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THE EFFECT OF HOME ENVIRONMENT ON ADOLESCENT SUBSTANCE USE AND DEPRESSIVE SYMPTOMS

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We used data from the screening phase and first two waves of a panel study to compare the home environments of families with a substance-abusing parent, families with a depressed parent, and families in a comparison group. We diagnosed substance use disorder and affective disorder by administering the Structural Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders (third edition, revised) to each participating parent. The data suggest that families in which parents display a substance use disorder are very similar to those in which parents suffer from affective disorder, in terms of negative life events and lower family cohesion. The results of structural equation modeling indicate that parental substance use disorder and parental affective disorder influence adolescent substance use and depressive symptoms. In addition, parental substance use disorder has a direct influence on adolescent substance use at the time the first-wave data were collected, but this effect does not persist over time.

Introduction

Parental psychopathology is a risk factor that increases the likelihood of maladjustment among children. Literature has documented that children with drug-abusing and alcoholic parents are at greater risk than their peers for alcohol use (Chassin et al. 1993), for drug use (Gross and McCaul 1990; Johnson et al. 1989; Weintraub 1990), and for depression (Clair and Genest 1987; Gross and McCaul 1990; Johnson et al. 1990-1991; Moos and Billings 1982; Roosa et al. 1988;

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reviewed in West and Prinz 1987). The results of offspring studies indicate a strong and consistent association between parental depression and depression in children (reviewed in Beardslee et al. 1983; Beardslee et al. 1993; Downey and Coyne 1990; Hammen 1992; Weissman et al. 1987). In addition, children of depressed parents show elevated rates of substance abuse compared to children of non-depressed parents (Beardslee et al. 1987; Hammen et al. 1987; Weissman et al. 1987; Weissman 1988).

At the same time, growing evidence suggests that children of substance-abusing and depressed parents vary greatly in terms of adjustment status and that many exhibit adequate functioning (Downey and Coyne 1990; for a review, see Sher 1991). Some researchers have suggested that family process variables are essential in understanding the adjustment of children whose parents have substance use or affective disorders. Although some family process variables are considered risk factors for offspring, others can offset the adverse effects of parental psychopathology. Family process variables that amplify or mediate these adverse effects include family discord, hostility, quality of caregiving, family cohesion, supportive parenting, and parental monitoring (Chassin et al. 1993; Clair and Genest 1987; Easley and Epstein 1991; Johnson et al. 1995; Reich et al. 1988; Roosa et al. 1993; Rutter and Quinton 1984; Werner 1986). However, very little research has investigated the mediating role played by these factors (Burk and Sher 1988).

Among the various theories designed generally to explain parental influences on adolescent development and behavior, there are two competing perspectives that address specifically the relationships between parental psychopathology and adjustment among adolescents. The first, which is often labeled the social learning theory, contends that the linkage between parental psychopathology and child's adjustment is explained mainly by social modeling, identification, and social reinforcement (Akers 1992; Akers et al. 1979). Empirical studies provided strong support for the association between parental substance use and adolescent substance-using behavior. For instance, Dishion and his colleagues (Dishion et al. 1988) demonstrated that parental drug use is related to early adolescent drug experimentation; similarly, Barnes and colleagues (Barnes et al. 1986) have shown that parents who are heavy drinkers are more likely to have adolescents who are also heavier drinkers. (However, Green and colleagues [1991] report a positive association between parental drinking and adolescent drinking for girls but not for boys.) Independent of its interference with parenting functions, parental psychopathology can directly influence an offspring's adjustment by becoming a behavioral model or a model of coping responses (Seilhamer and Jacob 1990). Furthermore, parental drug use may provide availability of substances and opportunities for substance use in the family setting. Previous studies also suggest that because of parental rejection and impaired parenting, offspring of depressed parents learn helplessness, self-depreciation, and social withdrawal instead of effective social skills, and that this makes them more susceptible to depressed moods (Seligman and Peterson 1986; Whitbeck et al. 1992).

The second of these competing viewpoints is called the stress-coping theory. It proposes that parental psychopathology—in this case, substance use disorder and affective disorder—increases an offspring's risk of experiencing other family stressors. It further assumes that family stressors associated with a substance-

abusing parent or a parent with an affective disorder alter family functioning, which in turn leads to negative outcomes in children (Seilhamer and Jacob 1990; West and Prinz 1987). According to this model, substance use among adolescents results from unfavorable family circumstances related to parents' substance abuse (Fawzy et al. 1983; Stein et al. 1987) or parent's depression, and adolescents use substances to cope with the problems caused by adverse family circumstances. Altermann and Tarter (1983) also suggest that the development of alcohol and drug abuse, and related problem behaviors, in offspring of substance abusers may be due to the combined influence of psychological and genetic vulnerability, and may be exacerbated by the adverse social and environmental conditions in which these offspring are often reared.

Families with a substance-abusing or alcoholic parent usually experience multiple negative life events, such as marital conflict and dissolution, social isolation, unemployment, financial hardship, and parent-child conflict (Clair and Genest 1987; Moos and Billings 1982; Reich et al. 1988; Rubio-Stipec et al. 1991; Roosa et al. 1988; Roosa et al. 1993; Velleman and Orford 1993; Werner 1986). Similar negative life events are also reported in families of depressed parents (Compas et al. 1986; Roosa et al. 1990; Swearingen and Cohen 1985). Research suggests that exposure to a greater number of negative life events is related to increased substance use (Bruns and Geist 1984; Hoffmann and Su 1997; Newcomb and Harlow 1986) or depressive symptoms in adolescents (Garrison et al. 1990; Roosa et al. 1990; Swearingen and Cohen 1985).

However, there is evidence that negative life events alone do not determine the adjustment of children and adolescents, and that family cohesion has an important role in accounting for some of the variability in their reactions to the negative life events that are associated with parental psychopathology (Clair and Genest 1987; Cronkite et al. 1990). Moreover, the degree of cohesiveness in a family environment can directly influence adolescent adjustment, independent of parental psychopathology (Seilhamer and Jacob 1990).

Recent theoretical work in developmental psychopathology suggests that the etiological pathways to adolescent depressive symptoms and substance use may be gender-specific, and that these differences may be the result of differential vulnerabilities between males and females to interpersonal depressive experiences—experiences that often stem from negative life events involving the self and/or others (Hops 1995; Leadbeater et al. 1995). Depressive vulnerability theory posits that adolescents with higher levels of interpersonal or self-critical vulnerability have heightened reactivity to stresses involving others or themselves. Research shows that females are more likely than males to demonstrate interpersonal depressive vulnerabilities. Female's greater sensitivity creates differences in perceived interpersonal contexts that affect both available social supports and the types of interpersonal events that are perceived as stressful (Gore et al. 1993). Literature also suggests that female adolescents appear to be more sensitive than male adolescents to parental influences (Kandel 1985), and that the family is a more salient context for female adolescents' adjustment than for male adolescents' (Johnson et al. 1995; Krohn and Massey 1980; Rosenbaum 1987; Su 1989). Therefore, one would expect girls to be more sensitive to parental psychopathology and negative life events.

Although there has been some progress in understanding the way parental psychopathology varies in its effects on offspring, most studies examining family processes and adolescent adjustment in families where parents have psychiatric disorders have at least four methodological limitations that compromise the interpretation of the research findings. First, most rely on small, clinically identified or treated samples that are unrepresentative of offspring of parents with psychiatric disorders, and that lack adequate comparison groups (Downey and Coyne 1990; Seilhamer and Jacob 1990). Second, researchers often use cross-sectional designs, or retrospective accounts of adult offspring of problem drinkers or alcoholics during their childhood and adolescence (West and Prinz 1987). Third, parental alcoholism, drug use disorder, or major depression are often determined by the offspring's reports, or defined by vague or inconsistent criteria. Finally, most of these studies have not examined the differential gender effects of parental psychopathology and family process variables on adjustment among adolescents.

The purpose of this study is to test and compare social learning theory and stress-coping theory as explanations of the link between parental psychopathology and adolescent substance use and depressive symptoms using a more rigorous methodology.¹ The social learning model posits that parental substance abuse has a direct effect on adolescent substance use and that parental depression has a direct effect on adolescent depressive symptoms. The stress-coping model hypothesizes that parental substance abuse and depression are associated with a greater number of negative life events; negative life events are in turn thought to attenuate family cohesion and ultimately lead to increases in adolescent substance use and depressive symptoms. (Figure 1 displays the models derived from social learning theory and the stress-coping theory.) We also test gender differences in the effects of parental psychopathology and family process variables using these two theoretical frameworks and, thus, directly test the theoretical work on gender-differentiated vulnerability.

Method

Sample and Data

The data used in this analysis are derived from the screening phase and the first two waves of a panel study of youth at high risk for substance abuse. The study began in 1991 with 600 families having at least one child aged 10 to 13. The study uses a prospective cohort design of a type that is common in epidemiologic and biomedical research (see Kelsey et al. 1986; Rothman 1986; Fleiss 1981). Having identified groups of children and adolescents with specific risk factors—one or more parents who are diagnosed with psychoactive substance use disorder (PSUD) or affective disorder—we compare these youths to a group of children and adolescents who do not have the same risk factors (i.e., parents are free from a diagnosis of PSUD or affective disorder). The study follows the groups over time, assessing their substance use, psychiatric and behavioral problems, and various other adjustment outcomes. The study was designed to identify children at risk for substance abuse, and to yield a large enough sample of families with a substance-abusing parent or a parent with an affective disorder to make it possible to observe the effect of parental substance use disorder and affective disorder on adolescent adjustment.

Figure 1
The Relationship Between Parental Psychopathology and Adolescent Adjustment
(Substance Use and Depressive Symptoms)

Sample

This study did not use probability-based sampling, as this type of sampling from such as rare population (i.e., families with a child of 10 to 13 years of age and with a parent who is clinically diagnosed with a substance use disorder or affective disorder, and no comorbid psychiatric disorders) would have been extremely difficult.² Approximately 44% of the representatives of the PSUD and affective disorder groups were recruited from a pool of adults who were being treated for PSUD or affective disorder at a major metropolitan medical center or at one of its six demographically diverse satellite clinics in the upper midwestern United States. To further build the sample, eligible families were enlisted from two sources: (1) outpatient Pediatrics, Family Medicine, and Internal Medicine clinics that were part of the same medical center, and which provide health services to youth 18 years of age or younger and their parents; and (2) the general community—via active recruitment in places where families with adolescent children visit, such as the YMCA, the YWCA, recreational centers, parks, and community centers where important events were held. The comparison group sample was also recruited from similar sources as the nonclinical substance use disorder and affective disorder group samples. Efforts were made to recruit families for the comparison group that matched the other families in terms of demographic characteristics.

Screening

Medical records of adults who were treated for PSUD (i.e., alcohol dependence and/or illicit drug dependence) or an affective disorder (i.e., major depression, dysthymia, and bipolar disorder) in the medical center and its satellite clinics were screened for eligibility. Screening questions were also developed to determine the demographic eligibility and the possible indication of substance use disorder and affective disorder (e.g., receiving treatment or counseling for alcohol or drug use or emotional problems) among individuals from the Pediatric, Family Medicine, and Internal Medicine clinics—i.e., diagnosis of PSUD or affective disorder at the time of sample recruitment, but no other comorbid disorders besides these, or no lifetime diagnosable psychiatric disorder—and who affirmed they had children 10 to 13 years of age were invited to participate in the study, together with their spouses or live-in partners, the focal child aged 10 to 13, and the focal child's older siblings aged 16 or younger at the time of sample recruitment. Biological parents who were not living with the focal child at the time of recruitment because of marital separation or divorce but had joint custody or regular visits with the focal child (e.g., weekends every 2 weeks or more) were also invited to participate in the study. (Studies have suggested that even when the substance-abusing or depressed parent no longer lives with the adolescent, episodes of substance abuse or depressive symptoms may directly affect their parenting practices during visits, or may affect the adolescents indirectly through the stressful life events that the impaired parent creates [Farrell et al. 1995].)

Recruitment and classification

Both the clinical and the community samples were recruited over approximately 18 months. About 16% of the total clinical group approached by study personnel met the eligibility criteria:³ about 85% of these agreed to participate in the study. Seven

percent declined to participate after the initial consent because of their spouse's or live-in partner's objection to their participation in the study; 5% failed repeatedly to show up for the screening interview after setting up appointments; and the rest refused to participate because they were overwhelmed with the stress associated with their PSUD or affective disorder. With the exception of marital status, subjects who participate in the study do not differ from those who declined to participate.

All parents and parent figures were administered the Structural Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders, third edition (DSM-III-R) (SCID) (Spitzer et al. 1990) between early fall of 1991 and late 1992. The SCID was conducted by intensively trained advanced graduate students in clinical psychology and family therapy who were familiar with the DSM-III-R classification and diagnostic criteria.⁴ Based on the SCID and the family history questionnaire (see detailed description in the *Measures* section), we classified parents and their adolescent children into three groups (i.e., substance use disorder group, affective disorder group, and the comparison group), irrespective of recruitment source. Families with one or both parents diagnosed with substance use disorder at the time of sample recruitment, but no comorbid affective disorder during their lifetime, were placed in the substance use disorder group; families with one or both parents diagnosed with affective disorder at the time of sample recruitment, but no comorbid PSUD during their lifetime, were placed in the affective disorder group. Families in which neither parent had a diagnosable psychiatric disorder during their lifetime were placed in the comparison group. As a result, 182 families were placed in the substance use disorder group, 134 families were placed in the affective disorder group, and 270 families were placed in the comparison group. (The remaining 14 families were placed in an "other psychiatric disorder" group and excluded from this analysis.)

Approximately 12 to 18 months after administering the SCID we scheduled a group appointment for the parent, spouse or partner, and their children to each complete a self-administered questionnaire, either at home, at the research office, or at some other arranged location. An interviewer was present during completion of most questionnaires, which took approximately 1 hour to complete. The second-wave interview was conducted about 12 months after the first-wave interview. Each responding family member received monetary compensation for completing the questionnaire each year. About 97% of the families completed the first wave questionnaires, and 98% completed the second wave.

In this analysis, we examine only the data from 820 children from families that completed the SCID screening phase and who participated during both waves of the study. The sample consists of 175 adolescents (78 boys and 97 girls) in the substance use disorder group, 233 adolescents (126 boys and 107 girls) in the affective disorder group, and 412 adolescents (215 boys and 197 girls) in the comparison group. Ninety-one% of the parents in this study are Caucasians, 6% African American, 1.5% Native American, 1% Hispanic, and 0.5% Asian. The ages of the fathers ranged from 28 to 56 (mean=40.11±5.55) at the time of sample recruitment and the ages of the mothers ranged from 27 to 53 (mean= 37.83±5.13). The ages of the children ranged from 10 to 16 (mean=12.06±1.61) at the time of the sample recruitment. About 89% of the fathers and 64% of the mothers were married; 8% of the fathers and 28% of mothers were separated or divorced. Forty-eight

percent of the fathers and 32% of mothers had graduated from college or completed a postgraduate degree. Family income ranged from \$30,000 or less (36.3%) to more than \$100,000 (5.4%), with a mean income range of \$30,001-40,000.

Measures

Parental PSUD and parental affective disorder were assessed using two versions of the Structured Clinical Interview for DSM-III-R (SCID), an assessment instrument designed to make DSM-III-R diagnoses. As part of screening, parents were provisionally identified as either potentially psychiatrically impaired or not. The patient edition (SCID-P) was used for parents who had been identified in screening as potentially psychiatrically impaired. For parents who had no apparent psychopathology, the nonpatient edition (SCID-NP) was used. Using these two versions of the SCID, interviewers elicited the sample parents' lifetime and current (during the past 30 days) PSUD and affective disorder.

In addition to the SCID, a family history questionnaire was administered to 6% of parents whose spouses or partners were unwilling to participate. This questionnaire was developed specifically to enable adult participants to provide information on their spouse's or partner's substance use disorder and/or affective disorder. It was adapted from the Family Informant Schedule and Criteria (Mannuzza et al. 1985), which is often used to obtain information on family members who are deceased or unwilling to participate in this type of research, or when the reliability and validity of informant versus direct information is of interest.

We created two dummy variables to represent parental substance use disorder and parental affective disorder. Adolescents in the substance use disorder group were scored 1 on the dummy variable representing parental substance use disorder, with 0 for all others; whereas adolescents in the depression group were assigned scores of 1 for the dummy variable representing parental affective disorder, with 0 for all others.

Family Cohesion for both parents and adolescents at Time 1 was calculated by a 16-item scale from the Family Adaptability and Cohesion Evaluation Scales, version 3, (FACES III) (Olson et al. 1985) that measures family functioning in terms of the degree of emotional bonding that family members have toward one another. Olsen and colleagues (1985) report test-retest correlations of .83 and internal consistency of .77 for family cohesion. The items for family cohesion in our sample showed an internal consistency reliability of .87 (alpha) for adult parents and .83 for adolescents.

Negative family life events for parents were assessed using a checklist of 34 life change events adapted from the Family Inventory of Life Events and Changes (McCubbin et al. 1981). Specifically, parents were asked to check off a list of 34 life events that happened to them or their families during the 12 months prior to the first-wave interview. The events included family conflict and violence, marital strain, child-bearing strain, family financial difficulty, work-related strain, strain related to caring for a seriously ill or injured family member, death of a family member or close friend, and family legal problems. The adolescents' stress was estimated using a checklist of 14 negative life events they had experienced during the 12 months prior to the first-wave interview. Adapted from the Junior High Life Experiences Survey (Swearingen and Cohen 1985), this instrument listed important events outside the

adolescent's control, including a family member's serious illness or injury, a family member's legal problems, a family member's becoming a victim of violence, death of a family member, family financial difficulty, increased parental conflict, change of parental marital status, and transition-related strain (i.e., stepparent or parent's partner joined the household, and/or family moved to a new home). Negative life events that may be adaptational outcomes to the stressors were excluded from the analysis to eliminate potential confounding effect; these included, for example, being suspended from school, repeating a grade, becoming pregnant or making someone pregnant, and running away from home. The total number of negative life events reported by parents at Time 1 ranged from 0 to 20, with an average of 5.69, and the number of negative life events reported by adolescents ranged from 0 to 9, with an average of 1.93.

Adolescent substance use was computed by combining frequency of use of alcohol and illicit drugs during the 12 months prior to each wave of interview. Adolescent respondents reported the frequency of use of alcohol and drugs across seven response categories: never used, 1 to 2 days, 3 to 5 days, 6 to 9 days, 10 to 19 days, 20 to 39 days, and 40 to 59 days during the last 12 months. The substances included alcohol (beer, wine or wine cooler, and liquor); marijuana, cocaine, crack, and other illicit drugs (i.e., sniffed glue, or took amphetamines, barbiturates or Quaaludes, hallucinogens including LSD and other psychedelics); tranquilizers; and narcotics (including heroin and other narcotics). The actual scales ranged from 0 to 22 at Time 1 and 0 to 47 at Time 2. (The higher end of the scales represents more extensive substance use.)

Adolescent depressive symptoms at Time 1 and Time 2 were measured by the revised Center for Epidemiological Studies Depression Scale (CES-D scale—Radloff 1977), a summated 20-item scale designed to assess the frequency of depressive symptomatology in a typical week during the last 30 days. Adolescent respondents responded on a four-category scale: less than 1 day, 1 to 2 days, 3 to 4 days, and 5 to 7 days. The reported scores ranged from 0 to 49 at Time 1, and 0 to 57 at Time 2. The CES-D is strongly associated with other self-report measures of depressive symptoms (see discussion by Avison and McAlpine 1992). Avison and McAlpine (1992) reported an internal consistency reliability of .90. The items in our sample showed an internal consistency reliability of .82 at Time 1 and .85 at Time 2. The higher end of the scores indicates more frequent presence of depressive symptoms.

Analytic Strategies

We conducted analyses using one-way analysis of variance to determine whether family members (fathers, mothers, male adolescents, and female adolescents) of the substance use disorder group differ from their counterparts in the affective disorder and comparison groups in terms of stressful life events and family cohesion. In addition, we used one-way analysis of variance with a priori contrast tests (Scheffé) and to examine the differences in adolescent substance use and depressive symptoms between pairs of groups (substance use disorder group versus affective disorder group, affective disorder group versus comparison group, and substance use disorder versus comparison group). To examine the potential gender differences we conducted these analyses for male and female adolescents separately.

We also used structural equation modeling (LISREL VIII) to test the direct and indirect effects of parental psychopathology, with parental substance use disorder and parental affective disorder at screening phase as exogenous variables and, as endogenous variables, negative family life events at Time 1, adolescent's perceived family cohesion at Time 1, adolescent substance use at Time 2, and adolescent depressive symptoms at Time 2. We controlled for adolescent's age, family income, adolescent substance use at Time 1, and adolescent depressive symptoms at Time 1.⁵ Mother's reported negative family life events were used in the structural equation modeling. In single-father families, father's reported negative life events were chosen in place of mother's reporting. Parent's reported family life events were used in place of adolescent's reporting because they include more items that address chronic life difficulties, such as financial strains and employment status, that have an impact on parents as well as adolescents. Furthermore, the full sample was divided into two gender groups, and the proposed model was imposed on the two groups to test whether the direct and indirect effects of parental substance use disorder and parental depression were the same for male adolescents and female adolescents. Specifically, we used structural equation modeling to answer the following questions.

- To what extent does parental psychopathology (i.e., parental substance use disorder and parental affective disorder) have an impact on family negative life events?
- What, if any, is the impact of parental psychopathology on perceived family cohesion among adolescents?
- Does parental psychopathology have a direct effect on adolescent substance use and depressive symptoms?
- Is the influence of parental psychopathology on adolescent children mediated by negative life events and family cohesion?
- Does the relationship between parental psychopathology and adolescent adjustment differ by the adolescent's gender?
- Are there common etiological pathways to adolescent substance use and depressive symptoms?

We began by specifying and fitting the proposed model using the full sample. To examine the effects by gender, we divided the full sample into adolescent males and adolescent females, and imposed the model on the two gender groups to test whether the structural relationships between parental psychopathology and adolescent adjustment were the same for male adolescents and female adolescents. We relied on existing theory of gender differences and Lagrangian multiplier tests supplied by LISREL VIII to modify the multi-group structural equation models. Finally, we tested the parameters of the common paths for equality.

We assessed the goodness-of-fit of the structural equation models through several statistical measures. First, we used a chi-square statistic to gauge the concordance of the theoretical model with the covariance structure found in the data. Chi-square tests also allow various alternative models to be compared to the proposed theoretical model. However, because chi-square tests are highly sensitive to sample size, we use two alternative measures of model fit. Our second assessment relies on the traditional adjusted goodness-of-fit indicator adjusted for sample size (labeled AGFI in the figures). The value converges to 1.00 as covariance structure and the theoretical

structure reach perfect agreement. Third, we used the root mean square error of approximation (RMSEA) test (Browne and Cudeck 1993; Jöreskog 1993) to further clarify the fit of the model. A value below 0.05 indicates a highly successful model fitting exercise.

Results

Table 1 shows means and standard errors for two indices of home environment and the significance of the main effect of family diagnostic group (F-values). As indicated in table 1, parents as well as adolescents in the substance use disorder and affective disorder groups tended to experience more negative life events and to perceive a lower degree of family cohesion at Time 1 than their counterparts in the comparison group. No significant differences in the number of negative life events and degree of family cohesion were observed between parents and adolescents in the substance use disorder group and those in the affective disorder group.

Table 2 presents the ranges, means, and standard errors of adolescent substance use and depressive symptoms at Time 1 and Time 2 for male and female adolescents by three family diagnostic groups. Females in the substance use disorder group reported significantly more substance use both at Time 1 and Time 2 than females in the comparison group, while males in the substance use disorder group used more substances relative to males in the comparison group at Time 2 only. Additionally, females in the substance use disorder group reported more substance use at Time 1 relative to their counterparts in the affective disorder group. Finally, females in the substance use disorder group manifested more depressive symptoms both at Time 1 and Time 2 than their counterparts in the comparison group. Females in the affective disorder group were also more likely to have a greater number of depressive symptoms at Time 1 compared to their counterparts in the comparison group. No significant difference in depressive symptoms was found among males across the three groups.

Figures 2 and 3 display the results of the structural equation models that estimate simultaneously the proposed relationships between parental psychopathology and adolescent adjustment over time for the full sample. The numbers associated with each path represent standardized regression coefficients. Only paths significant at the .05 probability level are displayed. Two modifications to the proposed theoretical model (i.e., the stress-coping model) were necessary to obtain the best fitting models. First, in support of social learning theory, parental substance abuse has a significant direct effect on adolescent substance use at Time 1. Second, parental depression has a modest attenuating effect on family cohesion.

As indicated in figure 2, the model estimating the direct and indirect effects of parental substance use and parental affective disorders on adolescent substance use has a good fit with the data. The RMSEA value is 0.026, suggesting a good approximation of the model in the population. The chi-square and AGFI are 16.56 (with 11 degrees of freedom) and .98, respectively, which indicate an adequate fit of the model with the data. As expected, having a parent with a substance use disorder or affective disorder was positively related to the number of negative life events experienced at Time 1. Higher levels of negative life events resulted in lower levels of family cohesion at Time 1, which in turn had a direct effect on adolescent substance use both at Time 1 and at Time 2. Adolescents with a depressed parent

were more likely to experience negative life events and lower family cohesion than their counterparts with a substance-abusing parent. On the other hand, adolescents with a substance-abusing parent tended to report more extensive substance use at Time 1. Moreover, parental affective disorder at the screening phase was associated with lower perceived family cohesion at Time 1, which in turn had a direct effect on adolescent substance use both at Time 1 and at Time 2. Adolescents with a depressed parent were more likely to experience negative life events and lower family cohesion than their counterparts with a substance-abusing parent. On the other hand, adolescents with a substance-abusing parent tended to report more extensive substance use at Time 1. Moreover, parental affective disorder at the screening phase was associated with lower perceived family cohesion at Time 1, which in turn negatively affected adolescent substance use both at Time 1 and at Time 2. Neither parental affective disorder at the screening phase nor negative life events at Time 1 had a significantly direct effect on adolescents' substance use.

The results of the structural equation model using adolescent depressive symptoms in place of adolescent substance use, displayed in figure 3, were almost identical to those shown in figure 2. Although the chi-square value is marginally statistically significant (23.33 with 12 degrees of freedom), both the RMSEA value (0.03) and the AGFI value (.98) indicate a reasonable fit. Contrary to hypotheses derived from social learning theory, the model did not display a direct effect of parental affective disorder on adolescent depressive symptoms.

Figure 4 presents the results of multi-group structural equation models for the relationship between parental psychopathology and adolescent substance use over time. Within-group standardized estimates are shown. The results of a test for equal structural forms, displayed in figure 4, suggests that the models for males and females had similar structural forms (chi-square=30.24 with 30 degrees of freedom, RMSEA=0.003, and AGFI=.99). An examination of the significance levels of the path coefficients showed that the relationships among parental psychopathology, negative life events, and family cohesion were generally equivalent for males and females. However, the direct path from parental affective disorder to family cohesion was significantly distinct: females' perceived family cohesion was affected by parental depression, but males' was not.

Figure 5 shows the results of a multi-group structural equation model of the relationship between parental psychopathology and adolescent depressive symptoms over time. Although the chi-square value achieves statistical significance (54.52 with 27 degrees of freedom), both the value of RMSEA (0.036) and AGFI (.99) indicate an adequate fit of the model with the data. We found similarities between males and females. However, female offspring of depressed parents tended to perceive significantly lower family cohesion than their male counterparts. In addition, we observed a significantly stronger association between family cohesion and adolescent depressive symptoms for females than for males, both at Time 1 and at Time 2.

Table 1
Comparison of Parent's and Adolescent's Perceived Home Environment At Time 1 By Family Diagnostic Groups

	Mean (standard error)			F-values	
	Substance Use Disorder	Depression	Comparison	Substance Use vs. Comparison	Depression vs. Comparison
No. of family stressful events					
Father	6.51 (0.44)	6.24 (0.39)	4.57 (0.23)	3.89***	3.65***
Mother	7.11 (0.34)	7.06 (0.31)	4.80 (0.18)	5.96***	6.35***
Adolescent boy	2.32 (0.18)	2.46 (0.21)	1.54 (0.12)	3.56***	3.81***
Adolescent girl	2.26 (0.17)	2.18 (0.22)	1.56 (0.12)	3.37***	2.54*
Perceived family cohesion					
Father	59.63 (0.89)	59.34 (1.13)	63.15 (0.56)	-3.36***	-3.03**
Mother	60.25 (0.71)	59.52 (0.86)	64.57 (0.47)	-5.09***	-5.16***
Adolescent boy	56.04 (0.93)	56.11 (1.07)	59.16 (0.66)	-2.74**	-2.42*
Adolescent girl	57.06 (1.05)	54.12 (1.17)	60.56 (0.77)	-2.69**	-4.58***

Notes: *** $p < .001$, ** $p < .01$, and * $p < .05$.
Fathers: $N = 90$ in substance use disorder group, $N = 62$ in depression group, and $N = 178$ in comparison group.
Mothers: $N = 148$ in substance use disorder group, $N = 130$ in depression group, and $N = 260$ in comparison group.
Adolescent boys: $N = 78$ in substance use disorder group, $N = 126$ in depression group, and $N = 215$ in comparison group.
Adolescent girls: $N = 97$ in substance use disorder group, $N = 107$ in depression group, and $N = 197$ in comparison group.

Table 2
Adolescent Substance Use and Depressive Symptoms By Family Diagnostic Groups

	Substance Use Disorder		Depression		Comparison	
	Boys (N=78)	Girls (N=97)	Boys (N=126)	Girls (N=107)	Boys (N=215)	Girls (N=197)
Substance use at Time 1 ^a						
Minimum-maximum	0 - 15	0 - 20	0 - 14	0 - 20	0 - 9	0 - 14
Mean (standard error)	.74 (.26)	1.44 (.37)	.45 (.15)	.67 (.23)	.27 (.06)	.47 (.13)
Substance use at Time 2 ^{abc}						
Minimum-maximum	0 - 13	0 - 20	0 - 11	0 - 12	0 - 11	0 - 47
Mean (standard error)	.95 (.28)	1.51 (.37)	.63 (.15)	.62 (.17)	.27 (.07)	.60 (.26)
Depressive symptoms at Time 1 ^{ad}						
Minimum-maximum	0 - 47	2 - 47	1 - 41	1 - 41	0 - 49	0 - 47
Mean (standard error)	13.84 (.95)	16.78 (.98)	13.79 (.73)	16.99 (.95)	12.89 (.49)	12.49 (.53)
Depressive symptoms at Time 2 ^a						
Minimum-maximum	0 - 38	0 - 52	0 - 36	1 - 41	0 - 33	0 - 40
Mean (standard error)	11.39 (1.06)	14.28 (1.18)	10.50 (.72)	16.99 (.95)	9.36 (.50)	10.28 (.58)

^a The difference between substance use disorder group and comparison group for girls is statistically significant at $p<.05$.
^b The difference between substance use disorder group and depression group is significant for girls at $p<.05$.
^c The difference between substance use disorder group and comparison group for boys is significant at $p<.05$.
^d The difference between depression group and comparison group for girls is statistically significant at $p<.05$.

Figure 2
Direct and Indirect Effects of Parental Substance Abuse and Depression on Adolescent Substance Use

Model Goodness of Fit: $\chi^2 (11df) = 16.56, p = .12$.
RMSEA = 0.026 (90% CIs: 0.01, 0.05)
AGFI = .98.

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

Note: The model controls for the effects of family income, gender, and age.

Figure 3
Direct and Indirect Effects of Parental Substance Abuse and Depression on Adolescent Depression

Note: The model controls for the effects of family income, gender, and age.

Figure 4
Direct and Indirect Effects of Parental Substance Abuse and Depression on Adolescent Substance Use, by Gender

* p < .05
** p < .01
*** p < .001

Multi-sample Model Goodness of Fit: $\chi^2 (30 \text{ df}) = 30.24, p = .45$
RMSEA = 0.003 (90% CI: 0.00, 0.028)
GFI = .99

Note: The models control for the effects of age and family income.

Figure 5
Direct and Indirect Effects of Parental Substance Abuse and Depression on Adolescent Depression, by Gender

In sum, the structural equation models shown in figures 2 and 3 support a causal relationship in that parental psychopathology is associated with adolescent substance use and depressive symptoms primarily through its effects on negative life events. The effects of the parental psychopathology variables in the model for adolescent adjustment both at Time 1 and Time 2, however, were very modest. The total effect of the parental substance use disorder on adolescent substance use was only .12 and .06 at Time 1 and at Time 2, respectively; the total effect of the parental affective disorder on adolescent depressive symptoms was only .04 (but significant at $p < .05$ level), both for Time 1 and Time 2. In comparison, family cohesion had much stronger effects on adolescent substance use and on adolescent depressive symptoms. The total effect of family cohesion on adolescent substance use at Time 1 and Time 2 was -.23 and -.19, respectively, and the total effect of family cohesion on adolescent depressive symptoms was -.34 at Time 1 and -.31 at Time 2.

Finally, the structural equation models shown in figures 4 and 5 indicate that no significant gender differences were found in the relationship between parental substance use disorder and adolescent substance use, nor between parental affective disorder and adolescent depressive symptoms. However, differential gender effects were observed in the consequences of parental affective disorder and in the effects of family cohesion on adolescent depressive symptoms. Specifically, parental affective disorder has a significant negative effect on perceived family cohesion for female adolescents but not for male adolescents. Further, female adolescents' depressive symptoms were influenced substantially more by family cohesion than was the case for male adolescents.

Discussion

Although prior research demonstrates that children of parents with psychopathology are at elevated risk for adjustment problems, most of these studies fail to investigate the mediating effects of family process variables on adolescent depressive symptoms and substance use (Seilhamer and Jacob 1990). The present study extends previous work by using a prospective longitudinal research design to test a causal model in which both direct and indirect effects of parental psychopathology on adolescent substance use and depressive symptoms are examined. The study also identifies parental substance abuse or affective disorder using structured diagnostic interviews of the parents, instead of relying on their responses to self-report questionnaires or on their children's accounts.

As expected, in both substance use disorder families and affective disorder families, parents and adolescents alike tend to experience more negative life events and to perceive a lower degree of family cohesion. This finding provides empirical support for the hypothesis that children of substance-abusing parents and those with depressed parents do not differ significantly in reported adverse family environment and in risks for maladjustment (Jacob and Leonard 1986; Rubio-Stipec et al. 1991). In addition, the results suggest that in families with substance-abusing or depressed parents, both the parents and the children see the family environment as adverse.

Parental substance use disorder and affective disorder increase the adolescent's risk of experiencing more negative life events. Negative life events further lower family cohesion, which in turn leads to adolescent substance use and depressive symptoms. In addition, family cohesion exerts a strong independent effect on

adolescent substance use and depressive symptoms, regardless of parental psychopathology and negative family life events. The results are similar to the findings reported in previous studies (Chassin et al. 1993; Roosa et al. 1993), and lend empirical support to a stress-coping model. The results from this study also demonstrate that family cohesion plays a dominant role in mediating the stress related to parental substance use disorder and parental affective disorder, when adolescent substance use and depressive symptoms are the outcome variables of concern. Parental substance use disorder not only exerts an indirect influence through the negative life events it generates and through its negative impact on family cohesion, but it also has a direct effect on adolescents' substance use. This finding is consistent with results reported by Green et al. (1991), and provides support for a social learning hypothesis in that the adolescent's substance use reflects the substance use patterns of the parents.

This study also reveals important gender differences in the impact of parental affective disorder on adolescents' perceived family cohesion, and in the effect of perceived family cohesion on adolescents' depressive symptoms. The data indicate that parental affective disorder exerts a strong direct effect on female adolescents' perceived family cohesion, but not on male adolescents'. Moreover, female adolescents appear to be influenced to a greater extent by family cohesion than their male counterparts, which is consistent with the results of previous studies (Johnson et al. 1995; Krohn and Massey 1980; Rosenbaum 1987; Su 1989). These results on gender differences support the hypothesis of Leadbeater et al. (1995) that the pathway to the development of psychopathology in adolescents—a path characterized by interpersonal depressive vulnerabilities, heightened sensitivities to stressful life events involving others or the self, and the occurrence of internalizing or externalizing disorders—is a more likely explanation of female adolescents' depressive symptoms than male adolescents'.

Finally, similar to findings reported by Cohen and colleagues (Cohen et al. 1987), the results of separate analyses for adolescent substance use and depressive symptoms show that there are common family environmental risk factors for adolescent substance use and depressive symptoms. However, we determine that family cohesion had stronger effects on adolescent depressive symptoms than on adolescent substance use.

In interpreting the findings from this study, it is important to recognize some of the limitations imposed by the sample. First, this research shares a problem common with many high-risk studies: namely, that the sample was not selected randomly and, therefore, the generalization of study findings to other samples may be limited. The study used a selective sample of substance-abusing or depressed parents who did not meet the DSM-III-R criteria for any other psychiatric disorders, and who lived with their children aged 10 to 13. As a result, adolescents in this study may be at relatively lower risk in terms of other life hardships and sources of social support, when we compare these subjects to those who have been placed in transitional living arrangements (such as with foster families or relatives) because their parents were too impaired to take care of them. Second, the subjects in this study were mostly in pre-adolescence or early adolescence at Time 1 and Time 2 and, thus, might not yet have experienced substance use and depressive episodes. Third, parental substance use disorder and affective disorder were treated as a unitary disorder—no distinc-

tions were made between alcohol and drug use and between different affective disorders—and the severity and chronicity of parental psychopathology could not be controlled. Recent research has noted that both substance use disorder and affective disorder are heterogeneous and highly variable in course, and are recurrent, episodic disorders with varying degrees of residual difficulties (Downey and Coyne 1990). Hence, longitudinal studies monitoring changes in parental psychopathology are needed to fully understand the processes that link parental psychopathology to adolescent adjustment. Finally, although previous research suggests that the risk of maladjustment increases substantially if both parents (rather than just one) are substance abusers or depressed (Orford and Velleman 1990; Weissman et al. 1984), we did not examine whether two substance-abusing or depressed parents, compared with only one, are more likely to produce offspring with substance use or depressive symptoms.

Nevertheless, this is the first study that simultaneously tests both social learning and stress-coping theories, along with a hypothesis of gender differences in adolescent vulnerability to family negative life events, to elucidate the complex pathways from parental psychopathology to adolescent maladjustment. We find that both theories play some role in explaining these pathways, although social learning theory provides a more tenuous explanation than stress-coping theory of the pathway from parental affective disorder to adolescent depressive symptoms. However, aspects of both social learning theory and stress-coping theory provide a good description of the path from parental substance abuse to adolescent substance use: parental substance abuse directly affects adolescent substance use, but it also attenuates family functioning and thus affects adolescent substance use indirectly. These results imply that efforts to treat substance abuse among adults who have children should be broad enough in scope to lessen stress and strengthen family ties. Without specific attention to how substance abuse affects family functioning, treatment programs may help the parent, but fail to attenuate the risk of substance abuse among children.

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Notes

1. We recognize that other possible theoretical perspectives may be used to explain the relationship between parental psychopathology and adolescent substance use or depressive symptoms, such as theories of genetic transmission. The design of this study, however, does not permit us to disentangle genetic versus environmental factors of familial transmission of substance use and affective disorders.
2. Based on data from the 1990 U.S. Census and the Epidemiological Catchment Area Survey (Bourdon et al. 1992), it is estimated that approximately 64 individuals in a population of 10,000 would meet the study's criteria for the substance use disorder group (i.e., diagnosis of substance use disorder and living

with own children of 10 to 14 years of age), and about 54 individuals in a population of 10,000 would meet the study's criteria for the affective disorder group (i.e., diagnosis of an affective disorder and living with own children of 10-14 years of age). The estimated numbers of individuals meeting study criteria will be substantially fewer if comorbidity is taken into consideration. A national or regional probability sample would have been optimal to ensure the generalizability of the study results. Nevertheless, a probability sample of a well-defined target population featuring the desired stratification by parental psychopathology and offspring's age would have been extremely expensive. Additionally, a randomly selected sample would have very little statistical power unless the sample size was enormous. The sampling strategy used in this study offers one viable way to rigorously study the effects of parental psychopathology on adolescent adjustment (cf. Chassin et al. 1993).

3. Among individuals who initially met our study criteria, about 20% became ineligible because they were no longer living with a child aged 10 to 13 due to marital separation or divorce, or were engaging in bitter disputes with their former spouses and had no access to their children. Approximately 3% became ineligible because of diagnosis of psychotic disorders as a result of the Structured Clinical Interview for DSM-III-R.

4. The inter-rater reliability for the diagnosis of affective disorders was .85, and for the diagnosis of PSUD it was 1.0 (Grove and Su n.d.).

5. Literature indicates that substance-abusing or depressed individuals are more likely to have a higher rate of marital disruption (for a review, see Downey and Coyne 1990). Parental marital disruption, however, was not used as a control variable because it was already included as one of the negative stressful life events.

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