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An Examination of Gender Patterns

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The primary goal of this study was to examine the relationship between parental psychopathology (specifically, alcohol dependence and depression) and adolescent psychopathology, by the gender of the adolescent and the gender of the parent. The sample included 426 13- to 17-year-old adolescents and their parents. All participants were administered the *SemiStructured Assessment for the Genetics of Alcoholism* to obtain clinical psychiatric diagnoses. Paternal psychopathology (depression and alcohol dependence) significantly predicted adolescent alcohol dependence, whereas maternal psychopathology did not. Maternal alcohol dependence did not predict any of the adolescent psychiatric diagnoses. In contrast, both paternal depression and maternal depression significantly predicted adolescent conduct disorder and depression. In addition, maternal depression significantly predicted adolescent anxiety. No significant interactions between parental psychopathology and adolescent gender were observed. Nevertheless, results from this study underscore the importance of considering the gender of the parent when examining the relationship between parental psychopathology and psychopathology in the offspring.

Many studies have established that children of alcoholic parents are at an increased risk for behavioral problems (Connolly, Casswell, Stewart, Silva, & O'Brien, 1993; Loukas, Fitzgerald, Zucker, & von Eye, 2001; Reich, Earls, Frankel, & Shayka, 1993). Children of alcoholic parents also are more likely to experience internalizing disorders such as depression and anxiety (Chassin, Pitts, DeLucia, & Todd, 1999; Reich et al., 1993) and externalizing disorders such as substance abuse problems in comparison to children who do not have alcoholic parents (Chassin et al., 1999; Chassin, Pitts, & Prost, 2002). Research also has shown that children of depressed parents are similar to children of alcoholic parents in regard to their psychological adjustment. More specifically, children of depressed parents have been found to have an elevated risk for both internalizing disorders (e.g., depression, anxiety) and externalizing disorders (e.g., conduct disorder, substance abuse; Downey & Coyne, 1990; Fergusson & Lynskey, 1993).

Although this research has yielded consistent results, the majority of research examining offspring of alcoholic or depressed parents has been conducted on children and adults. Relatively fewer studies have focused on adolescent offspring of alcoholic or depressed parents. Moreover, studies that have examined adolescents typically have included children and adolescents within the same sample, potentially masking the important developmental differences between childhood and adolescence. The lack of methodologically sound studies focusing on adolescent offspring of alcoholic or depressed parents is unfortunate, as adolescence is a time of immense change and challenge, when many transformations occur both within the adolescent and in the adolescent's contexts (Lerner, 2002; Steinberg, 2000). In addition, adolescence is the developmental period when experimentation with alcohol and drugs typically begins and the escalation of the use of these substances occurs (O'Malley, Johnston, & Bachman, 1998). Furthermore, the prevalence of psychological disorders such as depression and

anxiety also rises dramatically during this developmental period (Kessler, Avenevoli, & Merikangas, 2001; Lewinsohn, Clarke, Seeley, & Rohde, 1994).

The Gender of the Parent

It is important to note that many previous investigations examining the psychological adjustment of offspring of alcoholic parents and offspring of depressed parents have not considered the gender of the parent. Research focusing on offspring of alcoholic parents has overwhelmingly focused on fathers, whereas research examining offspring of depressed parents has tended to focus on mothers (Johnson & Jacob, 2000; Phares, 1992). Moreover, many theoretical models (e.g., attachment theory, Freudian theory) have emphasized maternal influences on offspring adjustment and have neglected to consider paternal influences. For example, socialization models typically infer that because mothers are more likely to spend more time with their children than fathers (Fagot, 1995; Holmbeck, Paikoff, & Brooks-Gunn, 1995), children should be more affected by maternal problems than paternal problems. However, the influence that fathers may have on their children should not be overlooked (Phares & Compas, 1992).

Recent research indicates that both mothers and fathers play an important role in their children's development and adjustment. Mothers and fathers appear to have differential effects on the adjustment of their children. For example, in a study conducted by Pederson (1994), paternal care was found to be a better predictor of adolescent anxiety and depressive symptomatology, whereas maternal care was found to be a better predictor of adolescent delinquency. Research specifically focusing on psychopathology has also indicated that psychological problems in the same-sex parent may be more closely associated with psychopathology in the offspring than psychopathology in the opposite-sex parent. For instance, in three separate studies examining adolescents and their parents (Crawford, Cohen, Midlarsky, & Brook, 2001; Davies & Windle, 1997; Fergusson, Horwood, & Lynskey, 1995), maternal internalizing symptoms (e.g., depressive symptoms) were associated with adolescent problems (depressive symptoms, conduct problems, and academic problems) for girls but not for boys. Unfortunately, none of these studies examined the link between *paternal* symptomatology or psychopathology and adolescent psychological adjustment. Therefore, results from these studies may simply indicate that girls are more affected by parental psychopathology than are boys. The absence of paternal data in these studies makes it difficult to discern which interpretation is more valid (i.e., girls are more affected by parental psychopathology and/or girls are more affected by maternal psychopathology, whereas boys are more affected by paternal psychopathology). Finally, it should be noted that all of these studies relied on the adolescents' and parents' *self-reports* of their *symptom* levels (clinical diagnoses were not assessed).

Only a handful of studies have examined *both* maternal and paternal *psychopathology* when examining the transmission of psychopathology. Consistent with the findings just discussed, Hops (1992) found maternal depression to be more closely related to daughters' depression, but paternal depression to be more closely associated with sons' depression. Luthar, Merikangas, and Rounsaville (1993) also examined the transmission of psychopathology by the gender of the parent. In their family study of opiod and cocaine abusers, maternal depression, but not maternal alcoholism, was significantly related to psychopathology in the offspring. In contrast, paternal alcoholism, but not paternal depression, was significantly associated with offspring psychopathology. However, the Luthar et al. study was limited because the data were retrospective and the participants were not directly interviewed. Moreover, offspring gender was not taken into account.

The Gender of the Offspring

As studies such as Hops's (1992) have indicated, the gender of the offspring also is important to consider when examining the relationship between parental psychopathology and adolescent psychopathology. Current theoretical models of human development suggest that the examination of adolescent gender differences should not be overlooked. For example, according to Gilligan's theory of human development (Gilligan, 1983), the development of the self for girls is closely tied to attachment and relationships with others, whereas for boys it is linked to emotionally separating from others. Research has supported and extended this theoretical framework by showing that parents reward girls more for dependent and affectionate behavior than boys, whereas boys are rewarded more for instrumental/achievement-oriented and aggressive behaviors (Hops, 1995, 1996). Research also has shown that girls show greater caring behavior and concern for the feelings of others in comparison to boys (Radke-Yarrow, Zahn-Waxler, Richardson, Susman, & Martinez, 1994). In essence, girls are taught and encouraged to be more oriented toward relationships with others than are boys. Accordingly, girls may be more enmeshed with the family than boys, especially during adolescence, when gender roles become even more pronounced and boys, not girls, are increasingly reinforced for their autonomous behavior. (Nolen-Hoeksema, 1994). Unfortunately for girls, their orientation to relationships with others and their failure to emotionally separate themselves from the problems of those that they care about may actually make them more vulnerable to family distress (Gore, Aseltine, & Colten, 1993). In contrast, boys may be protected from the harmful effects of family dysfunction and distress because they are rewarded for emotionally pulling away from the family.

Recent research is consistent with these hypotheses. For instance, Gore and colleagues (Gore et al., 1993; Gore & Colten, 1991) found that adolescent girls' higher interpersonal orientation and involvement were significantly related to their depression levels. In addition, Davies and Windle (1997) found that family discord was more consistently related to subsequent psychological difficulties (e.g., delinquency, alcohol problems) for girls than for boys during adolescence.

Consistent with this literature, maternal depressive symptomatology has been found to be related to the psychological adjustment of girls, but not boys, during adolescence (Crawford et al., 2001; Davies & Windle, 1997). Although this gender difference may partially be accounted for by gender differences in socialization, recent research also suggests that the genetic link between parental psychopathology and adolescent psychological problems is more robust for adolescent girls than for adolescent boys. For example, Jacobson and Rowe (1999) found that during adolescence, depressed mood was more strongly associated with genetic factors for girls than for boys. Moreover, these researchers found that the relationship between the family environment (i.e., family connectedness) and depressed mood was genetically mediated, particularly for girls.

Finally, it is important to note that when psychopathology occurs during adolescence, it may be differentially expressed according to the gender of the adolescent. Because girls are encouraged to be more dependent and self-depreciating (factors associated with internalizing disorders) and boys are encouraged to be more assertive and aggressive (factors more closely related to externalizing disorders), it is not surprising that girls are more likely to develop internalizing disorders during adolescence (Cohen et al., 1993; Kashani, Orvaschel, Rosenberg, & Reid, 1989), whereas boys are more likely to exhibit externalizing disorders (Kuperman, Schlosser, Lidral, & Reich, 1999). Moreover, research has suggested that family distress and dysfunction may be more strongly related to internalizing disorders for girls but to externalizing disorders for boys during adolescence (Crawford et al., 2001).

Goals of the Present Study

The present study sought to extend this literature by systematically examining the relations between parental psychopathology and adolescent psychopathology, by *both* the gender of the adolescent and the gender of the parent. More specifically, the following research questions were addressed: (a) Does the relationship between parental psychopathology (alcohol dependence and depression) and adolescent psychopathology differ depending on the gender of the parent? (b) Does the gender of the adolescent moderate the relationship between parental psychopathology (alcohol dependence and depression) and adolescent psychopathology? (c) Is the transmission of psychopathology specific to the particular disorder assessed?

Method

Participants

The COGA Project. All of the adolescents and their parents were participants in the ongoing Collaborative Study on the Genetics of Alcoholism (COGA) project (Begleiter et al., 1995). COGA is a large national study that was designed to follow alcohol-dependent men and women, their first-degree relatives, and their extended family members over time. The primary goal of COGA is to uncover susceptibility genes for alcohol dependence. However, the COGA project also provides a rich data set that allows for the examination of other important nongenetic research questions. Data collection occurs at the following sites: the University of Connecticut at Farmington, the State University of New York at Brooklyn, Indiana University in Indianapolis, Washington University in St. Louis, the University of Iowa in Iowa City, and the University of California in San Diego. Data for this specific study were collected at these sites between 1989 and 1994 (Phase I).

Index adult COGA participants were recruited as either probands (alcohol affected individuals) or controls (unaffected individuals). All immediate and extended family members of both the probands and the controls were asked to participate in the study. An average of nine people per family (including first- and second-degree relatives) participated in the COGA project. The adolescents (and many of the parents) in the current study were family members (immediate or extended) of the probands or controls. Of the adolescents in this study, 246 (58%) were from proband families; the remaining were from control families. Due to the family design of COGA, 42% of the adolescents had a sibling and/or first or second cousin participating in the present study. Although the majority of adolescents (58%) did not have another adolescent family member (sibling or cousin) participating, 29% had one participating sibling or cousin, 7% had two participating siblings or cousins, and 6% had three or more participating siblings or cousins (mean number of participating siblings or cousins = .63, *SD* = .98).

Index adult probands were recruited from inpatient and outpatient alcoholism programs. All probands met Feighner (Feighner et al., 1972) criteria for definite alcoholism and *Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised* (*DSM-III-R*; American Psychiatric Association [APA], 1980) criteria for alcohol dependence. Index adult controls were recruited through advertisements, dental and medical clinics, mailings to university students, and driver's license records. None of the controls met the diagnostic criteria for alcohol dependence. Participants were excluded from the study if they had a life-threatening illness, could not speak English, reported recent repeated use of intravenous drugs, or had fewer than five individuals in their immediate family who were willing to participate in the study.

The Sample. The COGA project includes 1,857 families. The present study included only families that had a 13- to 17-year-old adolescent at Phase 1 (n = 666 families). In addition, an inclusion criterion was that adolescents had to live with or have frequent contact (visited them at least once a week) with both their biological mother and their biological father (see Note 1). Four hundred twenty-six families met this criterion. Therefore, the sample for this study included four hundred twenty-six 13- to 17-year-old adolescents (52% girls) and their biological mothers and fathers. The majority of the adolescents lived with their biological parents (85% lived with their biological father, 96% lived with their biological mother, and 82%

lived with both of their biological parents). The mean age of the adolescents was 15.10 (*SD* = 1.42), and most of the adolescents were Caucasian (78%) or African American (17%). The mean level of family income was 5.09 (*SD* = 2.19) on a 9-point scale, ranging from 1 = *less than $10,000 per year* to 9 = *more than $150,000 per year.* This level was the equivalent of $40,000 to $50,000 per year.

Measures and Procedure

All of the Institutional Review Boards at the six COGA sites approved the study protocol. Prior to participating in the study, the adolescents and their parents provided informed assent and consent, respectively. All participants completed a battery of psychosocial surveys, neuropsychological testing, and family history questionnaires. A subset also provided blood samples for genetic linkage analysis. In addition, all participants were administered the *Semi-Structured Assessment for the Genetics of Alcoholism* (SSAGA; Bucholz et al., 1994) by trained research personnel. This measure was the focus of the present study. It is discussed in detail below.

SSAGA. All parents (regardless of whether they were probands, controls, or family members of probands or controls) were administered the adult version of the SSAGA to assess alcohol dependence and depression. The SSAGA is a clinical diagnostic psychiatric interview. It was designed to assess 17 Axis I psychiatric diagnoses and antisocial personality disorder (ASPD). A key feature of the SSAGA is that it allows for psychiatric diagnosis across multiple diagnostic systems. Current and lifetime *DSM-III-R* and *DSM-IV* (*Diagnostic and Statistical Manual of Mental Disorders–Fourth Edition*; APA, 1994) diagnoses from the SSAGA are derived by using computer algorithms. In the present investigation, maternal and paternal lifetime diagnoses of alcohol dependence, depression, and antisocial personality disorder were examined. Previous studies have demonstrated that the SSAGA is a reliable (Bucholz et al., 1994) and valid (Hesselbrock, Easton, Bucholz, Schuckit, & Hesselbrock, 1999) psychiatric diagnostic instrument. The reliability of the SSAGA has been examined in the COGA sample within and across the six centers. Test–retest reliabilities (kappas) range from .65 to .90 and from .74 to 1.00, within centers and across centers, respectively, for the diagnoses examined in the present investigation (Bucholz et al., 1994). The individual items of the SSAGA also demonstrate a high level of internal consistency in the COGA sample (Bucholz et al., 1994).

The SemiStructured Assessment for the Genetics of Alcoholism for Adolescents *(C-SSAGA-A).* The C-SSAGA-A was developed to assess psychiatric problems (using *DSM-III-R* diagnostic criteria) in 13- to 17-year-old adolescents. The format of the C-SSAGA-A is similar to the SSAGA's. Although the C-SSAGA-A includes a range of current and lifetime psychiatric diagnoses, the present study focused on the following lifetime diagnoses: alcohol dependence, marijuana dependence, major depression, anxiety, and conduct disorder. Because only a small minority of adolescents met the diagnostic criteria for the specific anxiety disorders assessed, a "general" anxiety diagnosis was created by collapsing the separation anxiety disorder, obsessive–compulsive disorder, and overanxious disorder diagnoses. The C-SSAGA-A has been shown to have good psychometric properties (Kuperman et al., 2001). For example, in the COGA sample, the test–retest reliability for a diagnosis of alcohol dependence is .86 and the average test–retest reliability for the eight lifetime Axis I non–alcohol dependence diagnoses (across a 1 week period) is .72 (*SD* = .17; Kuperman et al., 2001).

Analysis

A series of logistic regression models was conducted to examine the relations between parental alcohol dependence or depression and adolescent psychopathology. The dependent variables in these models were the adolescent psychiatric diagnoses (alcohol dependence, marijuana dependence, conduct disorder, depression, and anxiety). The independent variables were the parental psychiatric diagnoses (depression and alcohol dependence). Separate models were conducted for each of the adolescent diagnoses.

Because many of the adolescents were from high-risk families, family type (control vs. proband) was entered as a covariate in these analyses. Preliminary analyses (see the Results section) also indicated that parental psychopathology was significantly related to race; therefore, race was included as an additional covariate. Finally, it should be noted that 42% of the adolescents had sibling(s) or cousin(s) also participating in the study. Therefore, some of the cases were not independent of one another. To address this issue, a variable reflecting whether an extended family had more than one participating adolescent was created. This variable (case independence/dependence) was included as a covariate in the regression models to ensure that the nonindependent cases did not systematically differ from the independent cases.

Previous studies also have indicated that parental ASPD may be an important confounding variable to consider when examining the relationship between parental alcoholism and adolescent psychopathology (Moss, Baron, Hardie, & Vanyukov, 2001). Therefore, ASPD was included as a covariate in the logistic regression models (see Note 2).

In each of the logistic regression models, family type, race, case independence, and adolescent gender were entered as covariates in the first block. Next, the parental psychopathology variables were entered, followed by the parental psychopathology × adolescent gender interaction terms. Simple contrast coding was selected so that each category of the predictor variable could be compared to the reference category. The reference categories were as follows: control family for family type, non-Caucasian for race, independence for case independence/dependence, male for gender, and no diagnosis for all of the psychopathology variables.

RESULTS

Preliminary Analyses

Prior to conducting the primary analyses, we conducted analyses to examine whether the parental psychopathology variables systematically varied with the demographic characteristics of the sample. Maternal and paternal depression were not significantly related to the race of the adolescent, $\chi^2(4, N = 396) = 5.80$, p = ns; $\chi^2(4, N = 363) = 5.57$, p = ns, respectively. However, both maternal and paternal alcohol dependence were significantly associated with race, $\chi^2(4, N = 400) = 15.65$, $p < .01$; $\chi^2(4, N = 364) = 12.90$, $p < .05$, respectively. These results indicated that Caucasian adolescents were less likely to have a parent with alcohol dependence in comparison to African American and Hispanic adolescents. None of the parental psychopathology variables were significantly related to the age or the gender of the adolescent.

Adolescent Psychopathology

Table 1 shows the percentage of adolescents who met the diagnostic criteria for each of the psychiatric diagnoses, by the gender of the adolescent. As shown, 6.4% of the adolescents were diagnosed with alcohol dependence, 5.4% were diagnosed with marijuana dependence, 15.0% were diagnosed with conduct disorder, 20.4% were diagnosed with depression, and 12.2% were diagnosed with anxiety. Analyses were conducted to explore whether the adolescent diagnoses significantly differed by gender. Results indicated that boys were significantly more likely to be diagnosed with marijuana dependence, $\chi^2(1, N = 426) = 4.58$, $p < .05$, and conduct disorder, $\chi^2(1, N = 426) = 13.14$, $p < .001$, in comparison to girls.

Parental Psychopathology

Many of the parents also had experienced psychological problems. More specifically, 19.5% (n = 83) of the mothers and 44.8% (n = 191) of the fathers were diagnosed with alcohol dependence, and 39.2% (n = 167) of the mothers and 23.9% (n = 102) of the fathers were diagnosed with depression (see Note 3).

Logistic Regression Analyses

The results of the logistic regression analyses are presented in Table 2. As shown, paternal depression and paternal alcohol dependence significantly predicted adolescent alcohol dependence ($\beta = 1.30$, $p < .05$, odds = 3.67, *c.i.* = 1.11–12.10; $\beta = 2.73$, $p < .01$, odds = 15.37, *c.i.* = 2.92–81.06, respectively). These results indicated that adolescents with a depressed father were three times as likely to be diagnosed with alcohol dependence as those without a depressed father; and those with an alcohol-dependent father were 15 times as likely to develop alcohol dependence in comparison to adolescents with a non–alcohol-dependent father. In contrast, neither maternal depression nor maternal alcohol dependence significantly predicted child alcohol dependence.

Both paternal depression and maternal depression significantly predicted adolescent conduct disorder ($\beta = .81$, $p < .05$, odds = 2.24, *c.i.* = 1.03–4.85; $\beta = .94$, $p < .05$, odds = 2.56, *c.i.* = 1.18–5.59, respectively), indicating that adolescents with a depressed parent were about twice as likely to be diagnosed with conduct disorder in comparison to adolescents without a depressed parent. Paternal depression and maternal depression also significantly predicted adolescent depression ($\beta = .79$, $p < .05$, odds = 2.20, *c.i.* = 1.04–4.68; $\beta = .77$, $p < .05$, odds = 2.16, *c.i.* = 1.03–4.53, respectively). These results similarly suggested that adolescents with a depressed parent were approximately twice as likely to be diagnosed with depression as adolescents without a depressed parent. In contrast, parental alcohol dependence (paternal or maternal) did not significantly predict conduct disorder or depression.

Maternal depression also significantly predicted adolescent anxiety ($\beta = .75$, $p < .05$, odds = 2.12, *c.i.* = 1.02–4.40), in-

TABLE 1
Percentage of Adolescents Meeting Criteria for the Psychiatric Diagnoses

	Girls		Boys		Total	
Diagnosis	***n***	**(%)**	***n***	**(%)**	***n***	**(%)**
Alcohol dependence	12	(5.4)	15	(7.4)	27	(6.4)
Marijuana dependence[a]	7	(3.2)	16	(7.8)	23	(5.4)
Conduct disorder[a]	20	(9.0)	44	(21.6)	64	(15.0)
Depression	37	(22.0)	29	(18.6)	66	(20.4)
Anxiety	31	(14.0)	21	(10.3)	52	(12.2)

[a]Denotes a significant gender difference.

TABLE 2
Logistic Regression Models Predicting the Adolescent Psychiatric Diagnoses from the Parental Psychiatric Diagnoses

	Adolescent diagnosis									
	Alcohol		Marijuana		Conduct		Depression		Anxiety	
Variable	β	*SE*	β	*SE*	β	*SE*	β	*SE*	β	*SE*
Family type	−1.66*	.65	−1.24	.76	−.26	.40	.18	.45	−.08	.42
Case independence	.71	.58	.62	.58	−.11	.36	−.24	.40	−.81*	.40
Race	.40	.88	2.06	1.20	−.04	.48	−.26	.47	.49	.53
Adolescent gender	.46	.97	−4.02	50.24	−.98*	.45	−.29	.49	.33	.47
Paternal antisocial personality disorder	−.42	.84	1.42	.80	.17	.49	−.15	.56	.12	.53
Paternal depression	1.30*	.61	.58	.74	.81*	.39	.79*	.39	.43	.38
Maternal depression	.68	.61	4.72	16.91	.94*	.40	.77*	.38	.75*	.37
Paternal alcohol dependence	2.73**	.85	5.58	17.43	.69	.42	.56	.43	.56	.41
Maternal alcohol dependence	.39	.74	−5.25	24.30	.75	.43	.47	.49	.32	.46
Adol. gender × maternal depression	.35	1.19	9.26	33.82	.17	.78	.78	.74	.53	.72
Adol. gender × paternal depression	2.00	1.24	−.16	1.45	.66	.78	.18	.77	-.68	.75
Adol. gender × maternal alcohol dependence	2.07	1.48	9.54	48.54	.86	.83	−1.15	.94	.64	.91
Adol. gender × paternal alcohol dependence	−.32	1.58	8.15	34.84	−.23	.76	−1.13	.77	−.12	.73
R^2		.27		.35		.18		.15		.10
Model X^2		30.36**		40.45***		35.54**		23.77*		17.96

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

dicating that adolescents with a depressed mother were twice as likely to be diagnosed with an anxiety disorder as those without a depressed mother. None of the parental psychopathology variables significantly predicted adolescent marijuana dependence. In addition, none of the adolescent gender × parental psychopathology interaction terms were significant.

DISCUSSION

One goal of the present study was to examine whether the vulnerability to psychopathology is differentially transmitted depending on the gender of the parent. Results indicate that the gender of the parent is an important factor that should not be overlooked when examining the familial transmission of psychopathology. For example, in the present study, paternal psychopathology (alcohol dependence and depression) predicted adolescent alcohol dependence, but maternal psychopathology did not. There was a particularly strong link between paternal alcohol dependence and adolescent alcohol dependence, indicating that adolescents with an alcohol-dependent father were 15 times more likely to develop alcohol dependence in comparison to adolescents without an alcohol-dependent father. This finding is consistent with research demonstrating a high heritability (genetic influence) for the development of alcoholism (Prescott, 2002; Schuckit, 2000). Given this particularly robust finding for fathers, it is noteworthy that maternal alcohol dependence did not predict any of the adolescent psychiatric diagnoses. However, recent behavioral genetic and genetic epidemiological studies suggest that the heritability of alcoholism may differ by gender, with more consistent effects being observed for men in comparison to women (Prescott, 2002; Walters, 2002). Environmental influences also may account for the gender differences observed for mothers and fathers. Perhaps adolescents are more affected by their father's drinking than by their mother's drinking. For example, research has suggested that men tend to become more aggressive and violent when they drink in comparison to women (Schuckit, Daeppen, Tipp, Hesselbrock, & Bucholz, 1998). Therefore, differences in aggressive/violent behavior may partially account for the gender differences observed in relation to alcohol dependence. Female alcoholics also have been noted to be less likely to experience legal, personal, and employment-related problems in comparison to male alcoholics (Schuckit et al., 1998). In addition, female alcoholics tend to seek help earlier for their drinking problem than male alcoholics (Schuckit et al., 1998). In essence, maternal alcoholism may simply be less disruptive to the family than paternal alcoholism.

Consistent with the literature (Downey & Coyne, 1990; Fergusson & Lynskey, 1993), parental depression also predicted adolescent psychopathology in the present study. As mentioned previously, the majority of studies examining the transmission of depression have focused on mothers. Therefore, it is noteworthy that paternal depression also was found to consistently predict adolescent psychopathology. More specifically, in the present study, both paternal and maternal depression significantly predicted adolescent depression and conduct disorder. Paternal depression also significantly predicted adolescent alcohol dependence, and maternal depression significantly predicted adolescent anxiety. These results underscore the importance of examining both maternal and paternal factors when examining offspring psychopathology.

Results from the present study are strikingly consistent with those found in Luthar et al.'s (1993) study of families of opiod and cocaine abusers. Similar to findings from the present study, Luthar et al. found maternal depression, but not maternal alcoholism, to be consistently associated with offspring psychopathology. In addition, paternal alcohol dependence, but not paternal depression, was related to psychopathology in the offspring—but only to offspring substance abuse (as was found in the present study). The reason maternal depression, but not maternal alcoholism, was related to offspring psychopathology in these studies may reflect differences in the severity of the disorders (Luthar et al., 1993). Perhaps mothers are more severely affected by depression than by alcoholism. It also is possible that the differential findings for maternal depression and maternal alcoholism are due to a statistical artifact relating to power because maternal depression is more common than maternal alcohol dependence (in the present study, 167 mothers were diagnosed with depression, whereas 83 mothers were diagnosed with alcohol dependence). However, power issues do not appear to completely account for the differences observed for maternal depression and maternal alcohol dependence, as the βs and the odds ratios were consistently higher in the maternal depression models in comparison to the maternal alcohol-dependence models. Nevertheless, future studies including equal numbers of depressed mothers and alcohol-dependent mothers should be conducted to further address this issue.

As noted previously, paternal depression was consistently associated with offspring psychopathology in the present study. In contrast, paternal depression was not related to any of the psychiatric diagnoses assessed in the Luthar et al. (1993) study (depression, alcoholism, drug abuse, and ASPD). This difference may reflect differences in samples. Luther et al.'s sample included only adults, whereas the sample for the present study included only adolescents. Because adolescents are more likely to still be living at home with their families in comparison to adults, they are more likely to directly experience the effects of their fathers' psychopathology (e.g., on a daily basis). Subsequently, because of their more frequent exposure to their fathers' depressed behavior and the effect that it has on the family, adolescents also may be more likely than adults to be deleteriously affected by their fathers' psychopathology.

Another goal of this study was to examine whether the gender of the adolescent moderated the relationship between parental psychopathology (alcohol dependence and depression) and adolescent psychopathology. To address this issue, the statistical interactions between the parental psychopathology variables and the gender of the adolescent were examined. None of these interactions were significant. This null finding conflicts with current developmental theory (e.g., Gilligan, 1983) and recent research examining adolescent gender differences that indicate that girls are more interpersonally oriented than boys and, subsequently, more likely to be affected by family problems (including those that may result from parental psychopathology) than are boys (Gore et al., 1993). However, it should be noted that in the present study, adolescent gender effects were explored via statistical interaction terms. Therefore, the lack of significant findings pertaining to adolescent gender may be due to a relative lack of statistical power because more power is needed to detect significant interactions than significant main effects (Aguinis & Stone-Romero, 1997). Moreover, power tends to be further compromised if there is an unequal proportion of cases in the moderator (gender) groups, as was sometimes the case in the present study (e.g., 12% of girls and 28% of boys were diagnosed with conduct disorder). In essence, the examination of moderating effects through the use of interaction terms is likely to increase the risk of committing a Type II error, which may have occurred in this study. Therefore, it would be important for future research to attempt to replicate and validate the null results relating to adolescent gender with a larger sample.

In the present study, the transmission of psychopathology was not found to be specific to the particular disorder assessed (e.g., parental depression was not only related to offspring depression but also to offspring conduct disorder, alcohol dependence, and anxiety). Nevertheless, it is interesting to note that, in general, paternal psychopathology was more consistently related to externalizing disorders, whereas maternal psychopathology was more consistently related to internalizing disorders. These results are compatible with research that has shown paternal characteristics to be more strongly linked to offspring externalizing disorders than to internalizing disorders (Phares & Compas, 1992).

Although many intriguing results emerged in the present study, several caveats should be mentioned. One such caveat relates to the uniqueness of the sample assessed. All participants were involved in the COGA project. Whereas the COGA study design allows for the appropriate investigation of genetic hypotheses, the sampling strategy does not always provide ideal subsamples for the examination of other (e.g., nongenetic) hypotheses. In the present study, some of the adolescents were members of high-density alcoholic families, others had only an affected parent, and still others had no biological relatives affected with alcohol dependence. Not surprisingly, this sample had relatively high rates of psychopathology. Therefore, this sample is not representative of the general population. However, it should be noted that high rates of psychopathology are nec-

essary to obtain an appropriate level of statistical power needed to examine questions relating to the familial transmission of psychopathology.

It also should be noted that only adolescents who lived with or had frequent contact with both their biological mother and biological father were included in the present study. This criterion was necessary to ensure that offspring who had parents with alcohol dependence and/or depression experienced both the genetic and environmental effects of having a parent with psychopathology. However, as a result of this inclusion criterion, the percentage of adolescents living with or having frequent contact with their biological parents is greater in comparison to the general population. It also should be noted that the sample was primarily middle class Caucasian and included families who had rather close connections within the family. Therefore, the results of this study should be generalized with caution.

In addition, although the primary goal of the present study was to examine the relationship between parental psychopathology and adolescent psychopathology by gender, the more conceptually interesting hypotheses relating to the underlying factors involved in the relationship between parental psychopathology and adolescent psychopathology could not be explored. It is hoped that results from the present study will be used as a springboard for future investigations. For example, it would be important to examine *why* parental psychopathology is differentially related to offspring psychopathology, depending on the gender of the parent. It is not clear whether the differential relations that were observed in the present study are due to genetic factors, environmental factors, or both. Recent research suggests they are likely due to an *interaction* between genes and the environment. For example, in a study examining adolescent depression, Jacobson and Rowe (1999) found that the relationship between family connectedness and depressed mood was genetically mediated, particularly for girls. Future behavioral genetic studies need to be designed to further examine the link between parental psychopathology and offspring psychopathology, by the gender of the parent and the gender of the offspring.

Another limitation of the present study is that the direction of effect between parental psychopathology and adolescent psychopathology could not be addressed due to cross-sectional design and the use of lifetime psychiatric diagnoses. Longitudinal studies are needed to explore how parental psychopathology is related to offspring psychopathology as both offspring and parents progress through different developmental stages. Assessing offspring across time even within a particular developmental period, such as adolescence, would be valuable. For example, it would be important to follow offspring of alcoholic parents and offspring of depressed parents throughout the adolescent period as they confront different developmental tasks. It may be that early adolescence is an especially vulnerable period due to the sheer number of changes that occur within both the individual and his or her contexts. Longitudinal research also is needed to examine the influence that adolescent psychopathology has on parental psychopathology and the likelihood that both adolescent and parental psychopathology influence one another.

In light of these limitations, the present investigation contributed to the literature in many respects. One strength of this study is that it specifically focused on the relationship between parental psychopathology and *adolescent* psychopathology, a relatively neglected area in the literature examining offspring of alcoholics and offspring of depressed parents. Moreover, multiple psychiatric disorders were assessed in adolescents and their parents, including both internalizing and externalizing disorders. Most important, the influence of *both* the gender of the parent and the gender of the adolescent on this relationship also was *simultaneously* assessed, while taking parental comorbid psychopathology into account. Finally, the present study extended the findings of many previous studies that have assessed self-reported parent and offspring psychiatric symptomatology because psychopathology was assessed using clinically diagnostic, objective measures based on *DSM-III-R* and *DSM-IV* criteria. It is hoped that future research will replicate and extend the results from the present investigation by examining the relationship between parental psychopathology and adolescent psychopathology *over time*. Moreover, a critical step for future research will be to examine the *underlying mechanisms* involved in the relationship between parental psychopathology and adolescent psychopathology and the manner in which these mechanisms may differ by gender.

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Authors' Notes

1. The Collaborative Study on the Genetics of Alcoholism (COGA) (Principal Investigator: H. Begleiter; Co–Principal Investigators: L. Bierut, H. Edenberg, V. Hesselbrock, B. Porjesz) includes nine different centers where data collection, analysis, and storage take place. The nine sites and Principal Investigators and Co-Investigators are University of Connecticut (V. Hesselbrock); Indiana University (H. Edenberg, J. Nurnberger Jr., P.M. Conneally, T. Foroud); University of Iowa (S. Kuperman, R. Crowe); SUNY HSCB (B. Porjesz, H. Begleiter); Washington University in St. Louis (L. Bierut, A. Goate, J. Rice); University of California at San Diego (M. Schuckit); Howard University (R. Taylor); Rutgers University (J. Tischfield); Southwest Foundation (L. Almasy). Zhaoxia Ren serves as the NIAAA Staff Collaborator. This national collaborative study is supported by NIH Grant U10AA08401 from the National Institute on Alcohol Abuse and Alcoholism (NIAAA) and the National Institute on Drug Abuse (NIDA).
2. In memory of Theodore Reich, MD, co–principal investigator of COGA since its inception and one of the founders of modern psychiatric genetics; we acknowledge his immeasurable and fundamental scientific contributions to COGA and the field.

Notes

1. Adolescents who lived with or had frequent contact with (visited at least once a week) both their biological mothers and biological fathers were compared to adolescents who did not live with or have frequent contact with at least one of their biological parents on the demographic variables. No significant differences were found for age or gender. However, adolescents who lived with or had frequent contact with both biological parents were significantly more likely to be Caucasian than non-Caucasian in comparison to those who did not live with or have frequent contact with their biological parents.
2. Maternal ASPD was not included because there was an extremely low prevalence of ASPD in the mothers ($n = 7$; 1.6%).
3. Because COGA is an *extended* family study, some of the alcoholic fathers (74%) and mothers (87%) were from proband families, whereas other alcoholic fathers (26%) and mothers (13%) were from control families. The alcoholic fathers and mothers from control families were relatives of index control participants (who did not have a diagnosis of alcohol dependence).

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