa et al., 1988). Sample events included the following: parent arrested, parent lost job, family moved, close friend died, family had financial difficulty. Each informant's score was a count of stressful events that had occurred to the child. Correlations among reporters were as follows: mothers with fathers, .54; mothers with children, .49; and fathers with children, .38. For the structural modeling, the life stress variable was a multiple-reporter composite manifest variable created using factor score regression weights (see Results section for details).

*Child temperament: Emotionality and sociability.* Emotionality and sociability were measured using a modification of the adult version of the Emotionality, Activity, and Sociability Temperament Scale (EAS; Buss & Plomin, 1984). To make the content and wording appropriate for young adolescents, three items were deleted from the emotionality scale and one item was deleted from the sociability scale. Two items were added to the sociability scale: "I make friends easily" and "I tend to be shy." Children self-reported these items and parents also reported on the target child. Preliminary analyses indicated that two items (one on each scale) failed to adequately discriminate between the temperament constructs and were therefore deleted (Molina, Pillow, & Chassin, 1989). Coefficient  $\alpha$ s for the child, mother, and father were .72, .78, and .76 for emotionality and .46, .62, and .60 for sociability. Correlations among reporters for emotionality were as follows: mothers with fathers, .45; mothers with children, .27; and fathers with children, .25. Correlations among reporters for sociability were as follows: mothers with fathers, .48; mothers with children, .38; and fathers with children, .30. For the structural modeling, the temperament variables were multiple-reporter composite manifest variables created using factor score regression weights (see Results section for details).

*Child's negative affect.* Negative affect was measured using child self-report of internalizing symptomatology, self-derogation, and perceived loss of control in the past 3 months. Internalizing symptomatology was assessed with seven items from the Achenbach Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1981; coefficient  $\alpha = .78$ ). These were items that loaded on Achenbach's internalizing factor for both boys and girls, ages 12–16. Sample items included the following: felt lonely; cried a lot; and felt nervous, high-strung, or tense. Perceived loss of control was assessed by three items from Newcomb

and Harlow (1986; coefficient  $\alpha = .73$ ; e.g., "I felt I was not in control of my life"). Self-derogation was assessed using seven items from Rosenberg's (1979) scale (coefficient  $\alpha = .81$ ; e.g., "I feel I do not have much to be proud of"). Intercorrelations among the three dimensions ranged from .50 to .65. For the structural modeling, child's negative affect was a composite manifest variable created using factor score regression weights (see Results section for details).

*Associations with drug-using peers.* Children estimated how many of their friends used alcohol, marijuana, and other drugs both occasionally and regularly. These items were adapted from the Monitoring the Future study (Johnston, O'Malley, & Bachman, 1988) with response options ranging from *none* (0) to *all* (5) on a 6-point scale. Subjects also reported how their close friends would feel about their using marijuana, alcohol, and other drugs both occasionally and regularly. Response options ranged from *strongly disapprove* (1) to *strongly approve* (5) on a 5-point scale. Internal consistency (coefficient  $\alpha$ ) was .92 for the six-item peer substance use measure and .93 for the seven-item peer tolerance of substance use measure. Adolescents' perceptions of peer substance use and peer tolerance of substance use were highly correlated ( $r = .65$ ). For the modeling, each scale was standardized, and the two scales were averaged to represent the peer environment.

*Dependent measure: Adolescent substance use.* The dependent measure was the adolescent's self-reported frequency of substance use in the past 3 months. Subjects reported their frequency of consumption of beer or wine and hard liquor (two items), frequency of consumption of five or more drinks in a row (one item), frequency of getting drunk on alcohol (one item), and frequency of use of eight illicit drugs. All items were close-ended with response options ranging from (0) *not at all* to (7) *every day*. A single substance use score was calculated by summing the responses to the 12 items.

As with most research on adolescent substance use, the current study relied on self-report data. Previous studies have generally supported the validity of adolescent self-reported substance use (cf. Winters, Stinchfield, Henly, & Schwartz, 1991), although some data suggest that conditions of anonymity or perceptions that self-reports will be verified minimize underreporting (Murray & Perry, 1987). Anonymity is not feasible in a longitudinal interview study, and an atmosphere of "checking up" on the adolescent's reports was considered a threat to interviewer rapport and long-term subject retention in the current study. Accordingly, we attempted to maximize honest reporting by providing private interview conditions, by using the rapport established with the interviewer, and by reinforcing confidentiality through the DHHS Certificate of Confidentiality.

Also as expected with measures of substance use in early adolescence, the dependent measure was poorly distributed. Accordingly, a normalizing transformation was used to reduce skewness (skewness was 6.49 for the untransformed variable and 1.75 following the normalizing transformation).

## Results

### *Effect of Parental Alcoholism on Adolescent Substance Use*

Before testing the hypothesized mediating mechanisms, it was necessary to show that the predictor variable was related to the outcome (Baron & Kenny, 1986). That is, we first had to show that parental alcoholism was significantly related to child's substance use. Table 1 presents the rates of child's current alcohol and drug use and substance use–related social consequences or dependency symptoms for COAs and controls. We compared the groups using chi-squares and calculated relative risk indices to assess the magnitude of the effects. Compared

Table 2  
*Variables in the Current Model of Child Substance Use*

Construct	Reporter	Item pool	Construct formation
Parent alcoholism and associated psychopathology			
Biological mother's lifetime alcoholism	Mother or other custodial parent informant	DIS or FH-RDC	Diagnostic categorization: 0 = biological mother nonalcoholic, 1 = biological mother alcoholic (alcohol abuse or dependence)
Biological father's lifetime alcoholism	Father or other custodial parent informant	DIS or FH-RDC	Diagnostic categorization: 0 = biological father nonalcoholic, 1 = biological father alcoholic (alcohol abuse or dependence)
Parents' lifetime affective disorders	Parent self-report	DIS	Diagnostic categorization: 0 = neither parent has diagnosis, 1 = either parent has diagnosis
Parents' lifetime antisocial disorder	Parent self-report	DIS	Diagnostic categorization: 0 = neither parent has diagnosis, 1 = either parent has diagnosis
Parent current alcohol consumption			
Mother's quantity and frequency of alcohol use in past 3 months	Mother self-report	1 Quantity and 1 frequency of alcohol use item	Product of the 2 alcohol use items
Father's quantity and frequency of alcohol use in past 3 months	Father self-report	1 Quantity and 1 frequency of alcohol use item	Product of the 2 alcohol use items
Parental monitoring			
Mother's monitoring	Mother self-report	3 Maternal monitoring items	Mean of 3 items
Father's monitoring	Father self-report	3 Paternal monitoring items	Mean of 3 items
Current life stress			
Child life stress in past 3 months	Mother	22 Life event items	Factor score regression composite
	Father	22 Life event items	
	Child	26 Life event items <sup>a</sup>	
Emotionality and sociability			
Child emotionality	Mother	8 EAS emotionality items	Factor score regression composite
	Father	8 EAS emotionality items	
Child sociability	Child	4 EAS sociability items	Factor score regression composite
	Mother	4 EAS sociability items	
	Father	4 EAS sociability items	
Child			
Negative affect			
Child negative affect in past 3 months	Child	7 CBCL internalizing items 3 Perceived loss of control items 7 Rosenberg self-esteem items	Factor score regression composite



Table 2 (continued)

Construct	Reporter	Item pool	Construct formation
Peer substance use and peer tolerance of substance use			
Peer use and tolerance of substance use	Child	6 Perceived peer use items 7 Perceived peer tolerance of use items	Mean of 2 standardized subscale scores
Child substance use			
Child substance use in past 3 months	Child	2 Alcohol use items 2 Heavy alcohol use items 8 Drug use items	Sum of 12 items

Note. DIS = Diagnostic Interview Schedule; FH-RDC = Family History-Research Diagnostic Criteria (FH-RDC were used to determine diagnostic categorization for noninterviewed parents); EAS = Emotionality, Activity, Sociability Temperament Scale; CBCL = Achenbach Child Behavior Checklist.

<sup>a</sup> Four peer-related life event questions were administered to children only.

with children without an alcoholic parent, children with an alcoholic parent were 2.17 times more likely to use alcohol,  $\chi^2(1) = 13.55$ ,  $p < .001$ ; 3.96 times more likely to use illicit drugs,  $\chi^2(1) = 6.57$ ,  $p < .01$ ; and 3.33 times more likely to report a consequence or dependence symptom,  $\chi^2(1) = 5.89$ ,  $p < .05$ . Thus, parental alcoholism was significantly related to adolescent substance use, and the effects were of small to moderate magnitude. To precisely parallel the variables in the structural model,  $t$  tests were performed assessing the effect of maternal and paternal alcoholism diagnoses on the continuous measure of child substance use in the past 3 months. There were significant effects of both father's and mother's alcoholism diagnoses,  $t(325) = 2.85$  and 2.35, respectively, both  $ps < .02$ .

### Creation of Manifest Variables

Before estimating the hypothesized structural model, preliminary analyses were conducted to construct the variables used in the model. The exogenous variables were used exactly as described in the Method section (see Table 2). Four endogenous variables (child's substance use, peer substance use, and maternal and paternal monitoring) were used as described in the Method section. To correct for measurement error, their associated error terms were set to 1 – [the reliability  $\times$  the variance of the indicator] (Bollen, 1989). However, several endogenous variables involved either multiple reporters (environmental stress, child's emotionality, and child's sociability) or multiple indicators (child's negative affect). Inclusion of these as latent variables in the structural model was untenable given the required number of parameters and current sample size. Accordingly, manifest variables were created preserving information from the multiple reporters and multiple indicators.

**Multiple-reporter constructs: Environmental stress, child's sociability and child's emotionality.** To create manifest variables using information from multiple reporters, a multitrait-multimethod model was estimated<sup>4</sup> with stress, emotionality, and sociability representing the traits and mother, father, and child's report representing the methods. However, this model was numerically ill-conditioned as is common with many three-trait, three-method models with correlated factors (Marsh, 1990).

When this occurs, a close approximation of this technique is to estimate a measurement model without the methods factors and to correlate the relevant measurement errors across reporters (Marsh, 1990). This three-latent variable measurement model was estimated, and the model reproduced the observed covariance matrix very well,  $\chi^2(12) = 13.28$ ,  $p > .35$  (Bentler-Bonnet normed index [BBI; Bentler & Bonett, 1980] = .98, Tucker-Lewis Index [TLI; Tucker & Lewis, 1973] = .99). Models are generally considered to adequately reproduce the data when the BBI and TLI are between .90 and 1.00. The current findings thus indicate a very good fit of the model to the observed data. All indicators loaded significantly on the corresponding latent variables, and examination of the standardized residuals and modification indices reflected no problems in model fit. On the basis of the results of this model, multiple-reporter constructs of stress, emotionality, and sociability were formed. Factor score regression weights were taken from the measurement model and used to create multiple-reporter linear composite manifest variables representing stress, emotionality, and sociability (see Loehlin, 1987, for a more detailed discussion of factor score regressions).<sup>5</sup> Composite reliabilities were calculated for each manifest variable using the estimates of error and total variance produced by the measurement model (Lord & Novick, 1968, p. 86). The error terms for the manifest variables were then set to 1 – [the reliability  $\times$  the variance of the indicator].

**Multiple-indicator construct: Child's negative affect.** To construct a manifest variable for child's negative affect, a one-factor measurement model was estimated using the child's self-report of internalizing symptomatology, self-esteem, and perceived control as indicators.<sup>6</sup> The resulting factor score

<sup>4</sup> All measurement and structural covariance models were estimated using LISREL-7 (Jöreskog & Sörbom, 1989).

<sup>5</sup> Factor score regression weights and the sample covariance matrix can be obtained from Laurie Chassin.

<sup>6</sup> There were also multiple reporters available for the child's negative affect (reported by mother, father, and child). However, when this three-indicator latent variable was included in the measurement model with sociability, emotionality, and environmental stress, neither the mother's nor father's reports significantly loaded on this construct.

regression weights were used to construct a linear composite of the three negative affect indicators. The reliability of this composite was estimated using the method discussed earlier. The error term was similarly set to  $1 - [\text{the reliability} \times \text{the variance of the indicator}]$ .

### Structural Model

**Specification.** Before estimating the hypothesized model depicted in Figure 1, it was first necessary to account for any variance attributable to the background control variables (parent antisocial disorder, parent affective disorder, child's age, child's gender, and parents' education). To accomplish this, the hypothesized paths from the nonbackground variables were freely estimated whereas all paths from background variables were fixed at zero. This model was then run iteratively, and on each run the path for the background variable with the largest modification index greater than 5.0 was freed. This process was repeated until there were no modification indices for background variables greater than 5.0. This procedure resulted in freeing seven paths from the background control variables to the endogenous variables. These paths were as follows: parent antisocial disorder to father's monitoring; parent affective disorder to child's environmental stress and to child's emotionality; child's gender to father's monitoring; and child's age to mother's monitoring, to peer substance use, and to child's negative affect.<sup>7</sup> The final step before estimating the hypothesized model was to allow correlated structural disturbances among father's monitoring, mother's monitoring, environmental stress, child's emotionality, and child's sociability.

**Overall adequacy of the hypothesized model.** The hypothesized model depicted in Figure 1 was tested (including the seven aforementioned paths from the background variables) and resulted in a  $\chi^2(59) = 95.56$ ,  $p > .002$ , TLI = .92, BBI = .92. Both the BBI and TLI are above the .90 cutoff, indicating an adequate fit of the model to the data. Next, the modification indices were examined to determine if the estimation of additional structural paths would significantly improve the fit of the model. This was done because if the model is misspecified, the tests of specific path coefficients may be incorrect. Paths were added to the model if the modification indices revealed that the path would make a difference in the  $\chi^2$  of at least 5.0 and if the path was consistent with the theoretical flow of the model. Only three paths were found that fit these criteria: (a) a path from child's sociability to child's negative affect, (b) a path from mother's alcoholism diagnosis to father's monitoring, and (c) a path from father's monitoring to child's substance use. Estimation of all three paths resulted in a significant decrease in the overall model  $\chi^2$ . After these three paths were included, an examination of the modification indices and standardized residuals indicated no further specification problems. The final model depicted in Figure 2 resulted in a  $\chi^2(56) = 66.63$ ,  $p > .16$ , BBI = .95, and TLI = .98, thus indicating that the estimated model fit the observed data very well.

Significant paths (excluding those from the background variables) are shown in Figure 2. As can be seen in the figure, there was support for all three mediating mechanisms. In terms of parenting, mother's alcoholism diagnosis was associated with lower levels of parent monitoring (a significant relation with father's monitoring and marginally significant for mother's monitoring). Lowered parental monitoring, in turn, was related to the adolescent's affiliation with a drug-using peer group (significant effects for father's monitoring and a marginally significant effect for mother's monitoring). Associations with drug-using peers then significantly predicted adolescent substance use.

In terms of the stress mechanism, both mother's and father's alcoholism diagnoses were significantly related to elevated environmental stress which, in turn, was significantly related to elevated negative affect. Negative affect significantly raised risk for substance use outcomes both through a direct effect and an indirect effect (by raising risk for associations with drug-using peers).

There was mixed support for temperamental mediators. Mother's alcoholism was significantly associated with heightened emotionality, which was associated with higher levels of negative affect. However, there were no significant relations between parental alcoholism and temperamental sociability. As predicted, sociability was significantly related to increased associations with drug-using peers. Sociability was also negatively related to the child's negative affect.

Significant relations between parental alcohol variables and the mediating variables were generally found for alcoholism diagnoses but not for current alcohol consumption. However, there was a significant direct relation between mother's current drinking and child's substance use, such that mothers who drank at higher levels had adolescents with greater substance use.

Finally, significant effects of parental alcoholism involved maternal rather than paternal alcoholism (with the exception of a significant relation between paternal alcoholism and environmental stress). These findings should be considered in light of the larger multivariate model, particularly the effects of other parent psychopathology. There were significant effects of parent antisocial disorders on father's monitoring and significant effects of parent affective disorders on stress and emotionality. Thus, it must be remembered that paths from parental alcoholism diagnoses reflect only the unique effects of parental alcoholism above and beyond other risk factors.

In terms of the magnitudes of effect, the multiple  $R^2$  values are presented in Table 3. The final model accounted for a large amount of the variance associated with the child's substance use. However, this large magnitude of effect is expected given the inclusion of the child's age and peer drug use in the model (two strong influences on adolescent drug use). As shown in Table 3, the model accounted for very little variance associated with the child's sociability and mother's monitoring. These weak effects are also reflected in the lack of statistically significant

Thus, it was not possible to construct a multiple-reporter measure for this variable. Because the child was viewed as the best expert concerning his or her internal, affective state, only child reports were used.

<sup>7</sup> Although this procedure does capitalize on chance in estimating the effects of the background variables, it provides a stringent test of the theoretical variables of interest in the model.

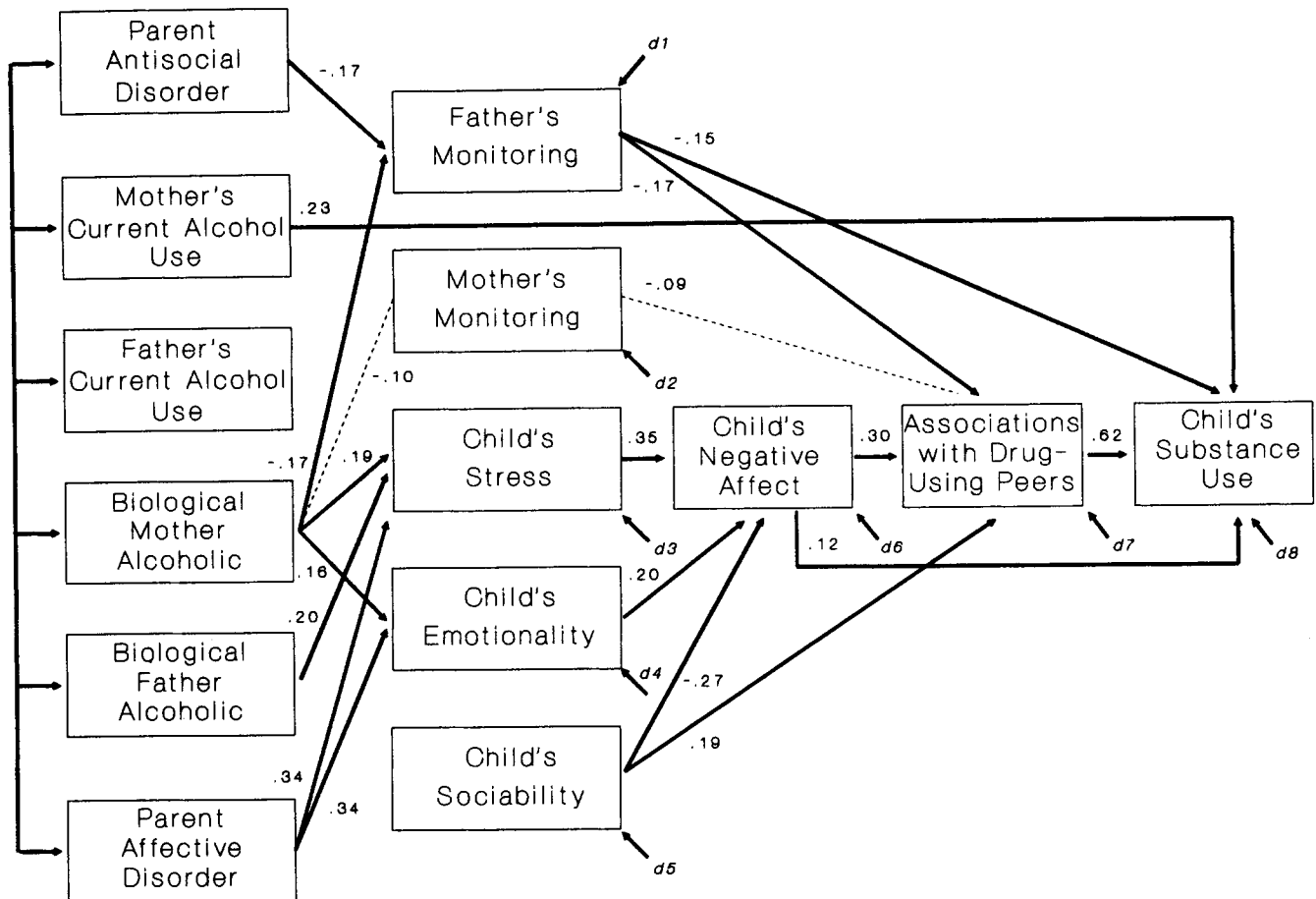


Figure 2. The obtained structural model representing the effects of parent alcoholism on child substance use through mediating mechanisms involving parent monitoring, stress-negative affect, and child temperament. (d1–d8 = structural disturbances. Standardized path coefficients are shown. Solid lines indicate significant effects at  $p < .05$ . Dashed lines indicate significant effects at  $p < .10$ . Because of space limitations and for presentational ease, several features of the estimated model are not shown. These include any nonsignificant pathways included in the final model, three background variables [child age, child sex, and average parent education level] and their associated pathways, and correlations between the structural disturbances [i.e., d1, d2, d3, d4, and d5].)

cant prediction of these variables described earlier. However, the model accounted for a moderate amount of variance associated with the remaining endogenous variables (father's monitoring, child's stress, child's emotionality, child's negative affect, and child's associations with drug-using peers).

**Testing the total indirect effects.** As described earlier, both maternal and paternal alcoholism showed significant mediational links to child substance use. However, significant individual links do not necessarily imply that the overall mediational pathways are statistically significant (Bollen, 1989). Thus, tests were performed to evaluate the statistical significance of the overall pathways.  $T$  ratios were computed for the total indirect effects of paternal and maternal alcoholism diagnoses. These  $t$  ratios were based on the estimates of the total indirect effects and the standard errors of these effects as provided by LISREL-7 (Jöreskog & Sörbom, 1989). There were statistically significant mediational pathways underlying the effects of both ma-

ternal and paternal alcoholism,  $t_s = 2.48$  and  $3.27$ , respectively, both  $p_s < .03$ .

#### Patterns of Missing Data

The sample used for the model tested consisted of families in which both parents and the child were interviewed. To determine whether these families were a biased subsample of the two-parent families, we compared families who were missing data for one (noninterviewed) parent ( $n = 89$ ) with families with data from both parents ( $n = 327$ ). In terms of parent characteristics, only two differences were found between the two groups. Families in which one parent was not interviewed were less likely to receive parental affective disorder diagnoses,  $\chi^2(1) = 5.85$ ,  $p < .05$ , than were families in which both parents were interviewed, and mother's self-reported lower educational attainment when the father was not interviewed,  $F(1, 391) =$

Table 3  
Multiple  $R^2$  Values for All Endogenous Variables

Variable	$R^2$
Father's monitoring	.10
Mother's monitoring	.04
Stress	.24
Emotionality	.17
Sociability	.01
Negative affect	.39
Associations with drug-using peers	.35
Child's substance use	.59

17.07,  $p < .01$ . To assess bias in child variables resulting from missing-parent data, a series of 2 (alcoholic parent vs. no alcoholic parent)  $\times$  2 (both parents interviewed vs. one parent interviewed) analyses of variance (ANOVAs) were performed. There were only three significant main effects or interactions involving missing parents out of 19 ANOVAs. First, children from families with noninterviewed parents reported lower levels of sociability,  $F(1, 412) = 5.83$ ,  $p < .05$ . Second, there was both a main effect and interaction effect obtained for the child's report of stress,  $F(1, 412) = 7.30$  and  $7.14$ , respectively, both  $ps < .01$ . Children who had both a noninterviewed parent and an alcoholic parent reported the highest level of stress.

*Estimating the model with imputed data.* Because of the potential bias introduced by using only families with complete data, the final structural model was reestimated on the total sample of two-parent families ( $n = 416$ ) using data imputation methods. Data imputation was possible in the present study because we had reports on almost every variable from multiple informants. This allowed us to formulate regression equations in which one informant's report of a mediating variable was regressed onto the reports of that variable by the two other informants. The data imputation process was accomplished using the BMDP (Version 5) AM program. This procedure computes initial estimates through a regression procedure followed by a maximum likelihood reestimation of the regression model until it converges (see also Little & Rubin, 1987).

After completing the imputation of missing data, the final structural model was estimated using the total sample of 416 two-parent families. Results largely replicated the findings reported for the sample of 327 families with complete data. The model with imputed data achieved a similar overall fit,  $\chi^2(56) = 93.86$ ,  $p > .001$ , BBI = .93, and TLI = .92. Using the criteria for adding paths discussed earlier, examination of the modification indices revealed that two paths would be added: a path from child's emotionality to peer substance use (such that high emotionality was associated with greater peer substance use) and a path from father's current quantity and frequency of alcohol use to child's negative affect (such that higher paternal consumption was associated with higher levels of child negative affect). Examination of the  $t$  values showed that, with only one exception, all paths reported earlier as significant remained so. The one change was that the path from child's emotionality to child's negative affect was nonsignificant in the model with the imputed data.

Two paths that were marginally significant in the original

model were significant in the model with imputed data. Both of these paths involved mother's monitoring of the child's behavior. In the model with imputed data, mother's alcoholism was significantly associated with lower levels of maternal monitoring, and lower maternal monitoring was associated with higher levels of peer drug use. Finally, one path that was nonsignificant in the original model was significant in the imputed model. In the model with imputed data, father's alcoholism was significantly associated with adolescent's heightened emotionality.

Thus, reestimating the model using imputed data for the total sample of two-parent families closely replicated the findings reported earlier. Results involving the parenting mediators and the stress and negative affect mediators were replicated. The only substantive change involved emotionality as a mediator, in that the data imputation suggested links between emotionality and paternal alcoholism (as well as maternal alcoholism as in the original model) and suggested that the effects of emotionality bypassed negative affect to predict associations with drug-using peers.

### Gender Differences in the Model

Because some researchers have suggested that there are gender differences in determinants of adolescent substance use, we tested for differences in the final model across gender. The sample was split by gender and all parameter estimates were specified to be invariant for boys and girls. The resulting fit,  $\chi^2(185) = 262.08$ ,  $p < .01$ , indicated significant gender differences.

Because of sample size constraints, a full series of cross-group comparisons was not feasible. However, examination of the modification indices revealed that two paths significantly differed for boys and girls: mother's current alcohol use to stress (when simultaneously freely estimated,  $b = .09$ ,  $p < .05$  for boys;  $b = -.07$ ,  $p < .10$  for girls) and age to negative affect (when simultaneously freely estimated,  $b = .19$ ,  $p < .01$  for boys;  $b = .04$ , n.s. for girls). Three paths that were not estimated in the overall structural model had high modification indices for boys: parent antisocial disorder to stress (when freely estimated,  $b = .16$ ,  $p < .01$ ), stress to associations with drug-using peers (when freely estimated,  $b = .22$ ,  $p < .01$ ), and parent affective disorders to associations with drug-using peers (when freely estimated,  $b = .23$ ,  $p < .01$ ). Finally, only one path not estimated in the overall structural model had a high modification index for girls: age to substance use (when freely estimated,  $b = .17$ ,  $p < .01$ ). These paths do not have important substantive implications for the current mediational hypotheses. However, complete cross-group comparisons with larger sample sizes might produce different findings.

### Discussion

The goal of this study was to test potential mediators of the effects of parental alcoholism on substance use during early to middle adolescence. The first notable finding is simply that there was a significant effect of parental alcoholism on adolescent substance use. This is in contrast to some recent reports that failed to find elevated alcohol use in adolescent COAs (e.g., Johnson et al., 1989; Pandina & Johnson, 1989). Elsewhere we

discuss the differences in sampling and methodology that may account for these discrepancies (Chassin et al., 1991). For present purposes, we conclude that when an actively recruited community sample is studied, risk is defined by parents' *DSM-III* alcohol abuse or dependency, and when parental alcoholism is directly assessed, parental alcoholism has a significant effect on adolescent substance use.

As in other studies of COA risk, the effects of parental alcoholism in the current study were of small to moderate magnitude (relative risk indices from 2.17 to 3.96). As Sher (1983) noted, the high-risk design (comparing COAs and non-COAs) may underestimate the magnitude of parental alcoholism effects because the COA group is presumed to be heterogeneous—to consist of individuals who are truly at risk and individuals who are not. This heterogeneity reduces the size of the differences observed between COAs and non-COAs, producing the small to moderate effect sizes in high-risk research.

Given that parental alcoholism does indeed raise risk for adolescent substance use, the current study investigated three potential mediating mechanisms of this effect. We hypothesized that risk for substance use was mediated through reductions in parent monitoring, through increases in environmental stress and negative affect, and through temperamental dispositions toward sociability and emotionality. With the exception of temperamental sociability, our findings supported these mediational mechanisms. In reaching that conclusion, it is important to note that these findings were produced even when the effects of co-occurring parental pathology and demographic background characteristics were controlled, that the data were provided by multiple reporters to minimize response biases, and that similar findings were produced using the subsample of families with complete data and the entire sample of two-parent families with imputation of missing data. These features increase confidence in the robustness of the conclusions. Our findings have implications for each of the mediating mechanisms.

### *Implications for Parenting and Socialization Mechanisms*

In terms of parenting, the findings suggest that decrements in parental monitoring mediate the relation between parental alcoholism and adolescent substance use. Alcoholic parents were less likely to monitor their adolescent's activities, and this lack of monitoring was related to the adolescent's affiliation with drug-using peers. A similar effect has been reported by Dishion et al. (1988). Interestingly, we found that father's monitoring was a statistically significant mediator whereas mother's monitoring was marginally significant in the main analysis and statistically significant in the analysis with imputed data and a larger sample. Many studies of parental socialization processes concentrate on mother-child dyads because of the convenience of maternal participation in research studies and because of theoretical beliefs about the primacy of mother's influence. The current data support the importance of father's influence as well.

It was also surprising that decrements in parental monitoring were related to lifetime clinical alcoholism diagnoses rather than to parents' current alcohol consumption. We might have expected more acute effects in which alcohol consumption pro-

duces short-term disruptions in parental monitoring. Instead, the current findings are consistent with a more cumulative mechanism. Such a cumulative effect could be due to particular parental characteristics that are both associated with lifetime alcoholism and that also affect parenting (e.g., some type of gene-environment covariation). Alternatively, such an effect could be due to some cumulative impact of historical alcoholism on the parent-child relationship in which past parental alcoholism had a lingering negative impact on the parent's ability to effectively control the child's behavior. Finally, it is possible that an acute mechanism does operate to influence parental monitoring but only above some threshold of "pathological" current parental alcohol consumption rather than through a linear relation between parental alcohol consumption and parenting behavior. Conceptually, of course, this notion of a pathological threshold of alcohol consumption is highly redundant with parental alcoholism diagnoses. Thus, it is possible that lifetime alcoholism diagnoses rather than current consumption is related to parental monitoring because these diagnoses capture a pathological level of consumption as opposed to "normal" use.

In contrast to lifetime diagnoses, current maternal alcohol consumption had a direct effect on adolescent substance use. This may reflect modeling mechanisms or increased availability of alcohol in the home, or it may reflect some unmeasured mediator that covaries with current maternal alcohol consumption. For example, perhaps some child sensitivity to the reinforcing effects of substances covaries with a type of maternal alcoholism that is particularly persistent and unlikely to remit. If so, these mothers would both continue to show high levels of current alcohol consumption and have children who were more likely to use drugs.

In terms of future research on parenting influences, there are many questions left unanswered by the current analyses. Most important, the current cross-sectional design cannot determine the directions of effect, and there are likely to be reciprocal relations between parenting behaviors and adolescent outcomes. Moreover, parenting is a multifaceted construct, and only the monitoring dimension was tapped in the current study. Further research on different aspects of parental support and control is warranted. Future research may also wish to consider the possibility of curvilinear models in which extremes of parent control (either highly authoritarian or highly permissive) are associated with negative child outcomes. Although preliminary analyses showed that these curvilinear effects were absent for the parental monitoring variables in the current data, other aspects of parenting have shown such effects (see Foxcroft & Lowe, 1991, for a review). Finally, interesting questions for future study emerge from considering parenting in the context of a larger family system. For example, it is possible that supportive and consistent parenting by one spouse could buffer the effects of impaired parenting by the other spouse or that aspects of the marital relationship could moderate the effects of parenting practices.<sup>8</sup>

<sup>8</sup> These potential "buffering" relations are beyond the scope of the current article and could not be tested within our complex multivariate model with the current sample size. However, other analyses of this data set (Curran, 1991) have reported that mother's characteristics had

### *Implications for the Stress and Negative Affect Mechanism*

In terms of stress and negative affect regulation, our findings supported a mechanism in which parental alcoholism was associated with increases in negative uncontrollable life events which, in turn, were linked to negative affect, to associations with drug-using peers, and to substance use. These findings not only replicate previous work on the link between stress and adolescent substance use (e.g., Newcomb & Harlow, 1986; Wills, 1986) but they demonstrate that the effects of this mechanism are unique, occurring over and above temperamental and parenting variables.

The ways in which negative affect were associated with substance use also has implications for negative affect regulation models because the data support two different interpretations. First, we found a direct path between negative affect and substance use. This is consistent with commonly described stress models that view substance use as a coping behavior used to manage the negative affect associated with stress events (e.g., Newcomb & Harlow, 1986). Although negative affect regulation motives have been more typically invoked to explain substance abuse or dependency than substance use initiation, some studies have shown direct relations between negative affect and overall adolescent substance use (Newcomb & Harlow, 1986; Paton et al., 1977). In the current design, because the outcome variable is overall adolescent substance use, predictors of substance use initiation and later-stage substance use cannot be clearly separated. Moreover, given the young age of the sample, there were few subjects who would be considered substance abusers. Thus, we can conclude that our data are consistent with a negative affect regulation model, but we cannot determine if the mechanism is operative in influencing the initiation, maintenance, or escalation of use.

We also found an indirect effect, in which the relation between negative affect and substance use was mediated by associations with drug-using peers. That is, adolescents who reported high levels of negative affect were also more likely to report associating with peers who modeled and tolerated substance use behaviors, and these peers were a powerful proximal risk factor for substance use. A similar result was recently reported by Swaim et al. (1989) with an older public school sample unselected by parental alcoholism. They found that the effects of emotional distress (particularly anger) on adolescent substance use were mediated by a drug-using peer group. This mediational pathway is also consistent with Kaplan's (1980) self-derogation theory. According to Kaplan (1980), adolescents with negative self-evaluations (a component of the current negative affect construct) seek out deviant peer groups as a way of obtaining more positive messages about the self. Affiliation with these peer groups then increases risk for delinquent behaviors.

independent positive effects above and beyond the negative effects of father's alcoholism. Because these maternal characteristics did not interact with father's alcoholism diagnosis, there was no special buffering effect of maternal characteristics on adolescent children with alcoholic fathers. Rather, maternal variables were important to outcomes for both adolescent offspring of alcoholic and nonalcoholic fathers.

The current data suggest that this type of mechanism may be operative in mediating the effects of parental alcoholism on adolescent substance use, and they underline the importance of the peer social context for adolescent substance use.<sup>9</sup>

For future research on the stress and negative affect mechanism, there are several useful directions. Here stress was operationalized simply as uncontrollable negative life events. Future studies might usefully distinguish between acute and chronic stressors, between major life events and daily hassles, and between different contents of stress events (e.g., loss events vs. danger events, see Monroe & Simons, 1991). Similarly, distinctions between different types of negative affect (e.g., anxiety vs. depression vs. anger) might prove useful. Moreover, further research should examine the ways in which adolescents' coping strategies (or other potential stress buffers) modify substance use outcomes for COAs. Finally, longitudinal analyses are necessary to illuminate the directions of influence between negative affect and adolescent substance use in COAs.

### *Implications for Temperament Mechanisms*

The current data supported temperamental emotionality but not sociability as a mediator of the relation between parental alcoholism and adolescent substance use. Adolescent COAs showed heightened levels of temperamental emotionality, which raised their risk for experiencing negative affective states which, in turn, increased the risk for substance use. Previous literature on adult samples has shown inconsistent support for a link between parental alcoholism and emotionality, and appropriate analyses of the mediating effects of temperament have been lacking (see Sher et al., 1991, for an exception). Interestingly, the link between parental alcoholism and adolescent emotionality was more robust for the effects of maternal alcoholism than for paternal alcoholism. (The effect of paternal alcoholism was significant in the analysis using imputed data only.) Emotionality was also linked to parental affective disorders. Perhaps inconsistent demonstrations of the relation between parental alcoholism and emotionality are due to varying prevalences of parental affective disorders in the samples or to varying prevalences of maternal alcoholism (although the effect has been reported in subjects with a family history of unigenerational male alcoholism, Finn & Pihl, 1987). Inconsistent

<sup>9</sup> The fact that associations with drug-using peers mediated the relation between negative affect and substance use also raises a caution about the specificity of the negative affect influences. The types of "internalizing" symptoms used to assess negative affect (e.g., depressed mood, anxiety, negative self-evaluations) are highly correlated with "externalizing" symptoms (e.g., aggression, delinquency, non-compliance; cf. Achenbach, McConaughy, & Howell, 1987). The current cross-sectional data cannot rule out the possibility that the influences of negative affect are actually due (at least in part) to preexisting externalizing symptomatology. Kaplan's (1980) longitudinal study found that negative self-evaluations (one component of our negative affect measure) prospectively predicted affiliation with deviant peers. However, because of the complex (and possibly bidirectional) relations among internalizing symptomatology, externalizing symptomatology, and drug use, the current cross-sectional data require caution in interpreting the influences of negative affect on adolescent substance use.

relations between parental alcoholism and emotionality may also be due to differing measures of emotionality. For example, Sher et al. (1991) found that neuroticism but not harm avoidance or social anxiety was related to a family history of alcoholism in college students.

Our data did not support temperamental sociability as a mediator of the effects of parental alcoholism because there were no significant links between parental alcoholism and adolescent sociability. This is consistent with some previous adult data (e.g., Finn & Pihl, 1987). As Tarter et al. (1985) suggested, previous speculation that COAs are particularly sociable may have confused high levels of disinhibition or behavioral undercontrol with being outgoing or socially skilled. That is, rather than sociability being the mediator of parental alcoholism effects, the actual mediator may be behavioral undercontrol. To test this hypothesis, future research is required to establish a clear theoretical specification of the dimensions comprising behavioral undercontrol; to disentangle measures of behavioral undercontrol as a temperament dimension from behavioral undercontrol as a pattern of antisocial behavior; and to test emotionality, sociability, and behavioral undercontrol as temperamental mediators of the effects of parental alcoholism (see Nathan, 1988; Sher, 1991; Windle, 1990). Consideration of temperament models such as Rothbart's (Rothbart & Derryberry, 1981), in which self-regulation is considered to be a basic component of temperament, is a useful step in this direction.

Although sociability did not mediate the effects of parental alcoholism, it did show interesting relations to adolescent substance use. In one way, sociability acted as a risk factor by increasing the likelihood of association with drug-using peers. Adolescents who are outgoing and extraverted may be more likely to find themselves in social peer contexts that support substance use behavior, and this social context increases their risk for use. However, sociability also acted as a protective factor by reducing the risk of experiencing negative affect. Thus, sociability acted as both a risk and a protective factor, reducing the risk of negative affect motives for use but increasing the risk of exposure to peer influences.

### Limitations and Conclusions

Although the current study corrected some of the methodological problems in earlier research, it is also important to recognize some of the study's limitations. First, the data are cross-sectional in nature and thus preclude definitive statements about the directionality of the obtained relations. Second, the current design cannot separate genetic and environmental pathways of risk. Third, alcoholism has been treated as a unitary disorder, and no attempts have been made to subtype particular forms of alcoholism or to consider the severity of alcoholism. Given speculations about gender differences in alcoholism subtypes (Cloninger, 1987), subtyping may be particularly important for future research in pursuing gender differences in mediating mechanisms. Although we found some evidence of gender differences, these differences did not have clear theoretical interpretations and they could not be adequately addressed with the current sample size. Fourth, as apparent from the frequently conflicting findings concerning COAs (Sher, 1991), the current results should be generalized

only with extreme caution. Different definitions of parental alcoholism, different operationalizations of the mediating constructs (especially a focus on different aspects of parenting or temperament), different ages of COAs (particularly inclusion of older adolescents with higher prevalences of substance abuse), different gender and ethnic compositions, or different recruitment strategies all would be likely to affect findings (Sher, 1991). Finally, the mediators that we tested are not the only possible mechanisms underlying the effects of parental alcoholism on adolescent substance use. Other possibilities include particular sensitivities to the reinforcing properties of substances or cognitive limitations that prevent adolescents from appreciating the potential negative consequences of use<sup>10</sup> (see Sher, 1991, for other examples).

In sum, the current study assessed three mediating mechanisms underlying the relation between parental alcoholism and adolescent substance use. The findings suggest that parental alcoholism affects substance use in adolescent children through stress and negative affect mechanisms and through impairments in parental monitoring, both of which increase the probability of associations with a peer network that supports drug use behavior. Parental alcoholism is also associated with higher levels of temperamental emotionality in adolescent COAs, which raises risk for experiencing negative affect. The data did not support a link between parental alcoholism and temperamental sociability. However, sociability did predict adolescent substance use, acting both as a protective factor (by decreasing the likelihood of negative affect) and a risk factor (by increasing exposure to a peer network that was supportive of drug use). These findings are clearly in need of replication with other samples and longitudinal designs.

<sup>10</sup> One way that COAs can show cognitive limitations is through fetal alcohol effects. Parents reported on the biological mothers' alcohol use during pregnancy with the target child. To assess possible fetal alcohol effects, we computed the number of subjects who drank more than twice weekly and who consumed three or more drinks per occasion during pregnancy (either by their own reports or by fathers' reports). Because only 14 subjects met this criterion, fetal alcohol effects were not considered.

### References

- Achenbach, T. M., & Edelbrock, C. (1981). Behavioral problems and competencies reported by parents of normal and disturbed children aged four through sixteen. *Monographs of the Society for Research in Child Development*, 46(1, Serial No. 188).
- Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psychological Bulletin*, 101, 213-232.
- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.
- Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, 51, 1173-1182.
- Bentler, P. M., & Bonett, D. G. (1980). Significance tests and goodness of fit in the analysis of covariance structures. *Psychological Bulletin*, 88, 588-606.

- Bollen, K. A. (1989). *Structural equations with latent variables*. New York: Wiley.
- Brook, J. S., Whiteman, M., & Gordon, A. S. (1983). Stages of drug use in adolescence: Personality, peer, and family correlates. *Developmental Psychology*, 19, 269-277.
- Brook, J. S., Whiteman, M., Gordon, A. S., & Cohen, P. (1986). Dynamics of childhood and adolescent personality traits and adolescent drug use. *Developmental Psychology*, 22, 403-413.
- Burnam, M. A., Hough, R. L., Escobar, J. I., Karno, M., Timbers, D. M., Telles, C. A., & Locke, B. Z. (1987). Six-month prevalence of specific psychiatric disorders among Mexican-Americans and non-Hispanic Whites in Los Angeles. *Archives of General Psychiatry*, 44, 687-694.
- Buss, A. H., & Plomin, R. (1984). *Temperament: Early developing personality traits*. Hillsdale, NJ: Erlbaum.
- 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, 316-320.
- Chassin, L., Rogosch, F., & Barrera, M. (1991). Substance use and symptomatology among adolescent children of alcoholics. *Journal of Abnormal Psychology*, 100, 449-463.
- Cloninger, R. (1987). Neurogenetic adaptive mechanisms in alcoholism. *Science*, 236, 410-416.
- Curran, P. (1991, April). *The relation between maternal characteristics and the development of the child of an alcoholic father*. Paper presented at the biennial meeting of the Society for Research in Child Development, Seattle.
- Dishion, T. J., Patterson, G. R., & Reid, J. R. (1988). Parent and peer factors associated with drug sampling in early adolescence: Implications for treatment. In E. R. Rahdert & J. Grabowski (Eds.), *Adolescent drug abuse: Analyses of treatment research* (NIDA Research Monograph No. 77, DHHS Publication No. ADM88-1523) (pp. 69-93). Rockville, MD: National Institute on Drug Abuse.
- Eysenck, H. J., & Eysenck, S. B. G. (1969). *Personality structure and measurement*. London: Routledge & Kegan.
- Finn, P. R., & Phil, R. O. (1987). Men at high risk for alcoholism: The effect of alcohol on cardiovascular response to unavoidable shock. *Journal of Abnormal Psychology*, 96, 230-236.
- Foxcroft, D. R., & Lowe, G. (1991). Adolescent drinking behavior and family socialization factors: A meta-analysis. *Journal of Adolescence*, 14, 255-273.
- Goldsmith, H. H., Buss, A. H., Plomin, R., Rothbart, M. K., Thomas, A., Chess, S., Hinde, R., & McCall, R. (1987). Roundtable: What is temperament? Four approaches. *Child Development*, 58, 505-529.
- Helzer, J. E., & Pryzbeck, T. R. (1988). The co-occurrence of alcoholism with other psychiatric disorders in the general populations and its impact on treatment. *Journal of Studies on Alcohol*, 49, 219-224.
- Herjanic, B., Herjanic, M., Penick, E., Tomelleri, C., & Armbruster, R. (1977). Children of alcoholics. In F. Seixas (Ed.), *Currents in alcoholism* (Vol. 2, pp. 445-455). New York: Grune & Stratton.
- Jessor, R., & Jessor, S. L. (1977). *Problem behavior and psychosocial development: A longitudinal study of youth*. New York: Academic Press.
- Johnson, S., Leonard, K., & Jacob, T. (1989). Drinking, drinking styles, and drug use in children of alcoholics, depressives, and controls. *Journal of Studies on Alcohol*, 50, 427-432.
- Johnston, L., O'Malley, P., & Bachman, J. (1988). *Illicit drug use, smoking and drinking by America's high school students, college students, and young adults, 1975-1987*. Washington, DC: U.S. Government Printing Office.
- Jöreskog, K. G., & Sörbom, D. (1989). *LISREL 7: A Guide to the program and applications*. Chicago: SPSS.
- Kandel, D. B. (1978). *Longitudinal research on drug use: Empirical findings and methodological issues*. New York: Wiley.
- Kaplan, H. B. (1980). *Deviant behavior in defense of self*. New York: Academic Press.
- Knop, J., Teasdale, T., Schulsinger, F., & Goodwin, D. (1985). A prospective study of young men at high risk for alcoholism: School behavior and achievement. *Journal of Studies on Alcohol*, 46, 273-278.
- Levenson, R. W., Oyama, O. N., & Meek, P. S. (1987). Greater reinforcement from alcohol for those at risk: Parental risk, personality risk, and sex. *Journal of Abnormal Psychology*, 96, 242-253.
- Little, R. J. A., & Rubin, D. B. (1987). *Statistical analysis with missing data*. New York: Wiley.
- Loehlin, J. C. (1987). *Latent variable models: An introduction to factor, path, and structural analysis*. Hillsdale, NJ: Erlbaum.
- Lord, F. M., & Novick, M. R. (1968). *Statistical theories of mental test scores*. Reading, MA: Addison-Wesley.
- Marsh, H. W. (1990). Confirmatory factor analysis of multitrait-multimethod data: The construct validation of multidimensional self-concept responses. *Journal of Personality*, 58, 661-692.
- Merikangas, K., Weissman, M., Prusoff, B., Pauls, D., & Leckman, J. (1985). Depressive with secondary alcoholism: Psychiatric disorders in offspring. *Journal of Studies on Alcohol*, 46, 199-204.
- Molina, B. S. G., Pillow, D., & Chassin, L. (1989, April). *Models of adolescent temperament: Measurement issues using multiple reporters*. Paper presented at the 1990 Annual Meeting of the Western Psychological Association, Los Angeles.
- Monroe, S. M., & Simons, A. D. (1991). Diathesis-stress theories in the context of life stress research: Implications for the depressive disorders. *Psychological Bulletin*, 110, 406-425.
- Murray, D. M., & Perry, C. L. (1987). The measurement of substance use among adolescents: When is the 'bogus pipeline' method needed? *Addictive Behaviors*, 12, 225-233.
- Nathan, P. (1988). The addictive personality is the behavior of the addict. *Journal of Consulting and Clinical Psychology*, 56, 183-188.
- National Institute on Drug Abuse. (1989). *Highlights of the 1988 National Household Survey on Drug Abuse*. Rockville, MD: National Institute on Drug Abuse.
- Newcomb, M. D., & Harlow, L. L. (1986). Life events and substance use among adolescents: Mediating effects of perceived loss of control and meaninglessness in life. *Journal of Personality and Social Psychology*, 51, 564-577.
- Pandina, R. J., & Johnson, V. (1989). Familial drinking history as a predictor of alcohol and drug consumption among adolescent children. *Journal of Studies on Alcohol*, 50, 245-254.
- Paton, S., Kessler, R., & Kandel, D. B. (1977). Depressive mood and adolescent illegal drug use: A longitudinal analysis. *Journal of Genetic Psychology*, 131, 267-289.
- Patterson, G. R., & Reid, J. (1984). Social interactional processes within the family: The study of moment by moment family transactions in which human social development is embedded. *Journal of Applied Developmental Psychology*, 5, 237-262.
- Robins, L. N., Helzer, J. E., Croughan, J., & Ratcliff, K. S. (1981). Natural Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics, and validity. *Archives of General Psychiatry*, 38, 381-389.
- Robins, L. N., Helzer, J. E., Ratcliff, K. S., & Seyfried, W. (1982). Validity of the Diagnostic Interview Schedule, Version II: DSM-III diagnoses. *Psychological Medicine*, 12, 855-870.
- Robins, L. N., & Pryzbeck, T. R. (1985). Age of onset of drug use as a factor in drug and other disorders. In C. R. Jones & R. J. Battjes (Eds.), *Etiology of drug abuse: Implications for prevention* (NIDA Research Monograph No. 56, DHHS Publication No. ADM 85-



- 1335) (pp. 178-192). Washington, DC: U.S. Government Printing Office.
- Rogosch, F., Chassin, L., & Sher, K. J. (1990). Personality variables as mediators and moderators of family history risk for alcoholism. *Journal of Studies on Alcohol*, 51, 310-319.
- Roosa, M. W., Sandler, I. N., Gehring, M., Beals, J., & Cappel, L. (1988). The children of alcoholics life-events schedule: A stress scale for children of alcohol-abusing parents. *Journal of Studies on Alcohol*, 49, 422-429.
- Rosenberg, M. (1979). *Conceiving the self*. New York: Basic Books.
- Rothbart, M. K., & Derryberry, D. (1981). Development of individual differences in temperament. In M. K. Lamb & A. L. Brown (Eds.), *Advances in developmental psychology* (Vol. 1, pp. 37-86). Hillsdale, NJ: Erlbaum.
- Sandler, I., Ramirez, R., & Reynolds, K. (1986, August). *Life stress for children of divorce, bereaved, and asthmatic children*. Paper presented at the annual meeting of the American Psychological Association, Washington, DC.
- Sher, K. J. (1983). Comment on platelet MAO activity in relatives of alcoholics. *Archives of General Psychiatry*, 40, 466.
- Sher, K. J. (1991). *Children of alcoholics: A critical appraisal of theory and research*. Chicago: University of Chicago Press.
- Sher, K. J., Walitzer, K. S., Wood, P. K., & Brent, E. E. (1991). Characteristics of children of alcoholics: Putative risk factors, substance use and abuse, and psychopathology. *Journal of Abnormal Psychology*, 100, 427-449.
- Swaim, R. C., Oetting, E. R., Edwards, R., & Beauvais, F. (1989). Links from emotional distress to adolescent drug use: A path model. *Journal of Consulting and Clinical Psychology*, 57, 227-231.
- Tarter, R. E., Alterman, A. I., & Edwards, K. L. (1985). Vulnerability to alcoholism in men: A behavior-genetic perspective. *Journal of Studies on Alcohol*, 46, 329-356.
- Tucker, L. R., & Lewis, C. (1973). The reliability coefficient for maximum likelihood factor analysis. *Psychometrika*, 38, 1-10.
- West, M. O., & Prinz, R. J. (1987). Parental alcoholism and childhood psychopathology. *Psychological Bulletin*, 102, 204-218.
- Wills, T. A. (1986). Stress and coping in early adolescence: Relationships to substance use in urban samples. *Health Psychology*, 5, 503-529.
- Windle, M. (1990). Temperament and personality attributes of children of alcoholics. In M. Windle & J. Searles (Eds.), *Children of alcoholics: Critical perspectives* (pp. 129-167). New York: Guilford Press.
- Winters, D. C., Stinchfield, R. D., Henly, G. A., & Schwartz, R. H. (1991). Validity of adolescent self-reports of alcohol and other drug involvement. *The International Journal of the Addictions*, 25, 1379-1395.

Received April 23, 1991

Revision received February 5, 1992

Accepted July 1, 1992 ■



## AMERICAN PSYCHOLOGICAL ASSOCIATION SUBSCRIPTION CLAIMS INFORMATION

Today's Date: \_\_\_\_\_

We provide this form to assist members, institutions, and nonmember individuals with any subscription problems. With the appropriate information we can begin a resolution. If you use the services of an agent, please do NOT duplicate claims through them and directly to us. **PLEASE PRINT CLEARLY AND IN INK IF POSSIBLE.**

PRINT FULL NAME OR KEY NAME OF INSTITUTION \_\_\_\_\_

ADDRESS \_\_\_\_\_

CITY \_\_\_\_\_

STATE/COUNTRY \_\_\_\_\_

ZIP \_\_\_\_\_

YOUR NAME AND PHONE NUMBER \_\_\_\_\_

MEMBER OR CUSTOMER NUMBER (MAY BE FOUND ON ANY PAST ISSUE LABEL) \_\_\_\_\_

DATE YOUR ORDER WAS MAILED (OR PHONED): \_\_\_\_\_

P.O. NUMBER: \_\_\_\_\_

PREPAID ☐ CHECK ☐ CHARGE ☐

CHECK/CARD CLEARED DATE: \_\_\_\_\_

(If possible, send a copy, front and back, of your cancelled check to help us in our research of your claim.)

ISSUES: \_\_\_\_\_ MISSING \_\_\_\_\_ DAMAGED \_\_\_\_\_

TITLE \_\_\_\_\_

VOLUME OR YEAR \_\_\_\_\_

NUMBER OR MONTH \_\_\_\_\_

*Thank you. Once a claim is received and resolved, delivery of replacement issues routinely takes 4-6 weeks.*

(TO BE FILLED OUT BY APA STAFF)

DATE RECEIVED: \_\_\_\_\_

DATE OF ACTION: \_\_\_\_\_

ACTION TAKEN: \_\_\_\_\_

INV. NO. &amp; DATE: \_\_\_\_\_

STAFF NAME: \_\_\_\_\_

LABEL NO. &amp; DATE: \_\_\_\_\_

SEND THIS FORM TO: APA Subscription Claims, 750 First Street, N.E., Washington, DC 20002

PLEASE DO NOT REMOVE. A PHOTOCOPY MAY BE USED.