

## Maternal Depression, Paternal Psychopathology, and Adolescent Diagnostic Outcomes

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The authors examined the relationship between maternal depression, paternal psychopathology, and adolescent diagnostic outcomes in a community sample of 522 Australian families. They also examined whether chronic family stress, father's expressed emotion, and parents' marital satisfaction mediated the relationship between parental psychopathology and adolescent outcomes. Mother's education, child's gender, and family income were covaried in all analyses. Results revealed that maternal depression and paternal depression had an additive effect on youth externalizing disorders. In addition, maternal depression interacted with both paternal depression and paternal substance abuse in predicting youth depression but not youth nondepressive disorders. Chronic family stress and father's expressed emotion appeared to mediate the relationship between parental psychopathology and youth depression.

There is a large body of research documenting an association between maternal depression and the presence of behavior problems in offspring. In particular, children of depressed parents have been found to be more likely to be depressed themselves (Downey & Coyne, 1990; Orvaschel, Walsh-Allis, & Ye, 1988; Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997). Children whose mothers are depressed also have been found to be at a greater risk for developing a wide range of behavior problems and psychopathology (Beardslee, Versage, & Gladstone, 1998; Cummings & Davies, 1994; Hammen, Burge, Burney, & Adrian, 1990; Orvaschel et al., 1988).

One area of crucial importance that has been overlooked in much of the literature on maternal depression and child psycho-

pathology is the role fathers play in the development of behavior problems in these high-risk groups (e.g., Phares & Compas, 1992)—both in terms of paternal disorders and quality of father-child relationships. Recently, research on the father's role in family functioning has increased significantly. However, much of the research on the father has focused on the father's contributions to normal child development and has ignored the possible relationship between paternal psychopathology and child behavior outcomes. A review by Phares and Compas (1992) found that between 1984 and 1991 only 26% of 577 empirical studies on psychopathology in parents and the effects on their children examined both maternal and paternal influences on psychopathology. Only a few studies of maternal depression have included specific reports of father psychopathology as well (e.g., Atkinson & Rickel, 1984; Beardslee, Schultz, & Selman, 1987; Merikangas, Prusoff, & Weissman, 1988).

Goodman and Gotlib (1999) distinguished among several ways in which fathers may contribute to the development of behavior problems in children with depressed mothers. One possibility is that a child's warm, consistent relationship with the father may serve as a protective factor for some of the negative effects of maternal depression. Previous studies have found support for this buffering role of the father in families with depressed mothers (Tannenbaum & Forehand, 1994; Thomas, Forehand, & Neighbors, 1995).

Another possibility is that paternal psychopathology increases the negative effect of maternal depression, such that children with depressed mothers and psychiatrically ill fathers would display more behavior problems than would children with depressed mothers and psychiatrically normal fathers. In support of this hypothesis, studies of offspring of two affected parents have found that these children may be at a significantly greater risk for the development of problems than are offspring who have only one or no affected parents (Dierker, Merikangas, & Szatmari, 1999; Meri-

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kangas, Dierker, & Szamari, 1998; Weissman et al., 1984). However, as Merikangas et al. (1998) noted, remarkably few high-risk studies have considered the role of the coparent in the connection between parental and child psychopathology.

The role of the father's diagnosis in predicting behavior problems in children becomes increasingly complex when one considers the relationship between depressed mothers and their spouses. According to the concept of assortative mating, depressed people tend to choose mates who also have a psychiatric illness or disorder or a family history of disorder and illness (Merikangas, 1984). Hence, children with depressed mothers may be at a greater risk for developing behavior problems both through a direct mechanism of transmission of maternal depression and, more indirectly, through psychopathology in the father, who may be more likely to have a disorder if the mother is depressed.

The interaction between maternal depression and the particular paternal diagnosis of depression may be important to consider when examining the outcome of depressive diagnoses in offspring. It has been noted that concordance for depression in married couples is quite high (Merikangas, Weissman, Prusoff, & John, 1988). In cases where both parents are depressed, there is some evidence for increased risk of offspring depressive outcomes. For example, in 1982 Goodwin found that whereas 15% of children who had one depressed parent evidenced depression themselves, this figure rose to 40% in cases where both parents were depressed. More recent studies that have examined youth outcomes in relation to the interaction of maternal and paternal depression have yielded mixed results. For example, whereas postpartum depression in mothers and fathers was found in one study to interact in the prediction of internalizing problems in young children (Carro, Grant, Gotlib, & Compas, 1993), in another study parental concordance for anxiety and affective disorders did not significantly increase rates of anxiety and affective disorders beyond that which was found in families with only one affected parent (Dierker et al., 1999). Additional research is necessary to gain more conclusive findings on how the presence of depression in both parents may be related to risk for depression in offspring.

Substance abuse is the second type of paternal disorder that may be of particular importance to consider in interaction with maternal depression. Again, depression in wives is associated with this particular disorder in husbands. In fact, Dierker et al. (1999) found the combination of maternal depression and anxiety and paternal substance abuse to be the most commonly occurring assortative mating pattern in their sample. In a recent meta-analysis of the contributions of maternal and paternal psychopathology to behavior problems in offspring, Connell and Goodman (2001) highlighted the relative infrequency of studies that consider such an interaction, and they suggested the importance of examining paternal substance abuse in particular. Results of the meta-analysis suggested that maternal depression is a stronger predictor than paternal depression and paternal substance abuse is a stronger predictor than maternal substance abuse of behavior problems in offspring. The contribution of the interaction of maternal depression and paternal substance abuse to this pattern of findings remains unknown. Connell and Goodman suggested that when studies have reported on the behavior problems seen in offspring with depressed mothers or substance-abusing fathers, they may in fact have been reporting on the same subpopulation, which may be exposed to a unique set of risk factors due to their dually affected

parents. These reviewers concluded that future studies should look more closely at the interaction between maternal depression and paternal substance abuse in relation to problematic youth outcomes.

It is important to examine both additive and interactive effects in the relationship between maternal depression, paternal psychopathology, and adolescent outcomes. In assessing for additive effects, one examines whether paternal psychopathology adds to the risk for negative outcomes above and beyond the effect of maternal depression on its own. In assessing for interactive effects, one examines whether the effect of maternal depression on adolescent outcome differs depending on the presence or absence of paternal psychopathology.

As stated earlier, children of depressed mothers are at risk not only for depressive outcomes but also for developing a wide range of internalizing and externalizing disorders (Downey & Coyne, 1990). In addition, whereas internalizing outcomes may be more tied to maternal psychopathology than to paternal psychopathology, externalizing outcomes appear to be related to both maternal and paternal diagnostic status (Connell & Goodman, 2001). For these reasons, it is important to examine the effects of maternal and paternal psychopathology on nondepressive internalizing and externalizing outcomes as well as on the specific diagnostic outcome of youth depression.

Given the paucity of research in this area, it is useful to examine whether maternal depression and paternal psychopathology have additive or interactive effects in relation to youth diagnostic outcomes. If such effects are noted, an important secondary question is how (or through what mechanisms) might this combination of parental disorders affect these youth outcomes? Clearly one possible mechanism is through genetic transmission. In addition, social or contextual mechanisms may help to explain how the combination of maternal depression and paternal psychiatric illness increases risk for negative youth outcomes. For example, the quality of the father-child interaction might be disrupted in cases where the father suffers from depression or substance abuse and the mother also suffers from depression. This disrupted father-child relationship might explain increased risk for youth diagnostic outcomes in these families. In a similar manner, the quality of the parents' marital relationship might be disrupted in cases of mother depression and concomitant father psychopathology. The negative marital interactions might be responsible for the increased risk of youth problems in these families. A family environment in which both parents suffer from a psychiatric disorder would likely be an extremely stressful environment for the child. General levels of family environment stress might be the mechanism through which maternal and paternal psychopathology increase the likelihood of youth diagnostic disorders. Previous research has found that disrupted parent-child relations (Cox, Puckering, Pound, & Mills, 1987; Harnish, Dodge, & Valente, 1995), marital conflict (Emery, Weintraub, & Neale, 1982; Fergusson, Horwood, & Lynskey, 1995), and family stress (Billings & Moos, 1983; Fendrich, Warner, & Weissman, 1990; Hammen, Burge, & Adrian, 1991) all act as mediators in the maternal depression and child outcome relationship. To date, no one has extended the examination of these potential mediators to the specific cases of families in which the father also suffers from a psychiatric disorder.

The present study sought to examine further the role of the father in the relationship between maternal depression and adoles-

cent diagnostic outcomes. In addition to examining the overall question of whether paternal depression or substance abuse has an additive or moderating effect on maternal depression in the prediction of youth outcomes, we also explored several potential mediators for this effect. Specifically, we tested whether (a) mothers with depression would be more likely to partner with men who had a history of depression or substance abuse disorders; (b) maternal depression and paternal psychiatric history would have additive and/or interactive effects on adolescent diagnostic outcomes, such that youths with both a depressed mother and a depressed or substance-abusing father would be more likely than the other youths in the sample to evidence depressive or other psychiatric disorders; (c) levels of family stress and marital conflict would be higher and the quality of the father-child relationship would be lower for those youths who had both a depressed mother and a depressed or substance-abusing father than it would be for other youths in the sample; (d) family stress, marital conflict, and father-child relations would act as mediators in the relationship between parental psychopathology and youth diagnostic outcomes.

## Method

### Participants

Participants were 522 families with children born between 1981 and 1984 at Mater Misericordiae Mother's Hospital in Brisbane, Queensland, Australia. The sample for the present study was drawn from a larger birth cohort ( $N = 7,775$ ) established in the context of the Mater University of Queensland Study of Pregnancy (MUSP; Keeping et al., 1989). The purpose of MUSP was to examine children's physical, cognitive, and psychological health as a function of pregnancy and obstetric conditions and psychosocial history. The MUSP cohort was predominantly Caucasian (92%), lower middle or working socioeconomic status. Mothers in the MUSP study completed interviews and questionnaires about themselves and their children at five different times: during pregnancy, 3 to 4 days after the birth of their child, 6 months after the birth of the child, when the child was 5 years old, and when the child was 14 years old.

### Sample Selection

In the context of MUSP, the mothers in the cohort completed self-report depression questionnaires during their pregnancy, 3 to 4 days after the birth of the child, when the child was 6 months old, and when the child was 5 years old. Data from these questionnaires were used to select a sample that included a large number of women with a history of depressive symptoms (varying in chronicity and severity) and a comparison sample of women who had no or few depressive symptoms. Further details concerning the selection of this high-risk sample were provided in Hammen and Brennan (2001).

High-risk and comparison families were contacted and asked to participate in a follow-up when their children were 15 years of age. They were informed that the purpose of the study was an examination of the relationship between maternal psychological and emotional functioning and youth behavioral and mental health outcomes. Families were included in the study if the mother and the child agreed to the interview; fathers and stepfathers were included where available, including a small number of cases of divorced parents in which the father had substantial contact with the youth (parental divorce status was unrelated to youth diagnostic status). All biological fathers who currently lived in the area were invited to participate in the interview, as were stepfathers who had lived with the child for 5 years or longer.

From the sample still available for follow-up (5,277, or 68% of the original MUSP sample), 991 families were targeted for inclusion in the present study. Of the 991, 816 consented and were included (82%); 68 families could not be located, 103 declined to participate in this phase, 3 included a child with a hearing or visual impairment that precluded participation, and 1 child had died. Children in the high-risk subsample ( $n = 816$ ) were not significantly different from the original birth cohort in terms of gender,  $\chi^2(1, N = 7,775) = 0.53, p = .48$ ; income,  $t(7147) = 0.81, p = .42$ ; or mother's education,  $t(7612) = 1.70, p = .09$ .

Of the 816 youth in the age 15 follow-up, 522 participated with both their mother and father in the in-home interview. These 522 youth and their parents composed the sample for the present study. Families in which only the mother and the youth were interviewed were excluded from the study because they lacked father self-reported psychiatric background, marital satisfaction, and expressed emotion. Excluded families were not significantly different from the high-risk subsample in terms of child gender,  $\chi^2(1, N = 816) = 0.17, p = .68$ , or mother's education,  $t(809) = 0.52, p = .60$ . Excluded families did have lower family incomes,  $t(765) = 5.48, p < .01$ , and higher rates of maternal depression (70% vs. 56%),  $\chi^2(1, N = 814) = 18.18, p < .01$ , than those families included in the final sample in this study. It is possible then that our results may reflect an underestimate of the effect of maternal depression and lower family income on adolescent diagnostic outcomes.

### Sample Characteristics

There were 262 boys and 260 girls, with a mean age of 15 years, 2 months ( $SD = 0.27$ ). The overall sample was 92% Caucasian, median family income was AU\$35,000–\$45,000, median mothers' education was Grade 10 (approximately equivalent to a U.S. high school graduate), and the mothers' mean age at the time of the youth's birth was 25.58 years ( $SD = 5.03$ ). Of the 522 fathers who were interviewed, 454 (87%) were the biological parents of the youth, 483 (93%) lived in the home with both the mother and the youth, and 501 (96%) reported that the youth lived with them at least some of the time.

### Procedure

Interviews were conducted in the homes of the families. Interviewers were blind to the mother's depression status or history, and a team of two interviewers conducted the parent and child interviews separately and privately. Between interviews, the participants also completed a battery of questionnaires. The mother, child, and father gave written informed consent (assent) and were paid for their participation, which lasted approximately 3.5 hr.

### Diagnostic Measures

**Maternal and paternal depression.** Mothers and fathers were classified as depressed if they qualified for a lifetime diagnosis of major depression or dysthymia on the Structured Clinical Interview for the *DSM-IV* (SCID; First, Spitzer, Gibbon, & Williams, 1995). In the 522 families, 200 mothers and 133 fathers were classified as depressed.

**Paternal substance abuse.** Fathers were classified as having a history of substance abuse if they qualified for alcohol abuse, alcohol dependence, drug abuse, or drug dependence diagnoses on the SCID. In the 522 families, 94 fathers were classified as such.

**Paternal diagnostic comorbidity.** A total of 37 fathers obtained criteria for diagnoses of both substance abuse and depression during their lifetime. Secondary analyses applying statistical controls for father diagnostic comorbidity yielded highly similar results to those presented.

**Timing of parental diagnosis.** In the majority of the cases of parental diagnoses (75% of the fathers' diagnoses and 93% of the mothers' diagnoses) the disorder was ongoing during the life of the child. Secondary



analyses examining only the effect of parental diagnoses occurring during the child's lifetime yielded highly similar results to those presented here.

**Youth depression.** Youths were classified as depressed if they met criteria for a lifetime diagnosis of major depression or dysthymia on the Schedule for Affective Disorders and Schizophrenia for School-Age Children—Revised for *DSM-IV* (K-SADS-E; Orvaschel, 1995). The instrument is a semistructured interview administered by trained clinical interviewers covering current and lifetime disorders. It is administered separately to the parent and the child; diagnostic decisions were reviewed by the clinical rating team with judgments made on the basis of all available information. Of the 522 youths in the study, 69 were classified as depressed.

**Youth nondepressive disorders.** Youths were classified as having a nondepressive externalizing disorder if they met criteria for attention-deficit/hyperactivity disorder ( $n = 38$ ), conduct disorder ( $n = 10$ ), oppositional-defiant disorder ( $n = 15$ ), or substance use disorder ( $n = 7$ ). They were classified as having a nondepressive internalizing disorder if they met criteria for anxiety disorders exclusive of specific and social phobia ( $n = 51$ ) or for eating disorders ( $n = 6$ ). Children may have had more than one diagnosis. There were 59 children classified as having nondepressive externalizing disorders; 24% of these youths had a diagnosis of depression as well. There were 40 children classified as having a nondepressive internalizing disorder; 45% of these youths were diagnosed with a depressive disorder as well.

**Reliability of diagnoses.** To determine interrater reliability for diagnoses, we randomly selected approximately 10% of the audiotaped SCID and K-SADS-E interviews to be scored by another clinician, who was blind to participants' diagnoses. Kappa coefficients for the SCID were .84 for maternal depression, .91 for paternal depression, and .92 for paternal substance abuse.

On the K-SADS-E the weighted kappa for youth depression was .73. Kappas for anxiety disorders, substance use disorders, disruptive disorders (conduct disorder or oppositional-defiant disorder), and disorders classified as other (primarily eating disorders) ranged from .72 to 1.0, with a mean of .81.

### Family Functioning Measures

**Chronic family stress (youth report).** To evaluate adolescents' experience of stress in the family, Hammen and colleagues developed a semistructured interview for adolescents from earlier versions of chronic strain-functioning measures for children (e.g., Hammen, 1991) and adults (e.g., Hammen et al., 1987). The adolescent version used in the age 15 follow-up consisted of six domains: social life, close friendship, romantic relationships, academic performance, school behavior, and relations with family members (the domain of focus in the present study). Interviewers probed each area with the youth, using standard general probes and follow-up queries where needed. Each domain was scaled on a 5-point scale with behaviorally specific anchors of 1 (*superior functioning*) and 5 (*severe difficulties*). Reliabilities were based on independent judges' ratings of audiotaped interviews, with sample sizes between 88 and 96 for individual items. The intraclass correlation for youth chronic stress experienced in their relationship with family members was .84.

**Father's expressed emotion.** In the course of the in-home interview, fathers completed the Five-Minute-Speech Sample (FMSS; Magana et al., 1986), which provides a measure of parental attitudes toward the child that may be represented as high or low expressed emotion (EE). In the FMSS, fathers were seated in front of an audiotape recorder and asked to speak unprompted and without interruption for 5 min about their adolescent child and how they get along together. The great majority of the fathers completed this task without a need for any prompts from the interviewer. Seven percent of the fathers were silent for a period and required prompts to continue speaking; however, rates of silence and prompts did not differ by paternal diagnostic group.

FMSS recordings were then coded by raters blind to all other information about the parents and their children as reflecting either low or high EE on the basis of criteria developed and validated by Magana et al. (1986). A categorical rating of high EE is based on a clear presence of either criticism or emotional overinvolvement in the content of the father's speech. Approximately 12% of the fathers were rated as high EE in this sample.

Raters for the study were trained by the group that developed the FMSS procedure. Interrater reliability of the raters with an expert from that group yielded EE criticism kappas ranging from .63 to .82 and EE emotional overinvolvement kappas ranging from .86 to 1.00 across raters. In this sample, FMSS data were not available for 39 fathers because of technical difficulties or inadequate sound quality.

**Marital conflict.** The Satisfaction subscale of the Dyadic Adjustment Scale (DAS; Spanier, 1976) was administered. Low satisfaction represents high frequency of quarrels, discussions of separation, and negative interactions. This subscale has been found to have high levels of reliability and validity and is useful as a measure of overall relationship quality (Kurdek, 1992). The scale was administered to both the mother and father, and these two reports were highly correlated ( $r = .93, p < .01$ ). The mean score on these reports was used as the measure of parental marital satisfaction in this study.

### Results

Mother's education, child's gender, and family income were included as statistical controls in all analyses. These particular controls were applied as they are commonly noted potential confounds in the literature on maternal depression and child outcome.

#### Paternal Psychopathology and Maternal Depression

Logistic regression analyses revealed that maternal depression was significantly related to paternal depression,  $\chi^2(1, N = 522) = 4.47, p = .03$ , but not to paternal substance abuse,  $\chi^2(1, N = 522) = 0.06, p = .81$ . Depressed mothers were more likely than nondepressed mothers to have a partner with a history of depression (31% vs. 22%), but they were not more likely to have a partner with a history of substance abuse (19% vs. 18%).

#### Main Effects of Maternal Depression and Paternal Psychopathology

Table 1 presents the results of preliminary logistic regression analyses examining the main effects of maternal depression, paternal depression, and paternal substance abuse disorders on youth diagnostic outcomes. As can be seen in the table, maternal depression was related to all types of youth diagnostic outcomes; however, paternal depression was only related to youth externalizing disorders, and paternal substance abuse was not significantly related to any youth diagnostic outcomes.

#### Additive and Interactive Effects of Maternal Depression and Paternal Psychopathology

Logistic regression analyses were also used to examine potential additive and interactive effects of maternal depression and paternal psychopathology in relation to youth diagnostic outcomes. Three types of youth diagnostic outcomes were examined—youth depression, youth nondepressive externalizing disorders, and youth nondepressive internalizing disorders. In these analyses, statistical controls, maternal depression, and paternal psychiatric status were

Table 1  
*Parental Psychopathology and Youth Diagnostic Outcomes*

Youth and parent diagnosis	Youth diagnosis		Logistic regression results	
	Parental disorder (%)	No parental disorder (%)	$\chi^2(1, N = 522)$	<i>p</i>
Youth depression				
Maternal depression	19	10	7.50	<.01
Paternal depression	16	12	0.40	.53
Paternal substance abuse	18	12	2.45	.12
Youth nondepressive externalizing disorders				
Maternal depression	19	7	15.82	<.01
Paternal depression	20	8	13.34	<.01
Paternal substance abuse	15	11	0.66	.42
Youth nondepressive internalizing disorders				
Maternal depression	12	5	5.55	<.05
Paternal depression	11	7	1.33	.25
Paternal substance abuse	13	7	2.94	.09

Note. The comparison group *ns* were as follows: 200 for maternal depression and 322 for no maternal depression, 133 for paternal depression and 389 for no paternal depression, and 94 for paternal substance abuse and 428 for no paternal substance abuse.

entered as a block in the first step (to assess for additive effects), and the significance of Maternal Depression  $\times$  Paternal Psychiatric Status was assessed in the second and final step.

**Youth depression.** Table 2 presents the logistic regression results for the outcome of youth depressive disorders. As can be seen in the table, there were no additive effects of maternal depression and paternal psychopathology on youth depression. However, maternal depression did interact with father depression in the prediction of youth depression. The percentage of youths with a depression diagnoses in each comparison group is presented in the top of Figure 1. Contrary to our hypotheses, post hoc chi-square analyses revealed that in cases where the father was not depressed, maternal depression was significantly related to youth depression,  $\chi^2(1, N = 389) = 12.50, p < .01$ , whereas in cases where the father was

depressed, the relationship between maternal depression and youth depression was nonsignificant,  $\chi^2(1, N = 133) = 0.14, p = .71$ .

Maternal depression was also found to interact with father substance abuse in the prediction of youth depression. As can be seen in the bottom half of Figure 1, the hypothesized pattern of results was noted. Post hoc chi-square analyses revealed that in cases where the father had no diagnosis of substance abuse, the relationship between maternal depression and youth depression was nonsignificant,  $\chi^2(1, N = 428) = 1.74, p = .19$ , whereas in cases where the father had a diagnosis of substance abuse, maternal depression was significantly related to youth depression,  $\chi^2(1, N = 94) = 11.20, p < .01$ .

**Youth nondepressive externalizing diagnoses.** Table 3 presents the logistic regression results for youth nondepressive externalizing disorders. As can be seen, paternal depression and maternal depression had an additive effect on youth nondepressive externalizing disorders (as displayed in Figure 2); however, no other additive or interactive effects were significant.

**Youth nondepressive internalizing diagnoses.** Table 4 presents the results of logistic regression analyses examining the potential additive and interactive effects of maternal depression and paternal psychopathology on youth nondepressive internalizing disorders. As can be seen from the table, there were no significant additive or interactive effects of parental psychopathology on these youth diagnostic outcomes.

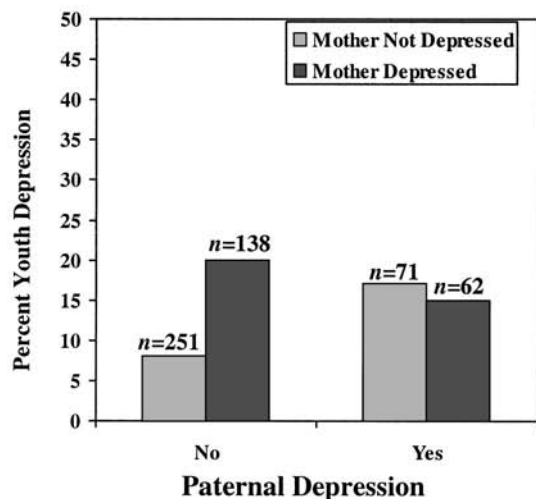
Approximately one third of the youth with nondepressive externalizing disorders and one half of the youth with nondepressive internalizing disorders also had a diagnosis of depression. Reanalysis of the parental psychopathology and youth nondepressive disorder relationship excluding these cases of youth comorbidity yielded similar results.

Because paternal psychopathology did not have an additive or a moderating effect on maternal depression in the prediction of youth nondepressive internalizing disorders, further analyses of mediator effects in relation to youth nondepressive internalizing disorders were unnecessary.

Table 2  
*Maternal Depression, Paternal Psychopathology, and Youth Depressive Disorders*

Paternal diagnosis	$\beta$	Wald	<i>p</i>
Depression			
Block 1			
Mother education	-0.12	0.99	.32
Gender	1.16	15.68	<.01
Family income	-0.06	0.18	.67
Maternal depression	0.72	7.23	<.01
Paternal depression	0.12	0.16	.69
Block 2			
Interaction term	-1.19	4.01	<.05
Substance abuse			
Block 1			
Mother education	-0.11	0.83	.36
Gender	1.18	15.97	<.01
Family income	-0.03	0.05	.83
Maternal depression	0.73	7.35	<.01
Paternal substance abuse	0.51	2.43	.12
Block 2			
Interaction term	1.48	4.38	<.05

## Paternal Depression



## Paternal Substance Abuse

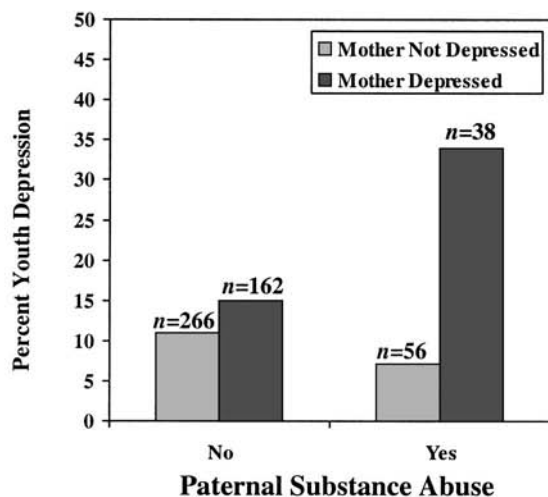


Figure 1. Parental psychopathology and youth depression.

### Maternal Depression, Paternal Psychopathology, and the Social-Familial Context

Next, we tested the hypothesis that levels of family stress and marital conflict would be higher and the quality of the father-child relationship would be lower for those youths who had both a depressed mother and a depressed or substance-abusing father than it would be for other youths in the sample.

**Chronic family stress.** Analyses of variance procedures revealed that paternal depression and maternal depression did not interact to predict chronic family stress,  $F(1, 522) = 0.18, p = .67$ . However, paternal substance abuse and maternal depression did

Table 3

Maternal Depression, Paternal Psychopathology, and Youth Nondepressive Externalizing Disorders

Paternal diagnosis	$\beta$	Wald	$p$
Depression			
Block 1			
Mother education	-0.07	0.26	.61
Gender	-1.63	21.82	<.01
Family income	-0.18	1.40	.24
Maternal depression	1.09	12.99	<.01
Paternal depression	1.03	11.25	<.01
Block 2			
Interaction term	-0.34	0.32	.57
Substance abuse			
Block 1			
Mother education	-0.06	0.20	.66
Gender	-1.58	20.92	<.01
Family income	-0.22	2.18	.14
Maternal depression	1.16	15.18	<.01
Paternal substance abuse	0.29	0.65	.42
Block 2			
Interaction term	0.10	0.02	.88

interact to predict this family-functioning variable,  $F(1, 522) = 7.12, p < .01$ . Post hoc Duncan's analyses revealed that the families with both a depressed mother and a substance-abusing father had a significantly higher rating of chronic family stress ( $M = 2.76$ ) than did those families with only a depressed mother ( $M = 2.30$ ), only a substance-abusing father ( $M = 2.29$ ), or neither ( $M = 2.19$ ).

**Father's EE.** Logistic regression analyses also revealed that Paternal Depression  $\times$  Maternal Depression significantly predicted father's EE,  $\chi^2(1, N = 483) = 4.85, p = .03$ . The top half of Figure 3 displays the percentages of children in each comparison group who were coded as having high levels of father's EE.

## Paternal Depression

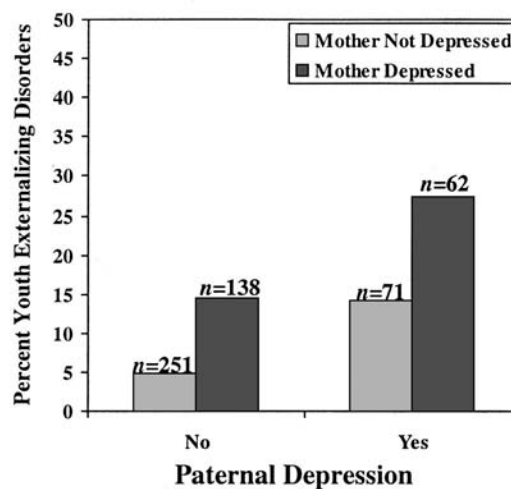


Figure 2. Parental depression and youth nondepressive externalizing disorders.

Table 4  
Maternal Depression, Paternal Psychopathology, and Youth  
Nondepressive Internalizing Disorders

Paternal diagnosis	$\beta$	Wald	<i>p</i>
Depression			
Block 1			
Mother education	-0.13	0.73	.39
Gender	0.79	4.99	<.05
Family income	-0.21	1.66	.20
Maternal depression	0.76	5.05	<.05
Paternal depression	0.34	0.92	.34
Block 2			
Interaction term	-0.51	0.51	.48
Substance abuse			
Block 1			
Mother education	-0.11	0.56	.46
Gender	0.81	5.19	<.05
Family income	-0.19	1.23	.27
Maternal depression	0.78	5.37	<.05
Paternal substance abuse	0.66	3.01	.08
Block 2			
Interaction term	0.50	0.43	.51

As can be seen from the figure, youths who had both a depressed mother and a depressed father had higher rates of father's EE than the other three comparison groups. This pattern is not the same as the pattern noted in rates of youth depression in these comparison groups (top of Figure 1). Logistic regression analyses also revealed that paternal substance abuse and maternal depression interacted in the prediction of father's EE,  $\chi^2(1, N = 483) = 3.88, p < .05$ . The bottom half of Figure 3 displays the percentage of children in each comparison group who were coded as having high levels of father's EE. As revealed in the figure, youths with both a substance-abusing father and a depressed mother had higher rates of father's EE than the youth in the other comparison groups. This pattern of results is very similar to that noted for the effect of paternal substance abuse and maternal depression on the outcome of youth depression (bottom of Figure 1).

**Marital conflict.** Contrary to our hypothesis, the results of analyses of variance tests revealed that neither Paternal Depression  $\times$  Maternal Depression,  $F(1, 519) = 0.80, p = .37$ , nor Paternal Substance Abuse  $\times$  Maternal Depression,  $F(1, 519) = 0.96, p = .33$ , significantly predicted marital conflict.

#### Family Functioning as Mediator in Predicting Youth Depression

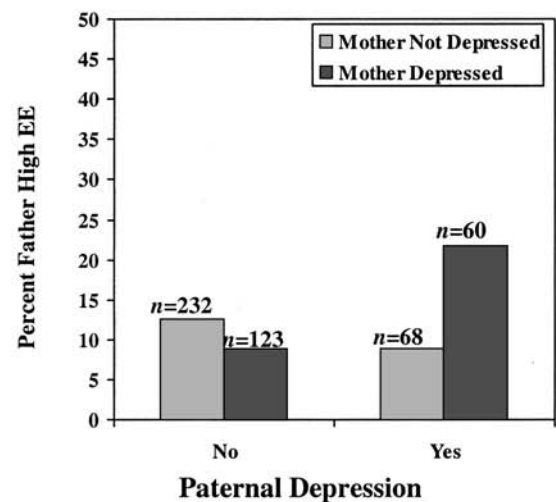
Maternal Depression  $\times$  Paternal Depression and Maternal Depression  $\times$  Paternal Substance Abuse were both significant in predicting youth depression. Next, we examined whether disrupted family functioning might help to explain the mechanism for this effect. Specifically, we followed the methods proposed by Baron and Kenny (1986) to assess whether family stress, father's EE, and marital conflict acted as mediators in the relationship between parental psychopathology and youth depression.

Baron and Kenny (1986) set forth the following criteria as necessary for a variable to qualify as a mediator: Criterion 1. The independent variable (in this case the interaction between maternal depression and paternal depression or substance abuse) must pre-

dict the potential mediator variable; Criterion 2. The potential mediator variable must significantly predict the dependent variable (with the independent variable included in the model); and Criterion 3. Statistical control of the potential mediator variable must reduce or eliminate the previously significant relationship between the independent variable and the dependent variable.

**Criterion 1.** As presented above, father's EE satisfied the first criterion as a mediator between the independent variable Paternal Depression  $\times$  Maternal Depression and the dependent variable of youth depression. That is, paternal depression and maternal depression interacted to predict father's EE. Both chronic family

### Paternal Depression



### Paternal Substance Abuse

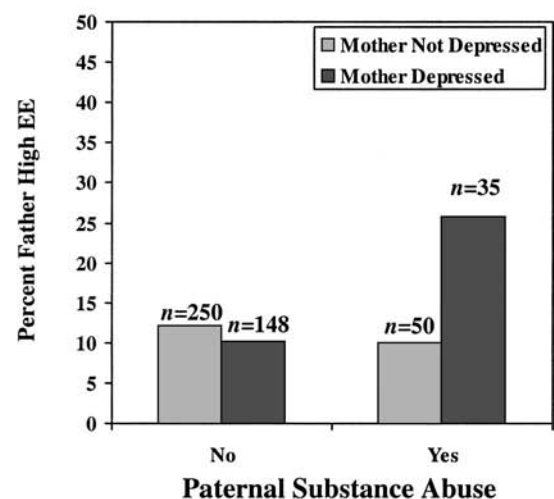


Figure 3. Parental psychopathology and father's expressed emotion (EE).



stress and father's EE also satisfied Criterion 1 as a potential mediator in the relationship between the independent variable Paternal Substance Abuse  $\times$  Maternal Depression and the dependent variable of youth depression. That is, paternal substance abuse and maternal depression interacted to predict both chronic family stress and father's EE. Marital conflict did not satisfy Criterion 1 for a mediator and so no further analyses were undertaken with this variable.

**Criterion 2.** For those variables that met the first criterion for a mediator, chronic family stress and father's EE, we next tested whether they met Criterion 2. Specifically, we examined whether each of these family-functioning variables (potential mediators) was related to youth depression (the dependent variable), while controlling for the effects of parent psychopathology (the independent variable). Logistic regression analyses controlling for the main effects and interaction of maternal depression and paternal depression revealed that father's EE significantly predicted youth depression,  $\chi^2(1, N = 522) = 11.88, p < .01$ . Further logistic regression analyses controlling for the main effects and interaction of maternal depression and paternal substance abuse revealed that both chronic family stress,  $\chi^2(1, N = 522) = 16.74, p < .01$ , and father's EE,  $\chi^2(1, N = 483) = 8.59, p < .01$ , were significant predictors of youth depression. Therefore, both chronic family stress and father's EE satisfied the second criterion for mediators.

**Criterion 3.** We next completed the final step in the test for a mediator effect of father's EE. We completed a logistic regression analysis in which demographic controls and main effects were entered in the first block, along with the father's EE, and the interaction term of maternal depression and paternal diagnosis was entered in the second step. Logistic regression analyses revealed that the inclusion of a statistical control for father's EE did not decrease the effect of Maternal Depression  $\times$  Paternal Depression on the outcome of youth depression,  $\chi^2(1, N = 483) = 5.71, p = .02$ . The inclusion of father's EE as a statistical control did, however, render the Paternal Substance Abuse  $\times$  Maternal Depression term nonsignificant in its prediction of youth depression,  $\chi^2(1, N = 483) = 2.42, p = .12$ . Father's EE, therefore, appears to mediate the relationship between paternal substance abuse, maternal depression, and youth depression but not the relationship between paternal depression, maternal depression, and youth depression.

Because chronic family stress was related to youth depression and was also significantly predicted by Maternal Depression  $\times$  Paternal Substance Abuse, we tested whether it satisfied the final criterion for a mediator in the parental psychopathology and youth depression relationship. Results of this analysis revealed that paternal substance abuse and maternal depression no longer significantly interacted to predict youth depression once chronic family stress was controlled,  $\chi^2(1, N = 522) = 3.33, p = .07$ . Chronic family stress, therefore, does appear to mediate the relationship between maternal depression, paternal substance abuse, and youth depression.

### *Family Functioning as Mediator in Predicting Youth Depression*

Maternal depression and paternal depression had an additive effect on the outcome of youth nondepressive externalizing disorders. In a final set of logistic regression analyses, we examined

whether family functioning might also mediate this parent psychopathology effect.

Both chronic family stress,  $\chi^2(1, N = 522) = 23.71, p < .01$ , and father's EE,  $\chi^2(1, N = 522) = 3.59, p < .05$ , were significantly related to youth nondepressive externalizing diagnoses when both maternal depression and paternal depression were controlled. Marital conflict,  $\chi^2(1, N = 522) = 2.97, p = .09$ , was not significantly related to youth nondepressive externalizing diagnoses. Therefore we tested whether either chronic family stress or father's EE might mediate the effects of maternal and paternal depression on youth externalizing disorders. Table 5 presents the results of these logistic regression analyses. As can be seen from the table, the addition of these family-functioning variables as statistical controls did not eliminate the significant additive effects of maternal and paternal depression on youth externalizing diagnostic outcomes.

## Discussion

The results suggest that fathers play an important role in determining whether maternal depression is related to youth depressive disorder outcomes. Paternal depression and paternal substance abuse both acted as moderators between maternal depression and youth diagnoses of depression. The specific moderator effect, however, differed according to the type of paternal psychopathology. In the case of paternal depression, maternal depression was only significantly related to youth depression in cases where the father did not exhibit the disorder. In this case, it seems that the independent presence of maternal depression or paternal depression increased the risk for youth depressive disorders to the same extent as the dual presence of these parental disorders. In contrast, in the case of substance abuse, the co-occurrence of maternal depression and paternal substance abuse had a potent effect on the risk for youth depression. Adolescents who had a depressed mother and a substance-abusing father evidenced significantly higher rates of depression than adolescents with only a depressed mother, only a substance-abusing father, or neither a depressed mother nor a substance-abusing father.

Maternal depression and paternal depression were found to be significantly related to one another in our study. In contrast, depressed women were no more likely than nondepressed women to have a partner with a diagnosis of substance abuse. This result

Table 5  
*Mediator Analyses of the Maternal Depression, Paternal Depression, and Youth Nondepressive Externalizing Disorders Relationship*

Variable	$\beta$	Wald	$p <$
Without mediators			
Maternal depression	1.09	12.99	.01
Paternal depression	1.03	11.25	.01
Controlling for father's EE			
Maternal depression	1.02	10.48	.01
Paternal depression	0.75	5.32	.05
Controlling for chronic family stress			
Maternal depression	1.05	10.61	.01
Paternal depression	1.01	9.65	.01

Note. EE = expressed emotion.



contradicts findings in the research on assortative mating in which women with depression have been found to be more likely to mate with substance-abusing men (Dierker et al., 1999). The reason for our contradictory result may be due in part to sample selection. Families were only included in this study if both the mother and the father agreed to be directly interviewed and the father had substantial contact with the youth. At the youth age of 15, substance-abusing husbands were likely lost to the sample because of these criteria. Moreover, it seems plausible that there would be higher rates of depression among the wives or ex-wives of substance-abusing fathers who did not participate in the study. In support of this supposition, family history data obtained from the mothers in the larger high-risk sample reveal a significant relationship between maternal depression and biological father substance abuse. Our sample selection therefore appears to have reduced cases of assortative mating for depressed women and substance-abusing men.

We found additive effects for maternal depression and paternal depression on youth nondepressive externalizing disorders. This result is consistent with the finding that paternal psychopathology is strongly associated with externalizing outcomes in youth (Connell & Goodman, 2001). Our results also suggested that chronic family stress and father's EE did not act as potent mediators for these additive parental psychopathology effects. It may be that other family-functioning variables, such as parental monitoring and discipline, act as the mechanisms through which maternal and paternal depression contribute to the increased likelihood of externalizing disorder outcomes in youth. Our data analyses did not assess for direction of effect between parental and youth diagnoses. Therefore it is also possible that youth externalizing disorders lead to parental depression rather than the other way around.

We did not find that paternal psychopathology moderated the relationship between maternal depression and youth nondepressive internalizing or externalizing disorders. In other words, our moderator findings appeared to be specific to the risk for youth depression. This finding stands somewhat in contrast to those of Merikangas, Prusoff, et al. (1988), who did not find any combination of maternal and paternal diagnoses that interacted to specifically predict youth depression. It should be noted, however, that Merikangas and her colleagues examined cases of "pure" youth depression, whereas our study did not exclude cases in which depressed youth also evidenced other disorders. Our finding that the results were specific to youth depression might suggest some genetic basis for our results; however, given the type of sample it is not possible for us to test for genetic effects.

Our study suggests that impairments in family functioning might be one of the mechanisms through which maternal depression and paternal substance abuse result in increased risk for youth depression. Specifically, the youth with substance-abusing fathers and depressed mothers experienced higher levels of chronic family stress and father's EE. These deficits in family functioning in turn were related to increased youth depression. In addition, these family-functioning deficits accounted at least in part for the Maternal Depression  $\times$  Paternal Substance Abuse effect on youth depression. These findings have direct implications for therapeutic intervention with families. They suggest that interventions targeted at reducing family stress and improving the father-child relationship in these types of families may be especially helpful in alleviating youth depression.

The combination of maternal depression and paternal depression was also related to higher levels of father's EE in our sample. Nevertheless, this family-functioning dynamic did not act as the mediating mechanism for the interactive effect of parental depression on youth depression. Risk for depression was similarly increased in children with either a depressed mother or a depressed father or both. It is possible that high levels of father's EE explain the increased risk for children who had both a depressed mother and a depressed father but that some other factors worked to increase risk for youth depression when only the mother or the father was depressed.

Marital conflict has been found to be an important mediator in the relationship between maternal depression and youth outcome (Cox et al., 1987; Emery et al., 1982; Fergusson et al., 1995). Our findings do not necessarily contradict the past research in this area, but they do suggest that marital conflict may not be an important mechanism whereby paternal psychopathology and maternal depression (together) lead to increased risk for youth depression. It should be noted, however, that our sampling method is likely to have resulted in conservative estimates of the potential mediating role of marital conflict. Simply stated, in cases of high marital conflict, fathers might have been less likely to participate in the interview. Further examination of the potential mediator effects of marital conflict in the parental psychopathology and youth depression relationship appear to be warranted, particularly in more heterogeneous samples.

Our hypotheses and conceptualization of the relationship between parental psychopathology, family stress and functioning, and youth diagnostic outcomes assume a specific direction of effects. That is, we surmise that parental psychopathology disrupts family functioning, which in turn leads to deleterious child outcomes. It is important to note, however, that we did not complete longitudinal analyses to confirm this direction of effect. It is also possible, for example, that the relationships that we found between parental psychopathology, family stress and functioning, and child outcome instead reflect the effect of child psychopathology on family and parental outcomes. In terms of clinical utility, knowledge that particular patterns of parental disorders, family interaction patterns, and youth outcomes tend to co-occur is still valuable and informative in the context of therapeutic intervention.

Because our sample consisted primarily of intact families or of cases where the youth had substantial contact with the father, our results may not be generalizable to the overall population of depressed mothers. In the Age 15 follow-up of the MUSP cohort, rates of maternal depression were higher in families where the father did not participate than they were in families where the father did (the sample for the present study). In addition, it is not clear whether our sampling method would overestimate or underestimate the moderating effects of paternal psychopathology in the relationship between maternal depression and youth outcomes. Perhaps paternal psychopathology has a greater effect on youth outcomes in cases of intact families or in cases where the father and the youth have substantial contact with one another. On the other hand, paternal psychopathology may have an equal or more potent effect on youth outcomes in cases where contact with the youth is more limited. The mechanism for the effect noted in our sample may or may not be the same mechanism that increases risk

for psychopathology when the father has less of a presence in the life of the child.

Our study presents results from a large community sample that contained adequate numbers of participants to examine the relationship between maternal depression, paternal psychopathology, and youth diagnostic outcomes. In addition, our measures of parent and youth diagnoses are based on extensive clinical interviews, and our measures of family functioning were gathered from multiple informants. Our high-risk sampling method using maternal self-reports of depression from early in the child's life suggests that maternal depression likely preceded youth depression temporally. Nevertheless, we did not specifically examine age of onset for parental and youth diagnostic conditions. Furthermore, our study is not an experiment, and therefore, we are not able to make causal inferences from our results. What does appear to be evident from our findings is that the father plays an important role in the relationship between maternal depression and youth diagnostic outcomes. In particular, the combination of paternal substance abuse and maternal depression is strongly related to high levels of youth depression. Moreover, chronic family stress and high levels of father's EE appear to be two mechanisms through which parental psychopathology is related to youth depression. These findings might be particularly valuable in the therapeutic context of family intervention and in prevention programs targeting high-risk youth.

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