Journal of Abnormal Psychology
1996, Vol. 105, No. 1, 70-80

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0021-843X/96/$3.00

The Relation of Parent Alcoholism to Adolescent Substance Use: A Longitudinal Follow-Up Study

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The current study tested parent alcoholism effects on growth curves of adolescent substance use and examined whether parent and peer influences, temperamental emotionality and sociability, and stress and negative affect could explain parent alcoholism effects. Longitudinal latent growth curve modeling showed that adolescents with alcoholic fathers, boys, and adolescents with drug-using peers had steeper growth in substance use over time than did adolescents without alcoholic fathers, girls, and adolescents without drug-using peers. Data were consistent with father's monitoring and stress as possible mediators of paternal alcoholism effects. However, the direct effects of paternal alcoholism on substance use growth remained significant even after including the hypothesized mediators in the model. This suggests that other (unmeasured) mediators are necessary to fully explain paternal alcoholism risk for adolescents' escalating substance use over time.

Parent alcoholism is a well-established risk factor for adult alcoholism, and recent data suggest that parent alcoholism also raises risk for alcohol and drug use during adolescence (Chassin, Rogosch, & Barrera, 1991; Hawkins, Catalano, & Miller, 1992). However, less is known about the mechanisms underlying this risk, particularly for adolescents. Theoretical speculations have included social environmental mechanisms, such as impaired parental monitoring and control and weak parent–adolescent bonds. More biologically based theories have focused on potentially heritable personality traits related to substance use and on potentially heritable individual differences in alcohol effects (e.g., tendencies to derive greater positive reinforcement from alcohol). Recently, these diverse theories have been integrated into heuristic models postulating links between biologically based individual differences and social environmental mechanisms that interact to determine risk (see Sher, 1991, for a review).

Despite theoretical speculation, however, there have been few empirical studies to test these models with adolescent children of alcoholics (COAs; although some studies have examined mediators of parent substance use effects in the general population, e.g., Wills, Schrebman, Benson, & Vaccaro, 1994). There is also a lack of longitudinal model tests. Moreover, research on parent alcoholism has been limited by methodological problems, including an overreliance on treated samples and failures to consider co-occurring risk factors, such as other forms of parent psychopathology (Sher, 1991; West & Prinz, 1987). To address these problems, we conducted a longitudinal study of a community sample, considering parent alcoholism in the context of other risk factors for adolescent substance use (Chassin et al., 1991). Previously, we tested a cross-sectional model of parent alcoholism effects on adolescent substance use that focused on three domains of mediators: (a) parental monitoring of the adolescent's behavior and adolescents' affiliations with drug-using peers, (b) adolescents' stress and experience of negative affect, and (c) adolescents' temperamental emotionality and sociability (Chassin, Pillow, Curran, Molina, & Barrera, 1993). These domains were chosen because they are linked (either theoretically or empirically) with both parent alcoholism and adolescent substance use. The current study provides a longitudinal test of this model.

Socialization Pathways: Parenting and Peer Affiliation

Deficits in parental support and ineffective parental control practices have been frequently identified as risk factors for adolescent substance use (Hawkins et al., 1992). For the present study, the most relevant work is Patterson's social interactional theory of adolescent conduct problems (including substance use). Dishion, Patterson, and Reid (1988) found a cross-sectional relation between parent drug use and early adolescent drug use that was both direct (interpreted as the result of parental modeling and availability) and indirect (mediated through impaired parental control). Parent drug use was associated with decreased monitoring of the adolescent's activities, and this decreased monitoring was associated with membership in a drug-using peer group that was the proximal pathway into adolescent drug sampling. Our cross-sectional data (Chassin et al., 1993) supported a similar mechanism. The current study tested whether such impaired monitoring predicted growth over

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This study was supported by National Institute on Drug Abuse Grant DA05227. We thank Kirsten Bech, Julie Kossak-Fuller, Polly Lewit, and Brooke Molina for coordinating the interviewing and data collection activities, and Jennifer Rose and Heather Montgomery for assistance in figure preparation. Many colleagues provided important advice on research design and on the latent growth curve modeling. We thank Kenneth J. Sher, Bengt Muthen, Phil Wood, David Francis, Jack McArdle, Mike Stoolmiller, Leona Aiken, Dave MacKinnon, Steve West, and Peter Bentler for their consultation on these issues.

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time in adolescent substance use and whether impaired monitoring mediated parent alcoholism effects on such growth.

Stress and Negative Affect Regulation Pathways

An alternative (but not mutually exclusive) pathway suggests that parent alcoholism is associated with environmental stress that produces negative affect. Negative affect can then lead to adolescent substance use in several ways. First, substance use may be adopted as a means of regulating negative affect. This mechanism is controversial with regards to adolescent substance use. Some researchers suggest that affect regulation may motivate substance abuse or adult substance use but has little impact on adolescent substance use initiation (Swaim, Oetting, Edwards, & Beauvais, 1989). However, other data link negative affect to earlier stages of adolescent substance use as well (Newcomb & Harlow, 1986; Paton, Kessler, & Kandel, 1977).

Perhaps more relevant for adolescent substance use is a mechanism that hypothesizes additional mediation through a drug-use-promoting peer group. Kaplan (1980) suggested that adolescents who suffer negative self-evaluations use deviant peer groups to restore damaged self-esteem. This peer group affiliation helps to repair self-image, but it also increases risk for delinquent behaviors. A similar mechanism may apply to adolescents who are experiencing negative affect.

Stress and negative affect pathways may help to explain the impact of parent alcoholism on adolescent substance use. Our cross-sectional data (Chassin et al., 1993) showed that parent alcoholism was associated with elevations in environmental stress that in turn were associated with negative affect. Negative affect had both a direct effect on use (consistent with negative affect regulation mechanisms) as well as an indirect effect, mediated through affiliations with drug-using peers (consistent with Kaplan's, 1980, self-derogation theory). In the current study, we tested whether a direct effect of negative affect (consistent with affect regulation) or an indirect effect of negative affect (consistent with self-derogation theory) or both, could explain the effects of parent alcoholism on growth over time in adolescent substance use.

Temperament Pathways: Emotionality and Sociability

Temperament factors may also mediate the effects of parent alcoholism on offspring substance use. Tarter, Alterman, and Edwards (1985) suggested that COAs were more likely to be high in activity, low in persistence, slow to soothe after stress, and emotionally labile and disinhibited. Other research suggests that these characteristics are associated with substance use as well (Hawkins et al., 1992; Watson & Clark, 1993; Wills, DuHamel, & Vaccaro, 1995). Moreover, cross-sectional studies of college student COAs (Sher, Walitzer, Wood, & Brent, 1991) support a mediational role for "behavioral undercontrol." Thus, adolescent COAs may be at risk for substance use because they are temperamentally emotionally reactive and underregulated.

We focus on temperamental emotionality and sociability as potential mediators of parental alcoholism effects. We chose these temperament dimensions because they are recognized by most theoretical models of temperament (Goldsmith et al., 1987), they map readily onto adult personality dimensions (e.g., neuroticism and extraversion; Eysenck & Eysenck, 1969), and they represent key elements of major models of adolescent substance use (e.g., sociability should be important for peer influence models, and emotionality should be important for affect regulation models).[1] Our cross-sectional data found that maternal alcoholism was associated with heightened emotionality that, in turn, increased the likelihood of adolescents' experiencing negative affect that raised risk for substance use (Chassin et al., 1993). Sociability, however, was unrelated to parental alcoholism. The current study tested whether these temperamental mediators predicted growth over time in adolescent substance use.

In sum, although research suggests that COAs' elevated risk for substance use may be mediated through multiple pathways, few empirical studies have tested mediational models with this population, and longitudinal studies are particularly lacking. Although our cross-sectional model showed that multiple mediators made independent contributions to adolescents' substance use outcomes, these data cannot establish the temporal precedence of the risk factors to the outcomes. The current study provides a longitudinal test of our mediational model over a 3-year period. Through the use of latent growth curve modeling, we examined predictors of individual differences in adolescents' rates of substance use growth. Specifically, we tested whether parent alcoholism predicted steeper growth in adolescent substance use and whether our hypothesized mediators could account for these parent alcoholism effects. We addressed methodological limitations of previous research by studying a community sample for whom parent alcoholism and co-occurring psychopathology were directly ascertained, and by using multiple reporters to minimize the impact of response biases.

Method

Participants

The total sample at Time 1 consisted of 454 adolescents, aged 10.5 to 15.5 years (M = 12.7, SD = 1.45) and their parents. COAs (n = 246) had at least one biological alcoholic parent who was also a custodial

[1] An important temperamental mediator missing from our model is behavioral undercontrol. Many characteristics have been considered under this rubric (e.g., impulsivity, aggression, sensation seeking, and overactivity). However, we did not focus on this construct because different theoretical models of temperament lack clear consensus concerning its structure and operationalization. Even for more clearly defined subcomponents such as activity level, data are conflicting about whether the operative risk factor is activity level per se or associated conduct problems and disregulation (Windle, 1990), and there are complex relations between activity level and other dimensions of behavioral undercontrol (Wills et al., 1995). Finally, many operationalizations of behavioral undercontrol rely on indicators of antisocial behavior and, under these circumstances, behavioral undercontrol may be an indicator of co-occurring deviant behavior rather than being a temperamental characteristic that predisposes an individual to substance use (Nathan, 1988; Windle, 1990). For these reasons, we did not consider behavioral undercontrol in the current model. However, future examinations of this construct are very important if clear operational definitions can be provided, and recent research has suggested some measurement directions (e.g., Martin et al., 1994; White et al., 1994).

parent, and controls (n = 208) had no biological or custodial alcoholic parents. Because the current study used adolescent, mother, and father reports, we excluded 38 single-parent families and 87 two-parent families without complete data from both parents at Time 1. Sample retention was high. Of the 329 two-parent families with complete data at Time 1, only 8 families did not provide complete data at all three time points. Finally, 5 participants were identified as influential outliers and were excluded,[2] leaving a final sample of 316 families in the analyses.

We compared the 138 participants who were dropped from the analyses to the 316 who were retained using all available Time 1 data (adolescent report, parent report, and spouse reports on non-interviewed parents). T tests and chi-square comparisons showed that, at Time 1, the groups did not significantly differ in age, gender, father's alcoholism, parent antisocial personality, parent affective disorder, stress, emotionality, sociability, or negative affect. However, those dropped from analysis had higher Time 1 substance use, less parent monitoring, more peer substance use, more Hispanic parents, less educated parents, and more alcoholic mothers (p values ranged from $< .10$ to $< .001$). Although the groups were largely comparable on the variables of interest, some caution is warranted in generalization.

Of the 316 adolescents in the current sample, 47% were female, 21% Hispanic, and 89% lived with both biological parents. COAs and controls did not significantly differ in these characteristics. However, COAs had less educated parents, more parents with lifetime diagnoses of affective disorders, and more parents with lifetime diagnoses of antisocial personality disorder (all $ps < .05$).

Recruitment

Recruitment procedures are presented in detail elsewhere (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). COAs had to be non-Hispanic Caucasian or Hispanic, Arizona residents, aged 10.5–15.5 years, and English speaking. Moreover, a biological and custodial parent had to meet the criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., *DSM-III*; American Psychiatric Association, 1980) for alcohol abuse or dependence or Family History—Research Diagnostic Criteria (FH-RDC; Andreasen, Endicott, Spitzer, & Winokur, 1977), based on spouse reports (if the alcoholic parent was not interviewed). Demographically matched controls were recruited using telephone interviews. Controls were screened to match the COA participant in ethnicity, family composition, age, and socioeconomic status. Neither biological nor custodial parents could meet *DSM-III* criteria (or FH-RDC criteria) for alcohol abuse or dependence.

Recruitment biases are discussed in detail elsewhere (Chassin et al., 1992; Chassin et al., 1993). We required that the alcoholic parent be custodial as well as biological (so that the adolescent had the potential to be exposed to this parent's influence). This requirement produced an overrepresentation of two-parent families. Also, those who refused participation were more likely to be Hispanic. However, the sample was unbiased with respect to alcoholism indicators that were available in archival records. In support of the representativeness of the alcoholic sample, their comorbidities were similar to those reported in the Epidemiological Catchment Area Study (Helzer & Pryzbeck, 1988). However, the underrepresentation of single-parent families and the higher refusal rate for Hispanics suggests caution in generalization.

Procedure

The procedures are described in detail elsewhere (Chassin et al., 1991). Data were collected using three annual computer-assisted interviews with the adolescents and their parents. Confidentiality was reinforced with a Department of Health and Human Services Certificate of Confidentiality.

Measures

Parent alcoholism and associated psychopathology. Lifetime *DSM-III* (American Psychiatric Association, 1980) diagnoses of alcohol abuse or dependence, affective disorder (major depression or dysthymia), and antisocial personality were obtained using a computerized version of the Diagnostic Interview Schedule (DIS, Version III; Robins, Helzer, Croughan, & Ratcliff, 1981). If only one parent was interviewed, alcoholism diagnoses for the other parent were made using spouse reports according to FH-RDC. For the current analyses, alcoholism diagnoses of the biological father and mother were each considered (separately) as dichotomous variables. Among the 316 families in the current analyses, 28 mothers and 151 fathers met these criteria.

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

Parent monitoring of the adolescent's behavior. Parents' monitoring of their adolescent'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 adolescent's] plans for the day"). Coefficient alphas over the three waves ranged from .74–.80 for father's monitoring and .77–.85 for mother's monitoring. A single score was computed for each parent using the mean of the three items.

Associations with drug-use-promoting peers. Adolescents estimated how many of their friends used alcohol, marijuana, and other drugs occasionally and regularly, using items adapted from Johnston, O'Malley, and Bachman (1988). They also reported how their close friends would feel about their using marijuana, alcohol, and other drugs occasionally and regularly. Coefficient alpha was .89–90 across waves for the six-item peer substance use measure and .89–.93 across waves for the seven-item peer tolerance of substance use measure. Because adolescents' reports of peer substance use and peer tolerance of substance use were highly correlated (r = .59–.63 across waves), the two scales were averaged to represent a drug-use-promoting peer environment.

Adolescent life stress. Parents and adolescents reported on negative, uncontrollable life events that had occurred to the adolescent within the past 3 months (e.g., friend moved away, parent lost job, parent arrested). Events were taken from the Children of Alcoholics Life Events Schedule (Roosa, Sandler, Gehring, Beals, & Cappo, 1988) and the General Life Events Schedule for Children (Sandler, Ramirez, & Reynolds, 1986), supplemented with items from other child life events schedules. Each informant's score was a count of reported stressful events. Correlations among reporters across waves varied as follows: mothers with fathers, .48–.54; mothers with adolescents, .36–.45; and fathers with adolescents, .32–.44. For the structural modeling, the stress variable was a multiple-reporter composite manifest variable created

[2] These outliers were 5 participants who reported extremely high use at Time 1 (more than 3.5 standard deviations above the mean) and steep decreases in use at Times 2 and 3. Their inclusion in the model produced estimation problems, with a large negative correlation between the intercept and slope factors and several models failing to converge. Although they may be a potentially important (albeit small) subsample when considering cessation of substance use, they cannot be appropriately modeled within our overall sample and thus were dropped from analyses.

using factor score regression weights.[3] Standardized factor loadings showed that each reporter's score loaded significantly, with parent data somewhat more heavily weighted (.75 for mother, .68 for father) than adolescent data (.57, averaged over waves).

Adolescent negative affect. Negative affect was measured using adolescent 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 α = .78–.79 across waves). Sample items included the following: cried a lot; felt nervous, high-strung, or tense. Perceived loss of control was assessed by three items from Newcomb and Harlow (1986; coefficient α = .72–.76 across waves; 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 α = .81–.87 across waves). Intercorrelations among the three dimensions ranged from .50 to .66 across waves. For the structural modeling, adolescent negative affect was a composite manifest variable created using factor score regression weights.[4] Standardized factor loadings showed that each indicator significantly loaded on the construct with slightly greater weight on perceived control (−.86) than internalizing symptoms (.72) or self-derogation (−.66, averaged across waves).

Emotionality and sociability. Emotionality and sociability were measured by parents' and adolescents' reports on a modification of the Emotionality, Activity, and Sociability Temperament Scale (Buss & Plomin, 1984). Coefficient alphas across reporters and waves ranged between .70 and .80 for emotionality and between .54 and .74 for sociability. Correlations between reporters for emotionality varied across waves as follows: mothers with fathers, .41–.45; mothers with adolescents, .21–.29; and fathers with adolescents, .19–.28. Correlations between reporters for sociability varied across waves as follows: mothers with fathers, .43–.46; mothers with adolescents, .38–.44; and fathers with adolescents, .22–.31. For the structural modeling, emotionality and sociability were multiple reporter composite manifest variables created using factor score regression weights.[5] Standardized factor loadings showed that each reporter's data significantly loaded for both constructs, with mothers' data weighing most heavily for sociability (.82) compared to father and adolescent (.56 and .53, respectively, averaged over waves), and parent data weighted more heavily (.69 for mother and .65 for father) than adolescent data (.39, averaged over waves) for emotionality.

Adolescent substance use. Adolescents self-reported their frequency of substance use in the past year (from none to daily use) on 12 items, including drinking beer–wine and hard liquor, drinking five or more drinks in a row, getting drunk on alcohol, and using eight illicit drugs. A substance use score was calculated by summing the responses to these 12 items.[6]

Because of the young age of the participants, the prevalence of substance use was generally low. Thus, the current study is best viewed as examining trajectories of substance use initiation (rather than substance abuse). However, by Wave 3 more than half of the COAs and one third of the controls used alcohol (with an average frequency among users of monthly use for COAs and occasional but less than monthly use for controls); 18% of the COAs and 6% of the controls used illegal drugs (with an average frequency among users of occasionally but less than monthly use for COAs and less than five times per year for controls), and 33% of the COAs and 13% of the controls had experienced a negative consequence of alcohol or drug use. Moreover, these prevalence rates should not be taken to minimize the significance of substance use in this sample, because substance use at these ages is prognostic of later substance abuse (Robins & Pryzbeck, 1985). For example, Robins and McEvoy (1990) found that, among individuals who retrospectively reported any use of illicit drugs before age 15, 87% reported a drug problem in adulthood. Among those who reported being drunk on alcohol before age 15, 84% reported an alcohol problem in adulthood.

Results

Testing for Growth in the Endogenous Variables Over Time

We first tested for growth in the endogenous variables over time using latent growth curve modeling (LGC; McArdle, 1988; Meredith & Tisak, 1984, 1990; Muthen, 1991). All models were estimated using EQS (Version 3.0; Bentler, 1989) based on the sample covariance matrix and a column vector of means.[7]

Step 1: FOCUS models. For each variable, we estimated a one-factor three-indicator Factor of Curves (FOCUS) model (McArdle, 1988), in which the indicators of the single latent factor were the Time 1, Time 2, and Time 3 measures of the variable. Eight models were estimated, one for each of the seven mediators and one for adolescent substance use. For each variable, an initial baseline model fixed all three factor loadings to 1.0 (representing no growth over time) while freely estimating the mean and variance of the latent factor. This no-growth model fit the data well for sociability, emotionality, and negative affect (i.e., all model chi-square test statistics were nonsignificant, all $ps > .15$). Because these three constructs showed no systematic change over time, they were represented in the structural model by their Time 1 scores.

However, this no-growth model did not hold for mother's and

[3] There were four endogenous variables that involved either multiple reporters or multiple indicators (stress, emotionality, sociability, and negative affect). These could not be used as multiple indicator latent factors because of the required number of parameters and the current sample size. To preserve maximal information, we created linear composite manifest variables using factor score regression weights. Longitudinal measurement models of each construct were estimated, with equality constraints placed on the loadings across time. Nested chi-square tests revealed no significant decrements in the model chi-square as a function of the imposed equality constraints, confirming that the constructs were structurally invariant over time. Accordingly, a single set of factor weights was used to create manifest linear composites for each construct at each time period. These manifest variables were corrected for unreliability (the measurement errors were set to [1-coefficient alpha] multiplied by the variance of the indicator; Bollen, 1989, p. 168).

[4] See Footnote 3.

[5] See Footnote 3.

[6] Because different types of adolescent substance use might have different determinants, we re-estimated our models predicting growth in alcohol use and heavy alcohol use separately. (The lower prevalence of illicit drug use precluded a separate test.) There were no substantive changes in the findings. We also tested whether a two-factor model (separating alcohol and drug use) would be a better fit to the data than a one-factor overall substance use model. The chi-square difference test showed no significant improvement in fit for the two-factor model; the two factors were highly intercorrelated, and there was a large cross-loading such that heavy alcohol use loaded on both factors. For these reasons, we used an overall substance use score as the dependent measure.

[7] The covariance matrix and vector of means are available from Patrick J. Curran.

father's monitoring, stress, peer substance use, or adolescent substance use. Thus, a series of nested models were estimated to ascertain the shape of the growth. Adolescent substance use and peer use showed significant linear increases over time. Mother's monitoring showed a significant linear decrease over time. Finally, stress and father's monitoring showed no changes from Time 1 to 2, but a decrease from Time 2 to 3. Because there were only three measurement points, the pattern of growth observed for environmental stress and father's monitoring could not be modeled further using an LGC framework. Accordingly, we used the Time 1 measures of stress and father's monitoring in the final model because they best captured the pattern over two of the three time points while also allowing for prospective prediction of adolescent substance use growth.

Step 2: Two-factor intercept and slope models. Because adolescent substance use, peer use, and mother's monitoring showed significant linear growth, for these constructs we estimated models that separated the intercept of the growth curve (which represents the starting point of the growth curve at Time 1) from the slope component of the growth curve (which represents the shape of the growth over time). For each construct, we estimated a two-factor, three-indicator model. The first factor was defined by fixing all three of the loadings from the Time 1, 2, and 3 measures of the construct to 1.0; thus, it represented the initial level or intercept of the growth curve. The second factor fixed the first loading to 0 (thus not allowing the Time 1 measure to load on this factor), the second loading was fixed at 1.0, and the third loading was fixed at 2.0. This second factor represented the linear slope of the growth curve. The mean of each factor represents the group parameter, and the variance of each factor represents the individual variation of each adolescent around the group parameter.

For both mother's monitoring and peer use, these models showed nonsignificant variances in the slope factor. This suggests that, over time, the entire sample experienced increases in peer use and decreases in mother's monitoring, but that these changes were uniform across individuals. Because of the lack of individual variation in growth, we used the Time 1 measures of peer use and mother's monitoring in the structural model. However, for adolescent substance use, there was significant individual variation in growth over time. Thus, the goal of all further analyses was to predict this individual variation in substance use growth.

Summary. Analyses suggested that the Time 1 scores for the predictor variables were appropriate for use in the structural model to predict adolescent substance use growth over time. This was true either because the predictors showed no growth over time (for sociability, emotionality, and negative affect); because they showed uniform growth without individual variation (for mother's monitoring and peer use); or because the pattern of growth could not be modeled with three time points (for stress and father's monitoring). However, because adolescent substance use showed both significant linear growth and significant individual variation in growth over time, it was represented in our model by a latent intercept factor (representing initial substance use levels) and a latent slope factor (representing rates of growth over time). Most important for the current study are predictors of the slope factor, because these are prospective predictors of individual rates of substance use growth over time.

Effect of Parent Alcoholism on Substance Use Growth

Our first question was whether parent alcoholism significantly predicted adolescents' substance use growth. To test this, we regressed the latent intercept and slope factors on mother's and father's alcoholism diagnosis, parent antisocial personality disorder, parent affective disorder, adolescent's age, and adolescent's gender (see Figure 1). A priori predicted paths were estimated from maternal and paternal alcoholism diagnosis to the substance use intercept and slope factors. This model was estimated and, on the basis of significant Lagrange Multiplier tests ($p < .01$), two paths from control variables were added: a path from adolescent age to substance use intercept, and from adolescent gender to substance use slope. This model fit the data well, $\chi^2(13, N = 316) = 24.5$, $p = .03$, Tucker–Lewis Fit Index (TLI; Tucker and Lewis, 1973) = .95, Comparative Fit Index (CFI; Bentler, 1990) = .98.

Both maternal and paternal alcoholism and adolescent age significantly predicted the substance use intercept factor. Thus, for COAs and older adolescents, the substance use growth curve started at a significantly higher level than it did for non-COAs and younger adolescents. More important, paternal alcoholism and adolescent gender significantly predicted the slope factor. That is, adolescents with alcoholic fathers and boys showed steeper substance use growth over time than did adolescents with nonalcoholic fathers and girls.[8]

Mechanisms Associated With Parent Alcoholism Risk

We next tested hypothesized mediators of parent alcoholism effects on substance use growth. Figure 2 presents the hypothesized model that considers the effects of parent alcoholism operating through elevations in environmental stress (which then act to increase negative affect), temperamental emotionality and sociability, and parent monitoring of the adolescent's behavior. We also tested whether negative affect influenced adolescent substance use both directly and indirectly (by increasing the likelihood of associating with drug-use-promoting peers). Paths from maternal alcoholism to father's monitoring and from father's monitoring directly to adolescent's initial levels of use (i.e., the intercept factor) were based on results from our earlier cross-sectional model (Chassin et al., 1993).

Before estimating the model, it was necessary to account for variance attributable to the control variables. Control variables were parent antisocial disorder, parent affective disorder, adolescent's age, and adolescent's gender. (Effects of ethnicity and

[8] The lack of maternal alcoholism effect on the slope factor was surprising. Because this effect might be due to the small number of alcoholic mothers in the subsample, we re-estimated this model using the full sample including single-parent families or those with noninterviewed parents. (This was possible because alcoholism diagnoses were available on all parents, even those who were not interviewed.) With the full sample, there was a marginally significant effect of maternal alcoholism on substance use slope ($p < .08$) such that adolescents with alcoholic mothers showed steeper substance use growth.

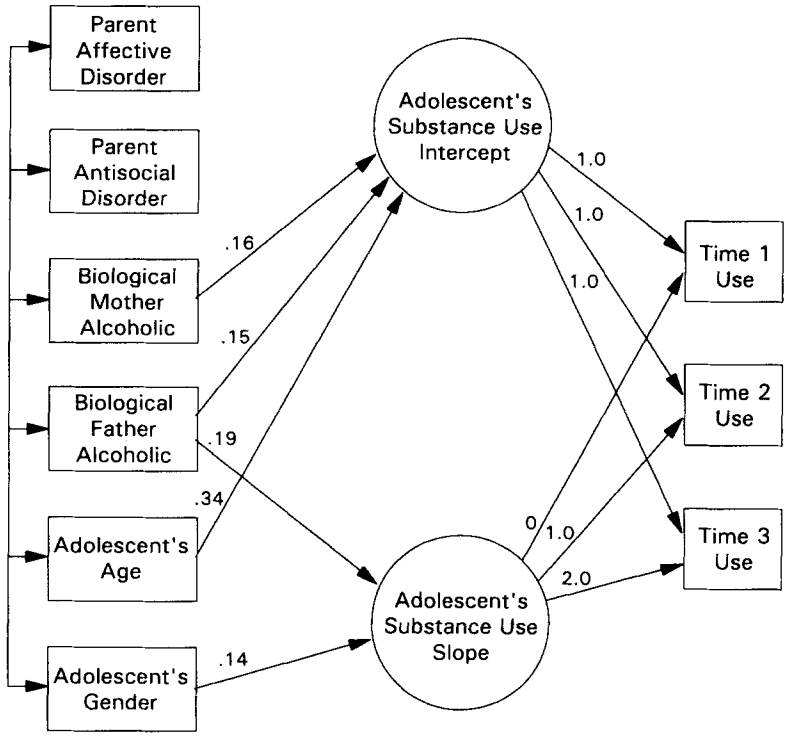

Figure 1. Direct effects model; $\chi^2(13, N = 316) = 24.5$, $p = .03$, Tucker–Lewis Fit Index = .95, Comparative Fit Index = .98. Only significant effects are shown. All coefficients are standardized and all ps $< .05$.

parent education were not considered because they were unrelated to the dependent measure at any wave of measurement.) To accomplish this, the a priori hypothesized paths from the noncontrol variables were freely estimated; the structural disturbances between maternal and paternal monitoring, stress, emotionality, and sociability were freely estimated; and all paths from the control variables were fixed to zero. This model was estimated, the path from the control variable with the largest Lagrange Multiplier was freed, and the model was re-estimated until no Lagrange Multipliers from control variables exceeded 6.6 ($p < .01$). This resulted in the freeing of six paths from control variables: parent affective disorder to stress and to emotionality; parent antisocial disorder to father's monitoring; adolescent age to negative affect and to peer use; and adolescent gender to the substance use slope factor. Although this procedure capitalizes on chance in estimating the effects of the control variables, it provides a stringent test of the theoretical variables of interest. The final model fit the data well, $\chi^2(62, N = 316) = 88.6$, $p = .01$, TLI = .95, CFI = .98; see Figure 3).

In terms of the parenting pathway, both maternal and paternal alcoholism were related to decreased paternal monitoring (although the relation was only marginally significant for father's alcoholism). In turn, adolescents whose fathers reported lower levels of monitoring were more likely to associate with drug-using peers, and these peer associations predicted increases in substance use over time. Adolescents whose fathers reported less monitoring of their behavior also had higher initial substance use levels.

In terms of the stress and negative affect pathway, maternal and paternal alcoholism significantly predicted higher levels of stress that in turn predicted higher levels of negative affect. Negative affect predicted greater associations with drug-using peers that in turn predicted both higher levels of initial substance use and steeper substance use growth. There was no direct effect of negative affect on substance use growth.

In terms of temperament variables, maternal alcoholism was marginally related to heightened emotionality, which in turn was significantly associated with elevations in negative affect and, thus, contributed to the negative affect pathway. High levels of sociability were significantly related to associations with drug-using peers. However, sociability showed no significant relation to parent alcoholism.[9]

[9] Our exclusion of single-parent families might have underestimated the effect of parent alcoholism on temperament, because this selection might have eliminated the most temperamentally "at risk" families. To assess this, we calculated correlations between parent alcoholism and the temperament variables separately for single-parent and two-parent families (relying on maternal and adolescent report of temperament because fathers in single-parent families were typically not interviewed). There were no significant differences in the correlations between parent alcoholism and sociability for either reporter, and no differences in the correlations between parent alcoholism and emotionality using adolescent report. Using the mother's report, there were stronger relations between parent alcoholism and emotionality in the single-parent families than in the two-parent families. Thus, the magnitude of the links between parent alcoholism and emotionality might be stronger if single-parent families were included (at least for the mother's report).

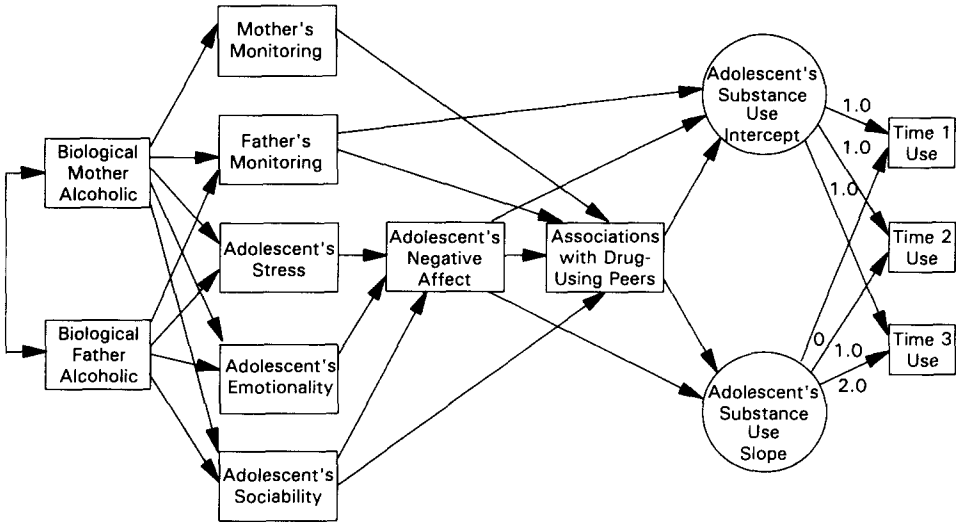

Figure 2. Hypothesized structural model.

Finally, the direct effect of paternal alcoholism on the substance use slope factor remained significant even after the inclusion of the hypothesized mediators in the model. Thus, the mediators could not completely explain the paternal alcoholism effect.[10] The final model explained a moderate amount of the variance in the substance use slope factor (R^2 = 13%) and a large amount of variance in the intercept factor (R^2 = 61%).[11]

Hierarchical Model Testing

To test whether a single domain of mediators was sufficient to explain substance use outcomes or whether multiple pathways were necessary, we estimated three nested models in which the regression parameters for each mediating path were set to zero. That is, one model fixed all paths associated with parenting to zero, another fixed all paths associated with temperament to zero, etc. Compared to the full model in which all hypothesized paths were freely estimated, the added restrictions in each of these three models produced a significant decrement in model fit. Thus, each of the three mediating pathways was necessary to best reproduce the observed data.

Mediation of Parent Alcoholism Effects

The direct effects models reported earlier showed that both maternal and paternal alcoholism predicted the intercept factor and that paternal alcoholism predicted the slope factor. To test whether our psychosocial variables significantly mediated these effects, we computed *z*-ratios for the total indirect effects in EQS. In predicting the intercept factor, the total indirect effects for both maternal and paternal alcoholism were significant (*z*-ratios = 2.34 and 2.17 respectively, both $ps < .05$). In predicting the slope factor, the total indirect effect for paternal alcoholism was marginally significant (*z*-ratio = 1.85, $p < .06$).[12]

Discussion

The current study provided a longitudinal test of parent alcoholism effects on adolescents' substance use growth and tested psychosocial mediators of these effects. As would be expected developmentally, our adolescent sample showed significant growth over time in their consumption of alcohol and illegal drugs. More important, however, latent growth curve modeling

[10] The use of lifetime diagnoses does not consider effects of the recency and severity of parent alcoholism (or of subclinical drinking problems in the control group). Accordingly, we re-estimated our model two other ways—operationalizing parent drinking problems as the number of alcohol-related consequences or dependency symptoms reported within the past year (among both alcoholic and control parents), and again considering the quantity–frequency of parents' alcohol consumption in the past year. All of the effects of paternal alcoholism were identical to those produced by the lifetime diagnoses. For maternal alcoholism, the links to father's monitoring, stress, and emotionality were weakened. Because we had only a small number of alcoholic mothers who were currently reporting drinking problems, this was a weaker test of maternal alcoholism effects. However, because only paternal alcoholism significantly predicted adolescents' substance use growth over time, these changes in the maternal alcoholism effects do not influence our conclusions. Parents' current use of other drugs might also be important. However, for current drug use (past 3 months) maternal use was at such low levels that analysis was not feasible, and paternal drug use did not significantly relate to the dependent variable over and above paternal alcoholism status. Accordingly, we did not consider parent current drug use in our multivariate model.

[11] Maximum likelihood estimation assumes that the observed data follow a multivariate normal distribution. Because our substance use scores were skewed, we re-estimated the model using manifest factor score regression composites of the intercept and slope of substance use, with robust maximum likelihood estimation from EQS (Bentler, 1989). No substantive differences were found.

[12] Maternal alcoholism showed a marginally significant indirect effect on the slope factor when the hypothesized mediators were included in the model (*z*-ratio = 1.89, $p < .06$). However, in the absence of a direct effect of maternal alcoholism on substance use growth, this indirect effect is not clearly interpretable and is likely due to suppressor effects of the mediating variables. Even when we re-estimated the direct effects model with the full sample, the direct effect of maternal alcoholism on slope was only marginally significant. Given the lack of a direct effect of

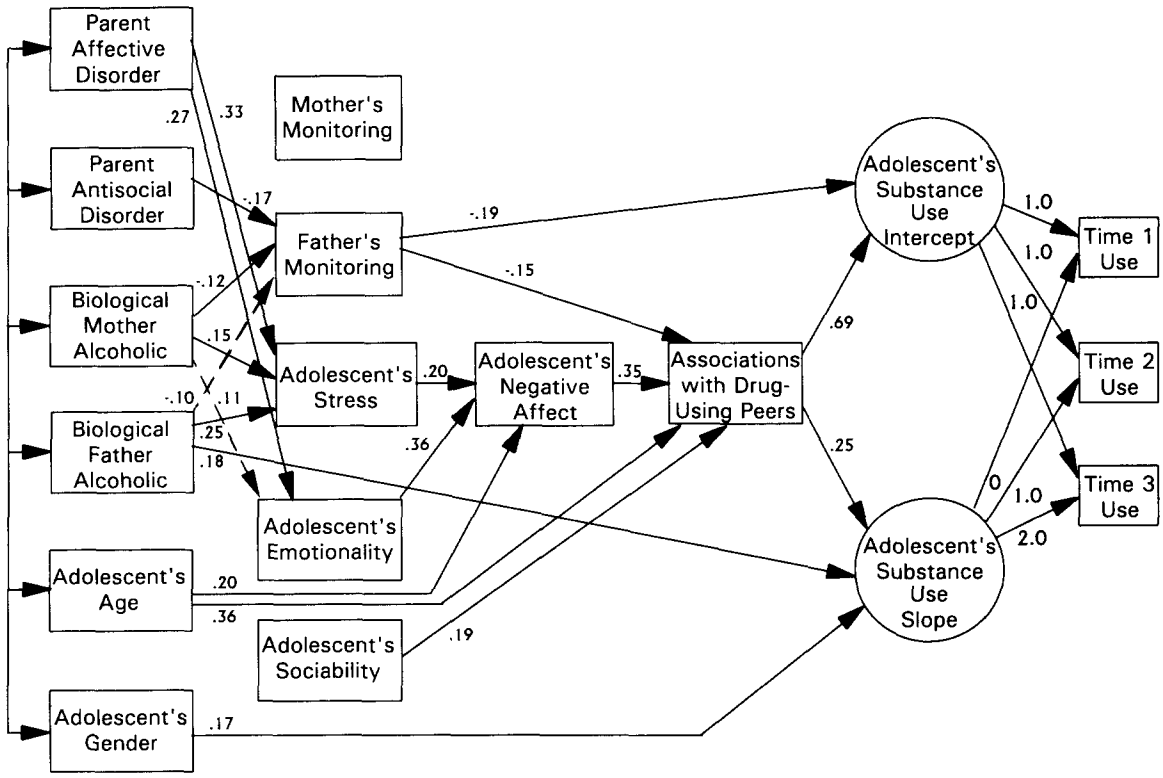

Figure 3. Final structural model. Standardized path coefficients are shown. For solid lines, $p < .05$; for dashed lines, $p < .10$; $\chi^2(62, N = 316) = 88.6$, $p = .01$, Tucker-Lewis Fit Index = .95, Comparative Fit Index = .98.

revealed significant individual differences in the rates of this growth, and paternal alcoholism significantly predicted steeper growth. Thus, adolescents with alcoholic fathers are not only more likely to use substances, but also increase their substance use at a more rapid rate than do their non-COA peers.

The finding that only paternal rather than maternal alcoholism predicted substance use growth over time was surprising. Because maternal alcoholism was associated with elevations in adolescents' initial levels of substance use, and because analyses of the full sample showed marginally significant prediction of substance use growth from maternal alcoholism, it would be premature to entirely rule out maternal alcoholism effects on adolescent substance use outcomes. Our ability to detect maternal alcoholism effects on substance use growth may have been weakened by the relatively small number of alcoholic mothers in the sample.

We also asked whether COAs' substance use outcomes could be accounted for by impaired parental monitoring, elevated environmental stress and negative affect, and elevated emotionality and sociability, all of which were hypothesized to be related to affiliations with drug-using peers. Our hierarchical model testing confirmed that no one domain of mediators could sufficiently account for COAs' substance use outcomes. Rather, it was necessary to consider all three domains. These results are consistent with recent theory that postulates multiple interrelated pathways to substance use (Sher, 1991; Zucker, 1994), and it underlines the importance of a simultaneous consideration of these multiple risk factors.

The central question, however, was whether these multiple risk factors could account for individual variation in substance use growth (i.e., whether they significantly predicted the slope of the growth curve). Whereas predictors of the intercept factor represent cross-sectional relations (with ambiguous directions of effect), predictors of substance use slope represent prospective predictors of changes in substance use over time. Of course, significant prediction of the slope factor does not rule out bidirectional effects in which adolescent substance use and the mediators show reciprocal relations. Indeed, theoretically, such reciprocal relations are quite likely, particularly for constructs such as peer affiliations or parent monitoring (e.g., Fisher & Bauman, 1988). However, our significant predictors of the slope factor can confidently rule out a unidirectional "reverse" direction of effect in which adolescent substance use influences the mediators without any reciprocal effects. Finally, as with all observational longitudinal research, our prospective prediction of the slope factor cannot rule out "third variables" underlying these relations. Thus, we can specify the temporal precedence between our predictor variables and adolescents' substance use growth, but this does not imply a causal relation.

maternal alcoholism on substance use growth, we have not interpreted the marginally significant indirect effect.

Our model accounted for 13% of the variance in this slope factor. This can be considered a moderate effect size (in Cohen's, 1988, terms) and is comparable to other prospective studies of adolescent substance use (Kandel, Kessler, & Margulies, 1978; Windle, 1990). Here, we discuss the implications of our findings for each of the hypothesized pathways, confining our interpretations to prediction of the slope factor (for a discussion of cross-sectional prediction, see Chassin et al., 1993). Moreover, because only paternal alcoholism showed a significant direct effect on substance use growth, we discuss our mediators in terms of their ability to account for this paternal alcoholism effect.

Parenting and Socialization Pathways

There was some support for father's monitoring as mediating the effects of paternal alcoholism on substance use growth. Father's alcoholism was associated with less paternal monitoring of adolescent behavior, which, in turn, predicted associations with drug-using peers. These peer associations prospectively predicted adolescents' growth in substance use involvement over time. These findings support family socialization models of adolescent substance use (Dishion et al., 1988; Hawkins et al., 1992) in suggesting that COAs are at risk for substance use growth in part because of impairments that occur in family socialization and behavioral management. The fact that the father's monitoring had unique effects (above and beyond a consideration of the mother's monitoring) is noteworthy because many studies of adolescent socialization consider only the mother's role in these parenting behaviors. Our data support the importance of the father's parenting behaviors as well.

However, this family socialization pathway could not fully explain the effects of the father's alcoholism on adolescents' substance use growth. Significant direct effects of the father's alcoholism remained, even when the hypothesized mediators were included in the model. This may be because monitoring is only one component of parenting thought to be important to adolescent substance use. More powerful effects might have been found if we had examined multiple aspects of parenting, including rule enforcement and parental supportiveness. Alternatively, our brief self-report measure of parent monitoring may have underestimated its importance.

Moreover, peers as well as parents are important socializing influences, and our findings showed that adolescents who (at the beginning of the study) had more friends who used drugs or tolerated the use of drugs also showed the steepest increase in substance use. This finding is in contrast to a recently reported longitudinal study (Farrell & Danish, 1993) that found no prospective effects of peer influences on subsequent gateway drug use in adolescents. The differences in findings may reflect the different ethnic composition of the samples. Farrell and Danish (1993) had a predominantly African American sample, and peer influences have been reported to be particularly small for this group (Farrell & Danish, 1993).

Stress and Negative Affect Models

The current findings also support stress and negative affect pathways as mediators of paternal alcoholism effects on adolescents' substance use growth. Paternal alcoholism was associated with elevations in environmental stress, which, in turn, were associated with heightened levels of negative affect. Negative affect was related to affiliation with drug-using peers, which significantly predicted increases in substance use over time. These findings support mechanisms such as Kaplan's (1980) self-derogation theory, which suggest that adolescents who experience low self-esteem (or lower levels of perceived control and higher levels of negative affect) are more likely to affiliate with deviant peer groups. This peer affiliation raises risk for delinquent behaviors, including substance use. As with socialization models, however, these mechanisms could not fully explain the effect of paternal alcoholism on substance use growth.

Unlike our cross-sectional results, there was no unique direct path from negative affect to substance use growth. Such an effect might have been predicted by a simple negative affect regulation model of adolescent substance use. As other researchers have suggested, negative affect regulation motives may be more important for later stages of substance "abuse" than for early adolescent substance use (Swaim et al., 1989). Alternatively, our annual measurement intervals may not be optimal for capturing simple negative affect regulation mechanisms that may operate over much briefer time windows. Nevertheless, that we found a unique direct path from negative affect to substance use cross-sectionally but not longitudinally raises the possibility that our cross-sectional finding represents a "reverse" direction of effect. That is, adolescent substance use may act to increase adolescents' levels of negative affect (Hansell & White, 1991). The most parsimonious interpretation of our longitudinal findings are that they support self-derogation mechanisms involving stress and negative affect rather than simple negative affect regulation models of use.[13]

Temperamental Emotionality and Sociability

Our findings did not support emotionality or sociability as mediators of paternal alcoholism effects because father's alcoholism was not significantly related to either construct. In terms of sociability, as Tarter et al. (1985) have speculated, previous notions of COAs as particularly sociable may have confused an outgoing, extraverted style with disinhibition (an indicator of behavioral undercontrol). Perhaps behavioral undercontrol is the "true" temperamental mediator of paternal alcoholism effects. Recent cross-sectional findings with college student COAs support this hypothesis (Sher et al., 1991). Temperamental bases for behavioral undercontrol could also be exacerbated by harsh or inconsistent parenting among alcoholic parents. However, recent adoptee data raise the possibility that behavioral undercontrol (as measured by

[13] It is also possible that direct negative affect regulation motives operate only within a subgroup of adolescents—those who do not have effective alternative strategies for coping with negative affect. However, other analyses of the current data show this moderating effect for measures of behavioral and cognitive coping strategies in cross-sectional but not longitudinal analyses (Hussong, 1995). Other measures of coping (e.g., drinking coping motives) might identify a subgroup of adolescents for whom direct negative affect regulation mechanisms are operative, but these effects were not found with our measures of behavioral and cognitive coping.

aggression and antisociality) is not uniquely tied to parent alcoholism but rather mediates the effects of co-occurring parent antisocial personality (Cadoret, Yates, Troughton, Woodworth, & Stewart, 1995). This is consistent with our baseline data that found adolescents' externalizing symptoms to be predicted by parent antisocial personality rather than uniquely related to parent alcoholism (Chassin et al., 1991). Thus, although indicators of behavioral undercontrol and antisociality may be important predictors of adolescent substance use outcomes, more research is needed to determine whether these characteristics mediate the effects of parent alcoholism or of parents' co-occurring antisociality.

In terms of emotionality, the current study found links to maternal but not paternal alcoholism. However, our data may have underestimated the role of emotionality because of our exclusion of single-parent families, among whom links between parental alcoholism and emotionality were particularly strong. Thus, samples that include greater number of single-parent families might find a significant mediational role for emotionality.

Direct Effects of Paternal Alcoholism

Perhaps the most surprising finding was that (unlike our cross-sectional model) the direct effect of paternal alcoholism on substance use growth remained significant even after the inclusion of our psychosocial mediators. Thus, although these hypothesized mediators play a role in COAs' substance use, they could not fully explain the risk for escalating substance use that was associated with paternal alcoholism. How then can we interpret the direct effect of paternal alcoholism on growth curves of adolescent substance use? One possibility is that important mediators of paternal alcoholism effects were unmeasured by our study. For example, previous research has suggested that COAs may experience greater pharmacological benefits from substance use (Newlin & Thomson, 1990; Sher, 1991), and these benefits may mediate the effect of paternal alcoholism on substance use growth. Indeed, these pharmacological benefits would be more likely to determine substance use growth than adolescents' first use of substances because they require some experience with the substance to be operative. Both behavioral undercontrol and the pharmacological effects of substances are worthy of further investigation as potential mediators of parent alcoholism effects on adolescents' substance use growth.

Finally, although the current study corrected many of the methodological problems in earlier research (e.g., by using a longitudinal design, a community sample, multiple reporter data, and direct ascertainment of parent alcoholism), it is also important to recognize its limitations. First, the small number of alcoholic mothers in the sample limits our ability to detect maternal alcoholism effects. Second, the study included only three time points, limiting our ability to model patterns of growth for two of the psychosocial variables. Third, alcoholism was treated as a unitary disorder, and no attempt was made to subtype particular forms of alcoholism. Similarly, substance use was treated as a unitary variable, and models focused on specific substances or specific transitions between different stages of substance use involvement might produce different findings. Fourth, our use of two-parent families and our higher refusal rates of Hispanic subjects suggests caution in generalization and may have underestimated the importance of temperamental emotionality.

In sum, the current study provided a longitudinal test of parent alcoholism effects on adolescent substance use growth and tested hypothesized mediators of these effects. Findings showed that boys, adolescents with drug-using peers, and adolescents with alcoholic fathers had steeper substance use growth trajectories. Data were consistent with father's monitoring, stress, and negative affect as mediators of this paternal alcoholism effect. However, because the direct effect of father's alcoholism on substance use slope remained significant even after considering the hypothesized mediators, it is likely that other (unmeasured) mediators are necessary to fully explain paternal alcoholism risk. Mediators of interest for future research include behavioral undercontrol and COAs' psychopharmacological experiences of substance use effects.

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Received June 22, 1994
Revision received June 16, 1995
Accepted August 17, 1995 ■