Published in final edited form as:
Clin Psychol Rev. 2013 December ; 33(8): 1205–1217. doi:10.1016/j.cpr.2013.10.004.

Preventing postpartum depression: A meta-analytic review

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Abstract

This meta-analysis assessed the efficacy of a wide range of preventive interventions designed to reduce the severity of postpartum depressive symptoms or decrease the prevalence of postpartum depressive episodes. A systematic review identified 37 randomized or quasi-randomized controlled trials in which an intervention was compared to a control condition. Differences between treatment and control conditions in the level of depressive symptoms and prevalence of depressive episodes by 6 months postpartum were assessed in separate analyses. Depressive symptoms were significantly lower at post-treatment in intervention conditions, with an overall effect size in the small range after exclusion of outliers (Hedges' g = 0.18). There was a 27% reduction in the prevalence of depressive episodes in intervention conditions by 6 months postpartum after removal of outliers and correction for publication bias. Later timing of the postpartum assessment was associated with smaller differences between intervention and control conditions in both analyses. Among studies that assessed depressive symptoms using the EPDS, higher levels of depressive symptoms at pre-treatment were associated with smaller differences in depressive symptoms by 6 months postpartum. These findings suggest that interventions designed to prevent postpartum depression effectively reduce levels of postpartum depressive symptoms and decrease risk for postpartum depressive episodes.

Keywords

Postpartum depression; Prevention; Meta-analysis

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1. Introduction

While the goal of treatment is to alleviate symptoms among individuals experiencing a given disorder, preventive interventions are intended to avoid the initial onset of disorder. Emotional and behavioral difficulties are commonly identified and treated only after the onset of illness, but prevention of these disorders can significantly reduce the human and economic costs associated with mental illness (National Research Council & Institute of Medicine, 2009). A recent review of progress that has been made in the field of depression prevention identified the implementation of interventions with strong evidence of effectiveness as a major goal for ongoing research in this area (Muñoz, Beardslee, & Leykin, 2012). In order for this goal to be reached, it is necessary to identify characteristics of effective preventive interventions.

Postpartum depression is a specific mental disorder for which preventive interventions could yield dramatic benefits. Depression is one of the most common complications of childbearing; a meta-analytic review found that approximately 13% of women will experience a major depressive episode during the first postpartum year (O'Hara & Swain, 1996). According to the World Health Organization, depression is the leading cause of disability worldwide (World Health Organization, 2012). Beyond the distress and impairment experienced by women with depression during the postpartum period, research also indicates that postpartum depression has negative consequences for the children of depressed mothers. Postpartum depression limits a woman's ability to function effectively in the maternal role: depressed mothers provide less responsive caregiving, are more likely to discontinue breastfeeding early or have problems breastfeeding, are less likely to comply with recommended safety practices such as use of car seats, and their children have lower rates of preventive healthcare utilization and vaccination (Field, 2010). Infants of depressed mothers show impairments in social engagement and emotional regulation, increased negative emotionality, and high cortisol reactivity (Feldman et al., 2009). Impairments in mother–infant bonding, including severe disorders of the mother–infant bond that may include rejection of the infant or abusive behavior, are more common among women experiencing postpartum depression (Brockington, Aucamp, & Fraser, 2006). Postpartum depression is also associated with increased risk for long-term cognitive impairment, emotional difficulties, and behavioral problems (Grace, Evindar, & Stewart, 2003).

The context in which postpartum depression occurs provides unique opportunities for preventive interventions. Women with fewer financial resources may have greater access to healthcare during pregnancy than during other points in the lifespan; for example, in the United States, women are eligible for Medicaid during pregnancy and the first 60 days postpartum (Centers for Medicare & Medicaid Services, 2012). More generally, pregnancy is a time of increased healthcare utilization, which provides opportunities for screening and intervention. Research has identified demographic groups at high risk for postpartum depression, such as minority women and women of low socioeconomic status, which may be used to target women at increased risk for the disorder (Beck, 2001; O'Hara & Swain, 1996). Finally, there is some evidence that preventive interventions may be more acceptable than treatment for depression, particularly among African-American women (Crockett, Zlotnick, Davis, Payne, & Washington, 2008).

A wide range of interventions for preventing postpartum depression have been assessed in randomized controlled trials. Many preventive interventions have modified treatments demonstrated to be effective for postpartum depression. For example, psychotherapy – particularly cognitive-behavioral and interpersonal psychotherapy – and antidepressant medication have all been shown to be effective in the treatment of postpartum depression (Sockol, Epperson, & Barber, 2011). Some studies have assessed whether implementation of these interventions before the onset of a depressive episode can effectively prevent the disorder (e.g., Austin et al., 2008; Wisner et al., 2001; Zlotnick, Capezza, & Parker, 2011). Non-therapeutic social support and educational interventions have also been assessed as preventive interventions (e.g., Dennis et al., 2008). Other research has investigated whether modifications to standard postpartum care, such as having women attend their first postpartum checkup at 1 week instead of 6 weeks postpartum, can reduce the incidence of depression after childbirth (Gunn, Lumley, Chondros, & Young, 1998). Alternative biological treatments, notably dietary supplements and hormonal interventions, have also been assessed as potential preventive interventions for postpartum depression (e.g., Lawrie et al., 1998; Llorente et al., 2003). Given the wide range of approaches that have been utilized in prevention research, a comprehensive review of the research in this area is needed to provide clinicians and researchers with important information regarding the absolute and relative efficacy of these interventions.

While a great number of reviews of the literature on the prevention of postpartum depression have been published, most of these reviews are qualitative in nature. Boath, Bradley, and Henshaw (2005) reviewed evidence from twenty-one randomized controlled trials, including both psychosocial interventions and biological interventions. Overall, this review found that there is evidence for their short-term effectiveness, particularly for psychosocial interventions, but that there was no evidence for long-term effectiveness. Dennis has published two reviews evaluating the evidence for biological (Dennis, 2004a) and non-biological (Dennis, 2004b) interventions. In the first review (Dennis, 2004a), she described seven studies of biological interventions including antidepressant medications, hormonal therapy, thyroid therapy, DHA, and calcium supplementation. Most of these studies found no significant group differences; however, Dennis noted that given the methodological limitations of the included studies no recommendations for clinical practice can be made on the basis of existing evidence. In the second review (Dennis, 2004b), she described twenty-nine studies of non-biological interventions including psychotherapy, psychological debriefing, educational classes, social support, continuity of care and modifications to postpartum care, and relaxation. This review found that the evidence for the efficacy of these interventions is mixed and there is insufficient evidence to recommend any specific intervention, particularly given the methodological limitations of many of the reviewed studies.

Several quantitative systematic reviews have attempted to synthesize prior findings in this area. Lumley, Austin, and Mitchell (2004) reviewed studies initiated during pregnancy and the postpartum period; their meta-analysis found that only indicated postnatal interventions were associated with decreased risk for postpartum depression. This metaanalysis did not assess possible moderators of effect sizes. In another quantitative review, Dennis (2005) conducted a meta-analysis of 15 psychological and psychosocial interventions for

preventing postpartum depression. These analyses found that prevention programs did not significantly reduce risk for postpartum depression. However, analyses of moderators suggested that interventions were more effective when they targeted women at increased risk, when they included a postnatal component, and when they were administered individually. In their review of hormonal interventions for preventing and treating postpartum depression, Dennis, Ross, and Herxheimer (2009) identified only one study in which hormones were utilized as a preventive intervention. Similarly, a review of antidepressant prevention of postnatal depression identified only two studies in which medication was utilized for prevention, rather than treatment, of postpartum depression (Howard, Hoffbrand, Henshaw, Boath, & Bradley, 2005). A protocol for a review of dietary supplements for preventing postpartum depression has been published, but the review has yet to be conducted (Miller, Murray, Beckmann, Kent, & Macfarlane, 2011).

Overall, existing meta-analyses suggest that preventive interventions for postpartum depression may have limited efficacy. However, these analyses have several limitations. Each of these analyses was limited to a single type of intervention (e.g., psychosocial, hormonal, pharmacological), which precludes the comparison of these approaches. With the exception of the Dennis (2005) meta-analysis, these studies have not assessed elements of study design or interventions as potential moderators of the efficacy of these interventions. These studies also fail to specify the timing of the postpartum assessments that were used to calculate the effect sizes. A meta-analytic review of depression during the perinatal period found that the prevalence of this disorder decreases after seven months postpartum, which suggests that the timing of evaluation should be considered when evaluating the efficacy of prevention programs (Gavin et al., 2005). Finally, a number of new prevention trials have been published since these earlier meta-analyses were conducted.

The present meta-analysis addresses several limitations of the above studies. We included a wide range of interventions, which allows for the direct comparison of the efficacy of different approaches. We included interventions other than antidepressant medication and psychotherapy, as complementary and alternative approaches to the treatment and prevention of depression have high levels of acceptability among perinatal populations (Battle, Uebelacker, Howard, & Castaneda, 2010). In order to assess whether these alternative interventions are as effective as empirically supported treatments, we elected to include as wide a range of preventive interventions as was possible. We limited our analyses to those in which postpartum depression was assessed within the first 6 months postpartum. We assessed characteristics of included studies and interventions as potential moderators of effect size. We also included several studies that have been published since earlier metaanalyses were conducted. The goal of the current meta-analysis was to assess the efficacy of a range of preventive interventions for postpartum depression. We assessed both the level of depressive symptoms in treatment conditions compared to control conditions and the difference in the prevalence of depressive episodes by six months postpartum.

2. Method

2.1. Search procedures and selection of studies

Relevant studies were identified through searches of PsycInfo and PubMed through 2012 using *postpartum depression* and *prevention* as keyword search terms. The reference lists of existing meta-analyses, relevant reviews, chapters, and retrieved articles were inspected for further relevant studies. Clinical trial databases (including the Cochrane Pregnancy and Childbirth Group, Cochrane Depression, Anxiety and Neurosis Group, and the International Standard Randomised Controlled Trial Number Register) were also reviewed for eligible studies.

To be included in the meta-analysis, studies had to meet the following inclusion criteria:

- **a.** Study design included intervention and control group(s). Both randomized and quasi-randomized controlled trials were eligible for inclusion. Due to the need to compare separate treatment and control conditions for the calculation of effect sizes, single-case designs were excluded.
- **b.** Authors specified that the goal of the intervention was to reduce postpartum depressive symptoms and/or the prevalence of postpartum major depressive episodes. Interventions that did not explicitly target depressive symptoms, such as smoking cessation programs, were excluded, even if authors reported outcome data for depressive symptoms and/or major depressive episodes. Interventions in which maternal depression was not the primary outcome of interest, such as studies of infant development, were excluded. Interventions designed to treat postpartum depression were excluded. Interventions were classified as treatment studies if all subjects met criteria for a major depressive episode at pre-treatment or if all subjects had depressive symptoms above a cutoff indicative of clinically significant depressive symptoms at pre-treatment.
- **c.** Intervention was initiated during pregnancy or within 4 weeks of childbirth.
- **d.** Reported outcomes for depressive symptoms and/or prevalence of depressive episodes between 1 and 6 months postpartum using a validated self-report or clinician-administered measure.
- **e.** Reported sufficient outcomes to allow for the calculation of effect size(s).

A flow chart summarizing the search process and exclusion of studies is presented in Fig. 1. After removal of duplicates, the search procedure yielded 797 articles. Abstracts for these articles were reviewed and the full text of 117 potentially relevant articles were obtained and reviewed for inclusion. Of these 117 articles, 80 were excluded for the following reasons: 17 were excluded because the target outcome of the intervention was not depressive symptoms or depression diagnosis, 16 were excluded because they were not randomized or quasi-randomized controlled trials, 14 were excluded because they did not report outcome data or reported insufficient data for the calculation of effect sizes, 11 were excluded because the intervention was initiated after 4 weeks postpartum, 5 were excluded because they did not include a postpartum assessment between 1 and 6 months postpartum, 4 were excluded because they were treatment studies in which subjects were selected on the basis of

depressive symptoms and/or diagnosis, and 1 was excluded because the measure of depressive symptoms was not validated. Secondary manuscripts were identified for 12 studies; all original manuscripts provided sufficient information for coding and calculation of effect sizes so these articles were not utilized. The remaining 37 articles were eligible for inclusion in the metaanalysis. Twenty-four studies reported sufficient outcome measures for calculation of effect sizes representing the difference in depressive symptoms between treatment and control conditions by 6 months postpartum, and 28 studies reported sufficient outcome measures for calculation of effect sizes representing the difference in prevalence of depressive episodes by 6 months postpartum.

2.2. Coding of studies

All studies were coded for intervention type (dietary supplement vs. educational vs. hormonal vs. medication vs. modified care vs. therapy vs. social support). Interventions were classified as educational when the intervention consisted of providing information, either verbal or written, regarding postpartum depression and accessing treatment without actively engaging participants in activities designed to change behavior or mood. Interventions were coded as modified care whey they consisted of changes to standard obstetric care (e.g., increasing frequency or changing timing of postnatal appointments). Interventions were coded as therapy when they were clinician-led and participants were engaged in activities with a goal of modifying behavior, cognition, or mood. Interventions in which participants were provided with nonspecific support were coded as social support interventions. For moderator analyses, interventions were also coded as biological interventions (dietary supplement, hormonal, and medication) or psychosocial interventions (educational, modified care, therapy, and social support) and as established treatments for postpartum depression (cognitive-behavioral therapy, interpersonal psychotherapy, and antidepressant medication) and non-established treatments for postpartum depression (dietary supplements, educational interventions, hormonal interventions, modified care, other psychotherapies, and social support).

Studies were also coded for type of control group (active vs. educational vs. placebo vs. treatment-as-usual), timing of intervention (pregnancy vs. labor vs. postpartum), outcome measure, and timing of postpartum assessment (in weeks). The type of prevention study was classified using the criteria proposed by the Institute of Medicine report on prevention research (Mrazek & Haggerty, 1994): *indicated* interventions target individuals with subclinical symptoms who do not meet diagnostic criteria, *selected* interventions target individuals with risk factors for a disorder but without symptoms of the disorder, and *universal* interventions are administered to all members of a given population. Several studies included subjects who were either at-risk or exhibiting sub-clinical symptoms of depression; we classified these studies as *selected/indicated*. While a conservative definition of preventive interventions would have required us to exclude studies in which subjects were experiencing major depressive episodes at pre-treatment, over a third of the potential studies either did not assess for the presence of a major depressive episode at pre-treatment or did not exclude subjects on the basis of a positive screening. Given the large number of studies that would have been excluded on the basis of this criterion, we elected to include these studies and to assess this as a potential moderator of effect size (excluded subjects with

MDE at pre-treatment vs. did not assess/did not exclude subjects with MDE at pre-treatment). We also coded the average level of depressive symptoms at pre-treatment across treatment and control conditions.

Because studies did not consistently report sample characteristics (ethnicity, parity, and marital status), these variables were not coded.

The only intervention type for which enough studies were included to assess potential moderators of effect size was therapeutic interventions. These studies were also coded for therapeutic orientation (cognitive-behavioral therapy vs. eclectic vs. interpersonal psychotherapy), whether therapy was conducted individually or in a group format, and the number of therapy sessions.

Effect sizes were calculated using the study's designated primary outcome measure. When more than one postpartum assessment was conducted between 1 and 6 months postpartum, the latest assessment point was used.

2.3. Analyses

Two separate analyses were conducted. The first analysis compared the difference in depressive symptoms by 6 months postpartum between treatment and control conditions using the standardized mean group difference. While this effect size does not account for possible differences in depressive symptoms between treatment and control conditions at pre-treatment, too few studies reported pre-treatment depressive symptoms for effect sizes that take these potential differences into account to be calculated. Effect sizes were calculated by dividing the difference between treatment and control means by the pooled standard deviation, corrected for upward bias using Hedges' g (Hedges, 1981):

$$g = c_{\mathrm{m}} \left[\frac{M_T - M_C}{SD_P} \right]$$

where the pooled standard deviation is defined as

$$SD_P = \sqrt{\frac{(n_T - 1)SD_T^2 + (n_C - 1)SD_C^2}{(n_T + n_C - 2)}}$$

and c_m is defined as

$$c_{\mathrm{m}} = 1 - \frac{3}{4(n-1)}.$$

Effect sizes were calculated so that positive effect sizes represent lower scores in the intervention group compared to the control group.

The second analysis compared the prevalence of depressive episodes by 6 months postpartum between treatment and control conditions using the odds ratio:

$$OR=\frac{P_T(1-P_C)}{P_C(1-P_T)}.$$

Where P_T is the proportion of depressed subjects in treatment conditions and P_C is the proportion of depressed subjects in treatment conditions. Odds ratios less than 1 indicate lower rates of depression among treated conditions compared to control conditions.

The heterogeneity of effect sizes was examined using the *Q* statistic and the I^2 index. Significant *Q* statistics indicate that the observed range of effect sizes is significantly larger than would be expected based on within-study variance. The I^2 value indicates the proportion of variance in effect sizes accounted for by between-study variance. The index has a range from 0 to 100; Higgins, Thompson, Deeks, and Altman (2003) suggest that 25, 50 and 75% I^2 values indicate low, medium and high levels of hetereogeneity, respectively. When analyses indicated significant heterogeneity among effect sizes, exploratory analyses were conducted to assess for moderators of effect size. Categorical moderators were assessed using an analysis of variance (ANOVA) of mixed-effects models for each variable hypothesized to influence the effect size. Meta-regression analyses were conducted to assess the effects of continuous moderators.

Publication bias was assessed by visual examination of funnel plots, Duval and Tweedie's (2000) trim-and-fill procedure, and classic failsafe *N* values (Rosenthal, 1979). First, the effect size for each study was plotted against the study standard error. An asymmetric distribution suggests missing studies due to publication bias (Lipsey & Wilson, 2001). We used Duval and Tweedie's trim-and-fill procedure (2000) to identify asymmetric distributions of effect sizes. When this test indicated significant asymmetry in the funnel plot, the overall estimates for the model were calculated using the trim-and-fill correction (Duval & Tweedie, 2000). Using the fail-safe *N* value, we determined the number of studies with null findings that would be necessary to produce a nonsignificant overall effect size. Using Rosenthal's (1991) recommendation, a value of 5K + 10, where K is the number of observed studies, was used as the cutoff for an unlikely number of studies.

For each of these analyses, outliers were identified using the sample-adjusted meta-analytic deviance (SAMD) statistic (Huffcutt & Arthur, 1995). A conservative cutoff score of 2.58 was used to consider studies for exclusion from the analyses, since extreme values can result from either true population variability or error, and removing outliers whose effects represent true variability limits the ability to assess the role of moderators (Beal, Corey, & Dunlap, 2002). The SAMDs were rank-ordered and the scree plots examined to confirm the outlier status of studies with SAMDs above this cutoff. In cases where the SAMD value was greater than 2.58 but the scree plot suggested that the SAMD was not discrepant from the overall distribution, the study was retained to maximize the variance available to assess the role of moderators.

Calculations of weighted mean effect sizes, heterogeneity, and moderators were conducted using Comprehensive Meta-Analysis, version 2.2.046 (Borenstein, Hedges, Higgins, & Rothstein, 2005). We estimated overall effect sizes using random effects models, based on

the assumption that the included studies represent a distribution of true intervention effects. Considerable heterogeneity of effect sizes was expected given the differences in interventions and samples across the included studies. As the Q statistic is underpowered in cases of small sample size (Lipsey & Wilson, 2001), random effects models were estimated regardless of the observed heterogeneity.

3. Results

3.1. Characteristics of included studies

Table 1 presents characteristics of the studies included in the analyses. Studies included a wide range of intervention types, including therapy (n = 18), modified care (n = 6), social support (n = 6), antidepressant medication (n = 2), educational programs (n = 2), dietary supplements (n = 2), and hormonal interventions (n = 1). Control group types included treatment-as-usual (n = 24), educational programs (n = 7), placebo (n = 5), and a nonspecific active treatment (n = 1). Interventions were initiated during pregnancy (n = 23), the first four weeks postpartum (n = 13), or during labor (n = 1). Prevention types included indicated interventions (n = 3), selected/ indicated interventions (n = 9), selected interventions (n = 12), and universal interventions (n = 13). The timing of the postpartum assessment ranged from 4 to 24 weeks, with the average assessment taking place at 14.6 weeks postpartum (SD = 6.7).

3.1.1. Characteristics of therapy interventions—Eighteen studies assessed therapeutic interventions. One study assessed training in guided relaxation provided via videotape; this study was excluded from moderator analyses of therapy characteristics due to differences in the method of administration of the intervention. The remaining studies assessed cognitive-behavioral (n = 10), interpersonally-oriented (n = 5), and eclectic (n = 2) interventions. Studies included both group therapy (n = 10) and individually-administered therapy (n = 7). The average number of therapy sessions was 5.9 (SD = 3.0). Study therapists came from a variety of fields including midwifery (n = 4), psychology (n = 4), nursing (n = 2), social work (n = 2), occupational therapy (n = 1) and psychiatry (n = 1). There was also considerable variability in the level of educational attainment of therapists, which ranged from bachelors'-level research staff (n = 1), to graduate-level students (n = 4), to psychiatrists (n = 1). Many studies included therapists from multiple backgrounds. For example, the intervention developed by Austin et al. (2008) was implemented by a clinical psychologist with a midwife who acted as a co-therapist.

3.2. Methodological quality

Table 2 presents characteristics of the included studies related to methodological quality. Two studies were quasi-randomized trials; the remaining 35 studies were randomized controlled trials. 62% of studies reported results on the basis of intent-to-treat analyses. 95% of studies provided some information characterizing the included sample. 28% of studies excluded participants with current major depressive episodes. Of the 19 studies that included a clinician-administered measure, 63% reported that assessors were blind to treatment status. Of the 35 randomized controlled trials, 83% specified the method by which participants were randomized.

3.2.1. Methodological quality of therapy interventions—Eighteen studies included therapeutic interventions. One of these interventions was provided via videotape. Of the remaining 17 studies, 83% provided information about the therapists who provided the intervention, 56% indicated that an intervention manual was utilized, 78% indicated that therapists received training in the intervention, 67% indicated that therapists received supervision during the study, and 44% assessed sessions for adherence to the intervention.

3.2.2. Methodological quality of pharmacological interventions—Five studies included pharmacological interventions (antidepressant medication, dietary supplements, or hormonal interventions). For these studies, 80% reported that clinicians were blind to treatment status and 100% reported that participants were blind to treatment status.

3.3. Postpartum depressive symptoms

Table 3 presents the results of the random effects model for postpartum depressive symptoms, representing results from 24 studies. These effect sizes represent the difference between depressive symptoms at the postpartum assessment closest to 6 months postpartum; positive effect sizes indicate superiority of treatment to control conditions. Effect sizes (Hedges' g) ranged from −0.20 to 12.10; eight studies had significant effect sizes, all in favor of the treated condition. There was a significant overall effect of treatment (g = 0.37, 95% CI 0.15–0.60, $p < 0.001$). Two studies had SAMD values greater than 2.58. Visual inspection of the scree plot of the rank-ordered SAMD scores suggested that the SAMD values for the studies by Small, Lumley, Donohue, Potter, and Waldonstrom (2000) and Wolman, Chalmers, Hofmeyr, and Nikodem (1993) were discrepant with the overall distribution of SAMD scores. These studies were excluded from subsequent analyses; the average effect size excluding these outliers was g = 0.18 (95% CI 0.09–0.27, $p< 0.001$).

We also used meta-analysis to assess the average level of depressive symptoms by six months postpartum in treatment and control conditions. In the 14 studies that utilized the EPDS as a measure of depressive symptoms, the average EPDS score was 7.06 in treatment conditions, compared to 7.69 in control conditions. In the five studies that used the BDI-II as a measure of depressive symptoms, the average BDI score was 8.99 in treatment conditions, compared to 8.55 in control conditions. In the two studies that used the CES-D as a measure of depressive symptoms, the average CES-D score was 1.49 in treatment conditions, compared to 1.57 in control conditions.

Results of tests for publication bias were acceptable. The fail-safe N value was 129, which exceeds the tolerance value of 120. While the funnel plot was slightly asymmetric (see Fig. 2); trim-and-fill procedures suggested no missing studies. The Q statistic indicated that there was significant heterogeneity among effect sizes ($p < 0.05$). The I^2 value indicated a medium level of heterogeneity, with 37% of the variance in effect sizes attributable to between-study variance (Higgins et al., 2003).

3.4. Moderator analyses: postpartum depressive symptoms

Because both the *Q* statistic and I^2 index indicated significant heterogeneity of effect sizes, exploratory analyses of potential moderators were conducted. Subgroups including only one study were excluded from moderator analyses.

3.4.1. Study characteristics—Nine characteristics of the included studies were assessed as potential moderators: intervention type (general, biological vs. psychosocial, and EST vs. non-EST), control group type, timing of intervention, type of prevention, measure, whether the study excluded women with a current major depressive episode, timing of postpartum assessment, and average pre-treatment depressive symptoms (see Table 4). No categorical variables were significant moderators of effect size. There was a trend for later assessment timing to be associated with smaller effect sizes, indicating a smaller difference between treatment and control conditions at later assessment points; slope = −0.01, $p = 0.05$. In studies that assessed depressive symptoms using the EPDS, higher levels of depressive symptoms at pre-treatment were associated with smaller effect sizes, indicating a smaller difference between treatment and control conditions at later assessment points; slope = −0.07, $p < 0.01$. There was no relationship between depressive symptoms at pre-treatment and effect size in studies that assessed depressive symptoms using the BDI-II, slope = 0.01, $p > 0.05$.

3.4.2. Intervention variables—Three characteristics of interventions for studies assessing psychotherapeutic interventions were assessed as potential moderators: therapeutic orientation, method of administration, and number of sessions. There were not enough studies representing other types of interventions to assess moderators for these interventions. No categorical characteristics of psychotherapeutic interventions were significant moderators of effect size. There was a trend for studies with more therapy sessions to have smaller effect sizes, indicating a smaller difference between treatment and control conditions at later assessment points; slope = −0.04, $p = 0.06$.

3.5. Postpartum depression diagnosis

Table 5 presents the results of the random effects model for postpartum depression diagnoses, representing results from 28 studies. Odds ratios for individual studies ranged from 0.02 to 1.79. Odds ratios were significant for eight individual studies; seven in favor of the treated condition and one in favor of the control condition. There was a significant overall positive effect of treatment (*OR* = 0.72, 95% CI 0.0.56–0.94, $p = 0.01$), representing a 28% reduction in risk for postpartum depression in treatment groups compared to control groups. Nine studies had SAMD values greater than 2.58. Visual inspection of the scree plot of the rank-ordered SAMD scores indicated that the value for the studies by Kozinszky et al. (2012) and Small et al. (2000) were discrepant. These studies were excluded from subsequent analyses. The average effect size, excluding these outliers, was *OR* = 0.67 (95% CI 0.52–0.85, $p < 0.01$), which represents a 33% reduction in risk for treatment groups compared to control groups.

Results of tests for publication bias indicated potential bias in the included studies. The fail-safe *N* value was 147, which exceeds the tolerance limit of 140. The funnel plot was

asymmetric (see Fig. 3), and the trim-and-fill correction suggested 5 studies missing to the right of the mean. After correction for publication bias, the overall effect size was 0.73 (95% CI 0.56–0.95), which represents a 27% reduction in the risk for treatment groups compared to control groups. The Qstatistic indicated that there was significant heterogeneity among the effect sizes ($p < 0.01$). The I^2 value indicated a medium level of heterogeneity, with 46% of the variance in effect sizes attributable to between-study variance (Higgins et al., 2003).

3.6. Moderator analyses: postpartum depression diagnosis

3.6.1. Study characteristics—Ten characteristics of the included studies were assessed as potential moderators: intervention type (general, biological vs. psychosocial, and EST vs. non-EST), control group type, timing of intervention, type of prevention, method of diagnosing depression, whether the study excluded women with a current major depressive episode, timing of postpartum assessment, and baseline depressive symptoms (see Table 6). No categorical variables were significant moderators of effect size. Studies with later assessments had larger effect sizes, indicating a smaller difference between treatment and control conditions at later assessment points; slope = 0.02, $p < 0.05$. There was no relationship between depressive symptoms at pre-treatment and effect size in studies that assessed depressive symptoms using the EPDS, slope = 0.04, $p > 0.05$.

3.6.2. Intervention variable—Three characteristics of interventions for studies assessing psycho-therapeutic interventions were assessed as potential moderators: therapeutic orientation, method of administration, and number of sessions. There were not enough studies representing other types of interventions to assess moderators for these interventions. None of these variables was a significant moderator of effect size.

4. Discussion

Results of these meta-analyses suggest that a wide range of interventions may be effective in the prevention of depression during the first 6 months postpartum. These interventions result in small but significant reductions in depressive symptoms ($g = 0.18$) and the prevalence of depressive episodes ($OR = 0.73$). Although the magnitude of the effects of preventive interventions are modest compared to treatments for postpartum depression, which a previous meta-analysis found to be in the medium range ($g = 0.65$, Sockol et al., 2011), the efficacy of these interventions is comparable to, or exceeds, the efficacy of preventive interventions for anxiety and depression from other meta-analyses (Cuijpers, van Straten, Andersson, & van Oppen, 2008; Zalta, 2011). The overall level of depressive symptoms by six months postpartum in both treatment and control conditions were below generally accepted cutoffs for clinically significant depressive symptoms (Cox, Chapman, Murray, & Jones, 1996; Dozois & Dobson, 2002).

For both depressive symptoms and depression diagnosis, a later assessment was associated with a smaller difference between intervention and control conditions. This is consistent with the results of a meta-analysis of treatments for postpartum depression, which found that greater treatment length was associated with smaller effect sizes (Sockol et al., 2011). Moreover, it is consistent with evidence that postpartum depression tends to naturally remit over time (Heron, O'Connor, Evans, Golding, & Glover, 2004). Given that the natural

course of postpartum depression is for symptom severity to decrease over time, it is unsurprising that preventive interventions appear to be most efficacious when they are assessed early during the postpartum period. However, this should not be taken as an indication that preventive interventions are unnecessary. Given the adverse impact of depression on depressed women and their children (Grace et al., 2003), even a self-limiting depressive episode may be extremely distressing and increase the risk for long-term negative outcomes.

Higher levels of depressive symptoms at pre-treatment were associated with smaller differences in depressive symptoms by six months postpartum between treatment and control conditions in studies that used the EPDS as a measure of depressive symptoms. As this result was only found in one of our analyses, and for only one measure of depressive symptoms, this result should be interpreted with caution. However, if this finding represents a true difference in the efficacy of preventive interventions, this suggests that preventive interventions might be more effective for women who are not yet experiencing significant levels of depressive symptoms. The duration or intensity of preventive interventions may not be sufficient to prevent the onset of depressive episodes or worsening of symptoms among this population.

Interestingly, we found that intervention type was not related to the effectiveness of treatments for either reducing depressive symptoms or preventing depressive episodes. A lack of social support is an established risk factor for postpartum depression (Beck, 2001). It may be that nonspecific social contact and support is sufficient for reducing risk for depression among this population and that the specific active elements of treatment are less important. However, further research assessing the efficacy of less well-studied interventions is necessary to determine whether our failure to identify moderators simply results from a lack of sufficient evidence. Given the small number of studies representing antidepressant medication and non-traditional interventions, particularly dietary supplements and hormonal interventions, further research is necessary to establish whether these approaches are truly equally efficacious.

One limitation of this meta-analysis was the use of uncontrolled effect sizes. This raises the concern that differences at post-treatment may actually reflect pre-existing differences between treatment and control conditions. A separate meta-analysis was conducted assessing the average change in depressive symptoms between treatment and control conditions, controlling for pre-treatment symptom levels, using the 13 studies for which this effect size could be calculated (Morris, 2008). The fail-safe *N* for this analysis was 17, which is well below the tolerance value, so the results should be interpreted with caution. With this caveat, this analysis also found a small but significant difference in the reduction of depressive symptoms between treatment and control conditions at post-treatment, Hedges' $g = 0.15$, $p = 0.01$,95% CI 0.03–0.27. The results of this analysis suggest that our findings are unlikely to simply reflect preexisting differences between treatment and control conditions.

While the number of studies included in these meta-analyses is comparable to other meta-analyses of preventive interventions (e.g., Cuijpers et al., 2008; Zalta, 2011), moderator analyses assessed small subgroups of studies. Because of this, moderator analyses should be

interpreted with caution. This is particularly true for the analyses of intervention type. There were relatively few studies assessing antidepressant medication, dietary supplements, educational interventions, hormonal interventions, and social support programs. More research assessing the efficacy of these interventions is necessary in order to establish whether there are systematic differences between types of interventions. Similarly, psychotherapy was the only type of intervention for which enough studies were present to assess for potential moderation of specific aspects of the intervention. Further evaluation of other types of interventions would allow for similar questions to be asked of these interventions; for example, whether phone-based social support programs have comparable efficacy to in-person support groups. Due to inconsistencies across studies in the reporting of demographic characteristics, we were also unable to assess these as potential moderators. Future research should help clarify whether particular interventions are more effective for specific populations, especially women of low socioeconomic status, ethnic/racial minorities, and single women who are at higher risk for postpartum depression (Beck, 2001; O'Hara & Swain, 1996; Robertson, Grace, Wallington, & Stewart, 2004).

A significant concern raised by these analyses is the evidence that published studies are biased in favor of studies with significant positive findings. While the overall effect for preventive interventions remained significant even after correction for publication bias, there is no statistical approach that can take the place of real data for moderator analyses. While our analyses found no evidence that types of interventions or characteristics of interventions were associated with efficacy, it is possible that there are systematic characteristics of ineffective interventions that we were unable to assess because these results have not been published. This may have limited our ability to identify moderators of effect size. While the “file-drawer problem” is well-known, these analyses provide further evidence that null findings from well-designed prevention studies are vitally important to a full understanding of these interventions.

A major limitation of research in this area is that most studies do not report infant outcomes. As postpartum depression is associated with a range of negative infant and child outcomes (Grace et al., 2003), it is important to investigate whether interventions that prevent or reduce the severity of depressive symptoms during the postpartum are able to lessen the impact of maternal psychopathology on infant development. Only one study that was eligible for these metaanalyses reported infant outcomes. Armstrong, Fraser, Dadds, and Morris (1999) found no differences in breastfeeding rates, knowledge or practice of SIDS-preventing behavior, or use of health services by the mother or infant at six weeks postpartum. While some studies have shown that intervention may improve both maternal depression and infant outcomes (e.g., Field et al., 1996; Murray, Cooper, Wilson, & Romaniuk, 2003), others find that treatment for postpartum depression is not sufficient to improve infant outcomes (Forman et al., 2007). Future studies investigating preventive and treatment interventions for perinatal depression should endeavor to assess infant outcomes in order to determine whether these interventions result in improved infant outcomes.

In summary, these analyses suggest that a wide range of interventions are effective in the prevention of postpartum depression. By six months postpartum, these interventions are associated with a 27% reduction in the prevalence of depressive episodes and a reduction in

levels of depressive symptoms compared to control conditions. Effect sizes were larger in studies that assessed depression earlier in the postpartum period; this is consistent with natural remission of depressive symptoms over the course of the postpartum period. In these meta-analyses, we found no differences between types of interventions, and different types of psychotherapeutic interventions appeared to have comparable efficacy. There were few studies assessing antidepressant medication and other non-therapeutic interventions; more research is necessary to assess whether these interventions are effective and to establish whether characteristics of other intervention types are related to efficacy. Although more research is needed to confirm and extend the results of these meta-analyses, these results suggest that a wide range of interventions should be targeted for further investigation as preventive interventions for this disorder.

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HIGHLIGHTS

- The perinatal period is an opportune time to reduce psychological morbidity.
- Many types of interventions may prevent postpartum depression.
- These interventions result in significantly lower levels of depressive symptoms.
- These interventions significantly reduce the risk of major depressive episodes.

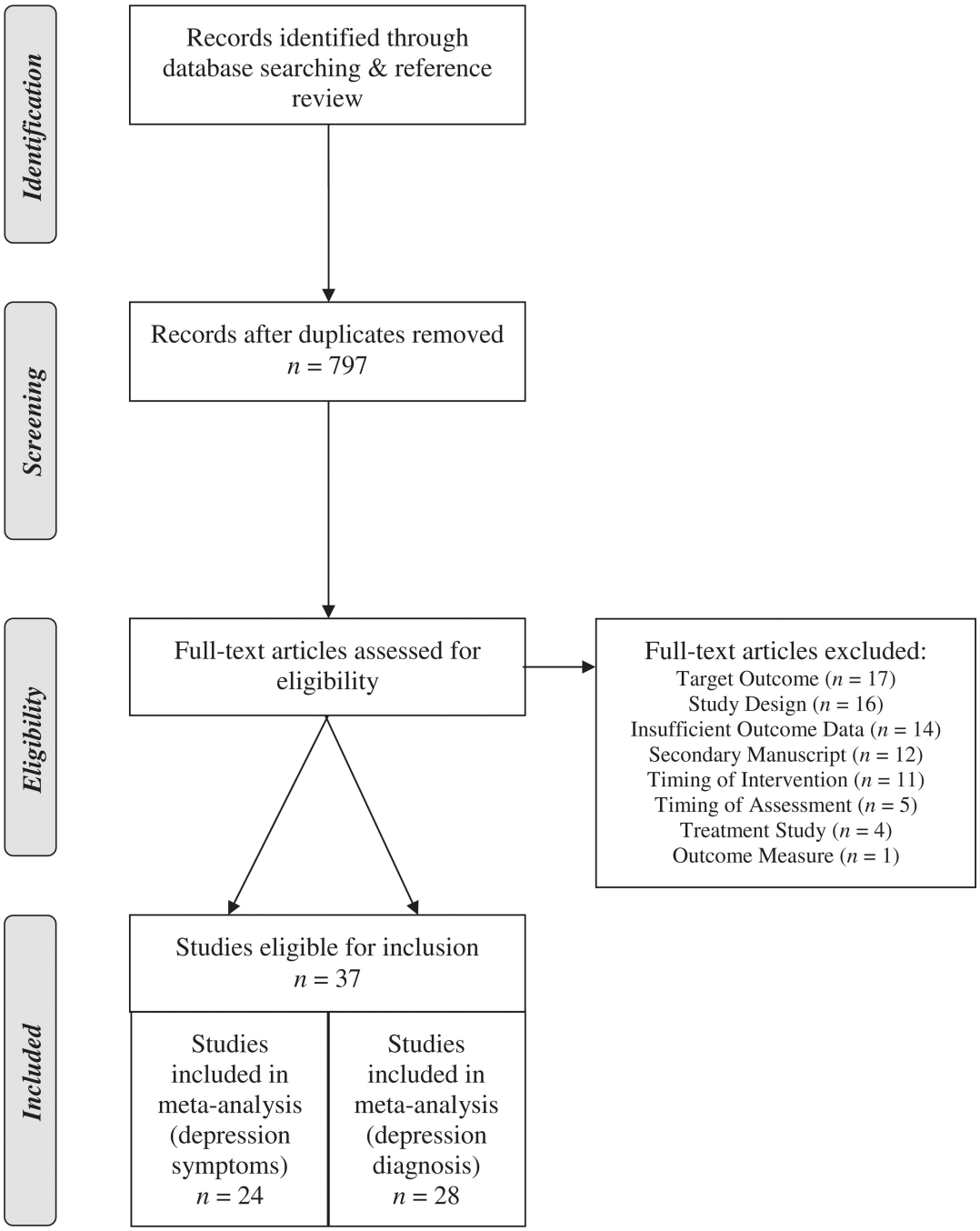

Fig. 1.
Flow chart illustrating identification of included studies.

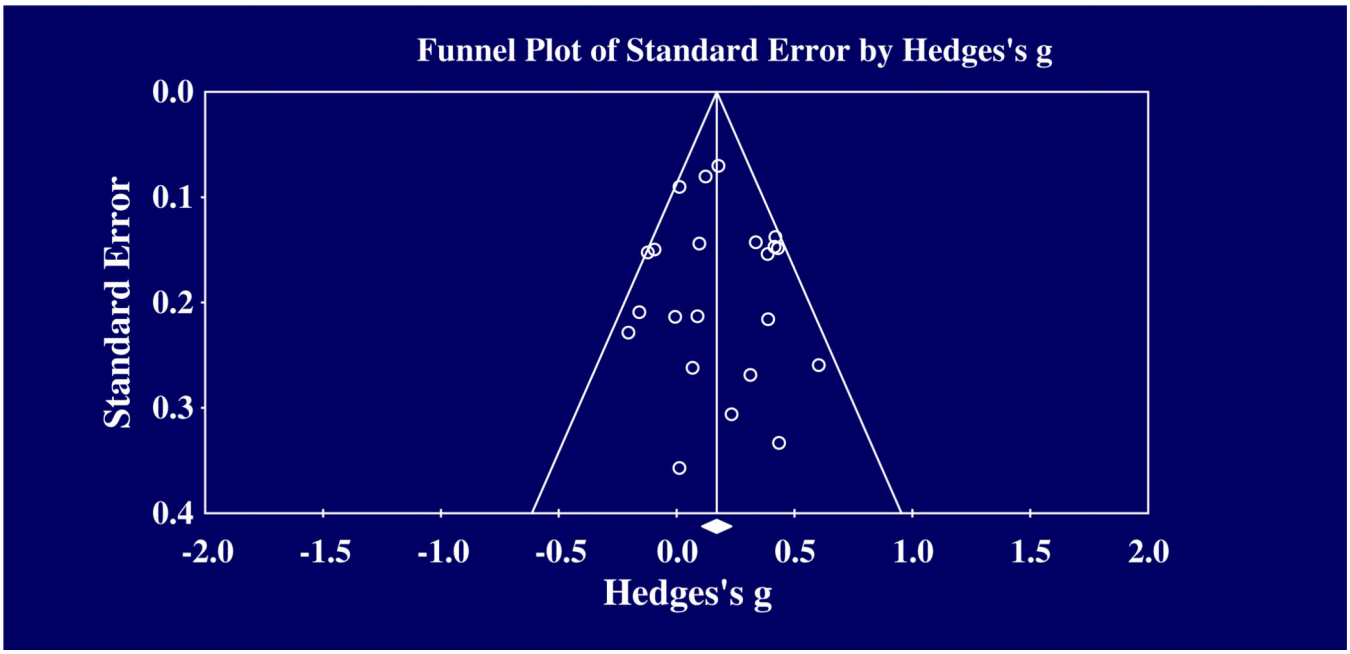

Fig. 2.

Funnel plot for studies assessing the difference between depressive symptoms between treatment and control conditions by 6 months postpartum. The asymmetric distribution of studies in the lower half of the funnel plot suggests that there are missing studies with negative effect sizes, in which control conditions would be superior to treatment conditions.

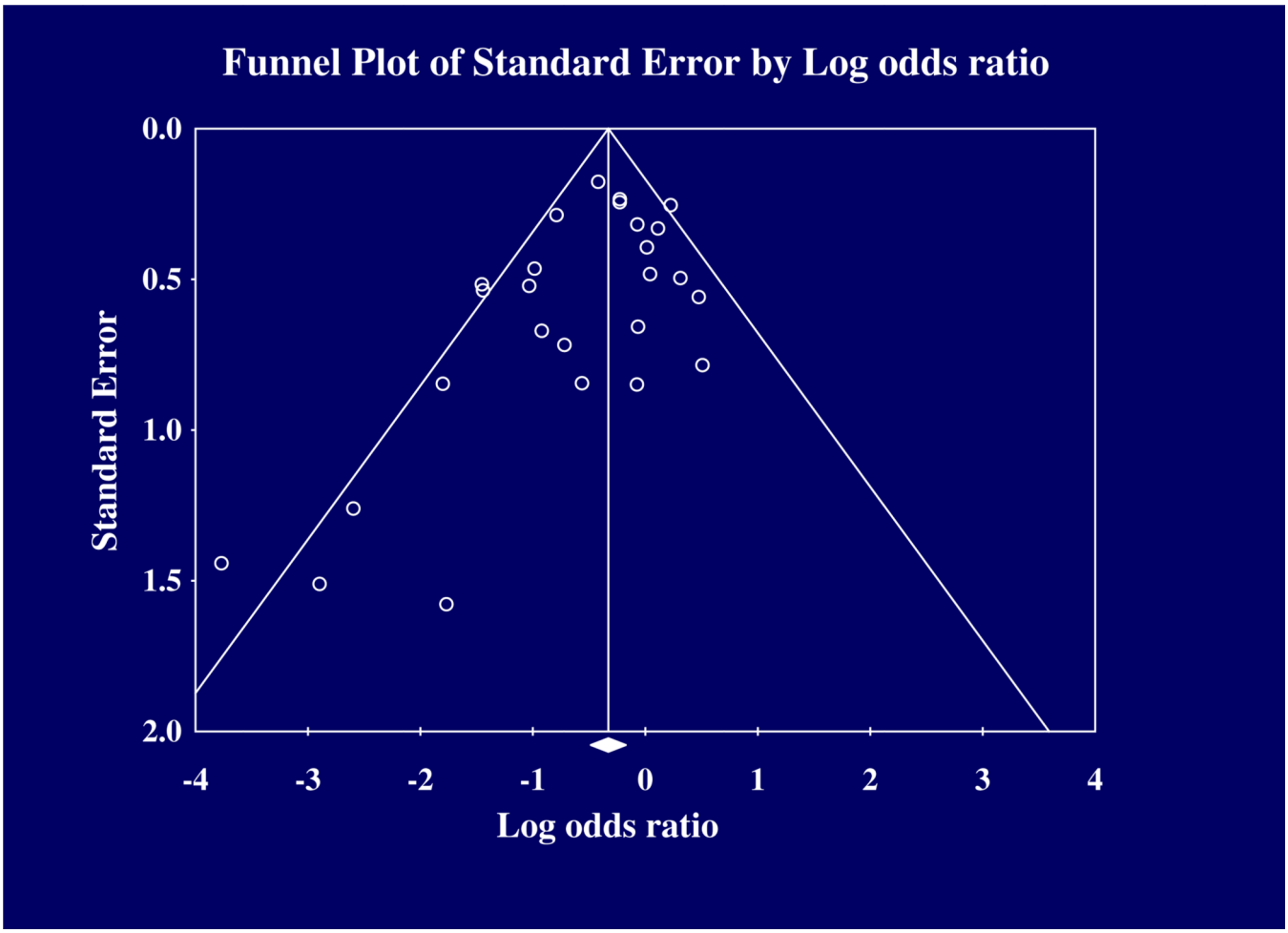

Fig. 3.

Funnel plot for studies assessing the difference in prevalence of depressive episodes between treatment and control conditions by 6 months postpartum. The asymmetric distribution of studies in the lower half of the funnel plot suggests that there are missing studies with odds ratios greater than 0, in which control conditions would be superior to treatment conditions.

Table 1

Characteristics of included studies.

Study	Country	Intervention type	Contra1 type	Intervention timing	Prevention type	Symptom measure	Diagnosis criteria	Postpartum assessment timing (weeks)	Psychotherapy orientation	Psychotherapy administration	Number of sessions
Armstrong et al. (1999)	AUS	Modified care	TAU	POST	Selected	EPDS	EPDS >12	6			
Austin et al. (2008)	AUS	Therapy	EDUC	PREG	Selected/indicated		MINI	16	CBT	Group	6
Bingha et al. (2000)	UK	Therapy	TAU	PREG	Selected/indicated		SADS	12	CBT	Group	6
Chabrol et al. (2002)	FR	Therapy	TAU	POST	Indicated	EPDS	EPDS > 11	4–6	CBT	Individual	1
Dennis, Hodnett et al. (2009)	CAN	Social support	TAU	POST	Indicated	EPDS	EPDS > 12	24			
Elliott et al. (2000)	UK	Social support	TAU	PREG	Selected		PSE	12			
Guo, Chan, and Sun (2012)	CHINA	Therapy	EDUC	PREG	Universal	EPDS		12	IPT	Group	2
Gorman (1997)	USA	Therapy	TAU	PREG	Selected/indicated	EPDS	SCID	24	IPT	Individual	5
Gunn et al. (1998)	AUS	Modified care	TAU	POST	Universal	EPDS	EPDS ≥ 13	24			
Hagan, Evans, and Pope (2004)	AUS	Therapy	EDUC	POST	Selected		SADS	24	CBT	Group	6
Hayes, Muller, and Bradley (2001)	AUS	Educational	TAU	PREG	Universal	POMS		16–24			
Hu et al. (2009)	CHINA	Educational	TAU	POST	Universal	EPDS		12			
Kozinszky et al. (2012)	HUN	Therapy	EDUC	PREG	Universal		LQ≥12	6–8	Eclectic	Group	4
Lara, Navarro, and Navarrete (2010)	MEX	Therapy	EDUC	PREG	Selected/Indicated		SCID	16–24	Eclectic	Group	8
Lawrie et al. (1998)	SAFR	Hormonal	PLA	POST	Universal	EPDS	EPDS ≥ 12	12			
Le, Perry, and Stuart (2011)	USA	Therapy	TAU	PREG	Selected/indicated	BDI-II	BDI-II ≥ 20	16	CBT	Group	8

Study	Country	Intervention type	Contra1 type	Intervention timing	Prevention type	Symptom measure	Diagnosis criteria	Postpartum assessment timing (weeks)	Psychotherapy orientation	Psychotherapy administration	Number of sessions
Llorente et al. (2003)	USA	Dietary supplement	PLA	POST	Universal	BDI-II		16			
Logsdon, Birkimer, Simpson, and Looney (2003)	USA	Social support	TAU	PREG	Selected	CES-D		6			
Marks, Siddle, and Warwick (2003)	UK	Modified care	TAU	PREG	Selected/indicated	EPDS	SCID	12			
Meeker (1985)	USA	Social support	TAU	PREG	Universal	BDI-II		7			
Milgrom, Schembri, Ericksen, Ross, and Gemmill (2011)	AUS	Therapy	TAU	PREG	Selected/indicated		BDI-II ≥ 14	12	CBT	Individual (Phone)	8
Mokhber et al. (2011)	IRAN	Dietary supplement	PLA	PREG	Universal	EPDS		8			
Munoz et al. (2007)	USA	Therapy	TAU	PREG	Selected/indicated	EPDS	MMS	24	CBT	Group	12
Nalepka and Coblentz (1995)	USA	Social support	EDUC	PREG	Universal		EPDS ≥ 10	12			
Ngai, Chan, and Ip (2009)	CHINA	Therapy	EDUC	PREG	Universal	EPDS		24	CBT	Group	6
Rees(1995)	USA	Therapy	ACT	POST	Universal	CES-D		4	Guided Relaxation	Individual (Home)	N/A
Shields and Reid (1997)	UK	Modified care	TAU	PREG	Universal	9 Item EPDS	EPDS > 13	7			
Silverstein et al. (2011)	USA	Therapy	TAU	POST	Selected		QJDS ≥ 11	24	CBT	Individual	4
Small et al. (2000)	AUS	Modified care	TAU	POST	Selected	EPDS	EPDS ≥ 13	24			
Stamp, Williams, and Crowther (1995)	AUS	Social support	TAU	PREG	Selected	EPDS	EPDS > 12	24			
Webster et al. (2003)	AUS	Educational	TAU	PREG	Selected		EPDS > 12	16			
Wisner et al. (2001)	USA	ADM nortriptyline	PLA	POST	Selected		RDC	17			

Study	Country	Intervention type	Contra1 type	Intervention timing	Prevention type	Symptom measure	Diagnosis criteria	Postpartum assessment timing (weeks)	Psychotherapy orientation	Psychotherapy administration	Number of sessions
Wisner et al. (2004)	USA	ADM sertraline	PLA	POST	Selected		DSM-IV	17			
Wolman et al. (1993)	SAFR	Modified care	TAU	BIRTH	Universal	PITT	PITT ≥ 35	6			
Zayas, McKee, and jankowski (2004)	USA	Therapy	TAU	PREG	Indicated	BDI-II		12	CBT	Individual	12
Zlotnick et al. (2011)	USA	Therapy	TAU	PREG	Selected	EPDS	LIFE	12	IPT	Individual	4
Zlotnick Johnson, Miller, Pearlstein, and Howard (2001)	USA	Therapy	TAU	PREG	Selected/indicated		SCID	12	IPT	Group	4
Zlotnick Miller, Pearlstein, Howard, and Sweeney (2006)	USA	Therapy	TAU	PREG	Selected	BDI-II	LIFE	12	IPT	Group	4

Note. AUS = Australia, CAN = Canada, CHINA = China, FR = France, HUN = Hungary, IRAN = Iran, MEX = Mexico, S AFR = South Africa, UK = United Kingdom, USA = United States, ACT = Active, EDUC = Educational, PLA = Placebo, TAU = Treatment As Usual, BIRTH = During labor, POST = Postpartum, PREG = Pregnancy, BDI-II = Beck Depression Inventory, CES-D = Center for Epidemiological Studies Depression Scale, DSM-IV = DSM-IV depression criteria, EPDS = Edinburgh Post-Natal Depression Scale, PITT = Pittsburgh Depression Inventory, LIFE = Longitudinal Interview Follow-Up Examination, LQ = Leverton Questionnaire, MINI = MINI-International Neuropsychiatry: Interview, MMS = Maternal Mood Screener, QJDS = Quick Inventory of Depressive Symptomatology, RDC = Research Diagnostic Criteria, SADS = Schedule for Affective Disorders and Schizophrenia, SCID = Structured Clinical Interview for DSM-IV, CBT = Cognitive-Behavioral Therapy, IPT = Interpersonal Psychotherapy.

Table 2

Methodological quality of included studies.

Study	ITT	Char sample	Excl curr MDE	Blind assess	Spec random	Therapy					Pharmacological	
						Spec ther	Manual	Training	Super	Adher	Blind clin	Blind pt
Armstrong et al. (1999)	−	+	−	+	+							
Austin et al. (2008)	+	+	−	+	+	+	+	+	−	−		
Brugha et al. (2000)	+	+	−	+	+	+	+	+	+	−		
Chabrol et al. (2002)	+	+	−	N/A	+	+	+	+	+	+		
Dennis, Hodnett et al. (2009)	+	+	−	N/A	+							
Elliott et al. (2000)	+	−	−	+	QR							
Gao et al. (2012)	+	+	−	N/A	+	+	−	+	+	−		
Gorman (1997)	−	+	−	+	+	−	+	−	−	+		
Gunn et al. (1998)	+	+	−	N/A	+							
Hagan et al. (2004)	−	+	+	+	+	+	−	+	+	−		
Hayes et al. (2001)	−	+	+	−	+							
Ho et al. (2009)	−	+	−	N/A	+							
Kozinszky et al. (2012)	+	+	−	+	+	+	−	+	−	+		
Lara et al. (2010)	+	+	+	−	+	+	+	+	+	+		
Lawrie et al. (1998)	+	+	−	+	+						+	+
Le et al. (2011)	+	+	+	−	+	+	+	+	+	+		
Llorente et al. (2003)	−	+	−	N/A	+						−	+
Logsdon et al. (2003)	−	+	−	N/A	+							
Marks et al. (2003)	+	+	−	−	+							
Meeker (1985)	+	+	−	N/A	−							
Milgrom et al. (2011)	+	+	−	N/A	+	+	+	−	+	+		
Mokhber et al. (2011)	−	+	+	N/A	−						+	+
Muñoz et al. (2007)	−	+	+	−	+	+	+	+	+	+		
Nalepka and Coblentz (1995)	−	+	−	N/A	+							
Ngai et al. (2009)	+	+	−	N/A	QR	+	−	+	−	−		
Rees (1995)	+	+	−	N/A	−	N/A	N/A	N/A	N/A	N/A		
Shields and Reid (1997)	−	+	−	N/A	−							

Study	ITT	Char sample	Excl curr MDE	Blind assess	Spec random	Therapy					Pharmacological	
						Spec ther	Manual	Training	Super	Adher	Blind clin	Blind pt
Silverstein et al. (2011)	+	+	−	+	+	+	+	+	+	+		
Small et al. (2000)	+	+	−	N/A	+							
Stamp et al. (1995)	+	+	−	N/A	+							
Webster et al. (2003)	+	+		N/A	+							
Wisner et al. (2001)	+	−	+	+	+						+	+
Wisner et al. (2004)	+	+	+	+	+						+	+
Wolman et al. (1993)	−	+	−	+	+							
Zayas et al. (2004)	−	+	−	N/A	−	+	−	+	+	−		
Zlotnick et al. (2011)	+	+	+	−	+	+	+	+	+	−		
Zlotnick et al. (2001)	−	+	+	−	−	−	−	−	−	−		
Zlotnick et al. (2006)	−	+	+	−	+	+	−	+	+	−		

Note. ITT = report intent-to-treat analyses, Char Sample = specify characteristics of sample, Excl Curr MDE = assess for depressive episode pre-treatment and exclude subjects who meet diagnostic criteria, Blind Assess = clinician-administered diagnostic measures conducted by independent evaluator blind to treatment condition, Spec Random = specification of method of randomization, Spec Ther = specify therapist characteristics, Manual = specify use of therapy manual, Training = describe therapist training, Super = describe therapist supervision, Adher = indicate therapy was assessed for adherence to manual, Blind Clin = clinician blind to treatment status, Blind Pt = patient blind to treatment status, + = Yes, − = No, N/A = Not Applicable, QR = Quasi-Randomized.

Table 3

Random weighted effect sizes (Hedges' *g*) comparing depressive symptoms between treatment and control conditions by 6 months postpartum.

Study	*n*	Hedges' *g*	95% CI	SAMD
Armstrong et al. (1999)	181	0.44**	0.14–0.73	1.97
Chabrol et al. (2002)	211	042**	0.15–0.70	2.05
Dennis, Hodnett et al. (2009)	600	0.13	−0.03–0.29	−0.17
Gao et al. (2012)	194	0.34*	0.06–0.62	1.39
Gorman (1997)	30	0.02	−0.68–0.72	−0.33
Gunn et al. (1998)	475	0.02	−0.16–0.20	−1.38
Hayes et al. (2001)	188	0.1	−0.18–0.39	−0.27
Ho et al. (2009)	168	0.39’	0.09–0.70	1.61
Lawrie et al. (1998)	168	−0.12	−0.42–0.19	−1.67
Le et al. (2011)	174	−0.09	−0.38–0.21	−1.52
Llorente et al. (2003)	89	−0.15	−0.56–0.26	−1.38
Logsdon et al. (2003)	109	−0.2	−0.65–0.25	−1.76
Marks et al. (2003)	85	0	−0.42–0.42	−0.65
Mokhber et al. (2011)	85	0.39	−0.03–0.82	1.15
Muñoz et al. (2007)	41	0.24	−0.36–0.84	0.30
Ngai et al. (2009)	184	0.42**	0.13–0.71	1.89
Rees (1995)	60	0.61*	0.10–1.12	1.78
Shields and Reid (1997)	788	0.18**	0.04–0.32	0.60
Small et al. (2000)[a]	917	−0.08	−0.21–0.05	−3.55
Wolman et al. (1993)[a]	149	12.10***	10.69–13.51	70.69
Zayas et al. (2004)	57	0.07	−0.44–0.59	−0.25
Zlotnick et al. (2011)	35	0.32	−0.21–0.85	0.86
Zlotnick et al. (2001)	86	0.44	−0.22–1.10	−0.22
Zlotnick et al. (2006)	54	0.09	−0.33–0.51	0.64
	k	Hedges' *g*	95% CI	*Q(df)*	I^2
Total (all studies)	24	0.37***	0.15–0.60	321.40(23)***	92.84
Total (outliers excluded)	22	0.18***	0.09–0.27	33.32(21)*	36.98

Clin Psychol Rev. Author manuscript; available in PMC 2014 July 21.

* $p < 0.05$.

** $p < 0.01$.

*** $p < 0.001$.

[a] Outlier excluded from subsequent analyses.

Table 4

Analyses of moderation for depressive symptoms by 6 months postpartum.

Moderator	*N*	Hedges' *g*	95% CI	*Q(df)*	*p*
Intervention type				2.73(4)	0.60
Dietary supplement	2	0.12	−0.42–0.65		
Educational	2	0.24	−0.04–0.53		
Modified care	4	0.16	−0.01–0.33		
Therapy	11	0.27***	0.14–0.40		
Social support	2	0.04	−0.25–0.33		
Intervention type				1.06(1)	0.30
Biological	3	0.02	−0.30–0.35		
Psychosocial	19	0.20***	0.11–0.29		
Intervention type				1.56(1)	0.21
EST	10	0.25***	0.12–0.38		
Non-EST	12	0.14*	0.03–0.25		
Control group type				4.89(2)	0.09
Educational	2	0.38***	0.18–0.58		
Placebo	3	0.02	−0.30–0.35		
TAU	16	0.16***	0.07–0.25		
Intervention timing				0.06(1)	0.81
Pregnancy	14	0.18***	0.09–0.26		
Postpartum	8	0.20*	0.03–0.36		
Type of prevention				2.17(3)	0.54
Indicated	3	0.22*	0.01–0.44		
Selected	4	0.18	−0.10–0.47		
Selected/indicated	5	0.03	−0.11–0.23		
Universal	10	0.19**	0.07–0.32		
Measure				4.34(2)	0.11
BDI-II	5	0.00	−0.19–0.18		

Moderator	*N*	Hedges' *g*	95% CI	*Q(df)*	*p*
CES-D	2	0.20	−0.60–.99		
EPDS	13	0.23***	0.12–0.34		
Exclude current MDE				0.32(1)	0.58
No	15	0.19***	0.08–0.29		
Yes	7	0.13	−0.02–0.29		
Psychotherapy orientation				0.06(1)	0.80
CBT	5	0.23	0.00–0.46		
IPT	5	0.27**	0.07–0.47		
Method of psychotherapy administration				0.39(1)	0.53
Group	6	0.23*	0.04–0.41		
Individual	4	0.31**	0.11–0.52		

* $p < 0.05$.

** $p < 0.01$.

*** $p < 0.001$.

Table 5

Random weighted effect sizes (odds ratio) comparing prevalence of depressive episodes between treatment and control conditions by 6 months postpartum.

Study	*n*	*OR*	95% CI	SAMD
Armstrong et al. (1999)	181	0.24**	0.09–0.65	−3.30
Austin et al. (2008)	277	0.94	0.50–1.76	1.79
Brugha et al. (2000)	190	0.49	0.12–2.02	−1.54
Chabrol et al. (2002)	211	0.46**	0.26–0.81	−1.95
Dennis, Hodnett et al. (2009)	600	0.80	0.49–1.31	1.00
Elliott et al. (2000)	99	0.38*	0.15–0.94	−1.73
Gorman (1997)	37	0.57	0.11–3.03	−0.43
Gunn et al. (1998)	475	1.26	0.76–2.09	5.63
Hagan et al. (2004)	192	1.02	0.47–2.23	2.04
Kozinszky et al. (2012)[a]	1719	1.79	1.30–2.48	18.00
Lara et al. (2010)	116	0.36	0.13–1.01	−1.95
Lawrie et al. (1998)	168	1.13	0.59–2.18	2.60
Le et al. (2011)	174	1.38	0.52–3.67	4.18
Marks et al. (2003)	87	1.05	0.41–2.73	1.51
Milgrom et al. (2011)	89	0.24**	0.08–0.69	−2.31
Muñoz et al. (2007)	41	0.17	0.01–3.82	−1.68
Nalepka and Coblentz (1995)	72	0.94	0.18–4.98	0.88
Shields and Reid (1997)	788	0.66*	0.47–0.94	−0.70
Silverstein et al. (2011)	42	0.40	0.11–1.51	−1.01
Small et al. (2000)[a]	917	1.26	0.88–1.80	7.39
Stamp et al. (1995)	121	1.62	0.54–4.89	4.79
Webster et al. (2003)	369	0.80	0.50–1.28	0.81
Wisner et al. (2001)	51	0.95	0.26–3.45	0.77
Wisner et al. (2004)	22	0.08*	0.01–0.90	−1.44
Wolman et al. (1993)	149	0.02**	0.00–0.40	−4.17
Zlotnick et al. (2011)	35	1.68	0.36–7.86	2.68

Study		*n*	*OR*	95% CI	SAMD
Zlotnick et al. (2001)		86	0.06	0.00–1.08	−3.00
Zlotnick et al. (2006)		54	0.17*	0.03–0.88	−2.01
	k	*OR*	95% CI	*Q(df)*	I^2
Total (all studies)	28	0.72*	0.56–0.94	74.83(27)***	63.92
Total (outliers excluded)	26	0.67**	0.52–0.85	45.95(25)**	45.60
Total (trim-and-fill correction)		0.73*	0.56–0.95	61.93	

* $p < 0.05$.

** $p < 0.01$.

*** $p < 0.001$.

[a] Outlier excluded from subsequent analyses.

Table 6

Analyses of moderation for depressive episodes by 6 months postpartum.

Moderator	*n*	*OR*	95% CI	*Q(df)*	*p*
Intervention type				1.13(3)	0.77
Medication	2	0.34	0.03–3.85		
Modified care	5	0.61	0.31–1.19		
Therapy	13	0.57**	0.38–0.84		
Social support	4	0.77	0.46–1.31		
Intervention type				0.02(1)	0.88
Biological	3	0.71	0.24–2.12		
Psychosocial	23	0.61**	0.50–0.84		
Intervention type				0.82(1)	0.37
EST	14	0.58**	0.39–0.87		
Non-EST	12	0.73*	0.54–1.00		
Control group type				1.13(2)	0.57
Educational	4	0.82	0.53–1.25		
Placebo	3	0.71	0.24–2.12		
TAU	19	0.62**	0.46–0.83		
Intervention timing				0.03(1)	0.87
Pregnancy	16	0.70	0.47–1.05		
Postpartum	9	0.67**	0.50–0.90		
Type of prevention				1.05(3)	0.79
Indicated	2	0.62	0.36–1.07		
Selected	10	0.60*	0.38–0.97		
Selected/indicated	9	0.60*	0.36–0.99		
Universal	5	0.84	0.48–1.46		
Criterion for diagnosis				0.03(1)	0.87
Clinical	12	0.64*	0.42–0.99		
Cutoff	14	0.67*	0.50–0.92		
Exclude current MDE				0.27(1)	0.60

Moderator	*n*	*OR*	95% CI	*Q(df)*	*p*
No	17	0.68**	0.53–0.89		
Yes	9	0.56	0.29–1.10		
Psychotherapy orientation				0.41(1)	0.52
CBT	8	0.63*	0.41–0.97		
IPT	4	0.40	0.11–1.51		
Method of psychotherapy administration				0.71(1)	0.40
Group	8	0.62	0.36–1.07		
Individual	5	0.46**	0.29–0.73		

* $p < 0.05$

** $p < 0.01$.