

A Longitudinal Study of Children of Alcoholics: Predicting Young Adult Substance Use Disorders, Anxiety, and Depression

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This study tested the specificity of parent alcoholism effects on young adult alcohol and drug abuse/dependence, anxiety, and depression, and tested whether adolescent symptomatology and substance use mediated parent alcoholism effects. Participants were from a longitudinal study in which a target child was assessed in adolescence and young adulthood with structured interview measures ($N = 454$ families at Time 1). Results showed unique effects of parent alcoholism on young adult substance abuse/dependence diagnoses over and above the effects of other parental psychopathology. There was some evidence of parent alcoholism effects on young adult depression and of maternal alcoholism effects on young adult anxiety, although these were not found consistently across subsamples. Mediational models suggested that parent alcoholism effects could be partially (but not totally) explained by adolescent externalizing symptoms.

Because parental alcoholism is a well-established risk factor for adult alcoholism, there has been great research and clinical interest in children of alcoholics (COAs) as a high-risk group (Sher, 1991). Recent reviews (McGue, 1994; Russell, 1990) have reported consistency among studies in finding that COAs are at elevated risk for adult alcoholism, although the magnitude of the risk ratios vary substantially across samples. Adult COAs are also at risk for drug abuse/dependence (Gotham & Sher, 1996), although data are conflicting about COA risk for anxiety disorders and depression (Merikangas, Stevens, & Fenton, 1996; Schuckit, 1996; Sher, 1997).

Despite the great research interest in COAs, there are limitations to our knowledge concerning COA risk, as well as methodological limitations to previous studies. First, the specificity of parent alcoholism as a risk factor is unclear. That is, increased risk for negative outcomes in COAs may be due to other forms of parental psychopathology that are associated with parental alcoholism rather than to the parent alcoholism itself (Sher, 1991; West & Prinz, 1987). For example, Zucker, Ellis, Bingham, and Fitzgerald (1996) found that risk among young COAs varies substantially as a function of associated paternal antisociality, and Finn et al. (1997) found that offspring characteristics differed among groups that were positive for a family history of alcoholism, but that varied in associated familial psychopathology. Moreover, parent alcoholism and other parental psychopathology may both influence offspring outcomes but in different ways. For example,

Cadore, Yates, Troughton, Woodworth, and Stewart (1995), using an adoption design, found that parental alcoholism had a direct effect on offspring substance abuse, whereas parental antisocial personality affected offspring substance abuse indirectly by raising risk for early conduct problems. These data suggest somewhat different mechanisms underlying the effects of parental alcoholism and associated parental antisocial personality. However, because studies have often lacked data on other parent psychopathology, the specificity of parent alcoholism risk is unclear.

Our knowledge of parent alcoholism risk is also limited by a lack of longitudinal studies that can track the developmental antecedents of parental alcoholism risk for young adult negative outcomes. Recent conceptualizations emphasize that alcoholism as well as other forms of psychopathology have predictable antecedents in earlier developmental stages (Caspi, Moffitt, Newman, & Silva, 1996; Newman et al., 1996; Tarter & Vanyukov, 1994; Zucker, 1994). However, little is known about how parent alcoholism risk influences the developmental unfolding of these disorders. One goal of the current study is to evaluate adolescent symptomatology and substance use as mediators of parent alcoholism effects on young adult disorders.

Some hypothesized mechanisms underlying parent alcoholism risk are expected to operate from an early age. For example, it has been suggested that parent alcoholism risk for later alcohol and drug abuse may operate in part by raising risk for early conduct problems or "externalizing" behaviors (Sher, 1991; Zucker, 1994). Offspring of alcoholic fathers have more conduct problems than do non-COAs, even in the preschool years (West & Prinz, 1987; Zucker et al., 1996). In turn, early conduct problems raise risk for later substance-use-related problems (Windle, 1990). For example, for boys, behavioral undercontrol at age 3 predicts alcohol problems at age 21 (Caspi et al., 1996). Similarly, early conduct problems and aggression are predictive of later drug abuse (Brook, Cohen, Whiteman, & Gordon, 1992; Robins & McEvoy, 1990). Thus, parental alcoholism effects on young adult substance abuse and dependence may be mediated through earlier externalizing problems.

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In addition to these externalizing pathways, offspring of alcoholics are thought to be at risk for poor emotional regulation, negative affectivity, and internalizing symptomatology (Colder & Chassin, 1997; Sher, 1991; West & Prinz, 1987), and these internalizing symptoms may also mediate parent alcoholism effects on young adult psychopathology. Internalizing symptomatology may raise risk for later substance abuse problems, either through a self-medication mechanism or by raising risk for affiliating with a deviant peer group (Kaplan, 1980). For example, Caspi et al. (1996) found that boys who were inhibited at age 3 were at higher risk for alcohol problems at age 21. Negative affectivity has also been linked to later drug misuse in adolescence and young adulthood (Pandina, Johnson, & Labouvie, 1992; Shedler & Block, 1990; Wills & Filer, 1996).

Taken together, these findings suggest that trajectories of risk for substance abuse and dependence among COAs may be initiated early in childhood and adolescence, and thus the effect of parental alcoholism may be mediated by these earlier adolescent levels of symptomatology. These two major mediated pathways, through elevations in externalizing and internalizing symptomatology, have been hypothesized in a variety of theories to represent two major risk pathways for the development of alcoholism, although the "internalizing" pathway has most often been associated with a late adult onset subtype of alcoholism (Cloninger, 1987; Zucker, 1994). The current study tested whether adolescent levels of internalizing and externalizing symptomatology mediated the effects of parent alcoholism and associated parent psychopathology on young adult alcohol and drug abuse and dependence.

Moreover, even as adolescents, COAs are more likely to use alcohol and drugs than are their non-COA peers (Chassin, Rogosch, & Barrera, 1991). These levels of adolescent alcohol and drug use themselves may be risk factors in the development of young adult psychopathology. Evidence is conflicting about whether substance use during adolescence is a risk factor for later mental health problems. Jessor, Donovan, and Costa (1991) found few detectable effects of adolescent substance use in young adulthood unless the use persisted through the young adult years. Newcomb and Bentler (1988) found that adolescent drug use was related to adult psychoticism and suicidal ideation, but was unrelated to other affective aspects of young adult mental health. Adolescent alcohol use was associated with lower levels of young adult depression. However, these studies did not examine clinical diagnoses. The current study tested the relation between adolescent alcohol and drug use and young adult anxiety and depression diagnoses.

Finally, previous research has suffered from a variety of methodological limitations. Studies have often relied on samples of convenience including clinical samples of treated alcoholic parents or college student COAs (Sher, 1991; West & Prinz, 1987). Clinical samples may overestimate pathology by focusing on more severely impaired parents, whereas college student samples may underestimate pathology by focusing on less affected COAs. Moreover, studies vary in whether they directly ascertain parent alcoholism or rely on offspring report, which may underidentify parent alcoholism (Sher, 1991). Finally, studies of adult COAs have often included wide age ranges (spanning early adulthood through middle age) within small sample sizes, limiting statistical power. The current study addresses these problems by using a large community sample, in which alcoholic parents were actively

recruited and directly diagnosed, and COAs were all young adults at the final wave of measurement. Because young adulthood is a peak age for substance abuse/dependence (Kandel & Logan, 1984), this age period is of particular clinical importance.

In short, the current study used a longitudinal design to address four questions: (a) Does parent alcoholism elevate risk for young adult psychopathology? (b) Is the risk specific to parent alcoholism above and beyond other parental psychopathology? (c) Is parent alcoholism risk mediated through adolescent internalizing and externalizing symptomatology? (d) Does adolescent alcohol and drug use contribute to risk for young adult psychopathology?

Method

Participants

Participants were from an ongoing longitudinal study of parental alcoholism (Chassin, Curran, Hussong, & Colder, 1996; Chassin, Pillow, Curran, Molina, & Barrera, 1993; Chassin et al., 1991). At Time 1, there were 246 adolescents with at least one biological alcoholic parent who was also a custodial parent (COAs) and 208 demographically matched adolescents with no biological or custodial alcoholic parents (controls). The initial study included three annual assessments of the adolescents and their parents, and a long-term follow-up was conducted 5–7 years after the initial assessment.

Details of sample recruitment and representativeness are reported elsewhere (Chassin, Barrera, Bech & Kossak-Fuller, 1992; Chassin et al., 1991). COA families were recruited using court records of driving under the influence (DUI) arrests ($n = 103$), health maintenance organization wellness questionnaires ($n = 22$), and community telephone screening ($n = 120$). One family was referred by a local Veterans Administration hospital. Screening and recruitment were done by research team members (or by participating agencies when required because of confidentiality concerns).

COAs had to meet the following criteria: parents who reported being either Hispanic or non-Hispanic Caucasian, with Arizona residency, age 10.5–15.5 years, English-speaking, and with no cognitive limitations that would preclude interview (e.g., severe mental retardation or psychosis). Finally, direct interview data had to confirm that a biological and custodial parent met *Diagnostic and Statistical Manual of Mental Disorders*, third edition (*DSM-III*; American Psychiatric Association, 1980), criteria for alcohol abuse or dependence (lifetime diagnoses using the Diagnostic Interview Schedule (DIS; Robins, Helzer, Croughan, & Ratcliff, 1981)) or Family-History Research Diagnostic Criteria (FH-RDC), on the basis of reports by the other parent (if the alcoholic parent was not interviewed). At Time 1, interviews were conducted with 75.6% of biological fathers and 86.6% of biological mothers. When families had multiple eligible children, the child closest to age 13 was selected.

Demographically matched control families were recruited using telephone interviews. When a COA participant was recruited, reverse directories were used to locate families living in the same neighborhood. Families were screened to match the COA participant in ethnicity, family structure, target child's age (within 1 year), and socioeconomic status (using the property value code from the reverse directory). Direct interview data were used to confirm that neither biological nor custodial parents met *DSM-III* criteria (or FH-RDC criteria) for lifetime diagnoses of alcohol abuse or dependence. At Time 1, interviews were conducted with 71.2% of biological fathers and 93.8% of biological mothers.

Recruitment biases because of selective contact with participants or participant refusals are discussed in detail elsewhere (Chassin et al., 1991, 1992). Analyses of participation bias found that the sample was unbiased with respect to alcoholism indicators that were available in archival records (e.g., blood-alcohol level at the time of the arrest, Michigan Alcoholism

Screening Test results). Moreover, the alcoholic sample showed similar rates of other psychopathology to those reported for a community-dwelling alcoholic sample (Helzer & Pryzbeck, 1988). These data support the representativeness of the sample. However, participants who refused participation were more likely to be Hispanic, and if there was an arrest record, more likely to be married at the time of the arrest (Chassin et al., 1992). Although the size of the bias was small and unrelated to archival indicators of alcoholism, some caution is warranted in generalization.

A long-term follow-up (hereafter referred to as Time 4) was conducted when the original adolescents were in young adulthood (ages 18–23, *mdn* age = 20). Sample retention was high and included 407 young adults (90% of the original targets) divided among 213 COAs (86.6% of the original sample) and 194 controls (93.3% of the original sample). Participant retention was unbiased by gender and ethnicity, but more COAs than controls were lost to follow-up, $\chi^2(1, N = 454) = 5.45, p < .02$. Retention was also successful for parents; 325 fathers (80% of those who had participated at least once, plus 24 first-time participants) and 390 mothers (88% of those who had participated at least once, plus 1 first-time participant) were interviewed at Time 4.

At Time 4, for the first time, full-biological siblings were included in the study if they were in the age range of 18–26. A total of 326 siblings (87% of those who were eligible, *mdn* age = 22) were interviewed. Although siblings' data could not be used to test questions requiring information from adolescence (i.e., questions concerning adolescent symptomatology), their data could be used to evaluate parent alcoholism effects on young adult outcomes. Combining data for the original adolescents and their siblings produced a Time 4 sample of 414 families with 732 young adults. This represents 91.2% of the original families. Of these, there were 230 families (420 young adults) for whom complete information was available on psychopathology in both biological parents (because both biological parents were interviewed at Times 1 and 4). Characteristics of the original young adult sample and their siblings at follow-up are shown in Table 1. Among the original targets, the COA and control groups did not significantly differ in gender, ethnicity, marriage rates, or rates of full-time employment. However, COAs were significantly more likely than controls to have had a child, $\chi^2(1, N = 406) = 5.58, p < .02$, and significantly less

likely than controls to be full-time students, $\chi^2(2, N = 406) = 16.56, p < .001$. Among the siblings, there were no significant differences between COAs and controls, although COAs were marginally less likely to be full-time students ($p < .10$).

Procedure

Data were collected through computer-assisted interviews with the adolescents and their parents, either at their residence or at the Arizona State University campus. There were three annual assessments during adolescence and one young adult follow-up. The measures were programmed onto laptop computers, and all skip patterns were automatically implemented. Trained interviewers read each item aloud. All responses were close-ended and entered directly into the computer. To minimize contamination, all members of the family were interviewed individually on the same occasion by different interviewers when possible. In cases in which a participant had moved out of state, an interviewer was recruited at a nearby university and administered a shortened paper-and-pencil version of the interview, and the computerized diagnostic interview was conducted by telephone. Interviewers were unaware of the group membership of the family and of the research questions (although the interview responses themselves revealed the extent of alcohol and drug use in the family). Interviews required 1–2 hr, and individuals were paid for their participation (up to \$65 over the waves).

To encourage honest responding, privacy and confidentiality were assured and reinforced with a Department of Health and Human Services Certificate of Confidentiality. To minimize the possibility of being overheard, participants had the option of entering their responses on the computer keyboard rather than making any verbal response.

Measures

The measures of interest were part of the larger interview battery.

Parent alcoholism and associated psychopathology. At Time 1, parents' lifetime *DSM-III* diagnoses of alcoholism (abuse or dependence), affective disorder (major depression or dysthymia), and antisocial personality were obtained with a computerized version of the DIS interview (Version 3; Robins, Helzer, Croughan & Ratcliff, 1981). At Time 4, parents' lifetime *DSM-III-R* (*Diagnostic and Statistical Manual of Mental Disorders*, 3rd ed., rev., American Psychiatric Association, 1987) anxiety disorder diagnoses (excluding simple phobia only) were obtained using a computerized version of the DIS (C-DIS III-R; Robins & Helzer, 1991). Moreover, for parents who did not meet lifetime criteria at Wave 1, C-DIS sections for alcohol abuse and dependence and depression were re-administered to allow for diagnosing new cases. Alcoholism in the biological mother and biological father were treated (separately) as dichotomous variables (lifetime diagnosis vs. no diagnosis). All other diagnoses were coded as dichotomous variables as either present (at least 1 biological parent met lifetime criteria) or absent (neither biological parent met lifetime criteria). Noninterviewed biological parents were considered not to meet criteria (except in the case of alcoholism for which FH-RDC were used to establish diagnoses based on spousal report).

Data imputation for noninterviewed parents. Making the assumption that noninterviewed parents do not meet diagnostic criteria may underestimate their psychopathology, and this could artificially inflate the importance of parent alcoholism effects. Accordingly, analyses were conducted both with the total sample and with the subsample in which both biological parents were interviewed at both Time 1 and Time 4 (for whom complete data on parental diagnoses were available). However, this subsample also has limitations in terms of generalizability and statistical power. Accordingly, analyses were also performed using data imputation methods (Little & Rubin, 1987) to assign noninterviewed participants a probability score of meeting diagnostic criteria for anxiety, depression, and antisocial personality. For each noninterviewed parent, this probability score was defined as

Table 1
Demographic Characteristics of the Target Sample at Follow-Up

Measure	Total	COA	Control
Original adolescents			
% female	46.9	47.8	45.9
% Hispanic	23.4	27.4	18.9
<i>M</i> age	19.9	19.9	19.9
% full-time students ^a	37.8	28.6	48.2
% ever married	14.0	11.7	16.5
% employed full-time	48.5	48.4	48.5
% had a child ^a	18.2	29.1	13.5
Siblings			
% female	50.0	48.3	51.4
% Hispanic	29.0	32.2	26.1
<i>M</i> age	21.6	21.8	21.5
% full-time students	26.3	24.5	28.0
% ever married	30.1	29.8	32.0
% employed full-time	53.7	57.0	50.9
% had a child	29.1	31.8	26.8

Note. Among original adolescents, *ns* vary from 381 to 407 because of missing data. Among siblings, *ns* vary from 310 to 326 because of missing data. COA = children of alcoholics.

^a Comparison between COA and controls was significant at $p < .05$.

the prevalence of the particular disorder in the subgroup of interviewed biological parents who were of the same gender and alcoholism status. A family level score was then created to reflect the presence of the disorder in at least one parent. If the one interviewed parent met diagnostic criteria, then the family was considered to meet criteria. However, if the one interviewed parent did not meet diagnostic criteria, then the family level score was the imputed value for the noninterviewed parent.¹

Recency of parental alcoholism. To assess recency of parental alcohol problems, at each wave of measurement parents self-reported alcohol dependency symptoms using nine items adapted from Sher's (1987) questionnaire. At Wave 4, parents self-reported these dependency symptoms occurring during the past 5 years (the approximate time period between Waves 3 and 4).

Adolescent symptomatology. In each of the three adolescent interviews, adolescents and their parents reported on the adolescents' symptomatology in the past 3 months using items from the Child Behavior Checklist (Achenbach & Edelbrock, 1981). For internalizing symptoms, parents and adolescents reported on 7 items that loaded on the Internalizing factor for both boys and girls ages 12–16. Internal consistencies (coefficient alpha) ranged from .63–.79 over waves and reporters. For externalizing symptoms, parents and adolescents reported on 21 items that loaded on the Externalizing factor for both boys and girls ages 12–16. Internal consistencies (coefficient alpha) ranged from .87–.90 over measurement waves and reporters. For the current analyses, each participant's score was computed (separately for child and parent reporters) by averaging across the available scale scores from Times 1–3. Internal consistencies of these aggregate measures ranged from .78 to .92. Because the parent and adolescent reports were only modestly correlated ($r = .30$ for internalizing and .48 for externalizing), the path analyses examining adolescent internalizing and externalizing symptoms as mediators of parent alcoholism effects on young adult diagnoses were performed separately for parent-reported symptoms and for child-reported symptoms.

For alcohol and drug use, at each measurement wave adolescents self-reported their frequency of consumption during the past year of alcohol (beer/wine and hard liquor) and drugs (marijuana/hashish, cocaine/crack, tranquilizers, barbiturates, amphetamines, hallucinogens, opiates, and inhalants). Synonyms for each substance, including street names, were given in each item, and response scales ranged from abstinence to more than daily use. Adolescents also self-reported their frequency of heavy drinking (5 drinks per occasion, times drunk). For the current analyses, an alcohol use score at each time of measurement was computed by averaging across the four past-year frequency items (use of beer/wine, hard liquor, 5 drinks at a sitting, and times drunk). At each wave of measurement, an illegal drug use score was computed by summing the frequency of marijuana/hashish use with the highest frequency of use for any other illegal drug. As with symptomatology, aggregate measures of adolescent use were created by averaging across available scores for the three waves of measurement (internal consistencies were .84 for alcohol use and .89 for drug use). Finally, at the last wave of adolescent measurement, parents reported adolescents' alcohol and drug-related problems using the Diagnostic Interview for Children and Adolescents—Parent Version (DICA-P) interview (Herjanic & Campbell, 1977). Adolescents' lifetime diagnoses of alcohol or drug disorders were considered as dichotomous variables ($n = 19$ diagnosed cases).

Young adult diagnoses. At Time 4, *DSM-III-R* diagnoses of alcohol abuse and dependence, drug abuse and dependence, affective disorder (major depression and dysthymia), and anxiety disorder (excluding simple phobia only) were made using the C-DIS III-R (Robins & Helzer, 1991). In analyses examining adolescent mediators of young adult disorders, we created a variable to reflect problems that were active in the past 5 years (reflecting problems since the last adolescent assessment). For the past-5-year diagnosis, only those who both met lifetime diagnostic criteria and

who reported a symptom within the past 5 years were considered to manifest the disorder.

Results

The first question of the study was simply whether parental alcoholism was associated with elevated risk for alcohol and drug diagnoses, anxiety, and depression. The prevalence of lifetime diagnoses of alcohol abuse/dependence, drug abuse/dependence, depressive disorders, and anxiety disorders are presented in Table 2 for the overall sample, for COAs, and for controls (separately for the original targets and their siblings). First, a series of parent alcoholism by target gender log-linear analyses were performed to determine whether there were significant interactions between parent alcoholism and the target participant's gender in predicting diagnosis. Because no significant interactions were found, prevalences are presented for both genders combined.² As shown in Table 2, compared with non-COAs, COA targets were at significantly elevated risk for diagnoses of alcohol abuse or dependence, drug abuse or dependence, and depressive disorder, and were marginally elevated in their risk for anxiety disorder. Among siblings, COAs were significantly elevated in alcohol and drug abuse/dependence, but there were no significant differences between COAs and controls in depression and anxiety disorders.

We next examined the effects of recency of paternal alcoholism (because there were insufficient numbers of alcoholic mothers to subdivide by recency). First, we selected a subsample of families in which there was paternal but no maternal lifetime alcoholism, and in which the biological father was interviewed at Time 4 ($N = 127$ fathers and 230 young adult targets and siblings). We divided alcoholic fathers into those who (at Time 4) did and did not report the occurrence of an alcohol dependence symptom within the past 5 years. Chi-square comparisons of the young adults' lifetime and past-5-year diagnoses of alcohol, drug, anxiety, and depressive disorders showed no significant differences (all $ps > .2$). Similar comparisons were performed for the adolescent phase of the study, comparing fathers who did and did not report dependence symptoms over the 3 years of study. There were no significant differences in young adults' diagnoses, except for a greater prevalence of lifetime drug diagnoses (and past-5-year drug diagnoses) among those whose fathers reported dependence symptoms during the adolescent study, both $\chi^2(1, N = 228) = 4.47, p < .04$ (preva-

¹ Other demographic variables (e.g., age, marital status, income, ethnicity) were not used in the data imputation because they were either uncorrelated with diagnoses or failed to make robust unique contributions to prediction of diagnoses after gender and parent alcoholism were considered. This was also true for the use of spousal "cross-diagnoses" (e.g., maternal anxiety predicting paternal depression). Spousal "within-diagnoses" (e.g., maternal anxiety predicting paternal anxiety) were not used, because if the interviewed spouse met criteria, the family was considered to meet criteria. Adjusting the imputed score for cases in which the spouse did not meet criteria left all results unchanged to the second decimal place.

² Although it is not a focus of the current article, the expected gender differences were also found in these analyses. That is, men had significantly higher rates of alcohol abuse/dependence than did women, and women had significantly higher rates of anxiety and depressive disorders than did men. These gender differences were marginal for the original target sample and significant for the sibling sample.

Table 2
*Prevalence of DSM-III-R Lifetime Diagnoses Among Young Adults
 As a Function of Parental Alcoholism*

Measure	Overall	COA	Control	Odds ratio	95% CI
<i>Original targets (n = 407)</i>					
Diagnosis					
Alcohol abuse/dependence***	39.6	52.6	25.3	3.28	2.15–4.99
Drug abuse/dependence***	15.2	21.1	8.8	2.79	1.54–5.06
Depression***	18.4	24.4	11.9	2.40	1.40–4.10
Anxiety disorder†	21.9	25.4	18.0	1.55	0.96–2.27
<i>Siblings (n = 326)</i>					
Alcohol abuse/dependence**	38.0	45.7	31.4	1.84	1.17–2.88
Drug abuse/dependence*	16.3	20.5	12.6	1.81	1.00–3.26
Depression	18.7	19.2	18.3	1.06	0.61–1.86
Anxiety disorder	23.6	23.8	23.4	1.02	0.61–1.71

Note. DSM-III-R = *Diagnostic and Statistical Manual of Mental Disorders* (3rd edition, revised); COA = children of alcoholics; CI = confidence interval.

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

lences of 14.7% and 25.9% for offspring of remitted and active alcoholic fathers, respectively). Accordingly, recency of parental alcoholism was not considered further.

Specificity of Parental Alcoholism Effects: Predicting Young Adult Diagnosis From Parental Alcoholism and Other Parental Psychopathology

A further question was whether parental alcoholism significantly predicted young adult psychopathology above and beyond other forms of parental psychopathology. To allow inclusion of both the original targets and their siblings in a single analysis while appropriately considering the nesting of observations within families, we tested this question with hierarchical linear modeling (HLM/2L software; Bryk, Raudenbush, & Congdon, 1996). In each case, the young adult's lifetime diagnosis (as a dichotomous variable) was predicted from his or her age and gender (as individual level variables) and maternal alcoholism, paternal alcoholism, parental antisocial personality, parental depression, and parental anxiety disorder (as family level variables). Interactions between parental diagnoses and young adult's age, and between parental diagnoses and young adult gender, were tested, and non-significant interactions were trimmed.

The magnitude of each significant unique effect can be seen in its associated odds ratio (OR; the odds of meeting diagnostic criteria for a young adult who is exposed to the risk factor divided by the odds of meeting diagnostic criteria for a young adult who is not exposed to the risk factor). The OR is an approximation to the relative risk statistic, and relative risk indices of three or higher have conventionally been considered strong (Ibrahim, 1985). Note that the ORs reported here reflect the magnitudes of unique effects (i.e., the effect of a given risk factor over and above the other factors in the model). Given dichotomous outcome variables, interpretations of coefficients in these HLM models are equivalent to those in logistic regressions.

Results are presented here for the total sample, with noninterviewed parents considered not to meet diagnostic criteria (except for alcoholism, which was assessed by spousal report). However, because missing data on noninterviewed parents might influence

findings, these models were also estimated using data imputation procedures, described earlier, to assign noninterviewed parents a probability score of meeting diagnostic criteria for anxiety, depression, and antisocial personality. To assess the robustness of the effects, models were also estimated using values two standard errors above and below the imputed score. Finally, models were estimated for the subsample of families with no missing data on parent diagnoses (because both parents were directly interviewed). Differences in findings across these methods and subsamples are noted.

Predicting young adult alcohol and drug abuse/dependence. The model predicting young adults' alcohol abuse/dependence showed significant unique effects of paternal alcoholism (coefficient = .73, $p < .001$, OR = 2.08), maternal alcoholism (coefficient = .55, $p < .05$, OR = 1.73), parental antisocial personality (coefficient = .93, $p < .02$, OR = 2.54), and gender (coefficient = .90, $p < .001$, OR = 2.46). Young adults with alcoholic mothers, with alcoholic fathers, with parents who met criteria for antisocial personality disorder, and males were more likely to develop alcohol abuse or dependence.

These effects were all replicated with data imputation procedures and at values two standard errors above and below the imputed scores, as well as in the subsample with two interviewed parents. The only change in the models with imputed data and in the directly interviewed subsample were additional significant effects of parental anxiety diagnoses (ORs between 1.51 and 2.24 across models, all $ps < .05$). In addition, in the subsample of two interviewed parents there was an interaction between parental antisocial personality and gender, such that the effects of parental antisocial personality were confined to females.

Prediction of young adults' drug abuse/dependence diagnoses showed significant unique effects of maternal alcoholism (coefficient = .73, $p < .02$, OR = 2.08), parental antisocial personality (coefficient = .95, $p < .02$, OR = 2.59), and a marginally significant effect of parental anxiety disorder (coefficient = .43, $p < .08$, OR = 1.53). Young adults with alcoholic mothers, with parental antisocial personality, and with parental anxiety disorder were more likely to develop drug abuse or dependence.

These results were all replicated across models using imputed data as well as values two standard errors above and below the imputed scores. However, the subsample with two interviewed biological parents replicated the effects for parental anxiety disorder, but not maternal alcoholism or parental antisocial personality. Rather, there was a significant effect of paternal alcoholism (coefficient = 1.31, OR = 3.69).

Prediction of young adult depression and anxiety disorders. For depression, variation at the family level was nonsignificant ($p > .50$), which reduces confidence in the appropriateness of the HLM models. Accordingly, logistic regression analyses were used to predict depression. Results showed significant main effects of maternal alcoholism ($\beta = .76, p < .01, OR = 2.13$) and gender ($\beta = -.71, p < .01, OR = .50$), such that individuals with maternal alcoholism and women were at higher risk for depression diagnoses. There was a marginal main effect of parental depression ($\beta = .37, p < .09, OR = 1.46$), a significant effect of age ($\beta = .12, p < .02, OR = 1.13$), and an interaction between parent depression and age ($\beta = -.24, p < .01$), such that parental depression was associated with greater risk for offspring depression among younger participants (1 *SD* below the mean, $\beta = .93, p < .01, OR = 2.52$) but not among older participants (1 *SD* above the mean, $\beta = -.18, ns$).

All of these effects were replicated in models with imputed data as well as values two standard errors above and below the mean of the imputed scores. However, the subsample with two interviewed biological parents did not replicate the maternal alcoholism effects. This subsample also showed a parent depression by gender interaction (such that parent depression effects were confined to males), an interaction between parent antisociality and age (such that parental antisociality was modestly associated with offspring depression among older participants, but not younger participants), and an interaction between paternal alcoholism and gender, such that paternal alcoholism was associated with depression among females but not among males.

For anxiety disorders, variation at the family level was nonsignificant ($p > .25$), reducing confidence in the HLM model. Accordingly, logistic regression analyses were used. Results revealed significant main effects of maternal alcoholism ($\beta = .75, p < .01, OR = 2.12$), parental anxiety disorder ($\beta = .51, p < .01, OR = 1.67$), and gender ($\beta = -.65, p < .01, OR = .52$), such that anxiety disorders were more prevalent in those with alcoholic mothers, in those with parental anxiety disorder, and in females.

These effects were all replicated in models with imputed data and at values two standard errors above and below the imputed scores. Prediction in the subsample of two interviewed parents replicated all effects except for maternal alcoholism, which was similar in magnitude to that in the full sample (coefficients in both samples = .75) but not significant in this subsample ($p < .11$).

Mediators of Parent Alcoholism Effects on Young Adult Diagnoses

The next question was whether parent alcoholism effects on young adult diagnoses were mediated by adolescent levels of internalizing and externalizing symptoms, and adolescent alcohol and drug use. Because these analyses required knowledge of adolescent symptomatology, only the 407 original target partici-

pants were considered. All models predicted dichotomous diagnoses of disorders that were active within the past 5 years.

Predicting substance abuse/dependence. In predicting alcohol and drug diagnoses, the exogenous variables were maternal and paternal alcoholism, parental antisocial personality, parental depression, parental anxiety disorder, and age. The three mediating variables were adolescent internalizing and externalizing symptoms, and either adolescent alcohol or drug use (see Figures 1 and 2). We hypothesized that adolescent internalizing symptoms would be predicted by maternal and paternal alcoholism, and parent depression and anxiety disorders. We hypothesized that adolescent externalizing symptoms would be predicted by maternal and paternal alcoholism, and parental antisocial personality. We hypothesized that adolescent alcohol (or drug) use would be predicted by maternal and paternal alcoholism, parental antisocial personality, and age. All exogenous variables and mediators were treated as predictors of young adult substance abuse/dependence.

Models were tested using path analysis with maximum likelihood estimation (using EQS 5.1 software; Bentler, 1995). Although LISCOMP software is the optimal method for modeling mediational relations with dichotomous outcomes, the current sample size was inadequate for this approach. Separate models were estimated for parent and child reports of adolescent symptomatology (because parent and child reports were only modestly correlated). Models were also estimated both with and without considering adolescent DICA-P diagnoses of alcohol or drug disorders as mediating variables.³ In all path analytic models, all exogenous variables were allowed to be correlated, and all covariances among the error variances of the three mediators were freely estimated.

Prior to model testing, we examined the presence of interactions involving child gender and the mediating variables using logistic regression analyses (SAS6.18 software). In predicting alcohol diagnoses, there was a significant interaction between internalizing symptoms and gender ($p < .01$). In predicting drug diagnoses, there was a significant interaction between adolescent drug use and gender ($p < .03$).⁴ Accordingly, in path analyses of alcohol and drug diagnoses, we stacked models on gender, and estimated

³ All mediational models and all tests of total indirect effects were also estimated with imputed data on missing parent diagnoses and at values two standard errors above and below the imputed score. Because all effects were maintained in models with imputed data and in models with values two standard errors above and below the imputed score, these models will not be discussed further. Because of sample size constraints, mediational models were not estimated for the subsample of two interviewed biological parents.

⁴ It was the interactions between child gender and the mediators that dictated the use of path analysis with maximum likelihood estimation rather than the use of logistic regression (which is designed for dichotomous outcomes). The path analytic technique allowed us to preserve the total sample size while estimating separate paths for men and women where necessary. Logistic regression mediational models would have required separate analyses for men and women, weakening statistical power. However, to assess the effect of the method of estimation, for each model, we performed logistic regressions (separately for men and women) and compared the pattern of findings with those produced by maximum likelihood estimation (path analyses separately for men and women). For the prediction of alcohol and drug abuse/dependence and anxiety, the patterns were identical. For predicting depression, logistic regression models replicated the path analyses and also produced a marginally significant direct effect of paternal alcoholism on young adult depression.

separate paths for men and women for all paths involving internalizing symptoms (for alcohol diagnoses) and all paths involving adolescent drug use (for drug diagnoses). This included all directional paths, error variances, and covariances involving those variables. All other paths, variances, and covariances were forced to equality for men and women.

Figure 1 presents the significant paths from the model predicting alcohol diagnosis. Young adult alcoholism was related to both adolescent externalizing symptoms and adolescent alcohol use. However, these mediators did not entirely account for the paternal alcoholism effect on young adult diagnosis, because they did not eliminate the significant direct effect of paternal alcoholism on young adult alcohol abuse/dependence. There was no significant effect of adolescent internalizing symptoms on alcohol abuse/dependence. However, the need to estimate separate paths for men and women from internalizing symptoms weakens the power to detect these effects.

We also tested a model in which adolescent diagnosis of alcohol abuse/dependence was included as a mediating variable. There were no changes in results. Finally, path analyses with parent reports of adolescents' internalizing and externalizing symptoms were performed. In models using parent-reported symptomatology, there was an interaction between externalizing symptoms and gender, such that higher levels of externalizing symptoms were related to offspring alcoholism only for boys ($p < .10$).

To assess mediational paths, we performed tests of unique indirect effects using the methods of Sobel (1986). There was a marginally significant indirect effect of maternal alcoholism on young adult alcohol abuse/dependence through adolescent externalizing symptoms (estimate = .02 for both men and women, $p <$

.10), a significant indirect effect of paternal alcoholism through externalizing symptoms (estimates = .04 for women, .03 for men, both $ps < .05$), and a significant indirect effect of parent antisocial personality through externalizing symptoms (estimate = .02 for both women and men, both $ps < .05$). These indirect effects through externalizing symptoms accounted for between 21% (for men) and 24% (for women) of the unique effect of maternal alcoholism; between 14% (for men) and 17% (for women) of the unique effect of paternal alcoholism, and for between 31% (for men) and 34% (for women) of the unique effect of parental antisocial personality on young adult alcoholism. Finally, there were significant indirect effects of both paternal alcoholism and parental antisocial personality on young adult alcoholism through adolescent drinking (estimates of .04 and .03, respectively, for both women and men, all $ps < .01$, accounting for between 29%–30% of the parent alcoholism effect for women and men, respectively, and for 37%–39% of the parent antisocial personality effect for women and men, respectively).

These significant indirect effects were all maintained when adolescent alcohol diagnoses were included in the model. However, in the parent-report model, for women, the indirect effects through externalizing symptoms were not replicated (because parent report of externalizing symptoms did not predict the outcome for women). For men, these parent-reported indirect effects were of similar magnitude to the child-report model, but because of lower power were not statistically significant.

Figure 2 presents the significant path estimates from the model predicting young adult drug diagnoses. There were significant effects of adolescent externalizing symptomatology, adolescent drug use (for women), and a marginally significant effect of

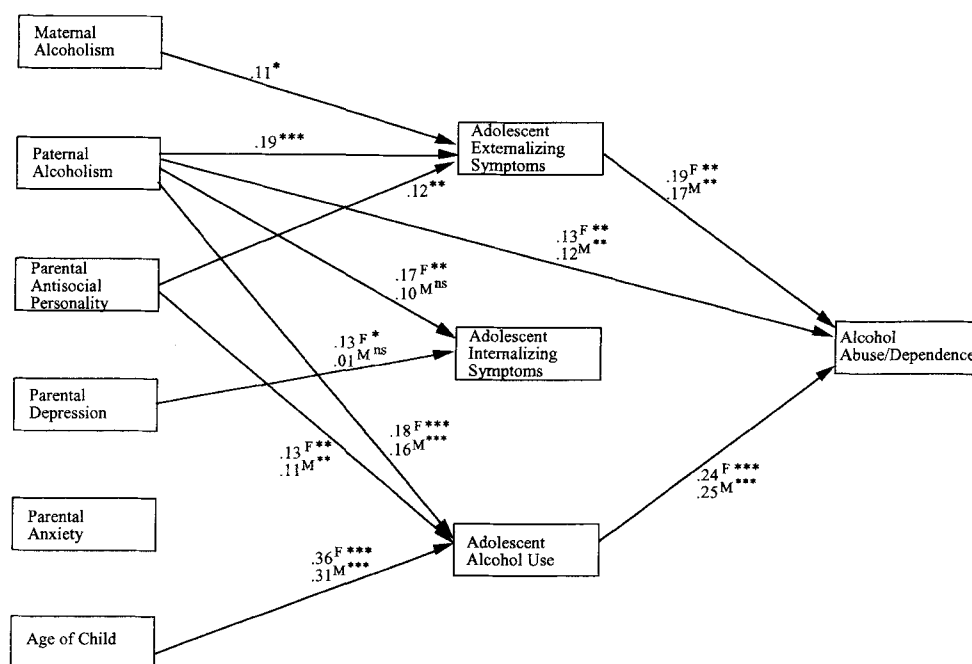


Figure 1. Standardized path coefficients from full model predicting alcohol abuse/dependence. For simplicity, nonsignificant paths, intercorrelations among the exogenous variables, intercorrelations among the mediators, variance estimates, and error variance estimates are not shown. F = path estimate for women; M = path estimate for men; ns = nonsignificant. * $p < .05$. ** $p < .01$. *** $p < .001$.

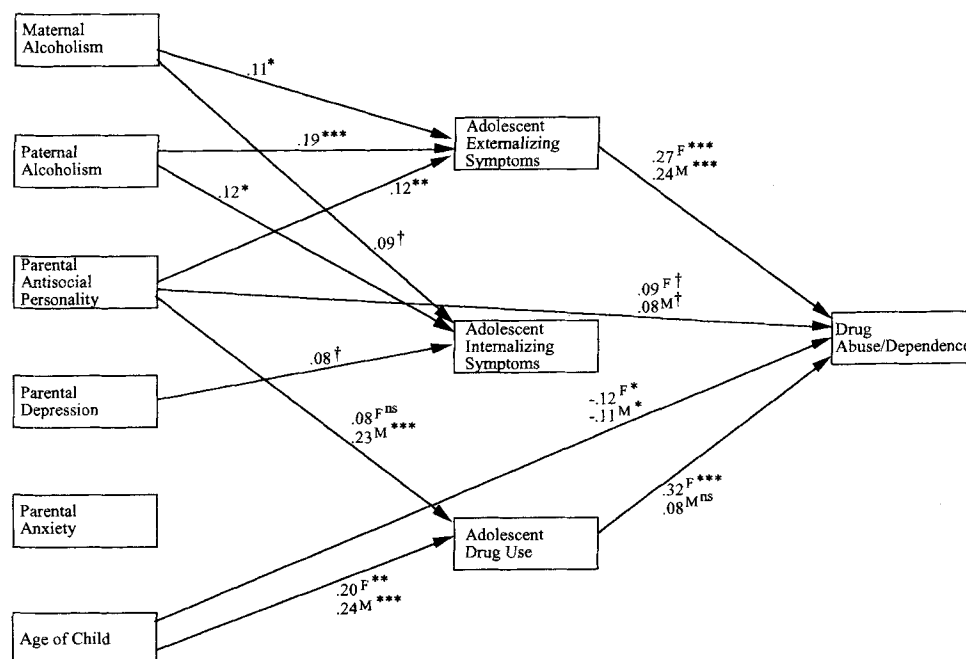


Figure 2. Standardized path coefficients from full model predicting drug abuse/dependence. For simplicity, nonsignificant paths, intercorrelations among the exogenous variables, intercorrelations among the mediators, variance estimates, and error variance estimates are not shown. F = path estimate for women; M = path estimate for men; ns = nonsignificant. † $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$.

parental antisocial personality on young adult drug abuse/dependence. All paths were replicated after including adolescent drug diagnosis as a mediator in the model. Moreover, except for the direct effect of age on drug diagnoses, all paths were replicated with parent report of adolescent internalizing and externalizing symptoms.

In predicting young adult drug abuse/dependence, there were significant indirect effects of maternal and paternal alcoholism and parental antisocial personality through adolescent externalizing symptoms (estimates ranged from .03 to .05, all $ps < .05$).⁵ The indirect effects through externalizing symptoms accounted for between 28% and 29% of the maternal alcoholism effect (for men and women, respectively), for between 56% and 59% of the paternal alcoholism effect (for men and women, respectively) and for 35% and 37% of the parental antisocial personality effect (for men and women, respectively). These indirect effects were found with and without including adolescent drug diagnoses and were replicated in the parent-report model.

Predicting depression and anxiety. Before predicting young adult depression and anxiety diagnoses, logistic regression analyses tested interactions between gender and the mediating variables. Because no significant interactions were found, gender was included as an exogenous variable (rather than estimating stacked models for men and women, as described for the substance use disorders). Gender was allowed to freely covary with the other exogenous variables and was treated as a predictor of adolescent internalizing and externalizing symptoms and of young adult depression and anxiety diagnoses. In predicting depression and anxiety, both adolescent alcohol and drug use were included as me-

diating variables. Because adolescent diagnoses were not available for depression and anxiety, adolescent status was not considered.

Figure 3 presents the significant paths from the model predicting young adult depression diagnoses. Young adult depression was predicted by both adolescent internalizing and externalizing symptoms, and marginally predicted by adolescent drug use. However, these mediators did not entirely account for parent alcoholism effects as seen in the significant direct effect of maternal alcoholism. There was also a significant direct effect of parent depression on young adult depression. However, models using parent report of adolescent symptomatology produced different findings. Parental reports of adolescent internalizing and externalizing symptoms were unrelated to young adult depression diagnoses.

There were indirect effects of maternal and paternal alcoholism on young adult depression through externalizing symptomatology (estimates = .02 and .04, respectively, both $ps < .10$, accounting for 14% of the maternal alcoholism effect and 27% of the paternal alcoholism effect). There was also a marginally significant indirect effect of paternal alcoholism through internalizing symptomatology (estimate = .02, $p < .10$, accounting for 3% of the paternal

⁵ An indirect effect of paternal alcoholism on young adult drug abuse/dependence through externalizing symptoms may appear inconsistent with our HLM models reporting no direct unique effect of paternal alcoholism. However, the HLM models predicted lifetime diagnoses, whereas the mediational models predicted lifetime diagnoses that are symptomatic within the past 5 years. Re-estimating the HLM model with the past-5-year diagnosis as the outcome variable showed a unique effect of paternal alcoholism (OR = 1.61, $p = .051$).

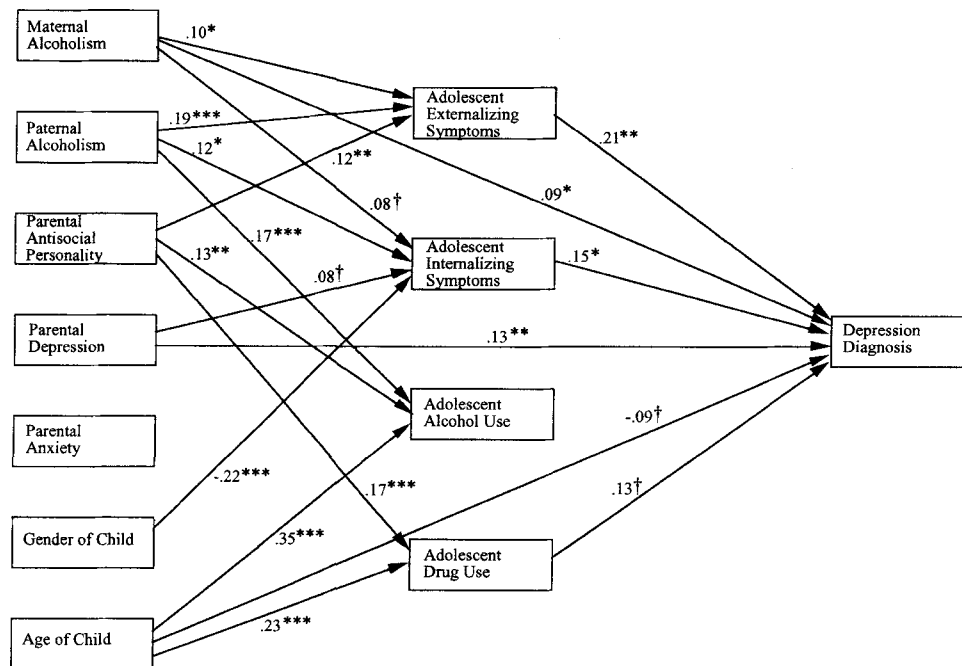


Figure 3. Standardized path coefficients from full model predicting depressive disorders. For simplicity, nonsignificant paths, intercorrelations among the exogenous variables, intercorrelations among the mediators, variance estimates, and error variance estimates are not shown. † $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$.

alcoholism effect). However, these indirect effects were not replicated in parent report (because parent-reported adolescent symptomatology was not significantly related to young adult depression).

Figure 4 presents significant paths from the model predicting young adult anxiety disorders. As shown in Figure 4, adolescent internalizing symptoms predicted young adult anxiety disorders, but did not entirely account for parental alcoholism effects (as reflected in the significant direct effect of maternal alcoholism). There was also a significant effect of parent depression. The model was also tested using parent report of adolescent symptomatology. In this model, there was no significant direct effect of parent depression on offspring anxiety disorders, but there was a significant effect of adolescent alcohol use. Finally, because of the high collinearity between adolescent alcohol and drug use, we tested each in separate models as a mediator. This produced a marginal effect of alcohol use on young adult anxiety ($p < .10$).

There were no significant indirect effects of parent alcoholism on young adult anxiety disorders. However, there was a marginally significant indirect effect of parent depression through adolescent internalizing symptoms (estimate = .02, $p < .10$, accounting for 36% of the parent depression effect). This significant indirect effect was replicated in the parent-report model, which also showed a marginally significant indirect effect of maternal alcoholism on anxiety disorder diagnosis through adolescent internalizing symptoms ($p < .10$).

Discussion

The first goal of the current study was to assess parent alcoholism effects on young adult psychopathology. Results showed that

COAs were more likely than were non-COAs to have a lifetime diagnosis of alcohol abuse or dependence (and drug abuse or dependence), and this result was seen both for our original targets and for their full-biological siblings. This finding is consistent with a large literature, documenting that alcoholism "runs in families" (see McGue, 1994; Sher, 1991, for reviews). The magnitude of the ORs are similar to those in recent studies of community samples of young adult COAs (Sher, Walitzer, Wood, & Brent, 1991). For example, Russell (1990) reviewed the literature and suggested that community studies typically produce risk ratios of 1.5 to 3. This suggests that parent alcoholism represents a moderate risk factor for young adult alcohol abuse/dependence in such samples.

It is interesting that COAs' risk for alcohol abuse/dependence in the past 5 years did not differ whether or not their fathers' alcoholism was active or remitted during the study period. This suggests that the mechanism by which paternal alcoholism influences offspring alcoholism is not dependent on active environmental modeling of problem drinking. This finding has several possible interpretations, which are not mutually exclusive. First, modeling may influence the beginning of an adolescent's risk trajectory prior to adolescence, but then drinking may become functionally autonomous and be maintained by other factors. Second, other risk factors that are correlated with parental alcoholism may not "recover" with parental recovery from alcoholism, but rather may continue to influence risk among COAs. For example, sibling drinking, affiliations with peers who drink, lowered socioeconomic status, and high levels of environmental stress may place adolescent COAs on a high-risk developmental trajectory regardless of whether parental alcoholism is active or remitted (Ellis, Zucker, & Fitzgerald, 1997). Finally, heritable individual differ-

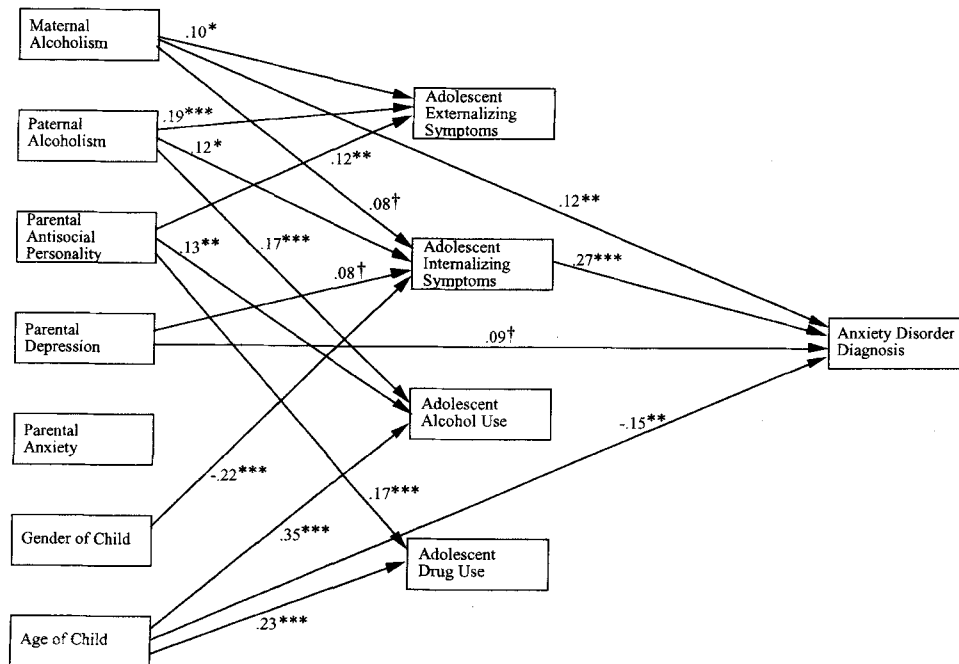


Figure 4. Standardized path coefficients from full model predicting anxiety disorders. For simplicity, nonsignificant paths, intercorrelations among the exogenous variables, intercorrelations among the mediators, variance estimates, and error variance estimates are not shown. † $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$.

ence factors such as personality or the pharmacological reinforcing effects of alcohol may elevate COA risk regardless of any environmental modeling of problem drinking (Schuckit & Smith, 1996; Sher, 1991). This is consistent with recent findings in an adoptee sample that the correlations between adolescent alcohol involvement and the problem drinking of their adoptive parents were small and nonsignificant (McGue, Sharma, & Benson, 1996). McGue et al. suggested that relations between the drinking behavior of adolescents and their biological parents may reflect shared genetic factors rather than shared environment factors.

The current data are in contrast to the findings of Moos and Billings (1982) who reported that children of recovered alcoholic parents were less impaired than were children of relapsed alcoholic parents (in terms of anxiety and depression). Because Moos and Billings recruited alcoholics from a treatment sample, successful treatment interventions may have altered the developmental trajectories of those children. Moreover, those children were considerably younger than the current sample (early to middle adolescence), so that their diagnostic status in young adulthood was not ascertained. It is interesting that children of recovered and relapsed alcoholic parents in the Moos and Billings study did not significantly differ in their cigarette smoking or other substance use. Because of their young age, it is unclear whether the lowered risk found among offspring of recovered alcoholic parents by Moos and Billings would extend to the domains of alcohol and drug use/abuse in young adulthood.

In terms of parent alcoholism effects on young adult psychopathology, the current data showed smaller effects for the sibling sample than for the original targets who had been followed since adolescence. In general, for COAs the prevalence of psychopa-

thology was somewhat lower among siblings than among our original targets. However, for controls, the prevalence of psychopathology was somewhat higher for siblings than for targets. This combination reduced the magnitude of the significant parent alcoholism effect on alcohol and drug abuse/dependence for the sibling sample, and failed to produce a significant parent alcoholism effect on anxiety and depression in the sibling sample. These findings are somewhat puzzling and difficult to interpret. Analyses suggested that reduced rates of psychopathology among COA siblings and increased rates of psychopathology among control siblings could not be explained by lower participation rates among COA siblings than among control siblings. However, it is possible that a more complex interplay between participation biases and the demographic composition of the samples (e.g., gender, ethnicity, and age) produced this pattern.

The current study also tested the specificity of parent alcoholism effects, that is, whether maternal and paternal alcoholism had significant effects on offspring psychopathology above and beyond other parental psychopathology. Here again, there were robust findings that parental alcoholism was associated with offspring alcohol and drug abuse/dependence above and beyond parental antisocial personality, depression, and anxiety disorders. These findings were produced in the total sample, and in the subsample of families with two interviewed biological parents (suggesting that they were robust to missing data concerning parental psychopathology).

There was also some evidence for parent alcoholism effects on offspring anxiety and depression. These findings were somewhat less consistent in that there was no overall parent alcoholism effect found in the sibling sample. However, when the unique effects of

maternal and paternal alcoholism were disaggregated, there were significant unique effects of maternal alcoholism on both offspring anxiety and depression (and additional analyses showed that maternal alcoholism was related to depression for women in the sibling sample). These effects were somewhat weakened in the subsample with two interviewed parents because of a low prevalence of maternal alcoholism in that subsample. Previous evidence has been conflicting with respect to the relation between parental alcoholism and offspring anxiety and depression, and Schuckit (1996) concluded that COAs are not at increased risk for major depression or anxiety disorders, whereas Merikangas et al. (1996) reported links between family histories of alcoholism and anxiety. Some inconsistency in the literature may be due to sample differences in parents' comorbid anxiety or depression. That is, some previously reported links between parent alcoholism and internalizing disorders in offspring may be due to associated parental anxiety and depression, rather than specific to the parental alcoholism itself. Moreover, the current data suggest that inconsistent findings across samples may also be due to sample differences in the prevalence of maternal alcoholism. Many studies of COAs examine paternal rather than maternal alcoholism, and it may be maternal alcoholism that is the more robust unique predictor of offspring anxiety and depression.⁶ However, given the young age of the current participants, they have not yet completed the age of risk for anxiety and depression. Further follow-ups at later ages are necessary to illuminate the effects of parent alcoholism on offspring anxiety and depression.

The next question of the study was whether the effects of parent alcoholism on young adult diagnoses were mediated by adolescent levels of internalizing and externalizing symptomatology. The current data supported the hypothesis that the effects of parent alcoholism on young adult substance abuse/dependence are, in part, mediated by earlier conduct problems or externalizing symptoms (Sher, 1991; Zucker, 1994). This is consistent with the notion that early onset substance abuse is associated with early antisociality and conduct problems, as well as with a family history of alcoholism (Cloninger, 1987). Moreover, support for this externalizing pathway was robust. It was detected over and above the effects of other parental psychopathology, replicated with multiple methods of model estimation, obtained both with and without a separate consideration of earlier adolescent substance abuse/dependence diagnoses, and (for men) obtained for both parent and adolescent reports of externalizing symptoms. The consistent support produces confidence in the reliability of this effect.

The current findings also replicate and extend Cadoret et al.'s (1995) report of two different pathways to substance abuse/dependence, one reflecting a direct effect of paternal alcoholism, and one reflecting indirect effects of parent alcoholism and parent antisocial personality mediated through externalizing behaviors. Prediction of young adult alcoholism in the current data precisely replicated this pattern and extended Cadoret et al.'s study by demonstrating these findings with prospective rather than retrospective data.

In both Cadoret et al.'s (1995) data and the current findings, there was a significant direct effect of parent alcoholism on offspring alcoholism that could not be completely explained by earlier levels of internalizing and externalizing psychopathology. This pathway likely reflects other unmeasured mediators. For example, it has been suggested that COAs experience greater

pharmacological benefit from alcohol than do non-COAs (Sher, 1991) or are less sensitive to the negative effects of alcohol (Schuckit & Smith, 1996). This differential sensitivity to alcohol effects may partly explain COAs' heightened risk for alcoholism (Schuckit & Smith, 1996) and may underlie the current direct effect of paternal alcoholism on young adult alcoholism.

It is interesting that by disaggregating alcohol and drug outcomes, our findings revealed a slightly different pattern than did Cadoret et al.'s (1995) for drug diagnoses, in which the effect of paternal alcoholism was completely mediated by externalizing symptoms, but a marginally significant effect of parent antisocial personality remained. The more specific link between drug diagnoses and parent antisociality may reflect the fact that drug diagnoses necessarily involve illegal behaviors, whereas alcohol diagnoses do not.

In contrast to the findings for the externalizing pathway, the current data did not support an internalizing pathway mediating parental alcoholism effects on offspring substance abuse/dependence. Consistent with theories of alcoholism subtypes, it might be that this pathway is operative for later onset forms of substance abuse/dependence, and thus is not detectable in young adulthood (Cloninger, 1987). The current analyses did produce a significant interaction between gender and internalizing symptoms in predicting alcohol abuse/dependence, suggesting that an internalizing pathway might be operative for women, but the effects of internalizing symptoms on alcohol abuse/dependence were not significant when men and women were considered separately. Future studies with larger samples of women might be able to detect internalizing pathways. Moreover, evidence of an internalizing pathway might have been weakened by the use of a broad construct of internalizing symptoms, and instruments that separate anxiety and depression might produce stronger support (Merikangas et al., 1996).⁷

The current study also produced some information concerning mediators of parent alcoholism effects on young adult anxiety and depression, although caution is needed in interpretation because the effects were not robust across reporters. Mediational models suggested that parent alcoholism effects on young adult depression could be partially explained by adolescent levels of internalizing and externalizing symptomatology. The link between adolescent externalizing problems and depression in adulthood may seem counterintuitive, but it has been reported in several studies either only for boys (Gjerde, 1995) or for both genders (Robins & Price, 1991; see Zoccolillo, 1992, for a review). However, adolescent symptomatology was not able to fully account for the maternal

⁶ Merikangas et al., (1996) noted that the relation between anxiety and alcoholism was stronger when alcohol dependence was considered separately from alcohol abuse. Accordingly, we examined the prevalence of target's lifetime diagnoses as a function of parent alcohol abuse only or parental alcohol dependence. Results showed no significant differences, although there was a consistent pattern of slightly higher prevalences of all diagnoses for offspring of parents with alcohol dependence compared with alcohol abuse.

⁷ An absence of support for the internalizing pathway could also be due to the use of an abbreviated measure of internalizing symptoms. However, models using a larger pool of Child Behavior Checklist items (reported by parents only) also failed to support this pathway.

alcoholism effect on young adult anxiety disorders and depression, suggesting the effects of unmeasured mediators.

The final study question was whether adolescent alcohol and drug use raised risk for young adult anxiety and depression. The current data found little evidence for unique effects of adolescent substance use on young adult diagnoses, although there were weak effects of adolescent alcohol use on anxiety diagnoses and of adolescent drug use on depression. In part, effects that are attributed to adolescent substance use may be due to correlated adolescent symptomatology or correlated parental psychopathology, or both. The current data cannot distinguish whether adolescent substance use itself was a cause or an effect of adolescent symptomatology. However, once adolescents already have elevated levels of internalizing and externalizing symptoms and already use alcohol and drugs, their substance use no longer shows strong unique effects on young adult anxiety and depression. Possibly, the effects of adolescent substance use were inadequately captured by the current frequency of use measures. Although beyond the scope of this article, more intensive investigation of changes over time in adolescent substance use or substance-use-related consequences might reveal stronger effects of adolescent substance use on adult mental health outcomes.

Finally, it is necessary to consider some limitations of the current findings. First, our passive longitudinal family design does not distinguish genetic from environmental effects and cannot address questions of gene-environment interaction or covariation (McGue, 1995; McGue et al., 1996). Alternative designs such as adoption studies (Cadoret et al., 1995; Ge et al., 1996; McGue et al., 1996) and twin studies (Pike, McGuire, Hetherington, Reiss, & Plomin, 1996) have suggested that both gene-environment covariation and gene-environment interactions are important to consider in the etiology of psychopathology in adolescence and young adulthood (see McGue, 1997; Rutter et al., 1997, for a fuller discussion of genetically sensitive designs). Second, the current study examined adolescent symptomatology and substance use as predictors of young adult diagnoses, but additional mediators should also be considered (e.g., pharmacological effects of alcohol and drugs). Moreover, more detailed consideration of adolescent symptomatology (e.g., disaggregating anxiety and depression, or considering escalations in substance use over time) might produce different findings. Third, although the sample is large in terms of high-risk designs, it was not able to address the current suggestions of gender differences in mediational pathways, particularly with techniques that are ideal for modeling dichotomous outcomes. Fourth, although the current study improves on previous research by examining maternal and paternal alcoholism and other parental psychopathology (and recency of paternal alcoholism), the heterogeneity of parent alcoholism suggests that results be generalized with caution. As is evident in the COA literature, samples that vary in the gender of the alcoholic parent, the density of alcoholism in the family pedigree, and so forth, may produce different outcomes. Fifth, the onset of the diagnoses cannot be precisely dated, so that the effects of the adolescent mediators may reflect both the continuation of and the new onset of disorders. In general, research on alcohol disorders points to potentially important heterogeneity as a function of age of onset and course. For example, early onset antisocial alcoholism is thought to differ from developmentally limited alcoholism (which remits in adulthood with the assumption of adult roles) and also differs from later onset alcoholism, which

is more strongly associated with anxiety and depression (Cloninger, 1987; Zucker, 1994). Further follow-ups of this sample into older age periods are necessary to distinguish the antecedents of these different forms of alcoholism.

In sum, the current study extended previous research by using a longitudinal study of families actively recruited from the community to test the magnitude and specificity of parent alcoholism effects on young adult outcomes, and to test whether adolescent symptomatology and substance use explained these effects. Results showed that parent alcoholism was a moderate risk factor for young adult alcohol and drug abuse/dependence. Parent alcoholism risk for young adult substance use disorders could, in part, be explained by heightened levels of adolescent externalizing symptoms. However, this externalizing pathway could not completely explain the effect of fathers' alcoholism on young adult alcoholism, and offspring of alcoholic fathers were still at risk, whether or not their father's alcoholism was remitted during the period of the study. Although there was little evidence for an internalizing pathway into young adult substance abuse/dependence, further studies at older ages (particularly of women) are necessary to investigate this hypothesis.

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