A Comparison of Mechanisms Underlying Substance Use for Early Adolescent Children of Alcoholics and Controls*

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ABSTRACT. The current study examined differences between children of alcoholics (COAs) and controls in parent monitoring, stress-negative affect, and temperament mechanisms underlying early adolescent substance use. Using structural equation modeling, we tested whether these mechanisms were equally predictive of substance use for both groups. We extended an earlier study that tested mediators of COA risk for substance use but did not examine COA status as a moderator of these mechanisms. Overall, we found no evidence of differential importance for COAs and non-COAs of the parent monitoring and negative affect mechanisms. Parental socialization and negative affect mechanisms significantly predicted adolescent substance use regardless of COA status. Differences did emerge regarding the effects of age and parent education on peer substance use and the effect of sociability on adolescent substance use. (*J. Stud. Alcohol* 55: 269-275, 1994)

FINDINGS that children of alcoholics (COAs) are at increased risk for adult alcoholism has stimulated interest in uncovering the mechanisms underlying this vulnerability (see Sher, 1991). Little is known, however, about alcohol and drug use among COAs during early adolescence which is a period of risk for substance use initiation (Johnston et al., 1988). Research has suggested that adolescent substance use may result from multiple mechanisms including peer modeling and social influence, deficits in parental socialization, control and support, parent modeling and tolerance of use, and attempts to cope with stress-induced negative affect (see Chassin, 1984, for a review). However, it is unknown if the mechanisms underlying substance use differ for high-risk adolescents (such as COAs) compared to the general adolescent population. If particular mechanisms were associated with substance use in a high-risk group, they would be of special interest for preventive intervention efforts. The current study tested whether mechanisms underlying early adolescent substance use differed for a sample of COAs and their non-COA peers.

Multiple pathways to substance use have been posited to account for the increased risk for substance use among COAs. These mechanisms have included differential sensitivity to the positive or negative effects of alcohol, parent modeling of alcohol abuse and exposure to alcohol, and use of alcohol and drugs to cope with tendencies to-

ward negative affective states (see Sher, 1991). Recently, Chassin and colleagues found that parental alcoholism affected early adolescent substance use through stress and negative affect mechanisms and through impairments in parent monitoring, both of which increased the probability of associations with a peer network that supported substance use behavior. Parent alcoholism was also associated with higher levels of temperamental emotionality in adolescents which raised risk for experiencing negative affect. These findings were produced using a community sample in which parent alcoholism was directly ascertained and data were gathered from multiple informants (Chassin et al., 1993).

However, Chassin et al.'s study did not include a test of whether the parent monitoring, negative affect and temperament mechanisms were equally predictive of substance use for COAs and controls. Such a test would examine the role of COA status as moderator of these mechanisms. The current study extended Chassin et al.'s work to address this issue: namely, are the processes that place adolescents at risk for substance use similar for COAs and non-COAs? The stress-negative affect mechanism was of special interest. Research suggests that young adult COAs derive greater stress response dampening benefits of alcohol use than do their non-COA peers (Levenson et al., 1987). Consequently, COAs may be more likely to use substances to cope with stress-induced negative affective states. This hypothesis suggests that mechanisms involving stress and negative affect may be more important to substance use among COAs than among non-COAs. To test this prediction, we used the same sample and measures reported in Chassin et al. (1993) to compare parameter estimates for the model in Figure 1 for COAs and non-COAs.
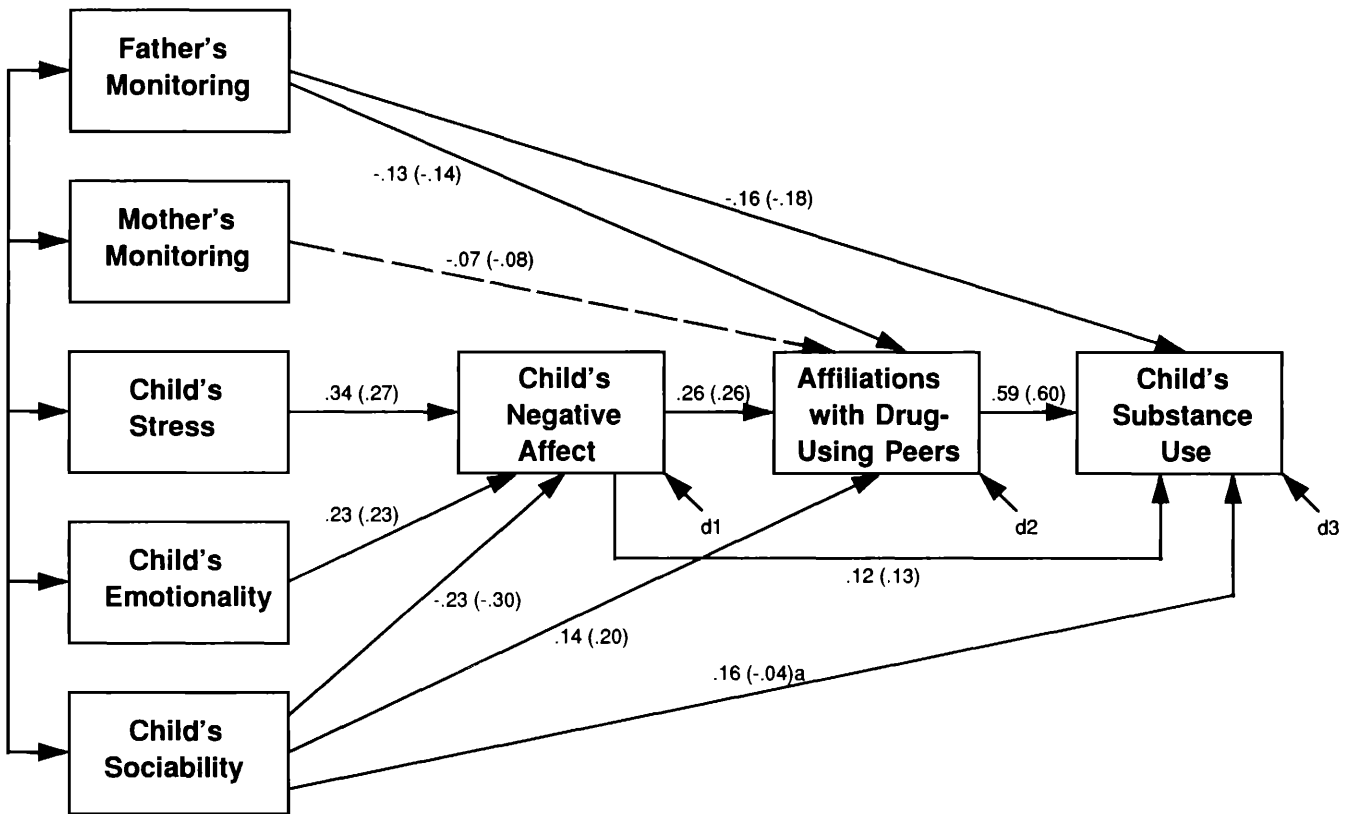
FIGURE 1.   The hypothesized structural model of mechanisms underlying early adolescent substance use. For presentational ease, the exogenous variables of age, gender, parent education, current parent alcohol consumption, and their associated paths are not shown. Path coefficient estimates are standardized within each group; estimates in parentheses are for controls and without parentheses are for COAs. d1 to d3 = structural disturbances. Solid lines indicate significant effects at $p < .05$ for both groups. Dashed lines indicate marginally significant effects for both groups. ([a]The path coefficient for sociability→substance use is significant for COAs [$b = .16$, $p < .05$] but not for controls [$b = -.04$, NS].)

Method

Subjects

Of a total sample of 454 adolescents and their parents, a subsample of 327 families with complete data (provided by two parents and the adolescent) was used for the analyses reported in the current study.[1] Adolescents in this subsample ranged in age from 10.5 to 15.5 years (mean age = 12.7 years) and approximately half were female ($n = 176$). In addition, 178 adolescents had at least one biological alcoholic parent who was also a custodial parent (COAs), and the remaining 149 adolescents were demographically matched controls with biological and custodial parents having no history of alcoholism. (For additional information, see Chassin et al., 1993.)

Recruitment

Recruitment procedures are presented in detail elsewhere (Chassin et al., 1991, 1992). COA families were recruited using court records (full sample = 103), well-

ness questionnaires from a health maintenance organization (full sample = 22) and community telephone surveys (full sample = 120). (One family was referred by a local VA hospital.) Demographically matched controls (matched on ethnicity, family composition, adolescent age within 1 year and SES) with no history of parent alcoholism were recruited over the telephone using reverse directories to identify families in the same neighborhoods as COAs. Adolescents had to be Hispanic or non-Hispanic Caucasian and 10.5-15.5 years old, English-speaking, and without cognitive limitations that would preclude interview. Analyses to detect participation bias determined that magnitude of bias was small and unrelated to archival indicators of alcoholism (see Chassin et al., 1991, 1992).

Procedure

Families were invited to participate in a study of adolescent development and substance use. Data were collected by trained interviewers using individual computer-assisted interviews with the adolescents and their parents. Interviewers were blind to alcoholism status of the family.

To minimize contamination, family members were interviewed individually on one occasion by different interviewers; privacy was emphasized. Confidentiality was assured and reinforced with a DHHS Certificate of Confidentiality.

Measures

The measures of interest were drawn from a larger interview battery. (A complete report appears in Chassin et al., 1993.) All variables were incorporated into the hypothesized structural model as manifest indicators and construction of these variables is described below.[2]

Demographic control variables. Child age, gender and average level of parent educational attainment were used as background control variables in the model.

Parent alcoholism. Lifetime DSM-III diagnoses of alcoholism (abuse or dependence) were obtained from parent interview using a computerized version of the Diagnostic Interview Schedule (DIS) (Robins et al., 1981).

Predictor variables in the hypothesized structural model. Mother and father monitoring of child behavior was assessed by parent self-report (3 items, e.g., "I had a pretty good idea of [the child's] plans for the day," Cronbach's α_{mother} = .79, Cronbach's α_{father} = .75). Child's current life stress was how many of 22 negative, uncontrollable events had occurred in the last 3 months as reported by parents and adolescents. Adolescents also reported on four additional peer-related events. Items were taken from the Children of Alcoholics Life Events Schedule (Roosa et al., 1988) and the General Life Events Schedule for Children (Sandler et al., 1986) supplemented with several items from other children's life events schedules. For the structural model, the life stress variable was a multiple reporter composite manifest variable using factor score regression weights. Child emotionality and sociability were measured using a modification of the adult version of the Emotionality-Activity-Sociability Scale (EAS) (Buss and Plomin, 1984). Coefficient alphas for the child, mother and father reports were, respectively, .72, .78 and .76 for emotionality (8 items) and .46, .62 and .60 for sociability (4 items). For the structural model, these two temperament variables were multiple reporter composite manifest variables created using factor score regression weights. Child's negative affect was measured using child self-report of internalizing symptomatology (seven items from the Achenbach Child Behavior Checklist; Achenbach and Edelbrock, 1981; α = .78), self-derogation (seven items from Rosenberg's 1977 scale; α = .81), and perceived loss of control (three items from Newcomb and Harlow, 1986; α = .73). For the structural modeling, child's negative affect was a composite manifest variable created using factor score regression weights. Affiliation with drug-using peers was measured using the adolescent's estimation of how many friends used alcohol,

marijuana and other drugs both occasionally and regularly (six items adapted from the Monitoring the Future study; Johnston et al., 1988; α = .92) and of how close friends would feel about the adolescent's use of these substances both occasionally and regularly (seven items; α = .93). The two scale scores were standardized and averaged to represent the proximal peer environment.

The dependent measure: Adolescent substance use. Adolescents self-reported their frequency of consumption of beer/wine and distilled spirits (2 items), five or more drinks in a row (1 item), getting drunk on alcohol (1 item), and eight illicit drugs (8 items) all in the past 3 months. Response options ranged from (0) not at all to (7) every day. A single substance use score was calculated by summing the responses to the 12 items. A normalizing transformation was used to reduce skewness (after transforming, skewness was 1.75).[3]

Results

Model specification and adequacy of fit for the full sample

Before testing for differences in path estimates between COAs and controls, the fit of the hypothesized model in Figure 1 was estimated for the full sample (n = 327).[4] To account for covariation among the background control variables and the endogenous variables, the model was first estimated with all paths from child age, gender and parent education fixed to zero; control paths with high modification indices ($\geq$ 5) were successively freed until none remained. Only two paths were significant: child age to child negative affect and child age to affiliations with drug-using peers. These two paths were included in the structural model.

The hypothesized, structural model fit the data very well (χ^2 = 18.75, 15 df, p > .22, TLI = .98, BBI = .98).[5] Moreover, examination of the residuals and modification indices further indicated that the observed variance/covariance matrix was adequately reproduced by the model in Figure 1. As described in Chassin et al. (1993), less parental monitoring was associated with greater adolescent affiliation with drug-using peers ($b_{father's\ monitoring}$ = −.17, p < .01; $b_{mother's\ monitoring}$ = −.09, p < .13) and with adolescent substance use ($b_{father's\ monitoring}$ = −.16, p < .01); affiliation with drug-using peers was in turn associated with increased adolescent substance use (b = .63, p < .01). Adolescent stress events were significantly related to adolescent negative affect (b = .38, p < .01) and negative affect was in turn significantly related to affiliation with drug-using peers (b = .29, p < .01). Negative affect was also marginally directly related to adolescent substance use (b = .11, p < .10). Adolescent emotionality was associated with higher levels of adolescent negative affect (b = .18, p < .05), while ad-

TABLE 1.   Comparisons of model parameters between COAs and controls

Model	χ^2 (df)	TLI	BBI	χ^2_{diff} (df)
1. All parameter estimates invariant	131.71[‡] (81)	.91	.85	–
2. Variances/covariances for exogenous variables, disturbances for endogenous variables, free within groups	51.44 (42)	.97	.94	80.27[‡] (39)
3. Age→peers free within groups	46.86 (41)	.98	.95	4.58* (1)
4. Parent education→peers added to model, invariant between groups	45.07 (40)	.98	.95	1.79 (1)
5. Parent education→peers free within groups	40.30 (39)	1.00	.95	4.77* (1)
6. Sociability→substance use added to model, invariant between groups	39.02 (38)	1.00	.96	1.28 (1)
7. Sociability→substance use free within groups	34.94 (37)	1.00	.96	4.18* (1)

Notes: "Invariant" refers to specified parameter(s) estimated as equal for COAs and controls. "Free within groups" refers to specified parameter(s) estimated as not equal for COAs and controls. Path coefficient parameter estimates are denoted by arrows surrounded by the relevant constructs (e.g., the effect of child age on affiliations with drug-using peers is denoted by age→peers). Peers = affiliations with drug-using peers. χ^2_{diff} indicates test of significant change in model fit from the previous model.

*p < .05.   [‡]p < .001.

olescent sociability was associated with less negative affect ($b = -.28$, $p < .01$) but also with greater affiliation with drug-using peers ($b = .19$, $p < .01$).[6]

Model fit for COAs and controls

An iterative series of cross-group analyses was conducted to determine the presence of significant differences between COAs and controls in overall model fit and in individual parameter estimates. First, the overall fit of the structural model in Figure 1 was compared for COAs and controls. A cross-group comparison was conducted with all parameter estimates specified as invariant across groups. The resulting fit, displayed in Table 1 for Model 1, was less than adequate and confirmed the presence of significant differences in overall model fit.

Second, differences between COAs and controls in individual parameter estimates were examined. The presence of multiple modification indices greater than five indicated that the variance/covariance matrix for the exogenous variables and the disturbances associated with the endogenous variables were significantly different across groups. These parameters were freed (1) to improve model fit for both groups in order to have increased confidence in path coefficient differences and (2) because differences in the variance/covariance and disturbance parameter estimates across groups were consistent with theory-based expectations (e.g., greater variability in stress among COAs

because of previously established links between parent alcoholism and environmental stress; Roosa et al., 1988). Model fit improved significantly and is displayed in Table 1 for Model 2.

The hypothesized differences between COAs and controls with regard to the relation between negative affect and substance use were not found. Modification indices associated with these two paths (negative affect to affiliations with drug-using peers, negative affect to substance use) were very low ($\leq$ 1) both before and after freeing the parameters described above, indicating a lack of significant differences in the paths from negative affect for the two groups. However, several differences between groups were identified by high modification indices. In Table 1 it can be seen that there was significant improvement in model fit after the path from child age to affiliations with drug-using peers was released from invariance (Model 3) and after the paths from parent education to affiliations with drug-using peers and from sociability to substance use were added to the model and released from invariance (Models 5 and 7, respectively). Final path coefficients for both groups are in Figure 1. Child age was more strongly related to affiliations with drug-using peers for COAs than for controls, parent education was more strongly related to affiliations with drug-using peers for controls than for COAs, and sociability was more strongly related to child substance use for COAs than for controls. There were no other indications of reliably significant differences between groups in any of the remaining path coefficient estimates.[7] Final multiple R^2 estimates for child negative affect, affiliations with drug-using peers and child substance use were .36, .38, .54 for COAs, and .43, .32, .51 for controls, respectively. These estimates each differed significantly between groups at $p<.05$ as indicated by significant increases in the model chi square when each estimate was individually specified as invariant (χ^2_{diff}, 1 df, ranged from 4.60 to 6.30, all $p<.05$). Overall, despite these differences in predicted variance, the findings suggested that the parent monitoring and stress and negative affect mechanisms were similarly related to substance use for COAs and controls.[8]

Discussion

The goal of this study was to consider possible differences between COAs and non-COAs in the mechanisms underlying early adolescent substance use. In particular, we hypothesized that negative affect would relate more strongly to substance use (both directly and indirectly via involvement with substance-using peers) for COAs than for non-COAs. Our findings did not support this prediction. Rather, our findings indicated that parent monitoring and stress and negative affect mechanisms were significantly related to substance use for both COA and non-COA adolescents, and the degree of association was not

different between the two groups. This finding is noteworthy in view of Chassin et al.'s (1993) finding that COA parents monitored their children less and COAs experienced significantly more stress and negative affect than controls. Thus, although early adolescent COAs show higher levels of substance use and higher levels of these particular psychosocial risk factors, these mechanisms underlying substance use appear to operate both for those with and without alcoholic parents. These findings suggest that programs aimed at teaching coping skills or at improving parent management could be useful regardless of parent alcoholism history.

Our finding that negative affect mechanisms operated similarly for COAs and controls may be due to the early ages of our adolescents. We purposefully sampled youth in the 10-15 year old age range with the aim of capturing the period of risk for substance use initiation (Johnston et al., 1988). This strategy resulted in our having few substance-abusing or substance-dependent adolescents— the population typically described with regard to negative affect regulation motives for substance use. Particularly strong negative affect regulation motives for use among COAs (compared to non-COAs) may not emerge until older ages and/or higher levels of use.

Evidence that girls, more than boys, report drinking to cope with stressors (Windle, 1991) suggests that gender rather than parent alcoholism may moderate the impact of stress-negative affect mechanisms on substance use. However, Chassin et al. (1993) did not find gender differences in the stress and negative affect mechanism when testing their mediational model for boys and girls separately. A moderating effect of gender may not emerge until later in adolescence.

Differences between COAs and non-COAs were found for the effects of child age and parent education on affiliations with drug-using peers, and for the effect of sociability on child susbtance use. Age was more strongly related to affiliations with drug-using peers for COAs than for controls. In other words, as they approach middle adolescence, COAs may escalate more rapidly into a high-risk environment marked by relatively greater immersion in a drug-tolerant peer network. Furthermore, for COAs, these associations were independent of socioeconomic status (as defined by parent education). For controls, however, a drug-tolerant peer environment was related to parent education, suggesting that broader sociocultural factors affect peer choice in the absence of a family history of alcoholism.

We expected sociability to exert all of its influence through affiliations with drug-using peers. Thus, our finding that sociability was directly (and more strongly) related to substance use for COAs than for non-COAs was surprising. Perhaps our operationalization of peer influence as friends who use drugs or who tolerate drug use was too narrow to capture all of the social interactions

that put COAs at risk for substance use. These interactions may include friends who are deviant in other ways (not directly reflected in their substance use) or interactions with drug-using individuals who are not perceived as "friends" (e.g., older relative, neighbors). Alternatively, sociability may not only elevate associations with deviant peers but also may serve as a marker for a disinhibited behavioral style that has been associated with the development of alcoholism, with substance use and with greater stress response dampening effects of alcohol (Sher and Levenson, 1982; for reviews, see Tarter et al., 1985; Windle, 1990). These mechanisms deserve future research attention in explaining the differential risk for adolescent substance use associated with parent alcoholism.

Overall, our findings suggested that parent monitoring and negative affect mechanisms significantly predicted early adolescent substance use regardless of parental history of alcoholism. Our failure to find a predicted stronger effect of the negative affect mechanism for COAs persisted independently of our consideration of current parent alcohol consumption. However, different definitions of parental alcoholism, different ages of COAs or different operationalizations of constructs might all affect findings (Sher, 1991), and our results are in need of replication with other samples and longitudinal designs.

Acknowledgments

Notes

1. When investigating the mechanisms underlying the relation between COA status and adolescent substance use, Chassin et al. (1993) tested their hypothesized mediational model using the subsample of families with complete data ($n = 327$) and the entire sample of two-parent families ($n = 416$) where data for one noninterviewed parent was imputed (BMDP AM program, Version 5; see also Little and Rubin, 1987). Similar findings were produced using the samples of 327 and 416 such that, with only one exception, all paths reported as significant using the sample of 327 remained significant using the sample of 416. The one change was that the effects of emotionality bypassed negative affect to predict associations with drug-using peers. In addition, several paths that were nonsignificant or marginally significant in the original model became significant using the imputed data: mother's alcoholism was significantly associated with lower levels of maternal monitoring, lower maternal monitoring was associated with higher levels of peer drug use and father's alcoholism was significantly associated with adolescent's heightened emotionality. Thus, re-estimating the model using imputed data for the total sample of two-parent families closely replicated the findings based on the sample of 327 two-parent families with complete data.

   In addition, comparisons were performed between two-parent and single-parent families (t tests and chi squares) to assess whether the exclusion of single-parent families (33 single mothers and 5 single fathers) introduced bias into the sample. The groups were compared on a variety of demographic variables as well as on all variables represented in the hypothesized mediational model tested by Chassin

et al. (1993). There were no significant differences between the single-parent and two-parent families for 18 of the 29 comparisons including, most importantly, the adolescent's alcohol- or drug-use outcomes. Of the significant differences that were found, the directions of the differences were not consistent in terms of putting either the single-parent or the two-parent family at higher risk. Moreover, the differences between the two groups were generally minor.

2. Composite manifest variables for constructs involving multiple reporters (stress, emotionality, sociability) or multiple indicators (negative affect) were created to preserve information from the multiple reporters and multiple indicators. The inclusion of these as latent variables was untenable given the required number of parameters and current sample size which would have exceeded Bollen's (1989, p. 268) suggestion to have at least several cases per free parameter. To create the multiple reporter variables, weighted linear composites of mother, father and child report of stress, emotionality and sociability were calculated using factor score regression weights taken from a single three-factor measurement model of these constructs. The negative affect variable was constructed similarly using weights taken from a single one-factor measurement model (see Loehlin, 1987, for a more detailed discussion of factor-score regressions). Composite reliabilities were calculated for each manifest variable using the estimates of error and total variance produced by the measurement model (Lord and Novick, 1968, p. 86). The error terms for these manifest variables, and for affiliations with drug-using peers and child's substance use, were then set to one minus the reliability multiplied by the variance of the indicator. (For additional details concerning these measures see Chassin et al., 1993).

3. The normalizing transformation was conducted for the full sample of 327 to retain a common metric for the substance use variable for COAs and controls—a requirement for comparing covariance structures across groups (Joreskog and Sorbom, 1989).

4. All analyses were based on the variance/covariance matrix in LISREL VII under maximum likelihood estimation.

5. The Tucker-Lewis (TLI) and Bentler-Bonnett (BBI) indices of incremental fit range from 0 to 1.00; Bentler and Bonnett (1980) recommend .90 as a minimal criterion for acceptable model fit.

6. Parameter estimates (b's) are standardized. When presented for cross-group analyses, b's are taken from LISREL's within-group standardized solution.

7. One final modification index equal to 6.22 remained for the path from emotionality to negative affect. When this path was freely estimated within groups, emotionality was significantly related to negative affect for controls ($b = .34, p < .01$) but not COAs ($b = .09$, NS) and model fit improved significantly ($\chi^2_{diff} = 6.52$, 1 df, $p < .05$). However, the estimated correlations between emotionality and stress exceeded 1.0 for both groups and differed slightly in magnitude between COAs and controls, prompting concern that the path coefficient difference was unstable and due to differential collinearity between groups. To test the possibility that over-correction for measurement error caused the out-of-bound correlation and, subsequently, the group differences in the path coefficient estimates, the final cross-group model was tested with the measurement errors for stress and emotionality set to zero ($\chi^2 = 31.49$, 37 df, $p > .72$). Statistically significant relations between stress and negative affect and between emotionality and negative affect were found for both groups, but the modification indices no longer suggested a difference between groups in the relation between emotionality and negative affect. Therefore, although the relations among stress, emotionality and negative affect were reliably significant both with and without correction for measurement error, the COA/non-COA difference in the relation between emotionality and negative affect was determined to be due to over-correction for measurement error and therefore unstable.

8. To determine whether our findings were independent of current parent drinking, the series of cross-group tests conducted above was repeated with current parent alcohol consumption as an additional exogenous variable affecting child's substance use. This single manifest variable was not included in the original model tested above because (a) Chassin et al. (1993) determined that effects of parental alcohol consumption were different for mothers and fathers, and (b) because inclusion of both mother and father alcohol consumption in the current article was untenable due to the required number of parameters and sample size. However, in an effort to approximate the effect of current parent drinking, a single manifest variable was created using parent self-report of alcohol quantity and frequency of use in the past 3 months. Quantity-frequency products were calculated separately for each parent and natural log transformations were used to reduce skewness below 1.00 as in Chassin et al., 1993. Of the two parent reports, the one reporting higher quantity-frequency was chosen in order to reflect current parental alcohol consumption. The predicted paths of theoretical importance (i.e., in Figure 1) were all replicated, with parent monitoring and stress and negative affect mechanisms significantly related to substance use for both COA and non-COA adolescents. Current parent drinking was significantly related to child's substance use for both COAs ($b = .14, p < .01$) and controls ($b = .13, p < .01$), and all of the group differences in path coefficients were confirmed. Final multiple R^2 estimates were: child's negative affect, .36; affiliations with drug-using peers, .38; child's substance use, .56 for COAs; and .43, .32 and .54, respectively, for controls; and these estimates differed significantly between groups ($p < .05$). The final model reproduced the observed variance/covariance matrix very well ($\chi^2 = 40.07$, 42 df, $p > .55$, BBI = .96, TLI = 1.00).

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