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Maternal Depression and Child Psychopathology: A Meta-Analytic Review

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Abstract Although the association between maternal depression and adverse child outcomes is well established, the strength of the association, the breadth or specificity of the outcomes, and the role of moderators are not known. This information is essential to inform not only models of risk but also the design of preventive interventions by helping to identify subgroups at greater risk than others and to elucidate potential mechanisms as targets of interventions. A meta-analysis of 193 studies was conducted to examine the strength of the association between mothers' depression and children's behavioral problems or emotional functioning. Maternal depression was significantly related to higher levels of internalizing, externalizing, and general psychopathology and negative affect/behavior and to lower levels of positive affect/behavior, with all associations small in magnitude. These associations were significantly moderated by theoretically and methodologically relevant variables, with patterns of moderation found to vary somewhat with each child outcome. Results are interpreted in terms of implications for theoretical models that move beyond main effects models in order to more accurately identify which children of depressed mothers are more or less at risk for specific outcomes.

Keywords Depression · Mothers · Children · Internalizing · Externalizing · Psychopathology · Positive affect or behavior · Negative affect or behavior · Meta-analysis · Moderators

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Introduction

The association between maternal depression and a range of adverse child behavioral and emotional outcomes has been documented in numerous studies and reviews (Goodman 2007; National Research Council and Institute of Medicine 2009). It is now well replicated that, by middle childhood, children with depressed mothers have significantly higher rates not only of mood disorders but also of other internalizing as well as externalizing problems and other difficulties in emotional development relative to children whose mothers are not depressed. In a meta-analytic review of this literature in 2002, Connell and Goodman found small effect sizes for the relations between depression in mothers and children's internalizing ($k = 78$; $r = .16$) and externalizing ($k = 79$; $r = .14$) problems, respectively. However, we also found substantial variability across studies. This variability highlights the need to extend our earlier work by examining the role of theory-based and research design features that vary across studies. Knowledge of the strength of these associations would

answer the question about the extent to which maternal depression accounts for the various adverse outcomes in children by revealing how much variance *is* and, conversely, *is not*, explained, after taking into account different levels of sampling error in studies of different sample sizes. Such information on the strength of associations would also indicate the extent to which other independent or correlated causal factors need to be considered in the elaboration of a model to explain the development of psychopathology and other adverse outcomes in children whose mothers have been depressed. The conclusions have important implications for theory (e.g., models of risk), research (e.g., high-risk designs), and practice (e.g., selective prevention). Thus, the first aim of this meta-analytic review was to determine the overall strength of the associations between maternal depression and child affective and behavioral outcomes. This review responded to the call to move beyond main effects models to explain risk for the development of psychopathology and to identify high-risk groups with greater precision by considering the role of potential moderators that enhance risk (Beekman et al. 2010; Cicchetti and Toth 2009; Kraemer 2003).

Maternal Depression and Multiple Aspects of Youth Psychopathology

It is likely that the strength of association with maternal depression differs across diverse aspects of child emotional or behavioral functioning. Knowledge of such differences is essential to further understanding of the specificity or breadth of risks for the development of psychopathology in children of depressed mothers, with implications for theory about risk as well as for pinpointing the targets of preventive interventions. Elevated rates not only of depression and other internalizing problems but also of conduct problems have been noted since the earliest studies on offspring of depressed mothers (e.g., Welner et al. 1977). From a theoretical perspective, externalizing disorders in children with depressed mothers are interesting because they may reflect problems with dysregulated aggression (Radke-Yarrow et al. 1992), a distinct pattern of inherited vulnerability perhaps related to behavioral disorders (e.g., alcoholism, substance abuse, or antisocial personality disorders) in first-degree relatives (Kovacs et al. 1997; Williamson et al. 1995), environmental risk such as the pattern of parenting that has been associated with conduct problems in general population samples, or particular interactions among genes, cognitive, affective, interpersonal, and other biological systems that lead to the emergence of externalizing rather than (or co-occurring with) internalizing disorders (Silberg and Rutter 2002). Thus, a second goal of this review was to determine the effect sizes for the association between maternal depression and a set of specific outcomes in the children and to compare them. Knowing whether the increased risk for externalizing problems is as great as for internalizing problems, for example, would motivate research to understand whether or how the risk of mothers' depression may operate differently for these two sets of problems in the offspring (Goodman 2003).

Although much of the literature has focused specifically on psychopathology in children with depressed mothers, a developmental psychopathology perspective suggests the need to examine a broader array of social and emotional functioning in youth in relation to depression in their mothers. The broadened view offers the further advantage of including infants and young children who may be too young to be reliably assessed for psychopathology. In particular, there are compelling theoretical reasons to be concerned about emotions and emotion regulation in children with depressed mothers (Cicchetti et al. 1995; Garber et al. 1991). Children of depressed mothers may have heightened negative emotionality and low positive emotionality, both of which may predispose them to the development of depression (Klein et al. 2009) or may be early signs of disorder. There are likely to be multiple complex pathways through which maternal depression is associated with both of these tendencies, including genetic (Plomin 1990), neurobiological (e.g., dysregulated stress regulation systems), and social (e.g., modeling) pathways. Thus, we included these outcomes in this review.

Moderators

In addition to potentially different models of risk for different child outcomes, moderators also have implications for these models. For example, if the strength of the association between maternal depression and a child outcome differs depending on a third variable, a possible moderator such as gender, then the causal pathway leading to that outcome may also differ depending on the value of that variable, in this example, being male or female. More broadly, without taking into account potential moderators, researchers may be over- or under-estimating the specific strength of associations between mothers' depression and child outcomes. Thus, a third goal of this review was to examine how the degree of association between depression in mothers and children's outcomes may vary depending on theoretically and methodologically relevant moderators (Goodman and Gotlib 1999), which meta-analysis is particularly well suited to examine. Greater understanding of the impact of these differences across studies on the magnitude of effects has the potential to inform refinement of theoretical models of the intergenerational effects of maternal depression. For instance, findings may highlight subgroups of children or families that are at greater risk

(e.g., infants/toddlers, children in single-parent families, or low socioeconomic status (SES) families), pointing to the need for studies elucidating the mechanisms underlying such heightened risk in detail.

Findings also have the potential to reveal methodological problems that may cloud understanding of the association between maternal depression and child outcomes, to the extent that differences in effect sizes across studies are due to methodological differences. For example, much of the literature on effects of depressed mothers on children has ignored the extensive knowledge of the nosology of depression in adults by, for example, grouping together mothers who vary in severity, chronicity, current levels, and history of depression within the target child's lifetime. A further aim, then, was to clarify how some of these aspects of depression in mothers may relate differently to child outcomes.

In particular, it is important to know whether the strength of the association between maternal depression and child outcomes varies by maternal self-report of depression symptoms versus depression disorder meeting Diagnostic and Statistical Manual-IV criteria (DSM-IV; APA 1994). In a review of epidemiological and other outcome differences between depression as a mood feature and depression as a diagnostic category, Harrington, Rutter, and Fombonne (1996) found this distinction to be important for a number of different aspects of clinical functioning. For example, clinically diagnosed depression has been found to have high heritability and a small role of common family environment. In contrast, the milder (i.e., subclinical) forms of depression, which are likely to characterize at least some portion of samples that are identified as depressed based solely on self-report, appear to be influenced predominantly by environmental factors (e.g., Kendler et al. 1995; McGuffin et al. 1993). Even so, researchers recognize that subclinical depression is important and that levels of psychosocial dysfunction may be no different from those of individuals who meet diagnostic criteria (Gotlib et al. 1995; Goodman and Tully 2009). Thus, determining whether diagnosed depression in mothers, relative to self-reported depression symptoms, is more strongly associated with children's functioning would contribute to as yet unresolved questions of the importance of this distinction (Ingram and Siegle 2009).

Similarly, the method of participant identification and recruitment may influence the results of studies. While most studies of depression in mothers rely on community samples, many studies identified samples of depressed women from various treatment settings. Such sampling decisions impose constraints on generalization of the findings, as clinical and community samples of mothers with depression may differ on important ways. For instance, most people with depression do not get treated. Kessler et al. (1999) and Kendler (1995) found that treatment-seeking in women with depression was associated with higher levels of education, being older, having a comorbid anxiety disorder, a higher level of impairment and more symptoms of depression. Moreover, after controlling for these variables, the history of major depression in one or more relatives was significantly associated with help-seeking. Similarly, W. E. Narrow (2002, personal communication) found that women with clinically significant depression were more likely to use specialty mental health services if they were white rather than Hispanic, had more education, were never married, and had suicidal ideation and more symptoms of depression. In addition, mothers who have been treated may be more likely to seek mental health services for their children (Goodman et al. 1997). In light of these findings, we expected that maternal depression identified through clinical samples would be more closely associated with children's functioning than maternal depression in community samples.

Another set of potential methodology-based moderators that we examined relate to the source from whom the data on the child outcome variables is obtained. We were particularly interested in determining whether the degree of association between maternal depression and child outcomes would be higher when the mother was the source of the data on the child, relative to other sources such as teachers, observers, clinicians, or the children themselves. Given the known associations between depression and negatively biased perceptions, it is not surprising that the maternal depression field has been mired in controversy over the potential negative bias of depressed mothers' reports of their children's psychological functioning. In a seminal review of the studies examining this question, Richters (1992) concluded that while there is little solid evidence for distortion, there is a need for studies to examine whether depressed mothers report more child behavioral problems than are reflected by independent, validated ratings of the same behaviors in the same setting. Following Richters' criteria, two studies used multiple raters (mother, teacher, and child) and structural equation models to estimate the extent to which the variance in mothers' ratings of their children that did not contribute to the latent variables was associated with maternal depression. Both Fergusson, Lynskey, and Horwood (1993) and Boyle and Pickles (1997) found small to moderate support for an association between higher maternal depression and mothers' tendency to over-report child behavioral problems, relative to the latent criterion variable. Based on these findings, we predicted that the association between maternal depression and child outcomes would be stronger when the depressed mother was the source of information on the child, relative to teachers or other sources or to child's self-report.

In addition to these methodology-based potential moderators, several theory-based potential moderators are

important to consider. Among these, children's age may influence the association between maternal depression and youth outcomes. Although researchers have argued that the degree of association between parental psychopathology and child functioning may be either positively or negatively associated with children's age (Rothbaum and Weisz 1994), Goodman and Gotlib (1999) concluded from their review that younger children's age at first exposure to their mothers' depression will have a stronger negative impact than later first exposure. Consistent with this expectation, Connell and Goodman (2002) found that the effect sizes for the association between maternal depression and children's internalizing and externalizing problems were negatively correlated with children's age ($r = -.29$ and $-.40$, respectively). Thus, we expected that the effect size for the association between maternal depression and children's psychopathology and emotional functioning would be stronger for younger children.

Gender of the child also may moderate the association between maternal depression and child outcomes, although neither theory nor research leads to a clear prediction. Goodman and Gotlib (1999) and Sheeber et al. (2002) explored theories and data relevant to gender-specific vulnerabilities in children of depressed mothers. Socialization theories, differential developmental vulnerabilities, and different contributions of genetic and nonshared environmental factors and the particular aspect of child functioning that is studied all may account for differential risk to sons and daughters of depressed mothers. Sheeber et al. (2002) argued that although there is little evidence that prepubertal sons of depressed mothers are at increased risk for externalizing behavioral problems, evidence converges to suggest that adolescent daughters of depressed mothers are at greater risk than sons for depressive symptoms. Thus, we expected a stronger association between maternal depression and internalizing problems for daughters than for sons, if only among older samples, and we examined gender as a moderator of associations with other aspects of child functioning in a more exploratory manner.

Although less often considered, sociodemographic variables could also be important, including race or ethnicity (minority), family income level (poverty), age of mother (an adolescent mother), and marital status (single-parent families). This set of variables contributes to a definition of the context of the lives of children and, when conceptualized as stressors, is likely to contribute significantly to the development of psychopathology in the children of depressed mothers (Goodman and Gotlib 1999). With regard to race/ethnicity, findings are mixed on whether rates of depression are higher in ethnic minority women, perhaps other than Latinos (Blazer et al. 1994; Jackson and Williams 2006). Nonetheless, being of minority ethnicity is often associated with a range of stressors, including discrimination, poverty, and limited access to health care and other resources (Sue et al. 2008; Krieger 1999). Thus, we expected a larger association between maternal depression and child outcomes among families who are ethnic minorities. Similarly, we expected to find larger associations between maternal depression and child outcomes in families with low SES than in middle or high SES families. The stress associated with poverty has been found to be a significant predictor, along with maternal depression, of problems in children (Belle 1982; Pound et al. 1985), and there is some evidence that poverty moderates the association between maternal depression and children's cognitive and motor development (Petterson and Albers 2001).

One might expect a similar pattern with regard to adolescent mothers. Women who give birth as teenagers have higher rates of depression symptoms (Deal and Holt 1998), poverty, single motherhood, and other stressors (Prodromidis et al. 1994) relative to older mothers. Although some studies of maternal depression are known to include large percentages of adolescent mothers in their samples, we are not aware of any studies that have looked at the differential association between maternal depression and child outcomes for adolescent as opposed to older mothers. Thus, we could only tentatively hypothesize that the association between maternal depression and child outcomes would be higher for samples of adolescent mothers than for older mothers given the associations between teen parenting and stress.

With regard to marital status, a few studies have found that among children with depressed parents, those whose parents were divorced are more likely to be rated by their teachers as under-controlled and lower on ego resiliency (Goodman et al. 1993) and to have a conduct disorder (Fendrich et al. 1990). It is possible that the elevated rates of behavioral problems in children of depressed mothers who have gone through divorce are related to the additional stresses of divorce or marital conflict on children. Conversely, it is possible that the presence of a healthy father may moderate the impact of maternal depression on children's functioning by decreasing the childcare burden on depressed mothers or by providing an alternative, potentially healthier parenting style for children. Although few studies have directly tested possible moderation by father presence/absence or involvement, findings on the effects of divorce suggested that the association between maternal depression and child outcomes would be stronger among samples with more families in whom the father is absent.

In sum, we tested the overall strength of association between maternal depression and a range of indices of child emotional functioning and psychopathology and examined several moderators. The focus is on the strength

of the association between maternal depression and these outcomes and methodological- and theory-based factors that may increase or decrease the strength of the associations. Although each of these tests addresses questions that are essential to answer in order to further an understanding of risk for the development of psychopathology in children of depressed mothers, the available body of literature imposes limitations on being able to address two other essential questions. First, questions on causality will not be answered by findings on the strength of these associations, given the limitations of the correlational designs typically employed. Second, an insufficient number of studies using genetically informed designs or testing mediation or transactional processes prohibited us from addressing questions on mechanisms or mediators, whether genetic, neurobiological, environmental, or their likely interactions and the possible pathways through which maternal depression is associated with adverse child outcome.

Method

Search Strategies

Several approaches were used to locate studies for inclusion in the meta-analysis. The principal method of location involved a search of computerized databases, including *PsycINFO*, *Dissertation Abstracts*, and *ERIC* (collectively covering 1888–2009), for studies presenting quantitative data on the association between maternal depression and the child outcomes of interest. All combinations of keywords in the following groupings were used: (mother, maternal, or mom), (depressed, depression), (children, toddler, boy, girl, or adolescent), and (behavioral problem, internalizing, depression, anxiety/anxious, withdrawn, shy, inhibit[ion], over-control, sad, fearful, happy, pleasant, cheerful, positive affect/behavior, negative affect/behavior, externalizing, conduct disorder, oppositional, delinquent, hyperactive, attention deficit, aggressive, angry, mental health, or psychopathology). Second, the ancestry method was used, in which references listed in review articles or empirical articles were retrieved. Third, correspondence was sent to the principal author of the studies identified by the first two methods requesting copies of any relevant unpublished or in-press articles. Finally, notices were sent to several internet-based discussion lists for researchers requesting copies of any relevant unpublished or in-press manuscripts. Although it is likely that other relevant studies exist that were not identified, the scope of the search makes it likely that these studies are at least a representative sample of the total body of potentially available research.

Inclusion/Exclusion Criteria

To be included in the meta-analysis, a study had to meet the following criteria. First, the study had to include data explicitly on depression in mothers. Studies that combined data from mothers and fathers (i.e., only included data on "parents") were excluded. Second, studies of adult offspring of depressed parents were excluded as those outcomes were considered beyond the scope of these analyses. In addition, studies relying on retrospective reports were excluded due to their questionable validity. Third, studies had to present data on the association between maternal depression (either as a continuous or as a categorical variable) and behavioral problems or positive or negative affect/behavior in children, operationally defined below. Fourth, we excluded studies in which the sample was exclusively clinically referred children or children selected for having psychopathology, among whom associations were examined with maternal depression. Inclusion of such samples would bias findings on the degree of association between maternal depression and child psychopathology given that those samples were selected for the presence of psychopathology in the children.

Information Extracted

Coders were trained to an acceptable level of reliability, and a randomly selected subset of 22% of the articles was coded by multiple coders. High inter-rater reliability was found between the first author and each of the other coders, with a percentage agreement of 95% for sample type and 93% for socioeconomic status. Inter-rater agreement for all other coding was 100%.

Child outcome variables. Five variables were coded to reflect the nature of the child outcome variable being assessed: (1) internalizing problems; (2) externalizing problems; (3) general psychopathology; (4) negative affect or behavior; and (5) positive affect or behavior.

Given high levels of co-morbidity among childhood disorders (Lewinsohn et al. 1991) and many researchers' reliance on symptom checklists that yield scores on internalizing and externalizing disorders as broadband constructs, we examined these broader constructs in children in relation to maternal depression. For internalizing problems, in addition to the broadband scores, we included symptom ratings of depressed mood, anxiety, or social withdrawal. Diagnoses of childhood depression and anxiety disorders were also included. For externalizing problems, again in addition to the broadband scores, we included symptom ratings of aggression, conduct problems, or delinquency and diagnoses of conduct disorder, oppositional defiant disorder, and attention deficit hyperactivity disorder. For general psychopathology, studies typically either used

symptom ratings that combined internalizing and externalizing symptoms or combined data from diagnoses of internalizing and externalizing disorders. Negative affect was operationalized as the expression of angry, sad, anxious, or fearful mood through behavior, facial expressions, verbalizations, or vocalizations. In contrast, positive affect involved the expression of happy, pleasant, or cheerful mood through behavior, facial expressions, verbalizations, or vocalizations. We expected maternal depression to be related to lower levels of positive affect and to higher levels of negative affect, internalizing problems, externalizing problems, and general psychopathology in children.

Mothers' depression measure. Studies were coded to reflect the manner in which maternal depression was assessed, that is, with either a diagnostic or a self-report symptom rating-based approach to assessment.

Sample type. Sample type was coded based upon the recruitment method used. Studies were coded as representing clinical samples when study participants were recruited from a clinical setting in which mothers were seeking or receiving services for themselves. Studies were coded as representing community samples when all participants were recruited from the general population (i.e., convenience samples or population-based samples).

Child assessment source. Child outcome variables were coded to indicate whether the source of measurement was a teacher, researcher (e.g., a trained observer), or clinician; the child's mother; the child (self-report); or both mother and child (i.e., for a clinical diagnostic assessment that combined the two sources of information).

Child age. The mean age of the children studied in each sample was coded. Moderation was examined using mean age as a continuous variable to examine the linear relationship between the mean age of children in the samples and the magnitude of effect sizes.

Child gender. When possible, separate effect sizes were calculated for boys and girls. When studies did not provide enough information to permit such calculations, child gender was coded as "mixed."

Race/ethnicity. Because very few studies provided separate analyses for different racial/ethnic groups, the percentage of Caucasian mothers in the sample was coded when available. Although the percentage of Caucasian mothers was highly skewed, with the majority of samples comprised mostly or entirely of Caucasian mothers, there were no meaningful cut points for designating samples as predominantly ethnic minority. So a decision was made to examine this moderator continuously.

Family income. Because we were interested in comparing samples of families who were living in poverty to others of middle or higher income or SES levels, we examined this potential moderator categorically as a two-level variable. Studies that specifically sampled families in poverty were considered as one group, the low SES group. Studies that sampled middle-to-high SES families or did not specify the SES of their samples comprised the second group.

Age of mother. Given our interest in comparing effects from studies of teen mothers with studies of older mothers, a categorical variable was created, with studies examining mothers with a mean age of 20 or lower coded as "teen mothers" and studies examining mothers with a mean age greater than 20 coded as "older than teenage." Studies that included both teenaged and older mothers were coded as "some teenage mothers." Studies of teen mothers typically only examined infants or young children, and measures of behavioral problems or psychopathology were rarely obtained. Thus, there were only enough studies of teen mothers to permit this comparison for general psychopathology, positive affect/behavior and negative affect/behavior and not for internalizing or externalizing problems. In order to ensure that we were comparing mothers with similarly aged children for these three analyses, we examined the ages of children in studies of teen mothers. Children from these studies ranged in age from 1 to 36 months ($M = 11.39$, SD = 10.33) for negative affect/behavior, from 3.38 to 36 months ($M = 13.53$, SD = 11.96) for positive affect/behavior, and from 28 to 82 months ($M = 52.80$, SD = 26.86) for general psychopathology. Thus, for each of these child constructs, a comparison group of studies was selected in which the mothers on average were older than 20 years of age and the children were within the range of ages of children of teenage mothers studied for each of these three constructs, and effects from these two sets of groups were compared.

Family composition. Because very few studies provided separate analyses for two-parent versus single-parent families, the percentage of two-parent families in samples was calculated when such information was provided. Although the percentage of two-parent families was skewed, with the majority of samples comprised mostly or entirely of two-parent families, there was no clear-cut point in the distribution. Thus, we examined this moderator continuously to test our hypothesis that samples with higher percentages of single-parent families would have stronger associations between maternal depression and child outcomes.

Meta-Analytic Method

We created a database using the Comprehensive Meta-Analysis program (Version 2; Borenstein et al. 2005), which has been used for the analyses of several published meta-analyses. The goals of the study-level analyses were to obtain an unbiased estimate of the population effect size and to examine the homogeneity of effect sizes within each

of these analyses. Separate study-level analyses were conducted for the relation between maternal depression and each of the five child outcome variables. When researchers only stated that no significant effects were found without providing statistics to permit the calculation of an exact effect size, the conservative strategy of assigning a correlation of .00 was adopted. Only results from analyses examining direct relations between maternal depression and the child variables were used; results from analyses controlling for the effects of other variables (i.e., multiple regression analyses, path analyses) were not used. Effect sizes assessing the same child construct within each study were averaged using Fisher's *r*-to-*z* transformation. Studies that involved the same outcomes in different publications were excluded. For studies that included data from more than one source of report on a particular child variable, we averaged effect sizes for use in all analyses except for tests of moderation by source, for which we treated them separately. For studies that included data from both diagnostic interviews and rating scales of mothers' depression, we followed the same procedure. Additionally, an a priori decision was made that in the case of longitudinal studies, only time one results were used. This conservative approach was chosen in order to capture the data for associations with the earliest time of exposure given that later time points of exposure might include effects of prolonged or repeated exposures to maternal depression.

The population effect size for these analyses is estimated by the average effect size, *r*, with each *r* weighted by its sample size. The resulting population effect sizes are interpreted using Cohen's (1988) recommendations that an *r* of at least .10 be termed a small effect, an *r* of at least .24 be termed a medium effect, and an *r* of at least .37 be termed a large effect. At the study-level, the homogeneity estimate (*Q*) follows a chi-square distribution and examines the likelihood that the variation in effect sizes within each analysis is different from that which would be expected to result simply from sampling error. A significant *Q*-value indicates heterogeneity of effect sizes, such that a moderator search is warranted. Additionally, Rosenthal's (1991) Fail-Safe *N* was calculated at the study-level, indicating the number of additional studies with null results that would have to be found in order to bring the mean effect size to 0 (see Table 1).

The goal of the construct-level analyses was to examine potential moderators of the relations between depression in mothers and each of the five child variables. Two types of moderator analyses were conducted, using procedures described by Cooper and Hedges (1994). For categorical moderating variables, categorical model testing procedures were used. These procedures are analogous to an ANOVA, with effect sizes grouped according to the levels of the moderator variable. These groups are compared to examine whether they differ significantly from one another. Categorical model testing yields two homogeneity estimates, a between groups *Q* (*Qb*) and a within-groups *Q* (*Qw*). A significant *Qb* indicates that the subgroups of effect sizes are significantly different from one another. A significant *Qw* indicates that the smaller group of effect sizes is heterogeneous, such that substantial variability among the effect sizes is still present. When an analysis yields a significant *Qb* but the subgroups of effects are not homogeneous, follow-up contrasts should be interpreted with caution, as there is still substantial variability within the subgroups of effects. In order to ensure adequate power for follow-up analyses, contrasts were only performed when subgroups were composed of 5 or more studies. As described by Hedges (1994), standardized contrasts (*g*) were calculated from the difference of effect sizes, and the significance of the contrast was examined by dividing *g* by the pooled variance and comparing the resulting value with the critical value of the Chi-square distribution at $p = .05$.

For the continuous moderator variables, weighted least squares regression procedures were used, as described by Hedges (1994), with effect sizes weighted by the inverse of the variance. Linear regression procedures were used to examine whether a significant relationship existed between the value of the moderating variables and the magnitude of the effect size. For these analyses, the correlation is interpreted as usual, and the *z*-test is a two-sided test of the null hypothesis that the regression coefficient equals zero.

Table 1 Study-level analyses for relations between mothers' depression and children's behavioral and emotional problems

Child variable	*k*	*N*	Weighted mean *r*	95% CI	Q	Fail-safe *N*
Internalizing problems	121	65,619	.23***$_{a,b}$	.22/.24	487.14	54,069
Externalizing problems	111	59,051	.21***$_{a}$	.20/.22	562.11	43,681
General psychopathology	39	9,754	.24***$_{b}$	.22/.26	127.64	4,070
Negative affect/behavior	44	4,818	.15***	.12/.17	226.59	1,033
Positive affect/behavior	29	3,523	−.10***	−.14/−.07	178.09	185

All effect sizes differ at $p < .05$ except as denoted by shared subscripts

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

Results

Study Sample

In total, results from 399 independent effect sizes, from 193 studies published or submitted for publication from 1982 to 2009,[1] met the inclusion criteria. Collectively, these studies included 80,851 mother–child dyads. Sample sizes ranged from 16 to 20,520 families ($M = 423.30$, SD = 1607.46). The mean age of the children in these studies ranged from 9 days to 20 years, with an overall mean of 7.13 years (SD = 5.08 years). In total, 71 (17.8%) effect sizes used clinical samples, and 328 (82.2%) used community samples. Overall, 118 (29.6%) effect sizes came from studies where mothers' depression was measured with clinical diagnostic tools, and 281 (70.4%) effect sizes were from studies relying on mothers' completion of depression symptom rating scales. Only 89 (22.3%) of the effect sizes we were able to calculate were reported separately for boys and girls. Details of these studies are shown in the Electronic Supplementary Material.

Out of concern that sample type and mothers' depression measure may be confounded, we ran a chi-squared test of the association between these two variables. Although statistically significant ($\chi^2 = 16.13$, $p < .001$), the analysis revealed that only about half of the clinical samples (49.3%) had measured mothers' depression with diagnoses while the other half (50.7%) measured depression with rating scales. On the other hand, the majority of community samples (74.7%) used rating scales. Given these distributions, we chose to continue to examine these two potential moderators separately, although we take this into account in the discussion.

In order to reveal the extent to which the theory-based moderators measuring context (% married, % minority, poverty, and teen mothers) were interrelated, we conducted a series of correlational and chi-squared analyses and *t*-tests. Results revealed that studies including a larger percentage of Caucasian families were likely to include a higher percentage of two-parent families ($r = .82$, $p < .001$) and families not in poverty (Spearman's rho = .63, $p < .001$), and the percentage of two-parent families was also positively related to the proportion of families not in poverty (Spearman's rho = .61, $p < .001$). Samples of teen mothers, compared to child age-matched samples of older mothers, had significantly greater percentages of minorities (t [177] = 7.04, $p < .001$), a lower percentages of married couples (t [151] = 6.97, $p < .001$, and were more likely to be in the low-income group (χ^2 [275] = 43.26, $p < .001$).

[1] Studies published before 1982 were considered but none met the inclusion criteria.

Study-Level Analyses

Results of the study-level analyses for the association between maternal depression and child outcome variables are shown in Table 1. All population effect sizes are small in magnitude, accounting for between 1 and 6% of the variance in child outcomes. Additionally, all analyses yielded results that are significantly heterogeneous, indicating the likely presence of moderating variables.

Follow-up contrasts were conducted to examine possible differences in the magnitude of effects across the five child outcome variables. Because the direction of effects differed across variables and we were interested in examining differences in the magnitude rather than direction of effects, contrasts were conducted on the absolute values of the weighted mean effect sizes. Maternal depression was no more strongly associated with internalizing than with externalizing problems in children. Maternal depression was more strongly associated with children's internalizing problems than with negative emotion/behavior ($g = .21$, $p < .001$) or positive emotion/behavior ($g = .30$, $p < .001$). In addition, maternal depression was more strongly associated with their children's general psychopathology than with their externalizing problems ($g = -.05$, $p < .01$) and than their negative ($g = .22$, $p < .001$) and positive affect/behavior ($g = .30$, $p < .001$). Finally, maternal depression was more strongly associated with their children's externalizing problems than with their negative ($g = .17$, $p < .001$) or positive affect/behavior ($g = .25$, $p < .001$) and more strongly associated with negative affect/behavior than with positive affect/behavior ($g = .08$, $p < .05$).

Construct-Level Moderator Analyses for Categorical Variables

Construct-level analyses tested for moderation of the associations between mothers' depression and each of the five child constructs on the set of categorical moderator variables. Results for these analyses, shown in Tables 2 through 6, are presented in turn.

Children's Internalizing Behavioral Problems

Results for categorical moderator analyses of the association between maternal depression and children's internalizing behavioral problems are shown in Table 2.

Among the method-based moderators, the analysis examining moderation by diagnostic versus symptom rating-based approach to mother assessment showed, as predicted, that studies in which the women's depression was diagnosed found significantly larger effect sizes than studies in which the women's depression was determined by a symptom rating scale ($g = .06$, $p < .001$). For the

Table 2 Construct-level moderator analyses for mothers' depression and children's internalizing behavioral problems

Level of moderator	*Qb*	*k*	Weighted mean *r*	95% CI	*Qw*
Assessment of mothers' depression	13.46**				
Diagnosis		31	$.25_{a}$	.23/.26	144.63***
Symptom rating		91	$.22_{a}$	.21/.23	339.16***
Sample type	5.51*				
Clinical		20	.25	.23/.27	113.08***
Community		101	.23	.22/.24	368.55***
Child assessment source	166.37***				
Rated by teacher/other		31	$.15_{c,d}$	.13/.17	150.35***
Rated by mother		68	$.25_{c,e,f}$	.25/.26	255.61***
Self-rating by child		36	$.17_{d,e}$	.15/.19	86.62***
Mixed mother/child report		10	$.15_{f}$	.12/.18	21.73*
Child gender	38.92***				
Boys		13	$.16_{g,h}$	.13/.18	32.68**
Girls		13	$.25_{g}$	.23/.28	33.67***
Mixed		95	$.24_{h}$	.23/.25	381.86***
Family income	9.39**				
Low		24	$.27_{i}$	.24/.29	75.78***
Mid/high/mixed		92	$.23_{i}$	.22/.24	399.24***
Age of mother	16.99***				
Older than teenage		83	.23	.23/.23	327.84***
Teenage		3	.26	.12/.39	7.01*
Some teenage		2	.003	−.10/.11	.37

Effect sizes that share subscripts differ at $p < .05$. Only subgroups containing at least 5 studies included in analyses of difference

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

analysis examining moderation by sample type, as predicted, effect sizes were significantly larger for clinical relative to community samples ($g = .05$, $p < .05$). For the analysis examining moderation by the source of data on the child's internalizing behavioral problems, as predicted, studies relying on mothers' reports found significantly larger effect sizes than studies relying on teachers' or others' reports ($g = .24$, $p < .001$), children's self-report ($g = .18$, $p < .001$), and on mother–child combined report ($g = .22$, $p < .001$).

Among the analyses of theory-based moderators, for the analyses of child gender, as predicted, the weighted mean effect sizes for studies of girls was significantly larger than for studies of boys ($g = .20$, $p < .001$). In order to determine whether this gender difference was primarily accounted for by studies with older samples, as hypothesized, we analyzed the distribution of child age within effect sizes separated by gender, finding that there was a distinct split, with 13 effect sizes from studies of children under 5 years old, and 21 effect sizes from studies of children over the age of 10. We performed independent samples *t*-tests to determine whether mean weighted effect sizes differed significantly by gender for both the younger and the older samples. As predicted, among the younger samples, there was no significant gender difference in mean weighted effect sizes, although the weighted effect size was higher for girls (t (11) = 1.01, two-tailed $p = .34$, $r = .29$). Contrary to prediction, there was also no significant gender difference among the older samples, although the mean weighted effect size was also higher for girls (t (21) = 1.71, two-tailed $p = .10$, $r = .37$). A comparison of the two effect sizes using Fisher *r*-to-*z* comparisons was also not significant ($p = .82$). That is, although the overall sample of studies showed a stronger association between maternal depression and internalizing problems for daughters than for sons, that gender difference was not significant in subsamples of studies with older or younger children. For family income, as predicted, the studies that sampled low-income (poverty) families yielded significantly larger effect sizes compared to studies that sampled middle-income or higher or mixed-income populations ($g = .08$, $p < .01$). We were unable to analyze moderation by age of mother because only three studies reported associations for samples of teenage mothers, our construct of interest.

Children's Externalizing Behavioral Problems

Results for these categorical moderator analyses are shown in Table 3.

Table 3 Construct-level moderator analyses for mothers' depression and children's externalizing behavioral problems

Level of moderator	*Qb*	*k*	Weighted mean *r*	95% CI	*Qw*
Assessment of mothers' depression	.25				
Diagnosis		29	.21	.20/.22	116.35***
Symptom rating		83	.21	.20/.22	453.881***
Sample type	.39				
Clinical		23	.22	.19/.24	134.31***
Community		88	.21	.20/.22	428.42***
Child assessment source	152.08***				
Rated by teacher/other		37	$.14_{a,b}$	.12/15	165.02***
Rated by mother		75	$.23_{a,c,d}$	.22/.24	392.42***
Self-rating by child		15	$.11_{b,c,e}$	.08/.14	16.39
Mixed mother/child report		6	$.14_{d,e}$	.10/.18	10.65
Child gender	2.14				
Boys		17	.22	.20/.24	50.09***
Girls		9	$.23_{f}$	.20/.25	22.08*
Mixed		85	$.21_{f}$	.20/.22	495.65***
Family income	52.27***				
Low		24	$.29_{g}$	.27/.31	76.12***
Mid/high/mixed		83	$.20_{g}$	.19/.21	434.36***
Age of mother	2.62				
Older than teenage		70	.20	.19/.21	412.69***
Teenage		2	.08	−.08/.23	.52
Some teenage		4	.20	.17/.23	29.82***

Effect sizes that share subscripts differ at $p < .05$. Only subgroups containing at least 5 studies included in analyses of difference

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

Among the method-based moderators, contrary to prediction, effect sizes were no larger among studies in which the women's depression was determined by meeting diagnostic criteria rather than by a symptom rating scale. Also contrary to prediction, effect sizes were not significantly larger for clinical samples relative to community samples. For the analysis examining moderation by the source of data on the child's externalizing behavioral problems, as predicted, results revealed that studies relying on mothers' reports found significantly larger effect sizes than studies relying on children's self-report ($g = .26$, $p < .001$), mother–child combined report ($g = .19$, $p < .001$), or teachers' or others' reports ($g = .20$, $p < .001$).

Among the theory-based moderators, the exploratory analysis of gender differences revealed no significant difference between the weighted mean effect sizes for studies of girls relative to studies of boys. As predicted, the studies that sampled low-income families yielded significantly higher effect sizes compared to studies that sampled middle-income or higher or mixed-income populations ($g = .19$, $p < .001$). We were unable to analyze moderation by age of mother because only two studies reported associations for samples of teenage mothers.

Children's General Psychopathology

Results for the analyses of categorical moderators of the association between maternal depression and children's general psychopathology are shown in Table 4.

Among the method-based moderators, results failed to support the expected larger effect sizes from studies in which the women's depression was diagnosed relative to studies relying on a rating scale ($g = .08$, $p < .05$). For the analysis examining moderation by sample type, consistent with predictions, effect sizes were significantly larger for clinical samples relative to community samples ($g = .22$, $p < .05$). For the analysis examining moderation by the source of data on the child's general psychopathology, there were sufficient numbers of studies to be able to compare three groups of studies: teacher or other reported, mother reported, and child reported. As predicted, results revealed that studies relying on mothers' reports found significantly larger effect sizes than studies relying on children's self-report ($g = .30$, $p < .001$) or than studies relying on teachers' or others' reports ($g = .30$, $p < .001$).

Among the theory-based moderators, with fewer than five studies of girls or of teen mothers, we were unable to examine child gender or teenage motherhood as moderators. As

Table 4 Construct-level moderator analyses for mothers' depression and children's general psychopathology

Level of moderator	*Qb*	*k*	Weighted mean *r*	95% CI	*Qw*
Assessment of mothers' depression	.08				
Diagnosis		18	.24	.20/.28	56.35***
Symptom rating		21	.24	.21/.27	71.21***
Sample type	6.03*				
Clinical		5	$.34_{a}$	.26/.41	10.44*
Community		34	$.23_{a}$	.21/.25	111.17***
Child assessment source	72.50***				
Rated by observer		13	$.13_{b}$	.10/.17	37.44***
Rated by mother		24	$.27_{b,c}$	.25/.29	43.99**
Self-rating by child		12	$.14_{c}$	.10/.17	52.59***
Mixed mother/child report		1	.04	−.22/.29	.00
Child gender	6.37*				
Boys		5	$.16_{d}$	.09/.23	12.48*
Girls		4	.19	.12/.27	6.19
Mixed		30	$.25_{d}$	.23/.27	102.60***
Family income	4.11*				
Low		8	$.30_{e}$	.24/.36	3.93
Mid/high/mixed		29	$.23_{e}$	.21/.25	112.04***
Age of mother	10.95**				
Teenage		3	.31	.27/.36	.57
Older		26	.22	.20/.25	87.83***

Effect sizes that share subscripts differ at $p < .05$. Only subgroups containing at least 5 studies included in analyses of difference

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

predicted, the studies that sampled low-income families yielded significantly higher effect sizes compared to studies that sampled middle-income or higher or mixed-income populations ($g = .15, p < .05$.

Children's Negative Affect and Behavior

Results for the analyses of categorical moderators of the association between maternal depression and children's negative affect and behavior are shown in Table 5. Analyses of method-based moderators revealed no significant moderation by approach to assessing mothers' depression or by sample type. We were unable to examine moderation by the source of data on the child's negative affect or behavior, because all of the studies relied on teacher or other reporter, typically a researcher observer.

Among theory-based moderators, we were unable to analyze the exploratory hypothesis of moderation by child gender because too few studies reported separate results by gender. As predicted, studies that sampled low-income families yielded significantly higher effect sizes than studies that sampled middle-income or higher or mixed-income populations ($g = .21$, $p < .01$). Contrary to prediction, studies that sampled teen mothers found significantly smaller effect sizes compared to studies that sampled older mothers and age-matched children ($g = .20$, $p < .001$).

Children's Positive Affect and Behavior

Results for the analyses of categorical moderators of the association between maternal depression and children's positive affect and behavior are shown in Table 6. Analyses of method-based moderators revealed no significant moderation by approach to assessing mothers' depression or by sample type. We were unable to examine moderation by the source of data on the child's positive affect or behavior, because all of the studies relied on teacher or other reporter, typically a researcher observer.

Among the theory-based moderators, we were unable to analyze moderation by child gender because too few studies reported separate results for boys relative to girls. Moderation by family income was statistically significant, and results were in the predicted direction of higher effect sizes for studies of low-income samples compared to others, although the two groups of studies did not significantly differ from each other. Contrary to prediction, results of analyses of moderation by age of mother (teen) were not significant.

Moderator Analyses for Continuous Moderator Variables

A series of weighted least squares regression analyses were conducted to examine whether the three hypothesized

Table 5 Construct-level moderator analyses for mothers' depression and children's negative affect/behavior

Level of moderator	*Qb*	*k*	Weighted mean *r*	95% CI	*Qw*
Assessment of mothers' depression	1.77				
Diagnosis		15	.12	.07/.17	25.04*
Symptom rating		29	.16	.12/.19	199.78***
Sample type	1.57				
Clinical		8	.20	.11/.30	20.05**
Community		36	.14	.11/.17	204.96***
Child assessment source	–				
Rated by observer		44	.15	.12/.17	226.59***
Child gender	9.62**				
Boys		2	.10	−.11/.30	.86
Girls		2	.45	.26/.60	14.43***
Mixed		40	.14	.11/.17	201.68***
Family income	8.86**				
Low		18	$.23_a$	.17/.29	71.30***
Mid/high/mixed		26	$.12_a$	.09/.17	146.42***
Age of mother	11.22**				
Teenage		9	$.10_b$	.05/.15	58.61***
Older-child—age matched		25	$.21_b$	.17/.25	125.36***

Effect sizes that share subscripts differ at $p < .05$. Only subgroups containing at least 5 studies included in analyses of difference

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

Table 6 Construct-level moderator analyses for mothers' depression and children's positive affect/behavior

Level of moderator	*Qb*	*k*	Weighted mean *r*	95% CI	*Qw*
Assessment of mothers' depression	3.06				
Diagnosis		10	−.06	−.12/.00	6.88
Symptom rating		19	−.12	−.16/−.08	168.14***
Sample type	.52				
Clinical		5	−.06	−.17/.05	3.37
Community		24	−.11	−.14/−.07	174.19***
Child assessment source	–				
Rated by observer		29	−.10	−.13/−.07	178.09***
Child gender	39.87***				
Boys		2	.15	−.06/.34	.89
Girls		2	−.63	−.74/−.48	58.64***
Mixed		25	−.09	−.13/−.06	78.69***
Family income	5.02*				
Low		12	−.18	−.25/−.10	137.18***
Mid/high/mixed		17	−.08	−.12/−.04	35.89**
Age of mother	.76				
Teenage		6	−.11	−.16/−.05	99.78***
Older than teenage—child age matched		12	−.14	−.20/−.08	59.72***

Effect sizes that share subscripts differ at $p < .05$. Only subgroups containing at least 5 studies included in analyses of difference

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

moderators that were continuously measured, the mean age of children in the samples, the proportion of married parents in the samples, and the proportion of ethnic minorities in the samples, were significantly related to the magnitude of effect sizes. Results for these analyses are shown in Table 7.

Table 7 Regression slope *b* (parameter estimates) between continuous study variables and the effect size for mothers' depression and children's emotional and behavioral problem and affect functioning

Child variable	Child age *k*	% Married *k*	% Minority *k*
Internalizing behavioral problems	−.0004*** (R^2 .06)	−.01 (R^2 .001)	.05*** (R^2 .05)
	120	63	75
Externalizing behavioral problems	−.0007*** (R^2 .09)	−.11*** (R^2 .09)	.09*** (R^2 .13)
	111	59	63
General psychopathology	−.0001*** (R^2 .12)	−.06 (R^2 .01)	.11 (R^2 .02)
	39	20	21
Negative affect/behavior	−.001** (R^2 .04)	−.32** (R^2 .37)	.20*** (R^2 .12)
	42	13	31
Positive affect/behavior	−.0001 (R^2 .0004)	.18 (R^2 .05)	−.11† (R^2 .02)
	27	8	20

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

The mean age of children in the samples was significantly negatively related to the magnitude of the effect sizes for the relation between maternal depression and children's internalizing problems, externalizing problems, general psychopathology, and negative affect/behavior. With all four child variables, as predicted, the younger the mean age of the samples was associated with stronger effects. Contrary to prediction, age was not related to effect sizes for positive affect/behavior.

The proportion of married parents in the samples was significantly negatively related to the magnitude of the effect sizes for the relation between maternal depression and two of the child variables: children's externalizing problems and negative affect/behavior. In both cases, as predicted, the lower percentage of married parents in the samples was associated with stronger effect sizes.

The proportion of ethnic minority parents in the samples was significantly positively related to the magnitude of the effect sizes for the relation between maternal depression and children's internalizing and externalizing problems and to negative affect/behavior and marginally significantly ($p = .06$) negatively related for children's positive affect/behavior. Given that positive affect is interpreted in the opposite direction as the other constructs, the results are consistent in showing that, as predicted, the higher percentage of ethnic minority parents in the samples was associated with stronger effect sizes for associations between maternal depression and children's greater internalizing and externalizing problems, higher levels of negative affect/behavior and (marginally) lower levels of positive affect/behavior, although not significantly related to strength of the effect sizes for general psychopathology.

Discussion

Although a burgeoning literature has documented the relation between depression in mothers and adverse child outcomes, little is known about the extent to which maternal depression accounts for these outcomes, how much the strength of associations with maternal depression may vary across different aspects of child psychopathology or affective functioning, what child or family characteristics might more precisely identify the groups at highest risk and what methodological variables may cloud an understanding of the strength of associations between maternal depression and child outcomes. Answers to these questions are essential for developing and testing theoretical models to explain the development of psychopathology in children of depressed mothers and for informing the design of preventive interventions (Beekman et al. 2010; National Research Council and Institute of Medicine 2009). We addressed these issues by determining the strength of the association between maternal depression and broad band constructs of child psychopathology and positive and negative affect/behavior, estimating the population effect sizes, and also examining the role of theoretically and methodologically relevant variables in moderating those associations.

Consistent with the developmental psychopathology notion of multifinality (Cicchetti and Rogosch 1996), study-level analyses confirmed that depression in mothers is associated not only with children's internalizing problems but also with externalizing problems and general psychopathology. The effect sizes for relations between maternal depression and children's internalizing and externalizing problems and general psychopathology were all statistically significant, albeit small in magnitude. Thus, the knowledge generated by this review of degrees of association between maternal depression and psychopathology-related outcomes furthers understanding that the risk to children encompasses psychopathology broadly.

Notably, the relation between maternal depression and internalizing problems was not significantly stronger than the relation between maternal depression and externalizing problems. An important caveat in interpreting this finding

is that it is *not inconsistent* with the notion of specificity in the mechanisms through which maternal depression may be related to internalizing relative to externalizing problems in youth. Thus, important next steps in this area of study are to examine mechanisms and developmental pathways whereby depression may be similarly or differently related to the emergence of internalizing relative to externalizing problems or to their co-occurrence in children and adolescents (Zahn-Waxler et al. 1990). For instance, there may be some specificity in genetic transmission, given distinct patterns of heritability of depression (or neuroticism) relative to externalizing problems (Kovacs et al. 1997; Williamson et al. 1995). Other mechanisms that may relate differently to internalizing relative to externalizing problems include particular patterns of parenting. Both withdrawn and harsh or inconsistent parenting have been found to be associated with maternal depression (Lovejoy et al. 2000), and harsh, inconsistent parenting in particular has been associated with children's externalizing problems (Patterson et al. 1992), suggesting specificity of outcomes to the children that may vary with the depressed mother's particular predominant parenting style. Next steps needed are for research to test theories of possible outcome-specific pathways to risk, including accounting for likely comorbidity, and for tests of more targeted preventive interventions, which themselves can serve as tests of theorized pathways. Researchers would also do well to employ genetically sensitive designs to permit a more rigorous test of gene–environment interactions (Rutter 2007), which may be especially relevant to children of depressed mothers given both heritability of depression and the stressors to which they are likely to be exposed (Hammen 2002).

Given a developmental psychopathology perspective, we extended the examination of child functioning beyond psychopathology per se to include studies of children's negative as well as positive affective functioning. Consistent with our hypotheses, we found statistically significant associations between maternal depression and both children's negative and positive affect/behavior, although the associations were significantly smaller than for internalizing, externalizing, and general psychopathology and significantly stronger for associations with negative affect/behavior than for positive affect/behavior. These findings are consistent with accumulating theory and research highlighting the importance of emotions and emotion regulation in children with depressed mothers (Cicchetti et al. 1995; Garber et al. 1991). Our findings of statistically significant relations between maternal depression and children's positive and negative affect/behavior are consistent with theories and research that depression disorders are characterized by the combination of high negative affect and low positive affect or anhedonia (Clark and Watson 1991). The findings underscore the need for research on the role that a dispositional tendency for high negative affect and low positive affect may play in predisposing children of depressed mothers to the later development of depression (Klein et al. 2009). Such tendencies may reflect genetic, neurobiological, or social learning mechanisms of transmission or their interaction. Most needed to clarify potential mechanisms are studies with genetically informed designs and that include measures of neuroendocrine stress levels (baseline and reactivity) and psychophysiological measures such as EEG asymmetry and vagal tone, as well as experimental designs to test the potential roles of learning. Studies of transactional processes are also needed given that mothers who are struggling with depression may be especially challenged by infants or children who exhibit little positive affect or enjoyment or high levels of negative affect.

Our meta-analyses also showed substantial variability across results, which was partially explained by the moderator analyses. Most importantly, the results of our theory-driven moderator analyses have implications for theoretical models as well as for sample selection in indicated prevention studies (Beekman et al. 2010). Our findings for most of the theory-based moderator analyses were consistent with our hypotheses, including those examining family context (poverty, single-parent households, and minority ethnicity, although not teenage motherhood) and child characteristics (age and sex). We discuss each of these in turn.

Consistent with our hypotheses, we found moderation by child age for associations between depression in mothers and children's internalizing and externalizing problems, general psychopathology, and negative (although not positive) affect/behavior. As predicted, effect sizes were stronger for younger children (effect sizes decreased as studies examined older children and adolescents). In line with Connell and Goodman's (2002) findings, we had hypothesized that the relation between maternal depression and child functioning would be strongest in studies examining younger children and had no reason to expect that this pattern would differ by the type of emotional or behavioral problem examined in the children.

Interpretation of this finding is constrained by studies rarely having selected samples based on children's history of exposure to maternal depression or imposing statistical controls for timing or extent of prior exposures. As a result, samples of older children with depressed mothers most likely included several subgroups: some children who had only recently been first exposed, others who had been exposed essentially continuously since early in their lives, and still others who had been exposed intermittently since early in their lives. Nonetheless, our findings are consistent with the notion of sensitive periods in that: (1) children

who are younger when first exposed to their mother's depression may be more vulnerable to the development of psychopathology than children not exposed until later; (2) children who are first exposed later in development may have experienced more years of healthy development prior to the exposure; (3) later in development, children are less exclusively dependent on their mothers, with fathers, teachers, and peers having more influence, potentially attenuating some of the effects of living with a depressed mother or having lived with a depressed mother in the past; (4) with increasing cognitive maturity, older children may be better able to understand their mothers' symptoms than younger children and may have developed better emotion regulation and social information processing skills (Crick and Dodge 1996; Grych and Fincham 1990). However, these interpretations of the finding that younger children are at greater risk are based on the premise that the age at which children were studied was their age of first exposure and thus must be considered tentative. Researchers providing such information, along with more longitudinal studies, will allow for mapping alternative courses of exposure and testing differential predictors of pathways to outcomes.

Gender effects were also consistent with our hypotheses, as maternal depression was more strongly associated with internalizing problems in girls than in boys. Contrary to Sheeber et al.'s conclusion (2002), this difference was not specific to samples of older children. The gender difference did not extend to externalizing problems or general psychopathology (for which maternal depression was equally associated with problems in both boys and girls). The findings raise interesting questions about how, regardless of age, girls may be more vulnerable and, conversely, boys less vulnerable to the development of internalizing problems when mothers are depressed. Overall, our pattern of findings could be explained by: (1) heritability of depression being substantially stronger in women than in men (Kendler et al. 2001); (2) gender-specific socialization mechanisms; (3) girls, relative to boys, both experiencing more stressors (especially in the interpersonal domain) associated with depression in mothers and also being more sensitive to the stress context often associated with depression in mothers (Hammen 2002; Hankin et al. 2007), or (4) particular styles of parenting (e.g., more aversive or less warm, responsive) that is either more often used with girls than boys or to which girls may be more sensitive than boys. In contrast, pathways to externalizing problems in children of depressed mothers appear to not be gender specific. Researchers are strongly encouraged to report findings separately by gender, to develop and test gender-specific models of risk to children of depressed mothers suggested by these findings, and to test whether or not they vary for older, relative to younger children.

In terms of family characteristics, consistent with predictions, effect sizes for associations between depression in mothers and children's internalizing and externalizing problems, general psychopathology, and negative and positive affect/behavior were stronger for studies that sampled families in poverty relative to studies of families in higher or mixed-income levels. Thus, poverty seems to be a broad-scale enhancer of risk in relation to depression in mothers, regardless of the aspect of child outcome assessed. Since poverty is associated with maternal depression (Liaw and Brooks-Gunn 1994), this is an important population to study further. Based on the Goodman and Gotlib (1999) model for the transmission of risk, important questions raised by these findings include whether poverty is associated with depression being more severe or chronic for women and whether the larger effect sizes can be at least partially explained by children living in poverty experiencing more stressors, including prenatal stressors, and fewer resources than children of depressed mothers who are not living in poverty. Our findings suggest the need for testing models of risk for the development of psychopathology in children of depressed mothers that are potentially specific to children living in poverty. The findings also provide strong support for one of the recommendations of the recent National Research Council and Institute of Medicine report (2009), to conduct research and design and test interventions on vulnerable populations.

In contrast to the finding that poverty was associated with stronger effect sizes regardless of the child outcome, findings for the other hypothesized family characteristic moderators revealed specificity depending on the particular child outcome. For example, studies with samples that included more single-parent households yielded higher effect sizes of association between maternal depression and children's externalizing problems and negative affect/behavior only and not for internalizing problems, general psychopathology, or positive affect/behavior. Studies with samples that included more ethnic minorities similarly yielded higher effect sizes of association between maternal depression and children's externalizing problems, but also for internalizing problems and positive affect/behavior and not for general psychopathology or negative affect/behavior. Although such outcome-specific findings were not predicted, they suggest potentially fruitful avenues of research for example, in exploring how father absence in families with depressed mothers may be associated specifically with greater risk for children's externalizing problems and negative affect/behavior relative to other outcomes.

In terms of teenage mothers, unfortunately, there were insufficient samples to test the role of this moderator for internalizing, externalizing or general psychopathology. Further, contrary to prediction, this moderator was not

significant for positive affect/behavior and was significant but in the opposite direction for children's negative affect/behavior. The particular pattern of findings was not expected and is difficult to interpret. The small number of studies that examined teen mothers separately also suggests caution in drawing conclusions about the role of teen parenting in associations between maternal depression and these child outcomes. Further, we found that samples of teen mothers, compared to child age-matched samples of older mothers, were characterized by higher percentages of ethnic minorities, single-parent households, and poverty. Thus, teenage mothers are likely to experience a range of stressors related to these contexts, which themselves might increase their rates of depression and interfere with their ability to provide good quality parenting. Given our findings on the limitations of the research to address such questions, important next steps in the research are tests of mechanisms of transmission of risk in this particularly vulnerable population (National Research Council and Institute of Medicine 2009).

Taken as a whole, our findings on theory-based moderators support moving beyond main effects models of the role of maternal depression and developing models that are specific to particular aspects of children's functioning. In particular, child gender (being female) needs to play a stronger role in models of internalizing problems associated with maternal depression, whereas child gender seems less relevant for these other outcomes in association with maternal depression. Similarly, father absence needs to play a stronger role in models of externalizing problems and negative affect/behavior in association with maternal depression, whereas father absence may play less of a role in the link between maternal depression and children's internalizing or positive affect/behavior. Important next steps are to design studies of these potentially population-specific causal pathways, explaining how these child or family characteristics enhance risk for the development of specific aspects of psychopathology. The results of our correlational findings suggest, not surprisingly, that samples of families in poverty typically also have higher percentages of single-parents, teen mothers, and ethnic minorities. Thus, future studies would benefit from exploring how, when mothers are depressed, particular combinations of risk factors work together to increase children's risk (e.g., Silberg and Rutter 2002), consistent with such theoretical considerations in regard to psychiatric disorders (Kraemer 2003) and developmental psychopathology (Masten 2001).

To the extent that future studies continue to yield findings that models for the risks to children of depressed mothers are specific to particular child and family characteristics and to particular child outcomes, these findings also have implications for the design of preventive interventions. Prevention researchers increasingly recognize that, despite the general promise of prevention relative to treatment, universal prevention programs are unlikely to be the most effective (Beekman et al. 2010). Thus, it is compelling to identify the subsets of children of depressed mothers who are at highest risk for the development of psychopathology in order to inform the design of indicated prevention. In particular, our findings are promising in being able to identify high-risk groups at risk for particular outcomes with greater precision when the typically limited resources may prohibit targeting interventions to all children of depressed mothers. Our findings are also sobering in their implications for designing prevention studies in that the criteria for such programs require a risk factor with strong and stable associations with the outcomes of concern. We thus suggest caution to ensure that designs of preventive interventions are based on the strength of the evidence.

Our findings are also helpful in addressing the question of how much variance in child psychopathology and affective functioning is accounted for by maternal depression and, conversely, how much is not. An effect size of the magnitude we found for internalizing problems, for example, indicates that about 68% of children of depressed mothers (diagnosed or high symptom scoring) were worse off than the average child of a nondepressed mother. Conversely, this magnitude of effect size also means that about 32% of the children whose mothers had been depressed scored similar to, or better than, those of children of nondepressed mothers. Moreover, the findings show a range, albeit restricted, of effect sizes when the moderators were taken into account. For example, with internalizing problems, although the overall weighted mean r was .22 (95% CI = .22–.23), the effect size was as small as .15 for the subsample of studies of boys and as large as .26 for the subsample of families in poverty. Despite the ranges, it is striking that, for any of the child outcomes, most of the variance is *not* accounted for by maternal depression even when the moderators are taken into account. Although the effect sizes are within the range identified in meta-analyses of other risk factors and across a range or other predicted associations and are clinically meaningful (Amato and Keith 1991; Kitzmann et al. 2003; Meyer et al. 2001), they underscore the importance of developing and testing models with multiple co-occurring risk factors. As Sameroff has proposed, cumulative or interacting effects are likely to be the most accurate predictors of child outcomes, although it is still important to develop and test theories for the specific mechanisms and pathways that lead to specific outcomes (Sameroff et al. 2003).

In terms of methodological sources of variance, we found limited support for the hypothesis that studies identifying depression in mothers by clinical diagnosis rather than by self-report of symptoms would yield larger effects.

This was true only for children's internalizing problems and general psychopathology and not for externalizing problems or for positive or negative affect/behavior. Although this particular pattern of findings does not have a clear explanation, the overall findings suggest less reason for concern about how depression is measured in mothers than had been suspected. Nonetheless, our findings suggest that at least for some outcomes, models of risk may benefit from taking into account potentially greater heritability or greater impairment that might be associated with diagnosed depression relative to high depression symptom ratings. These models could be tested in studies that specifically address these constructs, including genetically informed designs, tests of genetic polymorphisms as moderators, or tests of impairment as moderators.

Similarly, studies using clinical samples of women presenting for treatment of depression yielded larger effects than studies using samples of women recruited from the general community, although this predicted pattern was, like assessment method, supported only for children's internalizing and general psychopathology and not for externalizing or negative or positive affect/behavior. Although this particular pattern of findings is also difficult to explain, it is possible that the important differences that have been found between treated and untreated samples of adults with depression (Kendler 1995; W. E. Narrow 2002, personal communication) are related to only certain aspects of children's psychopathology. To test this notion, it will be important for researchers to examine what it is about differences between women suffering from depression who do versus do not present for treatment that, according to our findings, strengthens the associations between maternal depression and internalizing problems and general psychopathology.

In contrast to these mixed findings on the support of method-based moderators, strong and consistent support was found for the prediction that the relation between maternal depression and child outcomes would be strongest when depressed mothers provided the information on child outcomes, relative to teachers or laboratory observers or clinicians, relative to children's self-reports, and relative to assessments that relied on a combination of maternal and child report (such as with clinical diagnostic interviews that merge reports from mothers and children). Support for this hypothesis was found for internalizing and externalizing problems and general psychopathology, all three outcomes on which there were alternative sources of child assessment allowing for a test of this moderator. These findings suggest that depressed mothers may be more sensitive to signs of emotional and behavioral disturbances in their children than are other informants or may be negatively biased in their perception of their children. Our pattern of findings is consistent with the conclusions of Fergusson et al. (1993) and Boyle and Pickles (1997) who, with their use of statistical modeling techniques, concluded that any tendency to over-report child behavioral problems on the part of mothers with depression represents a significant but small contribution to the findings.

Limitations and Future Directions

Several limitations to both the current meta-analysis and the literature on which it relied should be acknowledged.

Tests of Moderation

First, we were limited due to the numbers of studies with data allowing tests of specific moderators. In particular, more research is needed to examine family contextual effects in more detail. For all of the contextual variables we examined, limited data were available to provide strong tests of contextual effects. Very few studies systematically examined the occurrence of depression in mothers from diverse social and economic backgrounds and the potential impact of such contextual differences, as most studies sampled largely homogeneous, middle- and upper-middle-income, predominantly Caucasian families.

We were also unable to examine the timing and course of mothers' depression, which is likely to be related to the degree of association with child psychosocial functioning (Goodman and Gotlib 1999). The mean age of children studied in the current analyses is only a rough proxy for the extent and timing of the children's exposure to depression in their mothers and masks what is likely to be large variability in timing and course. Findings from longitudinal studies support the notion that children of mothers with more chronic depression have worse outcomes such as: (1) higher rates of insecure attachment (Campbell et al. 1995; Teti et al. 1995), (2) lower school readiness and verbal comprehension at 36 months (NICHD Study of Early Childcare 1999), and (3) more severe behavioral problems and more impaired cognitive functioning at 5 years of age (Brennan et al. 2000). More studies are needed of age at first exposure.

Similarly, we were unable to take into account potential moderation in relation to psychiatric disorders that may have been comorbid with the depression in mothers. Comorbidity with maternal depression may convey greater risk to children than depression that occurs alone. Foley et al. (2001), in a large community-based twin sample, found that maternal depression alone was associated with a .15 increase in children's depression symptoms, whereas maternal depression comorbid with simple phobia was associated with a .44 increase in children's depression symptoms. Moreover, in associations with psychiatric disorders in the children, rather than symptoms of disorder,

maternal depression alone, i.e., not comorbid with another disorder, was not associated with significantly increased odds for any child disorder, including depression.

More tests are also needed of additional child variables, beyond age and gender, as potential moderators of the association between maternal depression and the development of psychopathology. Particularly promising are studies of differential susceptibility (Belsky et al. 2007) or the orchid hypothesis (Ellis and Boyce 2008), proposing that some children are more susceptible to both the adverse effects of negative and/or maladaptive parenting and the favorable effects of positive and/or adaptive parenting. Examples include the following: (1) infants with easier temperaments being less vulnerable to the inadequate parenting associated with maternal depression (Bates et al. 1985); (2) observed child noncompliance during family interactions at age two predicting concurrent elevated maternal depressive symptoms (although not the linear rate of change in maternal symptoms from ages two to four), which in turn mediated increases in youth internalizing and externalizing problems from ages two to four (Gross et al. 2008); (3) increases in children's depressive symptoms following increases in their parent's level of depressive symptoms found to be greater among children with depressogenic inferential styles, especially girls (Abela et al. 2006). More such tests promise to reveal not only which children are more vulnerable but also why or how.

Causal Processes

Second, these analyses do not address causal processes. The vast majority of the studies were cross-sectional and correlational, and we took the conservative meta-analytic approach to using only time one data from the subset of longitudinal studies. Thus, although the weight of evidence supports maternal depression being associations with children's emotional and behavioral problems, causation, and direction of association are not established (Kraemer 2003). It is also important to consider the child's role in exacerbating mothers' depression or even contributing to the causes of mothers' depression. More broadly, transactional models will be needed to explain the unfolding of the influences between maternal depression and child characteristics over time (Goodman 2007; Sameroff and MacKenzie 2003). To understand the alternative developmental pathways that children may follow in relation to exposure to depression in their mothers, researchers need to design longitudinal studies or experimental paradigms with developmentally sensitive measures of vulnerabilities and outcomes. Once a body of such knowledge accumulates, it will be important to apply quantitative and qualitative methods to summarize those findings.

Although many of the longitudinal studies that met inclusion criteria were limited by only two data points, a short interval between data collection points, or lack of control for earlier levels of symptoms, other more sophisticated longitudinal studies have been conducted, and these studies are worth highlighting in more detail for their potential to address causal processes. Among studies that utilized complex statistical techniques with longitudinal data to ascertain the direction of influence between maternal depressive symptoms and child outcomes: (1) Forbes et al. (2006), using cross-lagged modeling, detected specific mother-to-child directional effects for maternal depressive symptoms predicting an interaction of child frontal EEG asymmetry and child negative affect expression; (2) Gross et al. (2008), using latent growth curve modeling and structural equation modeling to create reciprocal models, found consistent effects for earlier maternal depression predicting later anti-social behavior in adolescent boys; (3) also using latent growth modeling, Garber and Martin (2002) found that maternal depression history was related to the initial level of offspring depressive symptoms in grade 6, but was not related to individual differences in the rate of change in adolescent depressive symptoms from grades 6 to 11; and (4) Leve, Kim, and Pears (2005) found that maternal depressive symptoms at age 5 related to the initial level of internalizing symptoms for boys (but not girls), as well as to greater growth in symptoms in both boys and girls from ages 5 to 17. Of note, no relations between age 5 maternal depressive symptoms and growth parameters for either boys or girls were found for externalizing symptoms, highlighting the importance of specificity in youth outcomes. One notable limitation of the latter two studies is that only baseline maternal symptoms were considered, although maternal depressive symptoms, themselves, are likely to change over time, and it is possible that differences in the trajectories of maternal depressive symptoms might predict variations in the growth trajectories of teens' symptoms over time. Overall, these findings are promising of the potential of longitudinal studies to reveal causal pathways and processes.

In addition to longitudinal studies, treatment studies provide quasi-experimental evidence of the importance of maternal depression in affecting youth functioning. Studies examining the extent to which treatments that improve maternal depression yield improvements in youth functioning (e.g., Pilowsky et al. 2008; Weissman et al. 2006) are consistent with the causal importance of maternal depression. However, such findings do not rule out alternative causal mechanisms such as genetic vulnerabilities, neurobiological dysregulation, temperament vulnerabilities, exposure to stressors, and inadequate parenting, many of which might be better addressed with experimental

studies (Garber et al. 2009). In a few recent examples of such an approach, (1) the effects of a parent training program for high-risk families of 2-year-old children on reducing early behavioral problem trajectories from age 2 to 4 were mediated by reduced depressive symptoms in mothers (Shaw et al. 2005); and (2) a group family-based cognitive intervention with families with depressed parents was effective in reducing rates of depression, anxiety, and other internalizing problems and, marginally, externalizing problems, in the children 12 months later, relative to controls (Compas et al. 2009). Such intervention studies underscore the potential for improving children's lives by reducing maternal depression.

Role of Fathers

Third, effects of paternal psychopathology should be taken into account in future studies of associations between maternal depression and child functioning, expanding beyond the mere presence/absence of fathers in the household. In light of assortative mating effects (Merikangas and Brunetto 1996), maternal depression is likely to co-occur with paternal psychopathology, and this co-occurring psychopathology is likely to play a role in explaining some of the variation in child outcomes. Paternal psychopathology could increase children's genetic risk for psychopathology as well as contribute to adverse qualities of the child-rearing environment. Conversely, a healthy father could protect the child by providing role models of healthy behavior, cognitions, affect, and interpersonal relationships, and the opportunity for the child to receive from at least one parent the qualities of parenting known to facilitate healthy development. In addition, the child could benefit if the depressed mother experiences support from the healthy father, which may facilitate the mother providing better quality parenting. Foley et al. (2001) found that maternal depression was associated with a significant increase in children's psychiatric disorders only when paternal psychopathology was also present. For children's levels of psychiatric symptoms, maternal depression co-occurring with paternal alcoholism was associated with increased levels of conduct disorder symptoms and oppositional defiant disorder symptoms, especially in male offspring. Similarly, Goodman et al. (1993) found that fathers' psychiatric status explained a significant proportion of the variance in the social and emotional competence of children of clinically depressed mothers. While a few studies have examined the role of fathers' psychiatric status as a moderator (e.g., Carro et al. 1993; Conrad and Hammen 1993; Eiden and Leonard 1996; Thomas and Forehand 1991; Weissman et al. 1984), more studies are needed of psychopathology and parenting involvement from fathers to understand both the extent and mechanisms of fathers' influences when mothers are depressed.

Although these are important limitations, this meta-analysis marks a significant step in both quantifying the strength of the associations between mothers' depression and multiple domains of children's emotional and behavioral problems and affective functioning and identifying the theoretically and methodologically relevant variables that play moderating roles in those associations. This review reveals the importance of developing theoretical models specific to aspects of child functioning. We also identified important areas for continuing research that promise to further reveal the mechanisms and moderators of risk for psychopathology in the development of children with depressed mothers and generated suggestions to enhance preventive interventions.

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