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ScienceDirect
Clinical Psychology Review 27 (2007) 986–1003
CLINICAL PSYCHOLOGY REVIEW

Examining the association between parenting and childhood depression: A meta-analysis ☆

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Received 20 December 2006; received in revised form 26 February 2007; accepted 2 March 2007

Abstract

Theoretical models posit that parenting plays a causal role in the development and maintenance of child psychological problems, yet meta-analytic findings indicate that parenting accounts for less than 6% of the variance in child externalizing problems and less than 4% of the variance in childhood anxiety. Extending the analysis to childhood depression, we conducted a meta-analysis of 45 studies testing the association between parenting and childhood depression. We found that parenting accounted for 8% of the variance in child depression. Parental rejection was more strongly related to childhood depression than was parental control. Moreover, various subdimensions of parenting were differentially associated with childhood depression, with parental hostility toward the child most strongly related to child depression. Analyses also revealed that methodological factors (i.e., how parenting and child depression was conceptualized and assessed) moderated the parenting–childhood depression association. Inconsistent findings within the literature are partially attributable to variations from study to study in measurement quality. Closer attention to the precise measurement of these two constructs in future studies may lead to a more accurate estimate of the association between parenting and child depression. In all, the modest association between parenting and childhood depression indicates that factors other than parenting may account for the preponderance of variance in childhood depression.

Keywords: Parenting; Childhood depression; Meta-analysis

Contents

☆ This paper was supported in part by a training grant from the National Institute of Mental Health (NIMH) awarded to Bryce D. McLeod (National Research Service Award; F31-MH64993) and grants awarded to John R. Weisz from NIMH (R01-MH49522) and the John D. and Catherine T. MacArthur Foundation (Initiative on Youth Mental Health, and Research Network on Youth Mental Health).

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doi:10.1016/j.cpr.2007.03.001

The debate over whether parenting affects the psychological well-being of children has challenged a longstanding assumption about childrearing — i.e., that parental childrearing practices play a major role in the development and maintenance of child[1] psychological problems (Harris, 2002; Kagan, 2003; Maccoby, 2002; Rutter, 2002). Meta-analytic studies examining the association between parenting and child psychological problems have contributed importantly to this debate; they have demonstrated that parenting accounts for a relatively small proportion of the variance in child externalizing problems and in at least some internalizing problems. In one meta-analysis, parenting was found to account for less than 6% of the variance in child externalizing problems and disorders (see Rothbaum & Weisz, 1994). In another meta-analysis, parenting accounted for only 4% of the variance in childhood anxiety (see McLeod, Wood, & Weisz, 2007). These findings run counter to the long-standing belief – among the lay public, professionals, and researchers – that parenting plays a highly significant role in the psychological adjustment of children (see Maccoby, 1992, for a review). The findings raise, in turn, the question of whether another prevalent condition of childhood and adolescence – i.e., depression – is or is not strongly related to parenting.

Depression in childhood and adolescence is a significant, persistent, and debilitating problem, undermining social and school functioning, and prompting substantial mental health service use (Angold, Messer, & Stangl, 1998; Clarke, DeBar, & Lewinsohn, 2003; Whitaker, Johnson, & Shaffer, 1990). By age 18, nearly a fourth of all children will have experienced clinically significant depressive symptoms, making such symptoms among the most prevalent psychiatric problems of young people (Clarke, Hawkins, Murphy, & Sheeber, 1993; Lewinsohn, Rohde, Seeley, & Fisher, 1993). Once they appear, depressive symptoms remain present and problematic for many youngsters throughout childhood, adolescence, and beyond (Achenbach, Howell, McConaughy, & Stanger, 1995; Garber, Braafladt, & Weiss, 1995; Kessler, Avenevoli, & Merikangas, 2001; Lewinsohn, Rohde, & Seeley, 1998; Luby, Todd, & Geller, 1996). Depression in children is therefore a serious, pervasive problem with an unfortunate developmental course.

Theoretical models have hypothesized that parenting plays a major causal role in the development and maintenance of child depression. Parental rejection and parental control are most often identified as the causal processes (e.g., Bowlby, 1988; Garber & Flynn, 2001b; Hammen, 1992; Lewinsohn et al., 1998). Parental rejection has been defined as encompassing excessive disapproval, criticism, and lack of contact with the child (e.g., Clark & Ladd, 2000; Maccoby, 1992; Rapee, 1997). Influential models hold that parental rejection is experienced as aversive by the child, and that it contributes specifically to the development of childhood depression (especially in the early years of life — see Downey & Coyne, 1990; Marton & Maharaj, 1993) by undermining self esteem, promoting a sense of helplessness, and prompting development of negative self-schemas, which some see as building blocks of depression (Garber & Flynn, 2001b; Hammen, 1992; Kaslow, Deering, & Racusin, 1994). Parental control has been defined as a cluster of parent behaviors including excessive regulation of children's activities and routines, encouragement of

[1] Throughout this paper, we use the terms "child" and "children" to refer to the age range encompassing children and adolescents.

children's dependence on parents, and instructions to children on how to think or feel (e.g., Barber, 1996; Steinberg, Elmer, & Mounts, 1989). Such parental control over children's activities is hypothesized to set the stage for child depression by reducing perceived mastery (Chorpita & Barlow, 1998), reducing perceived personal control (Weisz, Southam-Gerow, & McCarty, 2003), and inducing perceived helplessness (Garber & Flynn, 2001b; Kaslow et al., 1994), a well-documented risk factor for depression.

The question of whether parental rejection or control is linked to child depression has been examined in a number of studies. The most common methodology employed thus far has been to interview adults about childhood relations with their parents (Burbach & Borduin, 1986; Rapee, 1997). Such studies generally indicate that depressed adults report that their parents were high in rejection (Burbach & Borduin, 1986; Gerlsma, Emmelkamp, & Arrindel, 1990; Rapee, 1997) and high in control or "overprotection" (Burbach & Borduin, 1986; Gerlsma et al., 1990); however, two factors limit what such findings can tell us about the association between parenting and *childhood* depression. First, retrospective methodology may not provide a reliable measure of actual parenting (Holden & Edwards, 1989). Second, retrospective accounts of parenting may be influenced by an informant's current depressive symptoms (see Lewinsohn & Rosenbaum, 1987). Thus, findings generated by studies that have relied upon retrospective reports of depressed adults must be interpreted with caution.

Other studies have employed an approach less susceptible to bias — i.e., concurrent assessment of parenting and child depression. Review articles have generally concluded that these studies do show child depression to be related to parental rejection (Burbach & Borduin, 1986; Chiariello & Orvaschel, 1995; Kaslow et al., 1994; Marton & Maharaj 1993; Rapee, 1997) and control (Burbach & Borduin, 1986; Kaslow et al., 1994). But methodological limitations that characterized the studies (e.g., an over reliance on child-report) summarized in these articles have led authors to question the overall validity of the findings (Burbach & Borduin, 1986; Marton & Maharaj, 1993; Rapee, 1997). An additional problem is none of the reviews examining the concurrent association between parenting and childhood depression have used quantitative methods to assess effects. Consequently, the reviews have not been able to estimate the strength of the parenting–childhood depression association or examine the differential strength of association with parental rejection vs. parental control. Meta-analysts (e.g., Rosenthal, 1991; Schmidt, 1992) stress that the best way to examine associations between constructs, such as parenting and childhood depression, is to rely on effect size (ES) values examined within meta-analyses that synthesize ESs across a representative collection of studies. In the present paper, we present such a meta-analysis, relying on a highly representative set of studies and using stringent procedures to assess strength of association.

Such an approach is useful in assessing the extent to which variations in parenting are actually associated with child psychological problems. Traditional theoretical models assert that this association is strong, but empirical findings from sources other than the parenting literature suggest that nonparenting factors might exert a more powerful impact on childhood depression. For example, evidence suggests that the heritability of liability to major depression is approximately 40% (Boomsma, van Beijsterveldt, and Hudziak, 2005; Kendler, Gatz, Gardner, & Pedersen, 2006; Sullivan, Neale, & Kendler, 2000). Complementary evidence indicates that gene-by-environment interactions – in which an individual's response to stressful events is moderated by his or her genetic makeup – may explain why some individuals develop depression following stressful events (Caspi, Sugden, & Moffitt, 2003; Wilhelm, Mitchell, & Niven, 2006). Clearly, genetic and other biological factors account for a substantial proportion of the variance in childhood depression. The literature we cited earlier indicates that parenting also accounts for some of the variance in childhood depression (see also Boomsma et al., 2005). Meta-analytic methods can shed light on the relative impact of parenting within the array of factors by generating an estimate of the strength of association between parental behavior and childhood depression.

An additional purpose of the study was to capitalize on the potential of meta-analytic methods to detect moderators of theoretically important associations and, in the process, to help explain what appear to be inconsistent findings in the literature. First, given the long-standing focus of the parenting literature on parental rejection and parental control, we first assessed the relative strength of association between these two dimensions of parenting, on the one hand, and childhood depression on the other. In addition, because some theorists have argued that the broad parenting categories of rejection and control lack specificity (see Wood, McLeod, Sigman, Hwang, & Chu, 2003), we also assessed whether subdimensions of parental rejection (i.e., warmth, withdrawal, and aversive parental behavior) and control (i.e., autonomy-granting and overinvolvement) identified in prior literature were differentially associated with childhood depression. If the parenting subdimensions are differentially associated with childhood depression then the use of the broad parenting dimensions may have contributed to inconsistent findings in the

literature given variability in the particular parenting subdimensions various studies included in the broad constructs.

We also examined whether methodological variables moderated the relation between parenting and childhood depression. This seemed feasible, given previous findings indicating that the way parenting and childhood outcomes are assessed can influence the strength of relations among variables. As an example, previous meta-analyses have found a weaker association between parenting and child outcomes in studies that used questionnaire measures of parenting than in studies that used observational methods (see McLeod et al., 2007; Rothbaum & Weisz, 1994). We therefore examined whether the strength of the parenting–childhood depression association was moderated by the way in which parenting and childhood depression was assessed.

Finally, we examined whether the magnitude of the parenting–childhood depression association varied as a function of several study design factors that are often examined in meta-analyses related to child and adult psychopathology. For example, we assessed whether strength of association differed as a function of how parenting was assessed, how depression was assessed, and who provided the information on parenting and depression.

To summarize, influential theories posit that parenting has a powerful impact on child problem behavior, but two meta-analytic reviews suggest that parenting may actually account for relatively little variance in child anxiety (McLeod et al., 2007) and child externalizing problems (Rothbaum & Weisz, 1994). Here we investigated whether the same might be true of childhood depression. We examined studies assessing the parenting–child depression association, focusing on the two most theoretically central and widely studied parenting dimensions: rejection and control. To avoid a significant limitation of prior reviews, we excluded studies that used adult retrospective reports, including instead only those studies that assessed parenting and childhood depression concurrently. We used a meta-analytic approach that permitted quantification of the magnitude of parenting–child depression associations, and tests of potential moderators of that association that have been suggested in previous research.

1. Method

1.1. Selection of studies

We conducted a literature search for studies presenting quantitative data on the association between parenting and childhood depression involving mothers or fathers, or both, and children from infancy through adolescence. A computer based information search was conducted on the PsychInfo computer database, which indexes (with key terms) and abstracts articles. The search covered up to April 2006, and we used the following five depression-related key terms and synonyms: *Internaliz-*, *Depress-*, *Dysthy-*, *Sad-*, and *Suicid-*. These terms were crossed with the following parenting-related key terms: *Father-*, *Maternal*, *Mother-*, *Parent-*, *Paternal*, *Rearing*, or *Socializ-*. Relevant literature reviews (e.g., Burbach & Borduin, 1986; Chiariello & Orvaschel, 1995; Kaslow et al., 1994; Marton & Maharaj, 1993; Rapee, 1997; Sander & McCarty, 2005) were used to initiate reference trails to pertinent investigations, and issues of journals (e.g., *Child Development*) dated 1990 and later in which relevant studies were reported were hand-searched to locate studies not yet incorporated into PsychInfo. These steps produced a pool of 45 studies (published in 43 articles) that met inclusion criteria. Table 1 lists the sample characteristics, methods of assessment, and study ESs for each of the 45 studies.

1.1.1. Inclusion/exclusion criteria

To be included in the meta-analysis, a study had to meet the following criteria: First, the study had to include a measure of the parenting of one parent towards a target child, or separate measures of both parents' behavior toward the target child; second, the study had to include a measure of childhood depression (e.g., self-report) or the child participants had to be diagnosed with a depressive disorder (e.g., Major Depression); third, the relation between parenting and childhood depression had to be tested statistically (e.g., correlation); finally, the mean age of the child participants had to be below 19 years.

We required that studies include a direct measure of parenting (e.g., positive parental involvement with one target child), so we excluded studies that did not, including studies that employed measures of "attachment" and the "family environment". Although attachment status may arguably reflect an aspect of the parent–child relationship, traditional attachment measures are based on observations of a *child's* behavior with a parent, rather than a parent's behavior with

Table 1
Reviewed studies, sample characteristics, methods of assessment, and study ES

Study	*N*	Age (years)	Parenting measure			Dx.	Depression measure			
			Parent	Tech.	Inform.		TF	Tech.	Inform.	Mean *r*
Asarnow, Tompson, Hamilton, and Goldstein (1994)	56	10.43	M	I	P	Y	C	S	C	.26
Asarnow, Tompson, and Woo (2001)	156	12.31	M	I	P	Y	C	S	C	.00
Barber (1996)	158	12.00	F, M	O	O	N	C	O	P	.16
Baron and MacGillivray (1989)	144	15.20	F, M	Q	C	N	C	S	C	.19
Barrera and Garrison-Jones (1992)	94	14.60	F, M	Q	C	N	C	S	C	.00
Biggam and Power (1998)	125	18.80	F, M	Q	C	N	C, U	S	C	.18
Brage and Meredith (1993)	156	14.00	F, M	Q	C	N	C, U	S	C	.22
Corona, Lefkowitz and Sigman (2005)	111	13.20	M	O	O	N	C	S	C	.16
Elder, Conger, Foster, and Ardelt (1992)	76	12.00	F, M	O	O	N	C	S, O	C, O	.28
Field, Sandberg, and Goldstein (1987)	38	5.10	M	Q, O	P, O	Y	C	O	O	.10
Field, Lang, Yando, and Bendell (1995)	455	16.60	F, M	Q	C	N	C	S	C	.27
Forehand, Brody, Long, and Fauber (1988)	89	13.08	M	O	O	N	C	S	C	.23
Forehand, Brody, Slotkin, Fauber, McCombs, and Long (1988)	69	13.42	M	Q	C	N	C	S, O	C, O	.18
Furukawa (1992)	165	17.47	F, M	Q	C	N	U	S	C	.00
Furukawa (1997)	144	17.47	F, M	Q	C	N	U	S	C	.15
Galambas and Maggs (1990)	91	11.58	M	Q	C, P	N	U	S	C	.26
Gallimore and Kobak (1992)	35	13.63	F, M	Q	C, P	N	C	S	C	.22
Garber, Robinson, and Valentiner (1997)	240	11.86	M	Q	C, P	N	C	S, O	C, P	.19
Garber and Flynn (2001a)	240	11.86	M	Q	C, P	N	U	S	C	.11
Greenberger and Chen (1996)	84	13.10	F, M	Q	C	N	C	S	C	.56
	89	13.20	F, M	Q	C	N	C	S	C	.53
Harold, Fincham, Osborne, and Conger (1997)	146	12.83	F, M	Q	C	N	C	S	C	.60
	380	13.00	F, M	Q	C	N	C	S	C	.32
Heaven, Newbury, and Mak (2004)	276	15.34	F, M	Q	C, P	N	C	S	C	.18
Huntley and Phelps (1990)	76	7.78	F, M	Q	P	N	C	S	C	.15
Jacquez, Cole, and Searle (2004)	72	15.00	M	Q	C, P	N	C	S, O	C, P	.31
Kobak, Sudler, and Gamble (1991)	48	15.70	M	O	O	N	U	S	C	.22
Marmorstein and Iacono (2004)	249	17.50	F, M	Q	C	Y	L	S	C	.31
Martin and Waite (1994)	681	15.00	F, M	Q	C	N	L	S	C	.30
McClellan, Heaton, and Forste (2004)	493	15.50	M	Q	C	N	C	S	C	.12
McFarlane, Bellissimo, and Norman (1995)	801	17.10	F, M	Q	C	N	U	S	C	.28
Messer and Gross (1995)	20	10.10	F, M	Q	C, P	N	C	S	C	.29
Milne and Lancaster (2001)	59	15.70	M	Q	C	N	U	S	C	.41
Puig-Antich (1985)	92	9.43	F, M	Q	P	Y	C	S	C	.54
Puig-Antich, Kaufman, and Ryan (1993)	98	15.20	F, M	Q	P	Y	C	S	C	.67
Rogers, Buchanan, and Winchel (2003)	306	11.70	F, M	Q	C	N	C	S	C	.34
Rudolph, Hammen, and Burge (1997)	81	9.65	M	Q	C	N	C	S	C	.37
Sanders, Dadds, and Johnston (1992)	46	10.40	M	Q, O	C, P, O	Y	C	S	C	.26
Sheeber and Sorensen (1998)	52	15.50	M	Q	C, P	Y	C	S	C	.42
Shek (1989)	2150	16.00	F, M	Q	C	N	C, U	S	C	.26
Stein, Williamson, and Birmaher (2000)	68	10.50	F, M	Q	C	Y	C	S	C	.37
Stocker (1994)	85	7.92	M	Q	C	N	C, U	S	C	.25
Tesser and Forehand (1991)	147	13.00	F, M	Q	C	N	C	S	C	.11
Thompson and Zuroff (1999)	54	13.90	M	Q	C	N	U	S	C	.39
Whitbeck et al. (1992)	451	12.00	F, M	Q, O	C, P, O	N	U	S, O	C, P	.20

Note. Age is reported in mean years. For Parent, M = mother, F = father. For Parenting Measure, Tech = Measurement technology, Q = Questionnaire, I = Interview, O = Observation, Inform = Informant, C = Child, P = Parent, O = Observer. Dx. = Diagnostic status, Y = Yes, N = No. TF = Timeframe, C = Current symptomatology, L = lifetime symptomatology, U = unspecified. For Depression Measure, Tech = measurement technology, S = self-report, O = other-report, C = counts/behavioral observation, Inform = informant, C = child, P = parent, O = other.

a child. Similarly, studies of the family environment have examined childhood depression as an outcome of interest; however, the family environment (e.g., family cohesiveness and adaptability) involves a complex interactive process among all family members and is not a direct measure of parental behavior.

1.1.2. Study sample

The findings reported in the Results were derived from 45 cross-sectional studies published from 1985 to 2005. These 45 studies encompassed children ranging in age from 5.1 to 18.8 years (M = 13.26, SD = 2.87), totaled 9746 participants, and produced 193 correlations and 57 group-comparisons. Thirty-four studies reported on the ethnic composition of their sample; 28 studies were comprised mostly of Caucasian children (i.e., more than 50% Caucasian), one study was comprised primarily of Latino children, and five studies were composed primarily of Asian children. On average, the families had 2.90 members (SD = 1.21; based upon seven studies), 74% of the families were intact (SD = .24; based upon 30 studies), and the parents reported an average of 13.42 years of education (SD = 1.75; based upon 12 studies). Studies included in the meta-analysis are denoted with asterisks in the References section.

1.2. Coding of the studies

Information extracted from the studies was coded by the first and third authors. To assess intercoder agreement they independently coded a randomly selected sample of 14 studies. Coders were trained to an acceptable level of reliability, weekly meetings were held to prevent rater drift, and differences between coders were resolved through discussion. Following Fleiss (1981), kappas below .40 reflect "poor" agreement, kappas between .40 and .74 reflect "fair to good" agreement, and kappas .75 and higher reflect "excellent" agreement. Inter-coder agreement ranged from .62 to .98, with one of our codes in the fair to good range and the rest in the excellent range.

1.2.1. Information extracted

Information about the following variables was extracted from each study: (a) design features including ethnicity, family size, parent education, intactness of families, study location (i.e., United States vs. international), study setting (i.e., urban vs. suburban/rural), and setting in which study was conducted (i.e., laboratory, home, other); (b) child age; (c) gender of the child; (d) gender of the parent; (e) type of parenting behavior; (f) measurement technology of parenting measure (i.e., questionnaire, interview, observation); (g) parenting informant (i.e., child, parent, observer); (h) measurement technology of depression measure [i.e., self-report (i.e., rating of own depression), other-report (i.e., rating of someone else's depression), counts/behavioral observation (i.e., objective ratings of subject's depression)]; (j) depression informant (i.e., child, parent, other); (k) depression assessment strategy (i.e., continuous vs. categorical); (l) single vs. multiple informants; (m) whether the participants were diagnosed with a depressive disorder (i.e., yes, no); and (n) whether the assessment of child depression focused upon current or lifetime symptomatology [i.e., current (depressive symptoms in the previous 4 weeks), lifetime (depressive symptoms during the child's lifetime), or unspecified].

1.2.2. Childhood depression

We classified studies based upon the way in which childhood depression was assessed, namely, whether the study used continuous measures (e.g., self-report measure such as the Children's Depression Inventory; n = 33) or a diagnostic/extreme-groups approach (termed "group comparison"; n = 12). Studies were classified as *group comparison* designs if "normal" children (children with no depressive diagnosis) were compared to a sample of children that (a) was diagnosed with a depressive disorder, or (b) had clinically elevated depression (i.e., above a clinical cutoff on a questionnaire measure of depression).

Childhood depression was assessed using the following methods within the current study set. Regarding the measurement technology of the depression measures, 38 studies relied exclusively upon self-report measures and two studies relied exclusively upon other-report measures. Only five studies used multiple methods to assess depression and each one relied upon a self-report and other-report measure. Regarding the informant for the depression measure, 38 studies relied solely upon child-report, one exclusively upon parent-report. Two studies collected information from the child and other reporters; and three studies collected information from child and parent reporters. With regard to the timeframe of the child depression assessment (current vs. lifetime), 30 studies relied upon measures focusing on current depressive symptomatology, 2 studies relied on measures of lifetime depressive symptomatology, and 9 studies employed measures with an unspecified timeframe. Four studies included measures of current depressive symptomatology as well as measures with an unspecified timeframe.

1.2.3. Parenting

The same definition and codes for parenting described in McLeod and colleagues (2007) were used in the present study. Parenting was defined as behavior towards children (e.g., "rejection"), reported by children, parents, or observers, and was divided into two broad dimensions: *rejection* and *control.* In addition to classifying each parenting measure into rejection or control we also classified each measure into a subdimension of rejection or control. The subdimensions of parental rejection were: (a) *withdrawal*, defined as the lack of involvement between parent and child, lack of interest in the activities of the child, or lack of emotional support/reciprocity; (b) *aversiveness*, defined as parental hostility towards children; and (c) *warmth*, defined as a sense of positive regard expressed by the parent toward the child. The subdimension of parental control was *overinvolvement*, defined as parental interference with children's age-normative autonomy and emotional independence.[2] Parenting measures were classified into one of these subdimensions except when a measure could be classified into more than one subdimension, in which case it was only coded as parental rejection or control.

Regarding the measurement technology of the parenting measures, 35 studies relied exclusively upon questionnaire measures, two relied upon interview measures, and five utilized only observational measures. Only three studies used multiple methods to assess parenting, and each study employed a questionnaire and observational measure. Regarding the parenting informant, 24 studies relied exclusively upon child-report, five upon parent-report, and five solely upon observers. Eight studies relied upon child and parent report; one study relied upon parent and observers; and two studies relied upon child, parent, and observer report. Regarding parent gender, 45 studies examined the parenting–childhood depression association for mothers and 27 examined the association for fathers. Regarding child gender, seven studies examined the parenting–childhood depression association for boys, eight studies examined the association for girls, and 37 studies did not specify child gender. Finally, 24 studies relied solely upon a single-informant (one informant for parenting and childhood depression), 11 studies relied solely upon multiple-informants, and 10 studies relied upon single-and multiple-informants.

1.3. Meta-analytic method

Studies expressed the parenting–childhood depression association both in terms of Pearson's product-moment correlation (r) and mean difference between-groups. Following Rosenthal (1994), we used the ES r to express the association between parenting and childhood depression because it is more familiar and thus easier to interpret for most readers compared to d-type ES indices. We calculated ES values for each association of interest within each study — i.e., separate ES values were calculated within each study for all pairings between a parenting category and a depression measure. When investigators reported nonsignificant effects, there was sometimes insufficient information to compute an ES. In such cases, we contacted the authors in an attempt to gain access to the pertinent data. But when such efforts failed we used the common, conservative strategy of assigning a correlation of 0 (Pigott, 1994).

Once ES values were calculated within each study we analyzed data across studies. In these analyses we first analyzed data at the study-level and then at the construct-level. The goal of these analyses was to obtain an unbiased ES estimate and to examine the homogeneity of the ES estimates. We weighted each ES by the inverse of its variance (Shadish & Haddock, 1994) and thus adjusted for heterogeneity of variance across individual observations. The resulting ES estimates were interpreted following Cohen's (1988) guidelines: r is a "small" effect when at least .10, r is a "medium" effect when at least .24, and r is a "large" effect when at least .37. In addition, so that ES estimates for negative parenting dimensions (i.e., rejection, withdrawal, aversiveness, overinvolvement, and control) could be compared to ES estimates for the positive parenting dimension (i.e., warmth) we multiplied the latter by -1. Thus, for comparisons involving warmth, positive correlations mean that more of the parental behavior was associated with less childhood depression (e.g., more warmth is associated with less childhood depression).

The first goal of the study-level analysis was to produce an estimate of the population ES for the parenting–childhood depression association. To ensure independence of observations, each study contributed only one ES to the analysis by averaging across all parenting and childhood depression comparisons contained within each study. The second goal was to assess the homogeneity of the ESs. At the study-level, the homogeneity estimate (Q) approximates a chi-square distribution with $k-1$ degrees of freedom (Hedges, 1994). A significant effect indicates that the variation

[2] Autonomy-granting (i.e., parental encouragement of children's opinions and choices) is another important subdimension of parental control (see McLeod et al., 2007), but in the present study no parenting measures were coded as autonomy-granting.

may not be due to sampling error (i.e., that variation across weighted mean ESs is greater than chance) and that moderators may explain the variability. The final goal was to assess whether any sample characteristics accounted for variations in ESs across studies.

The goal of the construct-level analyses was to examine potential moderators of the association between parenting and childhood depression. To ensure independence of observations, each study was allowed to contribute only one ES to each moderator level by averaging across all parenting and childhood depression comparisons up to the level of analysis. For example, measures of different parenting dimensions were averaged except when the moderating effects of parental rejection and parental control were assessed. For these analyses, we first examined whether any theoretical variables moderated the parenting–childhood depression association. Then, we examined whether a series of methodological variables associated with the way in which parenting and childhood depression were measured moderated the parenting–childhood depression association. Because these variables were categorical we used procedures analogous to analysis of variance (ANOVA) for the analyses — i.e., ES values were grouped according to each moderator to test for differences between the levels (Hedges, 1994). At the construct-level, two homogeneity estimates were produced (Hedges, 1994); a between-groups Q (termed Q_b) was calculated to test for significant variability across groups (e.g., parental rejection vs. parental control), and a within-group Q (termed Q_w) was calculated to test for significant variability within each group (e.g., variation within the parental rejection category). For follow-up contrasts, standardized contrasts (g) were calculated from the difference in ES values (Hedges, 1994). The significance of each contrast was determined by first dividing the contrast value by the pooled variance, which produces a critical value equivalent to the chi-square distribution with one degree of freedom. The critical value for the contrasts was set at $p<.05$.

2. Results

2.1. Study-level analysis

Our study-level analysis focused on the association between parenting and childhood depression. The weighted mean ES for the parenting–childhood depression association ES was .28 and the 95% confidence interval did not include zero, which reflects a relation in which more negative parenting was associated with more childhood depression. The magnitude of this ES meets criteria for a medium effect and indicates that parenting accounted for almost 8% of the variance in childhood depression. Because the homogeneity analysis was significant, moderating variables are likely to exist ($Q=207.11$, $p<.01$). However, before preceding with the construct-level analyses we conducted analyses examining whether ESs varied according to child age, child gender, parent education (number of years), parent gender, ethnicity, family size, intactness of families, study location (i.e., United States vs. international), study setting (i.e., urban vs. suburban/rural), and setting in which the study was conducted (i.e., laboratory, home, other). Of these analyses, only the one for parent education was significant ($r=.72$, $p<.01$, $n=12$). Because the parent education finding was based on only 12 studies, we did not control for parent education in the subsequent construct-level analyses.

2.2. Construct level analyses: Theoretical moderators

We first examined whether theoretical factors moderated the association between parenting and childhood depression.

2.2.1. Parenting dimensions

Results for the construct-level analyses examining the moderating effects of the parenting dimensions and subdimensions are shown in Table 2. First, we examined the association between childhood depression and the parenting dimensions of rejection and control. Our results revealed a significant between-groups homogeneity statistic ($Q_b=8.14$, $p<.01$), indicating that the rejection and control dimensions differed significantly. For rejection, the weighted mean ES was .28, which meets criteria for a medium effect and indicates that parental rejection accounts for approximately 8% of the variance in childhood depression. For control, the weighted mean ES was .23, which meets criteria for a small effect and suggests that parental control accounts for almost 5% of the variance in childhood depression. Altogether, higher levels of parental rejection and control were associated with more childhood depression,

Table 2
Moderator analyses for parenting and childhood depression

Moderator	Q_b	k	Weighted Mean ES	95% CI	Q_w
Theoretical moderators					
Parenting dimension	8.14*				
Rejection		43	$.28_a$	.27–.31	210.14**
Control		16	$.23_a$	.20–.26	38.17**
Parenting subdimension	14.69**				
Warmth		23	$.28_a$	.25–.30	60.55**
Withdrawal		1	$.20_b$	.11–.29	0
Aversiveness		17	$.33_{a,b,c}$	.30–.36	122.87**
Overinvolvement		14	$.24_c$	.21–.27	29.63**
Methodological moderators for childhood depression					
Assessment strategy	16.67**				
Group comparison		12	$.37_a$	.32–.42	67.87**
Continuous		33	$.26_a$	.24–.28	122.57**
Depression diagnostic status	21.85**				
Yes		9	$.40_a$	.34–.45	61.26**
No		36	$.26_a$	.24–.28	124.00**
Depression assessment timeframe	13.34*				
Current		34	$.29_a$	.27–.31	179.86**
Lifetime		2	.30	.25–.36	.02
Unspecified		13	$.23_a$	.20–.26	20.19**
Informant	10.07*				
Child		43	$.28_a$	.26–.30	206.41**
Parent		5	$.26_b$	.20–.32	9.23
Other		3	$.15_{a,b}$	.07–.23	4.41
Methodological moderators for parenting					
Measurement technology	24.21**				
Questionnaire parenting measures		38	$.29_{a,b}$	.26–.30	185.33**
Interview parenting measures		2	$.08_a$	−.06–.21	3.12
Observational parenting measures		8	$.16_b$	.10–.22	3.62
Informant	13.92**				
Child		34	$.28_a$	.26–.30	117.66**
Parent		16	$.26_b$	.23–.31	117.13**
Observer		8	$.16_{a,b}$	.10–.22	3.62
Methodological moderator					
Single vs. multiple Informant	7.15*				
Single informant		34	$.29_a$	.27–.31	118.62**
Multiple informant		21	$.23_a$	.19–.27	128.72**

Note.Categories with the same subscript denote significant differences. Q_b = homogeneity for test of variation across groups; k = number of correlations; Weighted mean ES = average corrected (i.e., weighted) correlation; 95% CI = lower and upper limits of 95% confidence interval; Q_w = test of variation within group of individual effects.
*$p<.05$, **$p<.01$.

though parental rejection was associated with a greater proportion of the variance in childhood depression than parental control.

Because the homogeneity analyses for parental rejection and control were significant we examined whether the parenting subdimensions – warmth, withdrawal, aversiveness, and overinvolvement – were differentially associated with childhood depression. This analysis generated a significant between-groups homogeneity statistic ($Q_b = 14.69$, $p<.01$), which suggests that the parenting subdimensions are differentially associated with childhood depression. The weighted mean ES estimates for the parenting subdimensions ranged from .20 (withdrawal), a small effect, to .33 (aversiveness), a medium effect, and the percent of variance explained ranged from 4% to 11%. The associations between the parenting subdimensions and childhood depression were in the hypothesized direction, with lower levels

of parental warmth associated with more childhood depression, and higher levels of parental withdrawal, aversiveness, and overinvolvement associated with more childhood depression. Follow-up contrasts revealed that the weighted mean ES for aversiveness was significantly higher than the weighted mean ESs for warmth (g=.05, p<.01), withdrawal (g=.13, p<.01), and overinvolvement (g=.09, p<.01). Overall, these findings suggest that the parenting subdimensions were differentially associated with childhood depression, with parental aversiveness explaining the greatest proportion of the variance in childhood depression.

2.3. Construct-level analyses — Methodological moderators

Next, we examined whether methodological factors moderate the association between parenting and childhood depression.

2.3.1. Childhood depression

Results for the construct-level analyses examining the moderating effects of the methodological factors are shown in Table 2. For these analyses, we started by examining the moderating effects of measurement technology, informant, diagnostic status, and depressive assessment strategy. Results indicated that depressive assessment strategy, diagnostic status, assessment timeframe, and informant were moderators. The weighted mean ES for group comparison designs (.37) was significantly higher than the weighted mean ES for continuous measurement designs (ES=.26) (Q_b=16.67, p<.01). The weighted mean ES for studies that determined whether each child met criteria for a diagnosis of depression (ES=.40) was significantly higher than the weighted mean ES for studies that did not (ES=.26) (Q_b=21.85, p<.01). The between groups homogeneity statistic was significant for assessment timeframe (Q_b=13.34, p<.05), indicating that the weighted mean ES varied according to whether the assessment of childhood depression focused upon current symptomatology (ES=.29), lifetime symptomatology (ES=.30), or an unspecified timeframe (ES=.23). Follow up contrasts revealed that depression measures that did not specify a timeframe yielded statistically significantly lower ES than depression measures focused upon current (g=.06, p<.05) or lifetime symptomatology (g=.07, p<.05). Finally, the between groups homogeneity statistic was significant for childhood depression informant (Q_b=10.07, p<.05), indicating that the weighted mean ES varied according to whether children (ES=.28), parents (ES=.26), or other (ES=.15) reported on childhood depression. Follow up contrasts indicated that child-report was significantly higher than other-report (g=.13, p<.01), and parent-report was significantly higher than other-report (g=.11, p<.01). These findings suggest that the way in which depression was conceptualized and assessed affected the magnitude of the parenting–childhood depression association.

2.3.2. Parenting

Construct level analyses examining the moderating effects of measurement technology and informant of the parenting measures indicated that both were moderators (see Table 2). Results indicated that the between-groups homogeneity statistic was significant for measurement technology (Q_b=24.21, p<.01), revealing that the weighted mean ES varied according to whether questionnaire (ES=.29), interview (ES=.08), or observational (ES=.16) parenting measures were used. In the follow up contrasts, questionnaire measures were significantly higher than observational (g=.13, p<.01) and interview (g=.21, p<.01) parenting measures. Results also indicated that the between groups homogeneity statistic was significant for parenting informant (Q_b=13.92, p<.01), indicating that the weighted mean ES differed according to whether children (ES=.28), parents (ES=.26), or observers (ES=.16) reported on parenting. Follow up contrasts indicated that child-report (g=.12, p<.01) and parent-report (g=.10, p<.01) were both significantly higher than observer-report. Overall, these findings indicate that the way in which parenting was assessed impacted the magnitude of the parenting–childhood depression association.

2.3.3. Single vs. multiple informants

To test the hypothesis that the association between two questionnaire-based measures from the same source would yield inflated correlations due to shared method variance (Campbell & Fiske, 1959) we compared the weighted mean ESs from analyses employing the same informant for both parenting and childhood depression measures ("single informant studies"), and analyses employing different informants for the parenting and childhood depression measure ("multiple informant studies"). Findings revealed that the between groups homogeneity statistic was significant for single vs. multiple informants (Q_b=7.15, p<.01), indicating that the weighted mean ES for single-informant measures

(ES = .29) was significantly higher than the weighted mean ES for multiple-informant measures (ES = .23). These findings indicate that the association between parenting and childhood depression is stronger when a single informant reports on both depression and parenting.

2.4. Do methodological factors explain the findings for parental rejection and control?

Next, we examined whether the weighted mean ES estimates for parental rejection and control remained statistically different when the effects of the methodological variables identified as significant moderators were controlled (see Table 2). To avoid multicollinearity, we did not enter parenting informant, depression informant, or measurement technology of parenting measure into the regression equation. This is because a majority of the studies that used questionnaire-based measures used a single-informant (i.e., the child) so the significant effects noted previously for these three methodological variables are due to single informant methodology. Simultaneous weighted least squares (WLS) regression was used for this analysis. The WLS regression analysis yielded significant multiple $R^2 = .28$, $F(4, 80) = 5.28$, $p < .01$. Parenting type did not remain significant ($\beta = -0.15$, $p < .15$) when we controlled for the methodological variables; however, single vs. multiple informant did remain significant ($\beta = .38$, $p < .01$). These findings indicate that the differential ESs for parental control vs. rejection may be explained in part by methodological differences in the studies looking at linkages between these two constructs and childhood depression.

To examine the suppressive effect of single vs. multiple informant upon parenting type revealed in the regression analysis, we examined the two-way interaction between parenting type and single vs. multiple informant. Our analyses indicated that the interaction was significant ($Q_b = 3.96$, $p < .05$). In multiple informant studies, parental rejection had a stronger association with depression than did parental control (ESs = .25 vs. .12, $p < .05$), but effects for parental rejection were not significantly different from those for parental control in single informant studies (ESs = .29 vs. .25, ns). These findings suggest that the differential ESs for parental rejection vs. control only emerge when multiple informants are used.

3. Discussion

The main objective of the present study was to provide an estimate of the strength of association between parenting and childhood depression. Parental childrearing practices have traditionally been assumed to play a critical role in determining the development and maintenance of childhood depression (see Garber & Flynn, 2001b; Hammen, 1992; Kaslow et al., 1994); however, methodological limitations have obscured the strength of the parenting–childhood depression association and led authors to question the validity of past findings (Burbach & Borduin, 1986; Marton & Maharaj, 1993; Rapee, 1997). In the present study, the overall parenting–child depression sample weighted ES was .28, which meets Cohen's (1988) criteria for a medium effect and indicates that parenting explains approximately 8% of variation in childhood depression. Thus, these findings indicate that parenting is consistently, albeit moderately, associated with childhood depression.

More broadly, our findings contribute to a growing body of meta-analytic evidence that suggests parenting is related to child symptomatology (McLeod et al., 2007; Rothbaum & Weisz, 1994). Theoretical models including attachment theory (Ainsworth, Bell, & Stayton, 1974) and social learning theory (Dishion, Patterson, & Griesler, 1994) emphasize that parenting plays a role in childhood development. Our findings are in line with past meta-analytic findings showing that parenting explained approximately 6% of the variance in childhood externalizing behavior (Rothbaum & Weisz, 1994) and 4% of the variance in childhood anxiety (McLeod et al., 2007). Thus, these findings offer convergent evidence that parenting is associated with both childhood externalizing and internalizing symptomatology, which provides provisional support for theoretical models that emphasize the role of parenting in the development and maintenance of child symptomatology.

However, our findings also challenge the prevailing assumption that parenting plays a major role in determining the psychological well-being of children. The theoretical models referenced above all suggest that parenting plays a central role in childhood symptomatology, yet the available meta-analytic evidence suggests otherwise. Parenting appears to account for a relatively small proportion of the variance, at least in terms of a direct effect. In contrast, using behavioral genetic methods, twin and adoption studies find a large role for additive genetic variance in children's depression. Recent studies have found that genetic influences may account for 36–60% of the variance in childhood depression

(see Boomsma et al., 2005; Middeldorp et al., 2005). Evidence therefore suggests that genetic factors may play a particularly important role in the developmental psychopathology of depression.

When considering what role parenting may play in childhood depression, it is important to note that recent findings suggest that an *interaction* between a specific genotype (i.e., 5-HTTPLR) and stressful life events (e.g., exposure to aversive parenting) may influence the likelihood of experiencing an episode of major depression (Caspi et al., 2003; Wilhelm et al., 2006). These findings imply that the expression of depression is likely the result of a complex set of interactions between biological vulnerabilities and environmental influences. Even if parenting plays a small role in childhood depression on average, it could potentially play a catalytic role among a subgroup of children who are vulnerable to depression for other reasons.

An additional objective of the present study was to assess whether sample design characteristics or methodological factors moderate the parenting–childhood depression association. We began our examination of these factors with the way in which childhood depression was modeled and assessed. Our findings indicate that categorical assessment approaches (i.e., group-comparison designs or studies that measured whether each child met criteria for a diagnosis of depression) generate larger effects than continuous (e.g., self-report) assessment approaches. This finding is consistent with past meta-analytic evidence that found the parenting–childhood anxiety association was stronger with group-comparison designs (McLeod et al., 2007). There are at least four possible explanations for these findings. First, if childhood depression is best modeled as a categorical variable, then studies using group-comparison designs may represent a more accurate estimate of the parenting–childhood association and thus produce stronger effects. Second, "extreme-group" designs may artificially inflate effects compared to continuous designs (Preacher, Rucker, MacCallum, & Nicewander, 2005). Third, according to cognitive theory (Beck, 1967), negative schemas influence individuals' evaluations of themselves, their environment, and the future; thus, the studies comparing clinically depressed children to typically developing controls may have produced stronger effects because children with depression tend to view relationships with their parent more negatively. Fourth, the presence of clinically significant childhood depression may elicit parental frustration and criticism (Chiariello & Orvaschel, 1995; Coyne, 1976; Hammen, Burge, & Stansbury, 1990; Kaslow et al., 1994), which would explain the more pronounced pattern of effects in studies comparing children with and without a depression diagnosis. Hence, although growing evidence suggests that the parenting–childhood internalizing association is stronger for group-comparison designs, whether such designs produce a more accurate estimate remains an open question.

The way in which the key constructs were measured also moderated the association between parenting and childhood depression. Analyses indicated that single informant studies produced significantly stronger effects than multiple informant studies. Single informant studies tend to overestimate the magnitude of effects due to shared method variance (Campbell & Fiske, 1959). This effect may be particularly pronounced when a depressed child is the informant. Asking children to report on parenting and their own depressive symptoms can lead to inflated ES estimates since depressed individuals tend to perceive their relations with their parents (Lewinsohn & Rosenbaum, 1987) and their own life as more negative (Beck, 1967). Relying solely upon parents to report upon their parenting and their child's depressive symptomatology may also lead to inflated ES estimates. Mothers with high levels of depressive symptomatology tend to rate their children as more behaviorally disturbed compared to nondepressed mothers (Boyle & Pickles, 1997; Chi & Hinshaw, 2002; Chilcoat & Breslau, 1997). Thus, when relying upon parent report it may be important to take into consideration the parent's level of depressive symptomatology. Within the present study set, most of the studies that used single informants relied upon the child ($n=31$), as opposed to the parent ($n=3$). Furthermore, most studies used measures of child depression that focused upon current symptomatology ($n=34$). It therefore is plausible that within the current study set, single-informant studies that relied upon child report may have been biased by negative schemas activated by current depressive symptomatology. Together, these factors indicate that method variance likely contributed to the pattern of findings in the present study and that the measurement approaches less susceptible to bias (e.g., multiple informants or use of behavioral observations) produced a more accurate estimate of the parenting–childhood depression association.

Past authors have questioned the validity of the evidence linking parenting to childhood depression due to methodological limitations (Burbach & Borduin, 1986; Marton & Maharaj, 1993; Rapee, 1997). Our findings support these observations by demonstrating that the way in which parenting and childhood depression are measured impacts the magnitude of the parenting–childhood depression association. It is important to note that the kinds of method variance noted above would have the effect of spuriously inflating the estimated strength of association between parenting and child depression. Thus, if the forms of bias noted above do exist, then the true

population effect is likely to be even lower than what is reported herein. Given the number of methodological factors that were found to moderate the parenting–childhood depression association it may be best to consider the current ES estimate a preliminary estimate of the true population effect that may be revised in the future as researchers address some of the methodological limitations in the field. To address these limitations, researchers should consider using multiple informants to reduce the problem of shared method variance. When children are asked to report upon their depressive symptoms and parents are asked to rate their parenting, response bias and response sets on the part of one rater (e.g., the parent) are unlikely to be shared by the other rater (e.g., the child), contributing to a more conservative estimate of the correlation between the two constructs. Future studies would benefit from employing multiple informants and considering how a child's mood state may influence their responses on self-report measures.

Another key question addressed in this meta-analysis was whether the broad parenting categories of rejection and control were equally associated with childhood depression — a finding that was evident in studies employing measurement approaches less susceptible to bias (i.e., studies that utilized multiple informants). Our results indicate that the two broad parenting dimensions of rejection and control were differentially associated with childhood depression, with rejection (a medium effect) demonstrating a significantly stronger association than control (a small effect). This finding suggests that parental rejection may play a particularly important role in childhood depression — either as a cause of depression, or as a reaction to the child's depressive symptoms (Coyne, 1976; Garber & Flynn, 2001b). Parental rejection is hypothesized to engender negative feelings about self, which can form the basis for negative schemas that increase vulnerability to depression (Hammen, 1992). Conversely, the development of depressive symptoms in children, such as irritability or anhedonia, can lead to parental behavior that may appear rejecting resulting from the difficulty of interacting with a depressed child (Coyne, 1976). Our results lend support to theoretical models that emphasize the importance of parental rejection in the development and/or maintenance of childhood depression.

It is important to note that the way in which parenting was assessed impacted the pattern of findings for parental rejection and control. In studies that relied on a single informant, parental rejection and control were not significantly different, but in the subset of studies that relied on multiple informants stronger effects emerged for parental rejection. The inconsistent findings regarding the relation between the parenting dimensions and childhood depression noted in past reviews may therefore be at least partially explained by the use of single informant methodology. Our findings thus suggest that using rigorous methods may both produce a more accurate ES estimate and help clarify how parental rejection and control are related to childhood depression.

Turning to the subcategories that comprise parental rejection and control reveals that these subcategories are differentially related to childhood depression. Specifically, the *absence* of parental warmth and the *presence* of parental aversive behavior demonstrated the strongest linkages to childhood depression than the *presence* of parental withdrawal or overinvolvement. It is notable that the sub-categories of parental rejection emphasized in the theoretical literature (i.e., absence of warmth and aversiveness) were associated more strongly with childhood depression than parental withdrawal or overinvolvement. Theoretical models posit that a *lack* of positive parental behavior (i.e., low acceptance) and the *presence* of parental aversive behavior (e.g., criticism) contribute to the development, maintenance, and relapse of childhood depression (see e.g., Chiariello & Orvaschel, 1995; Kaslow et al., 1994; Marton & Maharaj, 1993). According to these models, *low* parental acceptance and *high* parental aversiveness promote a sense of helplessness and lowered self-esteem within the child that form the basis of negative self-schemas, and these negative self-schemas, in turn, are hypothesized to contribute to childhood depression (Garber & Flynn, 2001b; Kaslow et al., 1994). It therefore is plausible that specific parenting practices are associated with childhood depression.

An interesting finding emerged with respect to the role of parental education in the parenting–childhood depression linkage. A strong effect emerged in which studies comprised of parents with relatively higher levels of educational attainment yielded stronger correlations between parenting and childhood depression than did studies comprised of parents with lower average levels of education. Because parent education is an indicator of socioeconomic status (SES), it may be that aversive parenting practices have more adverse consequences for children's mood at higher SES levels than at lower levels. It has been established that families in lower SES strata experience more stressors than other families (Bolland, Lian, & Formichella, 2005). Thus, it is possible that even if parenting patterns such as hostility are stressors that can trigger depression among vulnerable children, children from more disadvantaged families are exposed to so many other stressors that whether they experience depression is less

dependent on parenting practices. If this is true, it could account for the apparently lessened effect of parenting on childhood depression in families with relatively lower SES (as indicated by lower average levels of parent education) in this meta-analysis. It is important to note, though, that this finding is based only on 12 studies, and thus cannot be considered well-established.

Certain clinical implications may follow from the findings reported herein. Specific parental behaviors may represent possible targets for psychosocial treatments. Thus far, clinical science has been slow to develop interventions that target parenting behaviors hypothesized to contribute to the maintenance of child depression (Sander & McCarty, 2005). In a recent meta-analysis of the psychosocial treatment literature for child and adolescent depression, less than a third of the treatments involved parents (Weisz, McCarty, & Valeri, 2003), and in most of those the parenting component entailed primarily teaching parents the skills their children were being taught (i.e., not primarily addressing parent behaviors that might increase risk of youth depression). Developing interventions to target parenting behaviors associated with childhood depression, such as hostility and rejection, may represent an avenue for future research that could improve clinical care for affected youth (Sander & McCarty, 2005). Of course, since the direction of effects linking parental behaviors to childhood depression has not been established, it is important for researchers and clinicians to be mindful that children with depression may also *elicit* negative parental behaviors to some extent. It is quite possible that negative parenting and childhood depression exert an escalating reciprocity which, over time, causes parenting patterns and children's depression to become interwoven (cf. Dishion et al., 1994; Maccoby, 1992). It has been posited that parents and children get stuck in interactive cycles whereby the child displays depressed behavior that elicits negative parental behavior (e.g., rejection), which helps maintain the child's depressive symptoms. This cycle is hypothesized to become stronger over time and eventually persist even when the child's depressive symptoms have abated (Biglan, Lewin, & Hops, 1990; Chiariello & Orvaschel, 1995; Coyne, 1976; Hammen et al., 1990; Kaslow et al., 1994). Our findings highlight several specific parental behaviors that may contribute to the maintenance of such a cycle and thus represent possible targets for psychosocial interventions.

A few limitations of the current study warrant attention. Though the present findings indicate that parenting and childhood depression are linked (albeit modestly), this body of literature is limited in what it can tell us about the direction of effects. There are very few prospective studies and no experimental studies in the literature base that would allow researchers to test the direction of effects. Indeed, it is difficult to imagine an experimental study that would be plausible in design and ethically defensible. As a result, the following explanations cannot be ruled out: (a) parenting may directly cause or elicit childhood depression; (b) children's depressive symptoms (e.g., irritability) may elicit particular patterns of parenting; (c) genetic similarity between children and their parents may act as a "third variable" accounting for both parenting as well as childhood depression; or (d) genetic traits, parenting, children's depression symptoms, and other risk or protective factors may reinforce or moderate each other in a feedback loop. In short, parenting could be correlated with childhood depression, and yet not serve as a causal factor directly contributing to childhood depression.

The findings of this meta-analysis raise questions about how significant a role parenting may play in the emergence of childhood depression. Parenting only explained a small proportion of the variance in childhood depression. Meta-analyses addressing other social influences in relation to children's mental health outcomes typically obtain effects similar in magnitude to those we reported here (e.g., Amato, 2001; McLeod et al., 2007; Rothbaum & Weisz, 1994). Perhaps better methodology (e.g., more precise parenting measures) is needed to obtain the larger expected magnitudes of effect in studies of ecological influences such as parenting on outcomes such as depression. Or, alternatively, perhaps theoretical models have actually overemphasized the role that parenting plays in the development of childhood depression. Clearly, other factors, such as genetic influence, account for a substantial proportion of the variance in childhood depression. Although the literature currently provides a preliminary answer to the question, "What is the role of parenting in childhood depression?" (the role is limited in magnitude but intriguing in its forms), the use of different methodologies and research designs in future studies may provide a more nuanced statement of this general conclusion.

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