

Mediating links between maternal depression and offspring psychopathology: the importance of independent data

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Background: Research examining intergenerational transmission of psychopathology is often limited by reliance on the same individuals for information on multiple constructs of interest. To counteract this limitation, data from a prospective, longitudinal study of at-risk youth were analyzed to test the hypothesis that parenting and family environmental factors mediate the association between maternal depressive symptoms and offspring psychopathology in late adolescence. **Method:** Data were taken from 184 families of the Minnesota Longitudinal Study of Parents and Children. Measures included the CES-D and Beck depression inventories, home environment ratings and a family conflict scale, and CBCL behavior problem checklist and K-SADS psychiatric symptom scores. Regression analyses were conducted to test for mediation of maternal depression effects by family environmental factors. **Results:** Analyses using a single informant and time point showed evidence for substantial mediation; however, in analyses spanning independent informants and multiple time points mediating effects were markedly reduced. Sex differences were found, in that parenting and family environmental factors related to psychopathology for males, whereas maternal depression was more directly related to psychopathology for females. **Conclusions:** Results emphasize the importance of independent data for testing mediational claims, and support claims that the processes involved in the intergenerational transmission of psychopathology are different for male and female youth. **Keywords:** Maternal depression, parenting, adolescents, family factors. **Abbreviations:** BDI: Beck Depression Inventory; CBCL: Child Behavior Checklist; CES-D: Center for Epidemiologic Studies Depression scale; K-SADS: Schedule for Affective Disorders and Schizophrenia, Child version; SFI: Self-Report Family Inventory.

Many studies have documented the variety of negative outcomes for children and adolescents associated with having a depressed parent (for extensive reviews see Cummings & Davies, 1994; Downey & Coyne, 1990; Goodman & Gotlib, 2002). Much of this area has focused on mothers, in part because of the generally higher prevalence of clinical depression in females as well as the specific risk of postpartum depression. Although paternal depression is worthy of further study, the scope of the present paper is restricted to depression in mothers. Research in this area has focused both on homotypic links between parental depression and child depression, as well as heterotypic connections between parental depression and other problems such as impaired cognitive development, insecure and/or disorganized attachment styles, difficult temperament, high stress reactivity, school problems, and externalizing psychopathology (e.g., Ashman & Dawson, 2002; Hay, 1997; Lee & Gotlib, 1989; Lyons-Ruth, Connell, Grunebaum, & Botein, 1990; Radke-Yarrow, 1998).

One clear starting point for research in this domain is genetic links between maternal and offspring depression. Behavior genetic research has emphasized the potential heritability of depression itself as well as of personality and cognitive factors (e.g., neuroticism, behavioral inhibition, low self-esteem) that might predispose to depression. Although some

studies have found high heritability of clinical depression in adults (e.g., Kendler et al., 1995), this finding generally has been restricted to major depression and not to subclinical depressive symptoms, and heritability of depression in childhood and adolescence is unclear (Goodman & Gotlib, 1999). Still, it remains clear that genetic factors are part of the intergenerational transmission of depression. Although there are inherent limitations to familial aggregation studies, Todd, Geller, Neuman, Fox, and Hickok (1993) reported an additive increase in risk for depression with increased number of depressed relatives using a child-proband design.

However, attention has turned more recently to specifying and testing various mechanisms that may mediate or moderate the relation between depression in parents and in their children. For example, reviews have highlighted the different ways in which parenting and the parent-child relationship are affected by depression, and how such factors in turn can impact psychopathology in offspring. Depressed mothers' increased negativity extends into the demands of parenthood; they tend to see themselves as less competent than other parents and direct more critical comments to their children (e.g., Webster-Stratton & Hammond, 1988). They experience more helplessness in the parental role and are more likely to feel that developmental outcomes are determined

by uncontrollable factors (Kochanska, Radke-Yarrow, Kuczynski, & Friedman, 1987). Observational studies of depressed mothers interacting with their children show evidence that compared to non-depressed mothers, depressed mothers display less positive affect and respond less rapidly and consistently to their children's actions. In one study, depressed mothers were less likely to reach compromise with their young children than were well mothers (Kochanska, Kuczynski, Radke-Yarrow, & Welsh, 1987), supporting findings that depressed mothers are generally more coercive in their interactions with their children (studies reviewed in Downey & Coyne, 1990).

Of course, researchers have become increasingly aware that the above factors (and others not discussed here) are only part of the picture, and there has been more recent attention to the ways in which certain risk factors and processes might interact. Goodman and Gotlib (1999) have proposed an integrative and developmental model for the transmission of psychopathology to children of depressed mothers, that incorporates: (1) heritability of depression, (2) dysfunctional early neuroregulatory mechanisms, (3) exposure to negative maternal cognitions, behaviors, and affect, and (4) exposure to a stressful environment as key mechanisms putting offspring on a path to psychobiological dysfunction, skills deficits and maladaptive cognitive styles, and subsequent depression. Under this model, intrusive and uninvolved parenting would fall under the third category. Goodman and Gotlib (1999) review a large body of research and find support ranging from moderate to strong for each of their four proposed mechanisms. Their review highlights timing and severity of maternal depression and age and gender of the child as important potential moderators of these mechanisms. For example, they note that postpartum depression is likely to have a more detrimental effect than maternal depression experienced later in the child's life, due to the immaturity of cognitive and emotion regulation systems in very young children (Dawson, Hessler, & Frey, 1994; Hay, 1997).

The issue of differential effects of maternal depression by child gender is of special importance given that during adolescence the sex ratio of clinical depression jumps from rough parity to approximately 2:1 in favor of females (Nolen-Hoeksema & Girgus, 1994). Although there have been conflicting findings in this area, there is some evidence to suggest that females are more susceptible to negative effects of parental depression than are males. For instance, in the Christchurch, New Zealand study Fergusson, Horwood, and Lynskey (1995) reported a correlation of $r = .44$ between maternal depressive symptoms and subsequent depressive symptoms in their adolescent daughters, but no significant correlation ($r = -.01$) between maternal symptoms and symptoms in

their sons. Davies and Windle (1997) found that maternal depressive symptoms were related to subsequent adolescent reports of depressive symptoms, conduct problems, and academic difficulties for 10th and 11th grade girls but not for boys. In addition, they found evidence that family discord (largely assessed through parent-report measures) mediated the effect of maternal depressive symptoms on their indicators of girls' social and emotional adjustment. Sheeber, Davis, and Hops (2002) review these and other studies and propose several explanations for gender-specific vulnerability to the effects of maternal depression, including the maternal modeling of depressive interpersonal behaviors and lack of effective coping strategies for girls, more conflictual mother-daughter relationships, and increased relationship orientation and gendered socialization of adolescent females.

From a developmental perspective, there is reason to believe that the most salient mediators of intergenerational transmission of depression would vary depending on the age and developmental status of offspring. The example of conflict in family relationships (and parent-child relationships specifically) highlighted above is likely to be especially salient in the adolescent years, as mothers and offspring engage in the process of negotiating family boundaries and acceptable behaviors. Younger children, as compared to adolescents, are presumably more susceptible to a variety of facets of the parenting climate such as responsiveness and stimulation.

Mediational role of parenting: recent studies

Recent research has sought to clarify the association between maternal depression and maladaptive child outcomes by examining formal mediational models. Two studies in particular have capitalized on comprehensive assessments of longitudinal data to shed light on the question of parenting's mediating effect on the intergenerational transmission of depression. Johnson, Cohen, Kasen, Smailes, and Brook (2001) present results from a community sample investigating the role of maladaptive parental behaviors in the intergenerational transmission of psychopathology. In their study, Johnson et al. (2001) found that associations between parental psychiatric disorders and increased offspring risk for disorder became nonsignificant after controlling for maladaptive parental behaviors. In fact, the overall risk gradient for offspring psychopathology (as a function of maladaptive parental behaviors) was virtually identical among offspring of parents with and without psychiatric disorder. The assessment of parental behavior in the Johnson et al. (2001) study included a wide range of behaviors, from harsh punishment and numerous interparental arguments to poor home maintenance and cigarette smoking in the presence of the child. With the exception of home maintenance

and maternal behavior during the interview (obtained by interviewer ratings), parental behavior was assessed via interview and self-report measures.

Bifulco et al. (2002) examined intergenerational transmission of both psychological disorder and psychological vulnerability in two longitudinal groups of mother-child pairs, a representative working-class sample as well as a sample of women screened for one of three 'psychological vulnerability' factors: conflict with a partner or child, lack of social support, and low self-esteem. Their study assessed psychiatric disorder in mothers and offspring, as well as a range of parenting experiences including antipathy, control, neglect, and abuse; all data were collected via mother and offspring interviews. Bifulco et al. (2002) found that a composite index of offspring neglect/abuse mediated the association between mother's history of depression and offspring psychiatric disorder. In addition, they found an interesting direct effect from maternal psychological vulnerability to offspring disorder that was not mediated by parenting factors.

These particular studies are significant in establishing how and to what extent parenting and parent-child relationships are mediating factors in the link between maternal depression and offspring psychopathology. However, as noted by the authors of both papers, they are limited to some extent by reliance on certain informants. Specifically, the issue of non-independence of measurement is raised when information on maternal depression or disorder, as well as parenting practices or family climate, comes from the same individual (the mother), or is a combination of information from mother and offspring. The problem of non-independence of data may be especially important in studies of depression, because it is plausible that depressed status of parent or offspring may color reports of child outcome or recollections of parenting (e.g., Chi & Hinshaw, 2002; Truetler & Epkins, 2003). Bifulco et al. (2002) note the demonstrated reliability and robustness of their measures but acknowledge the potential problem of reporting bias. Although their assessments of maternal and child experiences were made independently and were separated in time, the offspring in their study provided information on both present symptomatology and prior history of abuse and neglect. Johnson et al. (2001) note the lack of systematic observational indices of parental behavior as a limitation of their study. Thus, it is important to reexamine and replicate their substantive findings using independent assessments of maternal depression, parenting, and offspring psychopathology.

Goal of the present study

The goal of the present study is to test in an at-risk sample the mediational role of parenting and the family environment in the association between maternal depressive symptoms and offspring

psychiatric symptoms in late adolescence, and to examine whether mediational effects would differ based on gender of offspring. Our data were drawn from a unique sample of children at developmental risk due to poverty, and an additional goal of the present study was to provide a bridge between the generalizability of community samples and the needed prevalence rates of clinical samples. We were interested in determining to what extent findings from this high-risk sample would correspond to other studies in the literature, given the elevated level of risk present in the sample more generally. We chose to focus on offspring psychopathology more generally, rather than depression or anxiety in particular, in part to follow the prior literature on which this study is based (e.g., Bifulco et al. 2002; Johnson et al., 2001).

In addition, given the importance of potential bias associated with an individual informant noted above, this study focuses on two sets of analyses: the first using only a single informant and time point (mother assessment at child age 16), and the second using multiple time points and independent assessments of maternal depression, parenting and family environmental factors, and offspring psychopathology. We conceived the first set of analyses as a logical starting point based on the goal of replicating the studies reviewed above, and the second as a more stringent test of a mediational claim, due to the use of independent informants and time points. Both sets of analyses were conducted separately by gender, and both consisted of initial examinations of correlations among putative factors followed by regression analyses to test for presence and amount of mediation. Although the nature of our specific measures raises additional interesting questions, we believe that comparison of these sets of analyses is an important focus for the present paper, not least due to the continued employment of single informants for multiple constructs in tests of mediational models.

Method

Participants

Data for this research come from the Minnesota Longitudinal Study of Parents and Children, a study of children and families at developmental risk due to poverty (Egeland & Brunnequell, 1979). The current paper used data from the following assessments: child age 4 years (48 months), 6 years, 7 years, 8 years, 16 years, and 17.5 years. One hundred eighty-four mothers participated in the 48-month assessment; of these, 165 (90%) were still participating by age 17.5. Of the 184 children participating at child age 4, 98 were male (53%) and 86 (47%) were female. Sixty-four percent were White/Caucasian, 13% African-American/Black, 3% Native American/Indian, 17% mixed race, and in 4% of the cases data from the father were unavailable to determine ethnic background.

Analyses comparing the participants who continued in the study throughout the assessment periods covered by the present analyses with participants who dropped out of the study showed no differences in maternal IQ, maternal education level, child gender, or maternal depression at child age 4. There was evidence of selective attrition by ethnicity, with children from African-American and Native American backgrounds more likely to not be participating by age 17.5, $\chi^2(4) = 15.79, p < .01$.

Measures

Maternal depression. *Center for Epidemiologic Studies Depression Scale* (CES-D; Radloff, 1977). Mothers completed this twenty-item self-report depression symptomatology scale twice, when their children were 4 years and 16 years of age. The measure requires participants to report the number of days during the past week in which they felt or behaved in the manner described by each item. Sixteen of the items describe negative feelings or behaviors, while the remaining four items relate positive feelings or behaviors. The depression score is obtained by combining (reverse-scored) positive and negative items. The measure has shown adequate internal consistency reliability (.85) and test-retest reliability over a four-week period is also good (.67; Radloff, 1977). Validity, as determined by patterns of correlations with other self-report measures, correlations with clinical ratings of depression, and relations with other variables, was reported to be satisfactory (Radloff, 1977). Alpha reliability coefficients for the present sample were .90 at child age 4 and .74 at child age 16.

Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). Mothers completed the BDI when at child ages 7 and 8. The BDI is a unidimensional self-report measure of behavioral manifestations and depth of depression. It consists of 21 clinically derived 'symptom-attitude' categories judged to be characteristic of depressive patients (e.g., pessimism, self-dislike, fatigability), which the individual rates using a series of four-point ordinal scales. Individual category scores are summed to produce a total score. Beck and Beamesderfer (1974) reported an internal consistency reliability of .86 for the scale. Alpha reliability for the present sample was unavailable due to coding of total score rather than individual item data at time of initial data collection and entry.

Overall depression score. An overall maternal depressive symptom score was created for the second stage of regression analyses. For this score, the two CES-D measures and two BDI measures were z-scored, and the maximum z-score of the four assessment points was selected for each participant. This procedure followed checks to ensure that no one assessment point was unduly weighting the peak depression score (below). We chose to aggregate maternal depression data in this fashion in order to retain focus on a hypothesized 'trait-like' component of maternal depression, and in order to overcome partially the limited time-span (typically one week) covered by the depression scale measures. Given limitations of sample size and measures, detailed examination of the effects of timing of maternal depression, though desirable, were not possible in this

study. This approach of the current study parallels the aggregation of child depression measures used in a prior report from the Minnesota study focusing on differences between child- and adolescent-onset depression (Duggal, Carlson, Sroufe, & Egeland, 2001).

Parenting/family environment. Two measures of parenting/family climate were used in the present study: the HOME scale in the multiple-informants-and-time-points analyses, and the SFI-Conflict scale in the single-informant-and-time-point analyses.

HOME scale. When offspring were age 6 years, the elementary version of the HOME (Home Observation for the Measurement of the Environment; Bradley, Caldwell, Rock, Hamrick, & Harris, 1988; Caldwell & Bradley, 1984) was completed. The HOME is completed by trained interviewers during visits to the child's home setting and with the child and primary caregiver present. The version used in the present study consists of 59 items grouped into eight subscales: Emotional and Verbal Responsivity, Encouragement of Maturity, Emotional Climate, Growth-Fostering Materials and Experiences, Provision for Active Stimulation, Family Participation in Developmentally Stimulating Experiences, Paternal Involvement, and Aspects of the Physical Environment. Each item is scored as present or absent; the analyses reported below include total scores on the measure, summed across the eight subscales. Bradley et al. (1988) report total scale internal consistency of .90 and an inter-observer kappa coefficient of .88. Bradley et al. (1988) report expected patterns of correlations between the measure and key demographic variables, younger-age HOME versions, and academic achievement. HOME total scores were used in the present study in order to tap into broad areas of parental and family functioning likely to be related to adjustment of school-age children, such as a lack of stimulation/involvement and a negative emotional climate. Alpha in the present sample was .89.

Self-Report Family Inventory (SFI; Hampson, Beavers, & Hulgus, 1989). The SFI is a 36-item self-report questionnaire designed to measure several aspects of family functioning; its development was informed by family systems orientations in clinical psychology and family counseling. For the current project, the 12-item Conflict subscale (consisting of items related to fighting, blaming, arguing, and lack of constructive problem-solving) was selected for analysis. Focus on the Conflict subscale was desired due to developmental considerations relating to the time frame of the proposed mediator: family conflict is presumed to be more salient for adolescent development than overall structure and stimulation (tapped into more by the HOME variable). Each SFI item is scored on a five-point Likert scale, with positively-worded items reversed for the Conflict subscale. Item content taps into respondents' views of the entire family climate, with sample item wording such as 'We argue a lot and never solve problems' and 'When things go wrong we blame each other'. SFI scores from mothers were assessed when their offspring were 16 years old. Internal consistency for the SFI as a whole has been assessed at between .84 and .88, with adequate test-retest reliability over 30- and 90-day periods. Alpha reliability for the present study (Conflict subscale) was .82.

Adolescent psychopathology. Two measures of adolescent psychopathology were used in the present study: CBCL total problem scores in the single-informant-and-time-point analyses, and a symptom composite measure from the K-SADS in the multiple-informants-and-time-points analyses. For both of these measures, overall problem scores were used rather than subscales or scores based on specific psychiatric disorders. As noted above, this decision allowed us to follow the general measurement strategy of Bifulco et al. (2002) and Johnson et al. (2001). In addition, use of broader outcome scores seemed warranted given the lack of specific pathway predictions by type of disorder, and conserved statistical power by limiting the total number of comparisons made.

Child Behavior Checklist (CBCL; Achenbach, 1991). The CBCL inventory was completed by mothers when their children were age 16. The measure consists of 118 items rated on a 3-point scale, corresponding to whether the behavior in question is not true, somewhat or sometimes true, or very true or often true of the target child. The CBCL factor structure consists of eight narrow-band problem scales and two broad-band scales, internalizing problems and externalizing problems, and also gives a total problems score. CBCL T-scores are based on an extensive norming sample, have short-term test-retest reliability coefficients of approximately .89, and have demonstrated ability to discriminate between youths referred for mental health services and demographically similar non-referred youth (Achenbach, 1991). In the present sample, the correlation between the internalizing and externalizing scales on the CBCL (mother report, age 16) was $r = .67$ ($p < .001$), suggesting that use of the total problem score did not result in undue aggregation of divergent information.

K-SADS. The Schedule for Affective Disorders and Schizophrenia – Child version (K-SADS; Puig-Antich & Chambers, 1978) was used to assess symptoms of mental health disorder, and was given to participating adolescents at age 17.5. The K-SADS is a structured clinical interview developed and modified for use with DSM-III-R. For the purposes of the present analyses, a quantitative index of psychiatric symptomatology was desired. Therefore, from this interview the total number of symptoms within the domains of affective disorders, behavioral disorders and anxiety disorders were calculated. In the domain of affective disorders, symptoms were scored from the following syndromes: major depressive disorder (single episode), dysthymia, mania, and cyclothymia. In the domain of behavioral disorders symptoms were scored from attention-deficit hyperactivity disorder, oppositional defiant disorder, and conduct disorder. In the domain of anxiety disorders, symptoms were scored for the following syndromes: avoidant disorder, schizoid disorder, overanxious disorder, panic disorder, separation anxiety, phobia, obsessive-compulsive disorder, and post-traumatic stress disorder. Because some symptoms are considered in the diagnosis of more than one syndrome, a scoring language was written that would make sure that symptoms would not be double-counted. For example, if a participant reported depressed mood both for the diagnosis of major depressive disorder and dysthymia, depressed mood was only counted once.

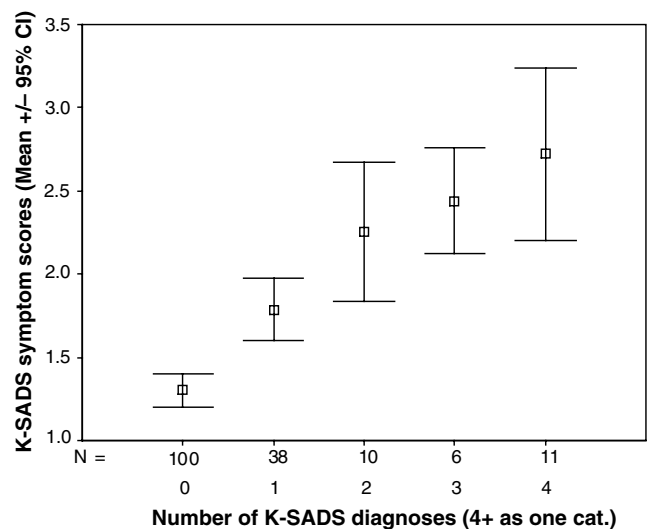


Figure 1 Transformed K-SADS symptom scores based on total number of diagnoses (offspring age 17.5 data)

Each symptom was scored 0/1, where a '1' was given if the behavior was mild/moderate or severe/extreme. A score of '0' was given if the symptom did not occur or only was present slightly. Scoring the symptoms in this manner reflects the K-SADS scoring manual where a symptom is considered to be present if it is either mild/moderate or severe/extreme. A summary score was created for three domains of psychopathology: behavior disorders, affective disorders, and anxiety disorders. Scores for each domain did not meet requirements for univariate normality, as indicated by skewness >2 and kurtosis >7 (Curran, West, & Finch, 1996), and therefore a natural log transformation was applied. Final composite symptom scores were obtained by averaging the transformed scores for each domain, resulting in a continuous overall symptomatology score from this data.

Since the algorithms used to form K-SADS diagnoses are complex and could result in different results than the transformed symptom score described above, we ran preliminary analyses to determine how symptom scores related to impairment rating and total number of diagnoses. Symptom scores were strongly negatively correlated with both overall adjustment (Global Assessment of Functioning) ratings made by interviewers ($r = -.56$) and were strongly positively correlated with total number of diagnoses (ranging from 0 to 6 in the present sample; $r = .62$). Figure 1 shows mean scores on the symptom measure based on total number of diagnoses where criteria were met. The bottom axis also shows the number of participants (out of 165) who met criteria for each number of total disorders.

Results

Descriptive statistics

Descriptive statistics for all major variables in the present study are presented in Table 1. For the measures of maternal depression and CBCL Total Problems, percentages of cases meeting clinical

Table 1 Descriptive statistics for measures of maternal depression, parenting/family environmental factors, and child behavior problems

	<i>M</i>	<i>SD</i>	Range	% Equal or above clinical cutoff	% Cases with Peak Depression Score
<i>Maternal depression</i>					
CESD – child age 4	14.42	10.78	0–51	35.9%	29.3%
BDI – child age 7	6.33	6.20	0–35	18.2%	23.4%
BDI – child age 8	6.25	6.26	0–33	19.9%	19.6%
CESD – child age 16	11.01	8.82	0–39	20.5%	27.7%
<i>Parenting / family environmental factors</i>					
HOME – child age 6	40.58	8.93	11–55		
SFI-Conflict – child age 16	1.96	.60	1.00–3.58		
<i>Child behavior problems</i>					
CBCL Total Problems T-score – child age 16, maternal report	53.76	11.45	23–88	19.5%	

Notes. Clinical cutoffs used = 16 for CESD, 11 for BDI, and 65 for CBCL. One-way chi square test for independence of peak maternal depression scale scores: $\chi^2(3) = 4.30, p = .23$.

Table 2 Zero-order correlations of all variables included in the study: chronological order by offspring age

	1.	2.	3.	4.	5.	6.	7.	8.
1. CES-D (Age 4)	—							
2. HOME (Age 6)	-.21**	—						
3. BDI (Age 7)	.22**	-.21**	—					
4. BDI (Age 8)	.31***	-.24**	.58***	—				
5. CES-D (Age 16)	.38***	-.20*	.30***	.28***	—			
6. SFI-Conflict (Age 16)	.09	-.09	.12	.07	.31***	—		
7. CBCL Total Score (Age 16)	.12	-.17*	.27**	.21*	.34***	.39***	—	
8. K-SADS Symptom Composite (Age 17.5)	.14	-.15	.20*	.21*	.29***	.23**	.25**	—

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

criteria are shown. In addition, for the maternal depression measures the percentages of cases in which that time point contributed to the peak score are also shown. Although more mothers in the sample as a whole were above cutoff at child age 4, a one-way chi-square test examining independence for frequencies of each time point contributing to the peak score was not statistically significant, $\chi^2(3) = 4.30, p = .23$. This suggests that of the points sampled, there was not one time interval ‘driving’ the peak maternal depression score for this sample.

Zero-order correlations

Correlations among all variables included in analyses are shown in Table 2, in chronological order by assessment point. As expected, the four indicators of maternal depressive symptoms (CES-D at ages 4 and 16, BDI at ages 7 and 8) were positively correlated with one another across the entire sample. The HOME measure was negatively correlated with all maternal depression indicators as well as with the CBCL problem outcome measure. SFI-Conflict scores were positively correlated with concurrent maternal depressive symptoms and both adolescent problem measures, but not with earlier depression indicators.

Table 3 Correlations among maternal depression, parenting/family conflict, and offspring emotional/behavioral problems: single informant

	CES-D	SFI-Conflict	CBCL Total Score
CES-D	—	.18	.33**
SFI-Conflict	.43***	—	.18
CBCL Total Score	.35**	.55***	—

Notes. ** $p < .01$. *** $p < .001$. Values for females appear above the diagonal; values for males appear below the diagonal. All data are from mother informant at offspring age 16.

Prior to conducting regression analyses to test mediating effects, correlation matrices were inspected to verify that for each analysis all three relevant variables (IV, mediator, DV) were significantly inter-correlated (as required for mediation; Baron & Kenney, 1986). Because of concerns with possible gender differences noted above, these comparisons were done separately by gender. Table 3 shows these correlations for the single informant and time point condition (mother assessment, child age 16). For comparative purposes, the parallel whole-sample correlations are as follows: between CES-D and SFI-Conflict, $r = .09, n.s.$; between CES-D and CBCL total scores, $r = .12, n.s.$; and between SFI-Conflict and CBCL total scores, $r = .39, p < .001$. As shown

in Table 3, the conditions for potential mediation were met only by families with male offspring. For families with female adolescents, maternal reports of family conflict were not related either to their own self-reported depressive symptoms or to reports of their daughters' emotional and behavioral problems. Thus, the single-informant-and-time-point regression analysis was conducted with only the male subsample.

Within-gender correlations among maternal depression aggregate scores (maximum out of four time points), HOME scores, and K-SADS symptom composite scores are shown in Table 4. For comparative purposes, parallel whole-sample correlations are as follows: between peak depression and HOME scores, $r = -.27$, $p < .001$; between peak depression and K-SADS scores, $r = .28$, $p < .001$; and between HOME scores and K-SADS scores, $r = -.15$, *n.s.* Examining Table 4, conditions for potential mediation were again met only by families with male children. For families with female adolescents, staff ratings of the home environment were not correlated with adolescent psychiatric symptom scores derived from clinical interview. Maternal depression, however, was correlated with both the psychiatric symptoms of adolescent females and the HOME ratings. As with the above analyses, given the failure to meet initial criteria for females, the multiple-informants-and-time-points regression analysis also was conducted using only the male subsample.

Following inspection of within-gender correlation tables to verify prerequisite associations for the mediation regression analyses, Fisher R-to-Z tests were conducted to test whether the magnitude of correlations differed significantly across gender. Significant differences were found for the correlation between SFI-Conflict and CBCL total scores at age 16 (.55 for males versus .18 for females; $p < .01$), but not for any other comparisons at the .05 alpha level.

Regression analyses

Mediation model: Single informant at child age 16. Table 5 shows the results of a two-step regression analysis testing mediating effects of SFI family conflict on the association between maternal depressive symptoms and maternal report of child emotional and behavioral problems. As shown in Table 5, the β weight for depressive symptoms dropped from .35 to .14 when including family conflict as a mediator. This change was significant at the $p < .01$ level by the Sobel test with third denominator term added (Baron & Kenney, 1986; Goodman, 1960). This implies that within constraints of a single informant and time point, for male adolescents there was substantial mediation of the link between maternal depressive symptoms and adolescent problems by factors related to family climate.

Table 4 Correlations among maternal depression, parenting/home environment variables, and offspring psychiatric symptoms: independent informants

	Maternal depression	HOME	K-SADS Symptoms
Maternal depression	–	-.24*	.33**
HOME	-.29**	–	-.03
K-SADS Symptoms	.24*	-.27*	–

Notes. * $p < .05$. ** $p < .01$. Values for females appear above the diagonal; values for males appear below the diagonal.

Table 5 Regression testing parenting as mediator of maternal depression–offspring problems association: single informant, males only

Step	Predictors	ΔR^2	b (SE)	β	<i>t</i>
1		.12**			
	CES-D		.48 (.14)	.35	3.44**
2		.20***			
	CES-D		.19 (.14)	.14	1.40
	SFI-Conflict		9.02 (1.85)	.49	4.87***
Total R ²		.32***			

Notes. ** $p < .01$. *** $p < .001$. Dependent variable = CBCL Total Score. All data from mother informant at offspring age 16. Listwise $N = 86$.

Table 6 Regression testing age 6 HOME as mediator of maternal depression–offspring symptoms association: independent informants, males only

Step	Predictors	ΔR^2	b (SE)	β	<i>t</i>
1		.06*			
	Maternal depression		.14 (.06)	.24	2.20*
2		.05*			
	Maternal depression		.11 (.06)	.19	1.71
	HOME		-.02 (.01)	-.23	-2.11*
Total R ²		.11*			

Notes. * $p < .05$. Dependent variable = K-SADS symptom composite. Listwise $N = 81$.

Mediation model: Multiple informants and time points. Table 6 shows the results of a two-step regression analysis testing mediating effects of age 6 HOME scores on the association between aggregate maternal depressive symptom scores and transformed K-SADS symptom scores. In this case, the β weight for depressive symptoms was reduced from .24 to .19 when the mediator was included in the equation. The depressive symptoms variable dropped from $p < .05$ to nonsignificant in this case, although the amount of mediation was not significant by the Sobel test. This finding suggests that with male adolescents, there was partial mediation of the association between maternal depressive symptoms and adolescent psychiatric problems by earlier home environment factors.

Discussion

This study focused on the potential mediating links of family environment factors in the link between maternal depressive symptoms and offspring psychopathology. More specifically, we sought to test whether mediating links that have been found in the literature would replicate using data gathered from independent sources, with an additional focus on gender differences in the links examined.

Turning first to the main question of mediating effects, our findings converge with the previous literature. When we used a parallel procedure to existing studies, that is, when measures of depression, parenting/environmental support, and offspring outcome all were based upon maternal report, strong evidence for family environmental factors mediating the consequences of maternal depression was obtained for the families of male adolescents.

When we then consider the findings in which maternal depression, child outcome and parenting/environmental support are independent and the parenting/environmental support assessment is based upon home observation, we find evidence for partial mediation. Two points are noteworthy about this finding: first, it does to some extent bolster the general case for mediation in this area, but second, it also underscores the need for independence of data.

It is noteworthy to draw attention to characteristics of the present sample when considering the results above. As noted in the introduction, we believe that using a high-risk sample is informative in this area of research because it can provide a bridge between clinical and community samples, allowing comparisons of results across samples with varying prevalence of disorder. In addition, however, the presence of multiple risk factors in the sample that were not included in the current study (e.g., abuse and neglect, poverty, negative life events) means that compared with a community study, here it is likely to be more difficult to identify the effects that were found. This is due to the interacting nature of risk factors generally; this sample under-represents certain adaptive 'quadrants' of distributions relative to others characterizing risk and maladaptation.

This study did not find mediating effects related to family context in sample members with female offspring. Like prior research in this area (e.g., Davies & Windle, 1997; Fergusson et al., 1995), the magnitude of association between maternal depression and psychopathology in daughters was greater than that for maternal depression and psychopathology in sons, although the R-to-Z comparison was not significant. This finding held for the multiple-informant-and-time-points data but not for the data collected from a single informant and time point, again highlighting the importance of independent information. However, we did not replicate the finding of Davies and Windle (1997) that found mediating effects of family conflict or discord for girls.

Although there are several possible explanations for the difference in findings, including nature of the mediator construct as well as time frame examined, it should be noted that family discord in the Davies and Windle (1997) study was comprised almost entirely of parental-report measures. Thus, it is possible that some of the associations observed in that study are due to informant variance.

Although the present study capitalized on longitudinal data involving independent informants, it is also limited in important ways. Most notably among these is the use of depressive symptom inventories in our assessment of maternal depression without access to clinical diagnostic status, and the consequent lack of attention to issues of timing, remission, relapse, and offspring 'exposure' to depressive behaviors and cognitions. It seems clear that most findings will differ depending on whether symptom measures or clinical diagnoses are the unit of analysis (Goodman & Gotlib, 1999). On the other hand, speaking specifically of parenting, there does not appear to be strong differences in the parenting styles of mothers with depressive disorder compared with mothers with milder depressive symptoms (Downey & Coyne, 1990). Also, although our choice of the HOME measure for the multiple-informants-and-time-points analyses gives us the strength of home observational data, it represents a broad-band score that does not allow for more fine-grained specification of mediating mechanisms. As with our analysis of total problem scores from the CBCL and transformed symptoms from the K-SADS, this provides a general test of mediation across differing informant structures rather than a detailed process-level exploration. Although use of such scores was appropriate for the modest goals of the present study, and though such scores retain validity and practical utility, future research in this area would ideally blend the comprehensive assessment of diagnostic and symptom status in clinical studies with the more detailed assessments of parenting and other aspects of family context seen in community and developmental studies.

An additional limitation of this study is the inability to disentangle fully effects of timing and content of mediator with issues of informant variance. More specifically, the analyses in this study compared a single informant and time point with multiple informants (and separate measures) across longitudinal time. The nature of our measures raises additional questions regarding effects of method and/or time point; for example, the lack of significant correlations between the SFI-Conflict measure at age 16 and earlier maternal depression indicators suggests that the reduced mediation in the second set of analyses may be partially related to the use of different time points. However, the overall goal of the present study was to explore evidence related to a developmental process of mediation. If this process is robust, as we believe it may be, evidence is needed

across different measures. This study is viewed as a step in elucidating such effects.

The present study can be seen as contributing to the growing research base demonstrating the importance of parenting behavior and family environmental factors in the intergenerational transmission of depression as well as the broader impact of maternal depression on offspring psychopathology more generally. Recent longitudinal work in this area (e.g., Bifulco et al., 2002; Johnson et al., 2001) has been valuable in the demonstration of mediating effects, but also has been limited to some extent by shared informant variance. The present study is of particular importance in demonstrating how certain results may change and others may stay similar when using data from independent informants, and how mediating pathways to psychopathology can be gender-specific. In addition to considering more detailed assessments of timing and course of maternal depression, future work could focus on delineating how the various components of the family climate and parenting context interact with each other and other factors (e.g., temperament, regulatory systems) in impacting the development of psychopathology in children and adolescents.

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