Meta-Analysis of the Effectiveness of Biological and Non-Biological Treatments for Postpartum Depression

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Meta-Analysis of the Effectiveness of Biological and Non-Biological Treatments for Postpartum Depression

Sarah Christian

A dissertation submitted to the faculty of Brigham Young University in partial fulfillment of the requirements for the degree of Doctor of Philosophy

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March 2013

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ABSTRACT

Meta-Analysis of the Effectiveness of Biological and Non-Biological Treatments for Postpartum Depression

Sarah Christian
Department of Psychology, BYU
Doctor of Philosophy

I provided an updated, comprehensive review of treatments for mothers diagnosed with postpartum depression. Studies included in this meta-analysis were single-group pre-posttest, non-randomized and randomized controlled studies published from 1986 to 2010 that included face-to-face psychotherapy and psychopharmacology as well as non-traditional methods such as exercise and nurse-assisted counseling. Fifty-three published studies were analyzed. The randomized studies showed a moderate to large effects ($\bar{d} = 0.72$ to $1.25$, $k = 9$) when postpartum interventions were compared to a control condition, and smaller effects ($\bar{d} = 0.3$ to $0.57$, $k = 13$) to treatment as usual. When postpartum interventions were compared to each other there was small to no difference in effect sizes ($k = 9$). All of the non-randomized comparisons showed no significant difference, except when therapy was compared to treatment as usual ($\bar{d} = 0.55$, $k = 2$). Pre-post studies showed large effect sizes for therapy ($\bar{d} = 0.95$, $k = 7$) and medication treatments ($\bar{d} = 4.30$, $k = 5$). Influence analyses suggest that two studies had a large effect on aggregate effect sizes and heterogeneity statistics. Moderator and multivariate analyses were largely underpowered. Publication bias was not significantly related to outcome. Clinical implications for postpartum depression treatments and directions for future research were identified.

Keywords: postpartum depression, treatment outcome, psychotherapy, pharmacology, exercise, meta-analysis.
ACKNOWLEDGEMENTS

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Through this program I have met wonderful colleagues and friends that have each helped me find meaning, inner strength, and value by offering a listening ear and unconditional support. I want to especially thank Julia Hubbard, Sasha Mondragon, Arjan Berkeljon, Brian Hansen, Lisa Takara, and Jill Walker.

Lastly, my family has been a remarkable source of absolute love and inspiration by showing me what it means to work hard, lend a helping hand, embrace opportunities, and nurture growth. Their generosity, compassion, drive, optimism and resiliency are qualities that I am forever grateful to have received by them.
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Meta-Analysis of the Effectiveness of Biological and Non-biological Treatments for Postpartum Depression

An estimated 20 percent of women suffer from Major Depressive Disorder at some point in their life making it a significant public health problem for women and their families (Seyfried & Marcus, 2003). The onset of depressive symptoms generally occurs during the childbearing years when women are having and raising children. Research has shown that the onset of depression “increases three-folds” within five weeks following delivery (Seyfried & Marcus, 2003, p. 231). Although the birth of a child is often a joyous occasion, it is also a time of great change in a woman’s life. The demands of infant care, changes in family dynamics, and the couple relationship as well as the impact a new child has on work, social activities, and finances can all lead to significant stress (Seyfried & Marcus, 2003). These stressors may negatively impact the development of the infant, impair mother-infant attachment, and impair familial relationships (Field, 2010).

Postpartum Criteria Defined

In the Diagnostic and Statistical Manual – Fourth Edition (DSM-IV-TR, American Psychiatric Association, 2000), Postpartum depression (PPD) is not currently recognized as a separate diagnosis. Instead, the DSM-IV-TR recognizes mood problems during the postpartum period using a “with postpartum onset” specifier to several mood disorders including, Major Depressive, Manic or Mixed Episode of Major Depressive Disorder, Bipolar I Disorder, Bipolar II Disorder and Brief Psychotic Disorder. The International Classification of Diseases- Tenth Edition (ICD-10, World Health Organization, 1992) recognizes PPD as a separate diagnosis that
occurs within six weeks after delivery and does not meet criteria for another mental disorder (Seyfried & Marcus, 2003).

Although the DSM-IV-TR defines PPD as a mood disorder that occurs four weeks after childbirth, research studies have defined PPD based on the standardized diagnostic criteria for depression (O’Hara & Swain, 1996). Hence, PPD is commonly referred to as a non-psychotic depressive episode that begins after delivery and persists for at least two weeks and results in some impairment in the woman’s functioning (Yozwiak, 2010). Specifically, during the two weeks, the woman experiences depressed mood, lack of interest in pleasurable activities, appetite changes, sleep disturbances, fatigue, feelings of worthlessness, difficulty concentrating, and suicidal ideation (American Psychiatric Association, 2000). Women with PPD may also become preoccupied with infant well-being, the intensity of which may range from over-concern to delusions (American Psychiatric Association, 2000). Maternal attitudes toward the infant are highly variable but can include disinterest in the infant, fearfulness of being alone with the infant, or over intrusiveness that inhibits adequate infant rest. Women with PPD often also experience severe anxiety and even panic attacks (American Psychiatric Association, 2000).

Other Postpartum Conditions

About 15 to 85 percent of new mothers experience “baby blues” or “postpartum blues” symptoms, a well-known postpartum syndrome that has a different onset, duration, and symptom profile than PPD (Pearlstein, Howard, Salisbury & Zlotnick, 2009). Mothers who feel the blues normally begin three to four days after birth and feel better by day 10, which differs from PPD mothers who feel depressed four weeks after delivery. “Baby blues” refers to a “transient disturbance in mood” commonly characterized by mood liability and tearfulness (Seyfried &
This syndrome is not diagnostically classified in the DSM-IV-TR Manual and no well-established measures have been developed to assess for this syndrome.

In addition, the DSM-IV-TR applies “postpartum onset” as a modifier for brief reactive psychosis and other mood disorders. Postpartum psychotic symptoms are consistent with an affective disorder, and include symptoms such as extreme euphoria, mood instability, disorganized behavior, and poor judgment (Pearlstein et al., 2009). Symptom onset is often sudden and unexpected, usually occurring within 48 hours to two weeks after giving birth (Doucet, Dennis, Letourmeau & Blackmore, 2009). The major difference between postpartum psychosis and PPD is their symptomatology. Postpartum psychotic symptoms are more complex at onset and decrease after 90 days following delivery (Seyfried & Marcus, 2003).

**Prevalence and Implications**

Previous research reported non-psychotic PPD prevalence rates ranging from 10 to 20 percent (O’Hara & Swain, 1996). Known risk factors for PPD include a history of depression and a history of PPD episodes which increase the risk of having a subsequent postpartum episode (Altshuler, Cohen, Moline, Kahn, Carpenter, Docherty & Ross, 2001).

In addition to the considerable negative personal impact that PPD has on women, PPD also negatively impacts marital relationships (O’Hara & Swain, 1996). Men with partners who have PPD feel more depressed, exhibit aggressive tendencies, and have more psychological impairments compared to men with partners who do not have PPD (Roberts, Bushnell, Collings & Purdie, 2006). In turn, strained relationships can prolong the duration of PPD symptoms (Roberts et al., 2006). PPD and marital distress appears to contribute to a negative cycle, where distress may facilitate the onset and duration of PPD symptoms and these symptoms may contribute to an increase of marital distress. This implies that not only does PPD play a major
role in marital relationships but the stability of the partnership is also necessary for PPD symptoms to improve.

PPD may also detrimentally affect mother-infant attachment, and infant social and cognitive development (Burke, 2003). Depressed mothers tend to show flat affect, provide less stimulation, and are less responsive than non-depressed mothers. They also react more negatively and are unsupportive to their child’s development (Burke, 2003). Thus, depressed mothers are less emotionally sensitive and less attuned to their infant’s emotional state. These behaviors negatively affect an infant’s cognitive development, mood regulation, and attachment to their mother. These serious implications make PPD an important mental illness to study because of the tremendous impact it has on the mother and support system, including child’s development and familial relationships.

Theories and Approaches of Treatment

In what follows, I provide a review of the PPD literature explaining the different interventions and theories of treatment for PPD. I specifically highlight those approaches that I coded in this meta-analysis.

Biological Interventions

Antidepressants. Psychotropic medications for PPD include selective serotonin reuptake inhibitors (SSRIs) (e.g., Sertraline, Paroxetine, Fluoxetine, Citalopram), tricyclics (e.g., imipramine and nortriptyline), and other antidepressant drugs. Female mice studies have shown that abnormal mood disturbances are associated with the inability of a neurotransmitter, GABA, to adapt to hormone fluctuations during the highly vulnerable postpartum period (Maguire & Mody, 2008). In order to improve postpartum mothering behaviors and well-being of child,
antidepressant medications are used to enhance mood-regulating neurotransmitters such as serotonin (Lerch-Haner, Frierson, Crawford, Beck & Deneris, 2008).

Although tricyclic antidepressants, SSRIs, and other antidepressant drugs have been shown to be effective in randomized control trials for the treatment of depression in men and in women of childbearing age, there are fewer well-designed studies among postpartum women. This may be due to concerns about potential adverse effects on the nursing infant when the mother uses an antidepressant medication in the perinatal or postpartum period. In a recent literature review conducted by Gjerdingen (2003), findings show that for most of the infants, drug exposure were either not detectable or very low. Seven out of seventy infants exposed to one of these drugs: Citalopram, Doxepin, Nefazodone, and Fluoxetine experienced adverse clinical outcomes (Gjerdingen, 2003). Given the various concerns regarding antidepressant treatment for breastfeeding women, the US Food and Drug Administration (FDA) has not approved any antidepressant for use during lactation.

**Estrogen therapy.** Another popular biological treatment is hormone therapy that treats PPD by increasing estrogen, estradiol, progesterone, and other female hormones levels in the mother. Women experience dramatic hormonal shifts with the birth of a child including decreased levels of endogenous glucocorticoids and estrogens, producing a transient hypoactivation of the hypothalamic-pituitary axis (HPA) that can last for months (Gjerdingen, 2003). Women who develop PPD usually have a more severe and prolonged suppression of the HPA (Cizza, Gold & Chrousos, 1997). In addition to the hypoactivation of the HPA, current research has shown that alpha and beta estrogen receptors located in the hypothalamus and other parts of the central nervous system may have benefits in controlling mood, cognition, and neuronal health (Studd & Panay, 2004). Research has shown that estrogen can increase the level
of serotonin and in turn, improve mood (Studd & Panay, 2004). Therefore, the postpartum administration of hormones might be useful in blunting hormonal and mood declines in women who suffer from postpartum mood disorders.

**Bright light therapy.** Whereas bright light therapy has been shown to be an effective treatment for seasonal affective disorder and nonseasonal depression, it has also been shown to improve depressive symptoms for pregnant mothers who have major depression with no adverse effects (Oren, Wisner, Spinelli, Epperson, Peindl, Terman & Terman, 2002). Bright light therapy is believed to work by suppressing melatonin and producing serotonin (Corral, Wardrop, Zhang, Grewal & Patton, 2007). Although this treatment approach is not a first-line option, this treatment strategy may be an option for depressed women who do not respond to traditional approaches.

**Exercise.** In addition to the traditional biological approaches, non-traditional physiological modalities such as exercise have been suggested for decreasing PPD symptomatology. In PPD literature, pram-walking is a popular method of exercising which entails a group of mothers walking together while they carry or push their infant in a stroller. According to the Surgeon General’s Report on Health and Physical Activity (1996), “regular participation in physical activity appears to reduce depression and anxiety, improve mood, and enhance ability to perform daily tasks throughout the lifespan” (p. 13). Physical activity has demonstrated effectiveness in helping to regulate the relationship between cortisol and adrenocorticotropic hormone (ACTH) which helps to regulate the HPA axis (Jolly, Elmore, Barnard & Carr, 2011). In addition, animal research has shown that exercise increases the frequency and rate of serotonin being “fired” within the brain and increases the level of tryptophan in the brain, which produces an amino acid used to manufacture serotonin (Jacobs,
High levels of serotonin are associated with an elevated mood, which helps to decrease mild depressive symptoms.

**Non-Biological Interventions**

**Individual therapy.** Psychological approaches are considered less controversial than medication regimes and therefore represent the first line of treatment for PPD. Psychotherapies that are formatted for one-on-one therapy were found to be an effective treatment modality for PPD in five randomized controlled trials (Dennis, 2004). These treatments focus on major role transitions that occur in the postpartum period such as caring for an infant. It may also include processing any feelings related to anger, shame, and guilt as well as resolving marital disputes. Further, therapy can help mothers challenge feelings of inadequacy or incompetency about mothering (Zuehlke, 2007).

Psychotherapies that target interpersonal and/or current psychological problems related to general depression have been shown to be more effective than long-term analytic psychotherapies (Dennis, 2004). In the PPD literature, the two most common types of individual therapy are interpersonal and cognitive-behavioral. Interpersonal therapy (IPT) suggests that the primary problem is disturbed interpersonal relationships. For mothers with PPD, IPT sessions often concentrate on relational disputes, such as conflict with spouses or other family members, the relationship with the baby, role transitions, concerns about returning back to work, and issues of grief and loss. Cognitive-behavioral therapy (CBT) is another empirically supported treatment for PPD (Dennis, 2004). CBT is based on the premise that individuals have a dysfunctional view of themselves, especially as new mothers. CBT interventions would address unrealistic expectations about a mother’s role change, motherhood, and attributional errors regarding the infant and their new self (Zuehlke, 2007).
**Group therapy.** Similar to individual therapy, group psychotherapy offers many of the same therapeutic advantages of individual therapy and also includes additional benefits. The group format encourages mutual support, reduces social isolation, fosters a sense of altruism, and promotes interpersonal learning (Yalom, 1995). Findings suggest that group therapy may be beneficial for PPD women who are seeking group support and who suffer from mild to moderate depression (Dennis, 2004).

In addition to a manualized group treatment approach, research demonstrated that peer support interventions are beneficial in treating mothers who have mild to moderate depression or for women who had no previous history of depression (Dennis, 2004). Social support is important to many new mothers who may lack an intimate confidant to talk to and feel socially isolated (Dennis, 2004). Research has shown that a lack of social support is a significant predictor of PPD. As such, a peer (mother-to-mother) support in a group modality for women experiencing PPD has been conducted in three treatment studies (Chen, Tseng, Chou & Wang, 2000; Fleming, Klein & Corter, 1992; Morgan, Matthey, Barnett & Richardson, 1997).

**Psychosocial counseling.** An alternative method to psychotherapy is psychosocial counseling by health visitors. The health professionals are trained in non-verbal encouragement and reflecting back the content made by the mother (Davies, Howells & Jenkins, 2003). Research has shown that having supportive relationships during the postpartum period could enhance a mother’s feeling of well-being (Dennis & Hodnett, 2007). Further, several studies showed that non-directive counseling from health professionals are effective in decreasing PPD symptomatology compared to a control condition (Armstrong, Fraser, Dadds & Morris, 1999; Holden, Sagovsky & Cox, 1989; Wickberg and Hwang, 1996).
Past Meta-analyses on Postpartum Depression

Six meta-analyses examined treatment outcomes for PPD, which are summarized in Table 1. The majority of the meta-analyses examined the effectiveness of psychological and psychosocial treatments, whereas one examined biological treatments and another studied the effectiveness of exercise in the management of PPD. All of these meta-analyses only included studies of non-psychotic PPD individuals. They also focused on both randomized and nonrandomized studies that compared a treatment to a control condition or alternative treatment.

Table 1

<table>
<thead>
<tr>
<th>Authors</th>
<th>Topic</th>
<th>Number of Studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ray &amp; Hodnett, 1998</td>
<td>Non-randomized studies comparing health visitor and professional support</td>
<td>2</td>
</tr>
<tr>
<td>Dennis, Ross, &amp; Herxheimer, 1999</td>
<td>Randomized Control Trials (RCTS) of estrogen and progestogen intervention</td>
<td>2</td>
</tr>
<tr>
<td>Bledsoe &amp; Grote, 2006</td>
<td>Pre/post and RCTs of psychotherapy, counseling, educational, group therapy, medication, medication plus therapy for PPD and depression during pregnancy</td>
<td>11</td>
</tr>
<tr>
<td>Cuijpers, Brannmark, &amp; van Straten, 2008</td>
<td>RCTs and non-randomized studies of social support, psychosocial, psychological interventions</td>
<td>17</td>
</tr>
<tr>
<td>Daley, Jolly, &amp; MacArther, 2009</td>
<td>RCTs and non-randomized studies of exercise</td>
<td>5</td>
</tr>
<tr>
<td>Dennis &amp; Hodnett, 2009</td>
<td>RCTs and non-randomized studies of psychosocial and psychological interventions</td>
<td>9</td>
</tr>
</tbody>
</table>
Findings From Past Meta-Analyses

Two meta-analytic reviews (Cuijpers, Brannmark & van Straten, 2008; Dennis & Hodnett, 2009) found psychosocial and psychological interventions are effective treatments for treating PPD. Psychological treatments when compared to a control condition has shown to have a moderate effect ($\bar{d} = 0.61, k = 14$) on depressive symptoms in women with PPD (Cuijpers, Brannmark et al., 2008).

The earliest meta-analysis contained two non-randomized trials comparing the effects of therapy with a psychologist or nurse to treatment as usual for depressed mothers (Ray & Hodnett, 1998). Results showed that depressive symptoms were significantly reduced in the groups receiving additional support (Odds-ratio (OR) = 0.34; 95% CI [0.17, 0.69]). Results of nine quasi-randomized studies showed professionally-facilitated support groups as well as structured psychological approaches such as CBT, IPT, and psychodynamic therapy were helpful in decreasing the likelihood of depressive symptomatology when compared to treatment as usual (Relative Risk (RR) = 0.70; 95% CI [0.60, 0.81], $k = 9$; Dennis & Hodnett, 2009).

However, there was no significant difference between one psychosocial intervention and another psychological intervention (Dennis & Hodnett, 2009). The group therapy outcome literature showed mixed results in regards to significant changes in symptom reduction for PPD, which may have resulted from differences in the structure and/or content of the group sessions. Thus, no definitive conclusions can be made regarding this modality (Gjerdingen, 2003).

Within the PPD literature, some researchers speculate whether alternative treatments to psychotherapy are effective treatment approaches. Given the hormonal and biological changes that occur after giving birth, some believe that biological causes are more prominent in PPD. However, few biological treatments have been conducted due to the medical concerns on infant
development. Cuijpers, Brannmark and van Straten’s (2008) meta-analysis examined psychological treatments to other interventions including pharmacological treatment, systematic care, and pram-walking. Results showed that the other interventions had superior effects than psychological treatments ($d = .86, k = 3$). However, heterogeneity was relatively high given that these interventions differed strongly from each other. Within the same meta-analysis, psychological treatments were compared to combined psychological and pharmacological treatments and showed no statistical difference in effect sizes ($d = -0.17; 95\% \text{ CI } [-0.62-0.28], k = 2$). Although there are no meta-analytic reviews focusing solely on psychopharmacology, there are several open-label and randomized trials that have shown antidepressant medication, especially SSRIs, to be effective in treating PPD (Dennis & Stewart, 2004). Aside from SSRIs, other antidepressant drugs such as Duloxetine, Buproprion, and Nortriptyline have demonstrated to be effective for PPD (Lanza di Scalea & Wisner, 2009). However, some randomized clinical trials compared medication to psychotherapy or a combination of medication plus psychotherapy showed mixed findings (Appleby et al., 1997; Misri et al., 2004).

One meta-analysis examined hormonal interventions. Findings showed estrogen therapy was associated with a greater improvement in depression scores than placebo among PPD women after four and 12 weeks post-treatment (Dennis, Ross & Herxheimer, 1999). Two recent reviews (Dennis & Stewart, 2004; Lanza di Scalea & Wisner, 2009) advocated for estrogen therapy with preliminary results from a recent pre-post study that demonstrated its effectiveness after eight weeks of treatment.

Finally, one meta-analysis examined the effectiveness of exercise for PPD (Daley, Jolly & MacArthur, 2009). Results showed that exercise reduced depressive symptoms when compared to a control group ($d = .81, k = 5$). However, the effect size decreased considerably
when the researchers excluded a trial that combined exercise with social support ($\bar{d} = .42, k = 4$). This suggests that exercise alone appears to reduce some depressive symptoms but combining it with social support produces more of a reduction in symptomology.

**Limitations of Previous Meta-Analyses**

Previous meta-analyses had five major limitations. First, four of the meta-analyses examined only one type of approach and modality (i.e. exercise or hormone therapy; Cuijpers, Brannmark et al., 2008; Daley et al., 2009; Dennis et al., 1999). This limitation makes it difficult to compare these interventions with alternative treatments and make any generalized statements about effective treatments for PPD. Second, only three meta-analyses included both randomized and non-randomized studies, as part of their inclusion criteria (Cuijpers, Brannmark et al., 2008; Dennis et al., 2009; Ray et al., 1998). Although using a strict research design such as randomized controlled design strengthens internal validity allowing for stronger conclusions about effect sizes, valid inferences using other types of designs can be made about the impact of a given intervention. Given that PPD outcome research is relatively new, non-randomized and pre-post studies are useful in strengthening results from randomized studies and by finding unique treatments that have not been mainstreamed. Third, one meta-analysis did not review all possible studies that met inclusion criteria but instead limited their search to a specific time frame (Bledsoe & Grote, 2006). They also broadly defined PPD to include pregnant mothers, which blurs the line that defines the onset of PPD after birth (Bledsoe & Grote, 2006). Fourth, three of the meta-analyses did not use a random effects analysis, perform tests of heterogeneity, perform moderator analyses, or investigate publication bias (Dennis et al., 1999; Dennis & Hodnett, 2009; Ray & Hodnett, 1998). In addition, one meta-analysis included randomized, non-randomized and pre-post studies, but did not perform separate analyses for the various research
designs or adjust for effect sizes that were dependent on other subsamples making the interpretation of the combined effect difficult to interpret (Bledsoe & Grote, 2006). Fifth, five of the meta-analyses reported effect sizes primarily on depression measures (Bledsoe & Grote, 2006; Cuijpers, Brannmark et al., 2008; Daley et al., 2009; Lumley, Austin & Mitchell, 2004; Ray et al., 1998). Although depression is an important factor to examine, PPD also impacts other parts of a mother’s life including parenting style, marital relationship, and health, which were overlooked in these meta-analyses.

Aims of Current Meta-Analysis

This meta-analysis was designed to address the limitations of previous meta-analyses in the PPD literature. Specifically, this meta-analysis has four aims:

[Aim 1] This meta-analysis updated the literature on PPD treatment outcome by including all primary studies from 1986 to 2010, which included biological and non-biological treatment approaches. This comprehensive approach to collecting PPD studies allowed me to identify which treatment interventions were most effective for PPD and make conclusions about which specific treatment characteristics (e.g. psychologist versus nurses) moderate the efficacy of interventions for mothers who suffer from PPD.

[Aim 2] This meta-analysis compared the outcomes of between-group designs that compare a treatment condition to a control or an alternative treatment as well as a single-group pre-posttest that examined changes in the treatment condition over time. Examining different research designs allowed me to broaden my results and infer on the effectiveness recent and/or common treatments have on treating PPD symptoms.

[Aim 3] This meta-analysis explored a wider range of moderators. I looked at participant characteristics (e.g. marital status), format of treatment, types of outcome measure (e.g.
depression, marital, infant development), and attrition between conditions. I also used up-to-date meta-analytic statistical procedures (e.g., random effects, tests of heterogeneity, moderator analysis). These methods allowed me to make a wider-range of conclusions integrating other study variables, which were not examined in previous meta-analyses.

[Aim 4] As a result this meta-analysis provided information that future PPD outcome research can use to improve and expand as we learn more about PPD. In addition, this meta-analysis also strives to become a resource and clinical tool for clinicians, nurses, and doctors who treat mothers with PPD.

Method

Literature Search

Selection of studies. I used two methods for identifying potential studies to include in this meta-analysis. First, studies were identified by performing an electronic search on Cochrane Library, MEDLINE, PsycINFO, Dissertation Abstracts, PsycEXTRA and Social Work Abstracts for randomized, non-randomized and pre-posttest designs published by December 2010. I used the following search terms: postpartum depression (e.g., postpartum depression OR PPD OR postnatal depression) and outcome studies (psychotherapy OR outcome OR psychopharmacology OR medications OR therapy OR intervention OR drug therapy OR medications). Second, I searched reference lists of previous literature reviews, meta-analyses, or studies of the treatments for PPD. However, I did not contact researchers for unpublished studies. I retrieved a total of 766 articles published in the years 1982 to 2010 and included 66 studies in this meta-analysis.

Inclusion criteria. To be included in the present meta-analysis, studies had to (a) compare the outcome of a psychological, psychopharmacological, physiological, psychosocial
and alternative interventions given to adult female participants with non-psychotic PPD against a control condition or another active intervention, or (b) measure an intervention across time (pre-post study). All included studies must have mothers meeting diagnostic criteria for PPD either using a clinical interview and/or standard self-report questionnaire. All included studies were in English.

**Exclusion criteria.** I excluded studies that included females with other postpartum conditions (e.g., postpartum blues) and comorbid diagnoses (e.g. affective, psychosis, alcoholism) to ensure that studies solely focused on PPD symptoms. Studies only focusing on infant development and interaction effects of medication on breastfeeding were not included. Retrospective case studies and prevention studies were also excluded. All decisions regarding which studies to be included in the present study were made before examining the outcomes of individual studies. I coded all outcome measures possible in terms of effect size and then aggregated them into categories. Figure 1 describes the selection of studies.
Figure 1. Flow chart describing the identification and selection of studies.

Coding Manual

Study level codes. I used seven items to code information at the study level (see Appendix A). The first two codes identify basic information about each study: (a) Year of Study, (b) Study Design: pre-posttest, non-randomized or randomized. The remaining codes distinguish participant characteristics based on method of recruitment that are unique to each study: (c) Initial Assessment of PPD: when was mother diagnosed with PPD, (d) PPD diagnostically defined for study recruitment: using self-report measures, screeners or clinical interview, (e) Method of Recruitment: how was the mother recruited to the study, (f) Proportion of Single
Mothers in sample and (g) Proportion of Married/Cohabiting Mothers in sample. These codes were used to provide a general demographic overview of the participants that were included in this meta-analysis. In addition, the proportion of single and married mothers in a sample was used as a moderator.

Comparison level codes. I used 18 items to code information at the comparison level. The following codes identify which treatments and treatment characteristics were most effective for treating PPD: (a) Treatment Type: theory or treatment used in the study, (b) Comparison Type: theory or treatment used as the comparison, (c) Format of Treatment in Treatment Condition/Comparison Condition: nature of the delivery of the treatment, (d) How was treatment administered in Treatment/Comparison conditions: who conducted treatment, (e) Number of subjects assigned to treatment condition, (f) Number of subjects assigned to comparison condition, (g) Attrition Number in Treatment/Comparison Condition, (h) Duration of Treatment in Number of Weeks in Treatment/Comparison Condition, (i) Duration of Treatment in Number of Sessions in Treatment/Comparison Condition, (j) Length of Sessions in the Treatment/Comparison Condition, and (k) Length of time participant was on medication in Treatment/Comparison condition.

Measure and effect size level codes. I used five items to code information concerning measures and effect sizes within a study. Specifically, examining measure rater (e.g., self, clinician report), timing of assessment (e.g., post-test or follow-up), timing of follow-up, outcome measures, and broad categorization of measures (e.g., depression, anxiety, infant development).
Inter-Rater Reliability

Inter-rater reliability was assessed by two raters (including the author) who independently coded the above codes using 10 randomly selected studies. Reliability was assessed using the Kappa statistic for categorical variables and a Pearson’s correlation for continuous variables. Five treatment-control and five pre-posttest comparison studies were selected to code all outcome measures and effect sizes from each study. Codes that had unacceptably low inter-rater reliability were identified, discussed, rewritten, and recoded on 10 additional studies, and their reliabilities were recomputed. This process was followed until sufficient reliability was found for each code (kappa and Pearson’s correlation were greater than .75). Inter-rater agreement for categorical variables using kappa ranged from .84 to 1.00. For continuous variables, the Pearson’s correlation between raters ranged from $r = .76$ to 1.00. Given that kappa and Pearson’s correlation were greater than .75, there is sufficient reliability among raters that goes beyond chance. Therefore, all variables in the coding manual were sufficiently reliable to proceed. All studies were coded by the raters independently and any disagreements were settled via mutual consensus.

Calculating Effect Sizes

Separate meta-analyses were conducted for: (a) single-group pre-posttest designs, (b) non-randomized designs and (c) randomized controlled trials, as shown in Table 2.
Table 2

Each research design is matched with a corresponding comparison that was computed for an effect size.

<table>
<thead>
<tr>
<th>Research Designs</th>
<th>Randomized</th>
<th>Nonrandomized</th>
<th>Pre-post designs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment 1 vs.</td>
<td>X</td>
<td>X</td>
<td>--</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment 1 vs.</td>
<td>X</td>
<td>X</td>
<td>--</td>
</tr>
<tr>
<td>Treatment 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time 1 vs. Time</td>
<td>--</td>
<td>--</td>
<td>X</td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

To calculate the effects of two independent groups, I used the standardized mean difference statistic. When possible, this statistic was computed directly using Hedges’ $g$ (Hedges & Olkin, 1985, Equation 3, p. 78):

$$g = \frac{\bar{X}_1 - \bar{X}_2}{S_p}$$

where $\bar{X}_1$ and $\bar{X}_2$ are the means for groups 1 and 2, respectively, and $S_p$ is the pooled standard deviation. The pooled standard deviation was calculated as follows (Hedges & Olkin, 1985, p. 79):

$$S_{pooled} = \sqrt{\frac{s_1^2(n_1 - 1) + s_2^2(n_2 - 1)}{n_1 + n_2 - 2}}$$

where $n_1$ and $n_2$ are the sample sizes and $s_1^2$ and $s_2^2$ are the variances for groups 1 and 2, respectively. For pre-post designs, a standardized mean gain effect size was calculated using Becker (1988) model:
\[ g = \frac{(X_{\text{post}} - X_{\text{pre}})}{SD_{\text{pre}}}, \]

where \( X_{\text{post}} \) and \( X_{\text{pre}} \) are mean scores on a single outcome variable, \( SD_{\text{pre}} \) is the pre-test standard deviation. When the means and standard deviations are not available, \( g \) was estimated by using methods described in Shadish, Robinson, and Lu (1999). Where results are reported only as not significant \( g = 0.00 \) was coded conservatively. Only seven studies did not report effect sizes for certain comparisons, and these comparisons were coded as zero. When results are reported only as significant, \( g \) was calculated assuming \( p = .05 \). Hedges and Olkin’s correction was applied to all effect sizes because it corrects for small sample bias, using this formula (Hedges & Olkin, 1985, p. 81):

\[ d = \left(1 - \frac{3}{4N-9}\right)g \]

The corrected effect size \( d \) rather than \( g \) was used in all of the analyses (Baldwin & Shadish, 2011). To ensure that each study only contributed a single effect size to each analysis I aggregated measures within a study.

**Analysis**

Once study-level effect sizes were computed, the effect sizes were aggregated across studies. Studies in a meta-analysis are drawn from populations of studies that systematically differ from each other, therefore a random effects model was used to combine, analyze, and generalize the resulting effect sizes allowing for generalizations beyond the studies included in the model. In this meta-analysis, I accounted for study variance and precision using a weighted average effect whereby larger studies influence the average effect size more than small studies because they produce more precise effect size estimates (Baldwin & Shadish, 2011). Forest plots were used to graphically represent both study-level effect sizes and aggregate information. In
addition to interpreting the average effect, $Q$ and $I^2$ was calculated for each weighted effect size in order to test for between-study heterogeneity and interpret the proportion of variance between studies, respectively (Baldwin & Shadish, 2011).

**Moderator analysis.** Between-study heterogeneity may be closely associated with a covariate variable (Baldwin & Shadish, 2011). For instance, attrition in a pharmacological treatment may be higher than attrition in a therapy intervention, which may impact the aggregated effect size. A moderator analysis can examine whether one variable influences the causal relationship between two other variables – treatment condition (e.g. hormonal treatment versus control) and treatment outcome (Baldwin & Shadish, 2011). A meta-regression model was used to test moderator hypotheses for continuous and categorical variables such as attrition between treatments, administration of treatment, and proportion of single to married females. In addition, a multivariate analysis was used to examine effect sizes for depression measures and all other broad measures.

**Publication bias.** I assessed publication bias using three methods. First, funnel plots and contour enhanced funnel plots were used to depict the potential presence of publication bias. When effect sizes are plotted against the standard error, a symmetrical funnel is usually formed in the absence of publication bias, while a skewed asymmetrical funnel suggest that small and negative effects were not present in the results (Baldwin & Shadish, 2011). Second, Begg’s test and Egger’s test were used to determine if there was a significant correlation between the effect estimates and their variances. Third, trim-and-fill analyses were used to provide an estimate of the number of missing studies as determined by the funnel plot, and subsequently inputting the presence of missing studies to yield an unbiased pooled estimate (Sterne & Harbord, 2004). In both the funnel plots and trim-and-fill analyses, asymmetry can sometimes occur for other
reasons such as heterogeneity between studies due to sample size or chance (Baldwin & Shadish, 2011). These methods have low sensitivity in a meta-analysis that has fewer than 10 trials; therefore, I analyzed comparisons that had more than 9 trials. All analyses, including the publication bias analyses, were conducted using Stata software.

Results

Study Characteristics

Of the 53 studies that were included in the analysis, 32 were randomized trials (60%), 8 were non-randomized trials (15%), and 13 were pre-post studies (25%). This meta-analysis included a variety of PPD treatments: 58% were psychological interventions (IPT, CBT, M-ITG); 14% were psychosocial interventions (non-directive counseling, support group); 10% consisted of a combination of treatments (exercise and social support); 9% focused on pharmacological treatments (estrogen, antidepressants); 6% were other biological interventions (bright light therapy, exercise); and 3% were specialized treatments (day hospital, repetitive TMS). The treatment format ranged from group therapy format (26%), individual therapy (24%), medication/hormone treatments (19%), combination treatments (11%), home visits (10%), exercise (7%), and phone therapy (3%). Treatment was administered either by a psychologist (48%), other professional (21%), nurse/midwife (9%), health worker (8%), psychiatrist (7.7%), self (5%), no person administered treatment (1%), and nursing student (0.3%). Length of treatment ranged from 4 to 44 weeks, with an average of 10 weeks. Duration of treatment in number of sessions ranged from 1 to 64, with an average of 12 sessions and each session averaging 68 minutes. Interpersonal therapy (IPT) was the most common psychological intervention \( n = 7 \), followed by Cognitive-Behavioral therapy (CBT) \( n = 5 \), Mother-Infant Therapy group (M-ITG) \( n = 3 \), and psychodynamic therapy \( n = 2 \). For individuals who were
taking medication, the length of treatment ranged from 42 to 168 days with a mean average of 87 days.

All of these studies included mothers who were diagnosed with PPD and were recruited into the studies by completing a clinical interview and/or self-report measures. 55% of the women with postpartum depressive symptomatology were assessed using only self-report measures such as Edinburgh Postnatal Depression Scale (EPDS), 42% completed a screening measure and clinical interview, 2% were assessed using a screening questionnaire over the phone, and 1% were assessed only by a diagnostic interview. Mothers were diagnosed with PPD as early as 6 days after giving birth and continued to exhibit PPD symptoms as late as 120 weeks after delivery, with an average of 16 weeks. 41% of mothers were recruited by a combination of referrals, followed by doctor (26%), hospital unit (24.7%), other referrals (6.9%), advertisement (0.9%), and midwife (0.5%). 39 studies reported information regarding the marital status of subjects. Single mothers were defined as mothers who were unmarried, living alone, separated or divorced, which ranged from 63% to 100% in these studies. 16% of all studies ($n=10$) only involved single mothers. Married or cohabiting mothers were a smaller percentage of the sample, ranging from 0% to 37% in these studies.

**Overall Aggregate Effects**

**All treatments versus control or placebo in randomized trials.** Nine studies randomized participants to either a PPD treatment condition or to a control (placebo) condition. Figure 2 displays a forest plot of the aggregated study-level effect sizes (across measures) and 95% confidence intervals for each study, along with the citation, confidence interval and numerical effect size. Study-level effect sizes are represented by squares, where the size of the
square represents the weight of the study in the analysis. When studies have a large sample size the square is larger than studies with smaller sample sizes.

![Forest plot and 95% confidence intervals for randomized studies in the therapy versus control, medication versus placebo and combination versus control meta-analyses.](image)

**Figure 2.** Forest plot and 95% confidence intervals for randomized studies in the therapy versus control, medication versus placebo and combination versus control meta-analyses.

A moderate to large effect was found favoring psychological therapies ($\bar{d} = 0.76, p<.01, 95\% \text{ CI } [.46-1.06]$), pharmacological or estrogen interventions ($\bar{d} = 0.72, p<.01, 95\% \text{ CI } [.36-1.07]$) and combination of exercise and social support interventions ($d = 1.26, p<.01, 95\% \text{ CI } [.29-2.22]$) when compared to a control or placebo condition. Homogeneity for psychological
therapies $Q(5) = 6.63$, $p = .28$, $\tau^2 = 0.03$ and $I^2 = 21\%$ and medication interventions $Q(1) = 0.01$, $p = .94$, $\tau^2 = 0$ and $I^2 = 0\%$ were accepted suggesting that the variability in effect size estimates is due to sampling error within studies. Homogeneity for combination treatments was not computed because there was only one study.

**All treatments versus treatment as usual in randomized trials.** Thirteen studies randomized participants to a PPD intervention or treatment as usual (TAU). Figure 3 displays the random effects weighted-average effect size comparing any treatment for PPD to TAU.

### Treatment vs TAU

<table>
<thead>
<tr>
<th>Study</th>
<th>ES (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Therapy vs. TAU</td>
<td></td>
</tr>
<tr>
<td>Cooper, Murry et al., 2003</td>
<td>0.39 (-0.05, 0.82)</td>
</tr>
<tr>
<td>Honey et al., 2002</td>
<td>0.09 (-0.50, 0.68)</td>
</tr>
<tr>
<td>Horowitz et al., 2001</td>
<td>-0.22 (-0.59, 0.15)</td>
</tr>
<tr>
<td>Milgrom et al., 2005</td>
<td>0.32 (-0.12, 0.76)</td>
</tr>
<tr>
<td>Mulcahy et al., 2010</td>
<td>0.57 (-0.00, 1.13)</td>
</tr>
<tr>
<td>Prendergast &amp; Austin, 2001</td>
<td>0.10 (-0.56, 0.75)</td>
</tr>
<tr>
<td>Tamaki, 2008</td>
<td>1.04 (-0.02, 2.10)</td>
</tr>
<tr>
<td>Wickberg &amp; Hwang, 1996</td>
<td>0.83 (0.14, 1.53)</td>
</tr>
<tr>
<td>Subtotal (I-squared = 46.4%, p = 0.070)</td>
<td>0.30 (0.03, 0.56)</td>
</tr>
<tr>
<td>Exercise vs. TAU</td>
<td></td>
</tr>
<tr>
<td>Daley et al., 2008</td>
<td>0.45 (-0.27, 1.17)</td>
</tr>
<tr>
<td>Dritsa, Da Costa et al., 2008</td>
<td>0.43 (0.01, 0.86)</td>
</tr>
<tr>
<td>Subtotal (I-squared = 0.0%, p = 0.966)</td>
<td>0.44 (0.07, 0.80)</td>
</tr>
<tr>
<td>Combination tx vs. TAU</td>
<td></td>
</tr>
<tr>
<td>Dennis, 2003</td>
<td>0.59 (-0.03, 1.21)</td>
</tr>
<tr>
<td>Heh, Huang et al., 2008</td>
<td>0.66 (0.15, 1.17)</td>
</tr>
<tr>
<td>Rojas et al., 2007</td>
<td>0.54 (0.28, 0.80)</td>
</tr>
<tr>
<td>Subtotal (I-squared = 0.0%, p = 0.918)</td>
<td>0.57 (0.35, 0.79)</td>
</tr>
</tbody>
</table>

*Figure 3.* Forest plot and 95% confidence intervals for randomized studies in the therapy versus TAU, combination versus TAU and exercise versus TAU meta-analyses.
The average effect size for therapy compared to TAU was $\bar{d} = 0.31$ ($p<.05$, 95% CI [.06-.55]). Homogeneity of effect sizes was not significant, $Q(8) = 13.37$, $p = .1$, $\tau^2 = 0.05$ and $I^2 = 40.2\%$, indicating that 40.2% of the variance among effect sizes is caused by heterogeneity between studies. There were three studies that compared TAU to a combination of interventions (e.g. telephone peer support, multicomponent intervention, and exercise plus social support). A moderate effect was found favoring combined treatments ($\bar{d} = 0.57$, $p<.01$, 95% CI [.35-.79]) as well as exercise ($\bar{d} = 0.44$, $p<.05$, 95% CI [.07-.80]) to TAU. Homogeneity of effect sizes for these two interventions was accepted and the between-studies variance component for both comparisons was $I^2 = 0\%$.

Eight studies reported follow-up data after 4 to 244 weeks post treatment. The weighted-average effect size comparing therapy to TAU showed a small effect ($\bar{d} = 0.28$). Homogeneity was not significant, $Q(5) = 7.51$, $p = .19$, $\tau^2 = 0.03$ and $I^2 = 33.5\%$, showing that 33% of the variance in effect sizes was between studies. There was a moderate effect favoring exercise to TAU ($\bar{d} = .44$), but there was no difference between combination treatments and TAU at follow-up. The treatments that seemed most effective were exercise (Dritsa et al., 2008), IPT therapy (Mulcahy et al., 2010), CBT group counseling, and CBT individual counseling (Milgrom et al., 2005).

**All treatments versus alternative treatments in randomized trials.** Sixteen studies examined different approaches to treating PPD against other PPD interventions. Figure 4 displays a forest plot of the study-level effect sizes and the aggregative effect size.
There were two comparisons that reported no significant difference: alternative interventions (e.g. exercise, bright light, baby massage) versus other treatments, and combination versus alternative treatment. Homogeneity of effect size was not significant in each of the comparisons. Combination treatment comparison had the largest between-studies variance component which was $I^2 = 46.2\%$ followed by alternative treatment comparison with $I^2 = 14.1\%$. Therapy versus medication comparison had one study that compared antidepressants to non-
directive counseling (Sharp et al, 2010), and found a significant effect size of \( d = 0.30 \) (\( p < .05 \), 95% CI [.02-.59]). Although this is a small effect, it suggests that women in the antidepressant group showed slight improvement compared to women in the listening visit group. Given that there was only one study, heterogeneity was not computed.

One study examined follow-up after 52 weeks post treatment. Results showed no difference found when baby massage was compared to support group after 52 weeks post treatment (O’Higgins et al., 2008).

Psychological interventions versus control, TAU, other therapies in non-randomized trials. Six studies were used to compare psychosocial and psychological interventions to either a control, TAU, or alternative therapy condition. The psychological and psychosocial interventions that were used in these studies included home visits, non-directive counseling, and Mother-Infant Therapy.
The comparison between psychological treatments and control groups showed no statistical significant difference (Figure 5). However, there was one study (Chabrol, 2002) that had an abnormally large effect size influencing the mean effect and homogeneity tests. This study had a prevention intervention that was conducted prior to mothers giving birth, and mothers who continued to exhibit PPD symptoms were given the home visit intervention. When I conducted a “leave-one-out” analysis the effect size was still not significant but the homogeneity test was not significant and the between-studies variance decreased significantly from $I^2 = 90.2\%$ to $I^2 = 0\%$, which showed that there was no noticeable between-studies variance.
The comparison between psychological treatments and TAU showed a moderate effect size difference with $d = 0.55 (p<.001, 95\% \text{ CI} [.28-.82])$. Homogeneity of effect size was not significant, $Q(1) = 0.45, p = .50$, although power is low with only two studies.

There was only one study that reported follow-up data. One follow-up study that compared therapy to TAU showed no significant difference after 35 weeks post treatment (Glavin et al., 2010).

**Medications versus control or therapy in non-randomized trials.** Two non-randomized studies examined PPD individuals who were in a pharmacological treatment or to either a control or psychological treatment. The effect size comparing medication to control was $d = -0.79 (p = .14, 95\% \text{ CI} [-1.84-.27])$, showing no difference between individuals in the drug treatment group or control group. In addition, there was no significant difference between therapy and medication with $d = 0.14, (p = 0.85, 95\% \text{ CI} [-1.38-1.66]$. Between-study heterogeneity could not be computed with only one study in each of these comparisons.

**Combination versus control or medication in non-randomized trials.** Two studies (Highet & Drummond, 2003; Pearlstein et al., 2006) examined individuals who were non-randomized to a combination of therapy and pharmacological condition or to either a wait-list control or medication condition. Both of these comparisons displayed no significant difference with the average effect size for control was $d = 0.41 (p=.21, 95\% \text{ CI} [-.24-1.05])$ and medication was $d = -0.52 (p = .5, 95\% \text{ CI} [-2.98-1.05])$. Since there was only one study for each comparison, between-study heterogeneity was not computed.

**Therapy in pre-post trials.** Seven single-group pre-posttest studies examined psychological interventions primarily CBT and IPT therapies in a group format (Craig et al., 2005; Morris, 1987; Muzik et al., 2001; Reay et al., 2006). Figure 6 shows a forest plot of
random effects weighted-average effect size for psychological intervention with $\bar{d} = 0.95$ ($p<.001$, 95% CI [.67-1.23]), showing a large effect post treatment. Homogeneity of effect size was not statistically significant $Q(6) = 5.53, p = .47$, $\tau^2 = 0$ and $I^2 = 0\%$, indicating that all variability in effect size estimates is due to sampling error rather than between study variability.

**Figure 6.** Forest plot and 95% confidence intervals for pre-post therapy studies.

Follow-up results from three studies (Craig et al., 2005; Muzik et al., 2001; Reay et al., 2006) after 6 to 24 weeks post treatment showed that therapy maintained its effectiveness with $\bar{d} = 0.99$ ($p<.01$, 95% CI [.25-1.73]).

**Medication in pre-post trials.** Five pre-posttest studies examined pharmacological interventions including hormone treatment, norepinephrine, SSRIs and other antidepressants. Figure 7 presents the random effects weighted-average effect size was $\bar{d} = 4.30$ ($p<.001$, 95%
CI [1.66-6.93], showing a significantly large effect post treatment. Homogeneity of effect size was significant, \(Q(4) = 82.04, p<.001\), indicating more between-study variability than what would be expected from sampling error. The between-studies variance component was \(\tau^2 = 8.05\) and \(I^2 = 95.1\%\), indicating that 95.1\% of the variability among effect sizes is between studies.

![Forest plot](image)

**Figure 7.** Forest plot and 95\% confidence intervals for pre-post medication studies.

As with the overall analysis, I used a “leave-one-out” analysis to identify highly influential studies. One study in particular (Akohas et al., 2001) had an unusually large effect size influencing both the mean effect size and the homogeneity tests dramatically. Although this study appears to have an appropriate methodological design, it was the only pre-post hormone patch study. When this study was left out of the analysis, the aggregate effect size was still significant with \(\bar{d} = 1.38 (p<.001, 95\% \text{ CI } [.55-2.22])\). The between-studies variance component decreased substantially to \(\tau^2 = 0.35\) and \(I^2 = 51.7\%\), indicating that half of the
variance is accounted for by that study. Homogeneity of effect size was not significant, $Q (3) = 6.21, p = .1$. None of these studies reported follow-up data.

**Moderator Effects**

**Univariate analyses.** I used meta-regression to explore whether the following study characteristics moderated effect size: attrition between conditions, administration of treatment, and proportion of single to married females. Attrition was calculated by using the difference score between two treatments. Tables 3, 4, and 5 provide descriptive statistics for the moderators. Moderator analyses were not computed for comparisons in non-randomized trials and pre-post trials because there were not enough observations to complete the analyses. Tables 6, 7, and 8 present the results of the moderator analysis for PPD treatments compared to a control, TAU or alternative treatment. All of these moderators were not significant predictors of effect size for any comparison.

Tables 3, 4, and 5 provide descriptive details about these moderators.

**Table 3**

*Means and Standard Deviations for Attrition as a Moderator Analysis*

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Tx Mean</th>
<th>Tx SD</th>
<th>Comp Mean</th>
<th>Comp SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Therapy vs. TAU</td>
<td>4.81</td>
<td>5.07</td>
<td>2.88</td>
<td>3.48</td>
</tr>
<tr>
<td>Therapy vs. control</td>
<td>3.33</td>
<td>4.5</td>
<td>3.33</td>
<td>3.32</td>
</tr>
<tr>
<td>Alt tx vs. Alt tx</td>
<td>1.25</td>
<td>1.5</td>
<td>1.75</td>
<td>2.36</td>
</tr>
<tr>
<td>Combination vs. Alt tx</td>
<td>4.16</td>
<td>3.11</td>
<td>3.08</td>
<td>2.16</td>
</tr>
</tbody>
</table>
Table 4

*Means and Standard Deviations for Proportion of Single to Married Females as a Moderator Analysis*

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Single Mean</th>
<th>Single SD</th>
<th>Married Mean</th>
<th>Married SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Therapy vs. TAU</td>
<td>0.887</td>
<td>0.083</td>
<td>0.113</td>
<td>0.084</td>
</tr>
<tr>
<td>Therapy vs. control</td>
<td>0.948</td>
<td>0.061</td>
<td>0.052</td>
<td>0.061</td>
</tr>
<tr>
<td>Alt tx vs. Alt tx</td>
<td>0.903</td>
<td>0.085</td>
<td>0.096</td>
<td>0.085</td>
</tr>
<tr>
<td>Combination vs. Alt tx</td>
<td>0.835</td>
<td>0.126</td>
<td>0.165</td>
<td>0.126</td>
</tr>
</tbody>
</table>

Table 5

*Means and Standard Deviations for Administration of Treatment as a Moderator Analysis*

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Tx Mean</th>
<th>Tx SD</th>
<th>Comp Mean</th>
<th>Comp SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Therapy vs. TAU</td>
<td>2.33</td>
<td>2.23</td>
<td>4.66</td>
<td>3.20</td>
</tr>
<tr>
<td>Therapy vs. control</td>
<td>3</td>
<td>2</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>Alt tx vs. Alt tx</td>
<td>6</td>
<td>2.31</td>
<td>6</td>
<td>2.31</td>
</tr>
<tr>
<td>Combination vs. Alt tx</td>
<td>3.75</td>
<td>2.87</td>
<td>3.5</td>
<td>3</td>
</tr>
</tbody>
</table>
### Table 6

**Moderator Analysis for Attrition in Randomized Studies**

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Coefficient</th>
<th>SE</th>
<th>p</th>
<th>95% CI</th>
<th>$I^2$</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Therapy vs. TAU</td>
<td>0.01</td>
<td>0.02</td>
<td>.49</td>
<td>-0.03-0.05</td>
<td>41.09%</td>
<td>9</td>
</tr>
<tr>
<td>Therapy vs. control</td>
<td>0.08</td>
<td>0.05</td>
<td>.15</td>
<td>-0.05-0.21</td>
<td>0%</td>
<td>6</td>
</tr>
<tr>
<td>Alt tx vs. Alt tx</td>
<td>0.17</td>
<td>0.19</td>
<td>.46</td>
<td>-.63-.97</td>
<td>23.98%</td>
<td>4</td>
</tr>
<tr>
<td>Combination vs. Alt tx</td>
<td>0.17</td>
<td>0.09</td>
<td>.20</td>
<td>-0.21-0.54</td>
<td>0.72%</td>
<td>4</td>
</tr>
</tbody>
</table>

### Table 7

**Moderator Analysis for Proportion of Single to Married Females in Randomized Studies**

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Coefficient</th>
<th>SE</th>
<th>p</th>
<th>95% CI</th>
<th>$I^2$</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Therapy vs. TAU</td>
<td>0.96</td>
<td>0.80</td>
<td>.28</td>
<td>-1.09-3.02</td>
<td>0%</td>
<td>7</td>
</tr>
<tr>
<td>Therapy vs. control</td>
<td>1.73</td>
<td>1.49</td>
<td>.33</td>
<td>-3.01-6.47</td>
<td>0%</td>
<td>5</td>
</tr>
<tr>
<td>Alt tx vs. Alt tx</td>
<td>-1.73</td>
<td>2.64</td>
<td>.63</td>
<td>-35.35-31.88</td>
<td>50.91%</td>
<td>3</td>
</tr>
<tr>
<td>Combination vs. Alt tx</td>
<td>0.74</td>
<td>1.35</td>
<td>.64</td>
<td>-5.06-6.54</td>
<td>60.12%</td>
<td>4</td>
</tr>
</tbody>
</table>
Table 8

*Moderator Analysis for Administration of Treatment in Randomized Studies*

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Coefficient</th>
<th>SE</th>
<th>p</th>
<th>95% CI</th>
<th>$I^2$</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Therapy vs. TAU</td>
<td>-0.02</td>
<td>0.035</td>
<td>.68</td>
<td>-0.11-0.08</td>
<td>0%</td>
<td>6</td>
</tr>
<tr>
<td>Therapy vs. control</td>
<td>0.29</td>
<td>0.14</td>
<td>.12</td>
<td>-0.14-0.73</td>
<td>0%</td>
<td>5</td>
</tr>
<tr>
<td>Alt tx vs. Alt tx</td>
<td>Not computed: due to collinearity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combination vs. Alt tx</td>
<td>-0.13</td>
<td>0.74</td>
<td>.88</td>
<td>-3.31-3.05</td>
<td>63.76%</td>
<td>4</td>
</tr>
</tbody>
</table>

**Multivariate analyses.** Table 9 presents the results of multivariate analyses comparing depression measures to other broad measures (e.g. health, marital relationship, maternal attachment, social adjustment). Given that within-study correlations were not provided among these measures, I repeated the multivariate analysis three times, setting the correlations to .25, .5, or .75. Because the analyses were not sensitive to the magnitude of the within-study correlation, I reported the analysis assuming a .5 correlation.
Table 9

Results of Multivariate Meta-analyses Comparing Depression Measures and Other Broad Measures assuming a 0.5 correlation.

<table>
<thead>
<tr>
<th>Comparisons</th>
<th>Depression Outcome</th>
<th>Other measures Outcome</th>
<th>Difference between measures</th>
<th>Omnibus test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Randomized Studies</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Therapy vs. Control</td>
<td>.93**</td>
<td>.63**</td>
<td><em>p</em> = .03</td>
<td>$X^2 (2, k=6) = 48.26, p &lt; .001$</td>
</tr>
<tr>
<td>Medications vs. Placebo</td>
<td>.71**</td>
<td>.76**</td>
<td><em>p</em> = .82</td>
<td>$X^2 (2, k=2) = 21.65, p &lt; .001$</td>
</tr>
<tr>
<td>Therapy vs. TAU</td>
<td>.22</td>
<td>.37**</td>
<td><em>p</em> = .41</td>
<td>$X^2 (2, k=9) = 13.51, p &lt; .001$</td>
</tr>
<tr>
<td>Exercise vs. TAU</td>
<td>.27</td>
<td>.50**</td>
<td><em>p</em> = .22</td>
<td>$X^2 (2, k=2) = 7.05, p &lt; .05$</td>
</tr>
<tr>
<td>Combination vs. TAU</td>
<td>.63**</td>
<td>.52**</td>
<td><em>p</em> = .56</td>
<td>$X^2 (2, k=3) = 30.36, p &lt; .001$</td>
</tr>
<tr>
<td>Alternative tx vs. Alt tx</td>
<td>.54**</td>
<td>.29</td>
<td><em>p</em> = .28</td>
<td>$X^2 (2, k=4) = 4.07, p = .13$</td>
</tr>
<tr>
<td>Combination Tx vs. Alt tx</td>
<td>.16</td>
<td>.14</td>
<td><em>p</em> = .99</td>
<td>$X^2 (2, k=4) = 0.43, p = .80$</td>
</tr>
<tr>
<td><strong>Non-Randomized Studies</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Therapy vs. Control</td>
<td>1.83**</td>
<td>.48</td>
<td><em>p</em> = .05</td>
<td>$X^2 (2, k=3) = 9.35, p &lt; .001$</td>
</tr>
<tr>
<td>Therapy vs. TAU</td>
<td>.65**</td>
<td>.35</td>
<td><em>p</em> = .97</td>
<td>$X^2 (2, k=2) = 8.65, p &lt; .01$</td>
</tr>
<tr>
<td><strong>Pre-post Trials</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Therapy</td>
<td>1.56**</td>
<td>.09</td>
<td><em>p</em> = .07</td>
<td>$X^2 (2, k=7) = 41.93, p &lt; .001$</td>
</tr>
<tr>
<td>Medications</td>
<td>4.91</td>
<td>3.72</td>
<td><em>p</em> = .40</td>
<td>$X^2 (2, k=5) = 2.95, p = .22$</td>
</tr>
</tbody>
</table>

*Note.** **p* < .01, this p-value tests whether the effect size differ from zero.*
The omnibus test for the multivariate analyses tests the null hypothesis that all average effect sizes between comparisons (e.g. therapy versus control) are zero. The omnibus test was significant across values of the within-study correlation for PPD treatments compared to a control and TAU condition. These results reject the null hypothesis and suggest that there is a difference in effect sizes between these comparisons on depression and all other measures. From these comparisons only two of them (therapy vs. control, combination vs. TAU) showed larger effect sizes for depression measures than for all other measures. However, in the combination treatments versus TAU comparison there was no statistically significant difference.

In the remaining randomized studies that compared a PPD treatment to a different PPD treatment, the omnibus test was not significant indicating that there is no difference in effect sizes between these two treatment conditions on measuring depression and all other measures. In addition, the effect sizes for depression and all other measures in each of these comparisons were not statistically significant from one another, suggesting that there was no difference of effect for PPD treatments on depression measures and all other measures.

Across the non-randomized comparisons, the effect size for depression measures and the effect size for other measures were statistically significant from zero (see Table 9). The effect size for depression measures was larger than the effect size for all other measures across values of the within-study correlation. However, the differences between the effect sizes were not statistically significant.

Therapy pre-post trials had a significant omnibus test, showing a difference between pre and post effect sizes on depression and all other measures (see Table 9). The effect size for depression measure was larger than the effect size for other measures; however, the difference between the effect sizes was not statistically significant. In contrast, medication pre-post trials
showed no significant difference between depression and all other measures. In addition, the effect sizes for depression and all other measures in each of these comparisons were not statistically significant from each other.

**Publication Bias**

Publication bias was examined in comparisons that had close to 10 trials in order to reduce sensitivity in the analyses. Given that almost all of the comparisons ranged from four to seven studies, I decided to choose the largest comparison that consisted of at least nine studies. Figure 8 presents the contour-enhanced funnel plot for randomized studies comparing therapy to TAU. The white, light gray and dark gray area of the plot represents regions of statistical significance with increasing confidence bands as it moves to the darker regions. The contour-enhanced plot show that most of the effect sizes are above zero and generally fall in the low statistical significance area, which argues against publication bias. However, the plot is asymmetrical.
Figure 8. Funnel plots for therapy versus TAU.

Table 10 presents the results of the rank correlation test (Begg’s test), regression test (Egger’s test), and trim-and-fill analysis. Both the rank correlation test and regression tests were not statistically significant. The results of the trim-and-fill analysis indicated three additional studies need to be included in order to make the plot more symmetrical. These three studies reduced $d$ to 0.16 (49% reduction).

Table 10
Results of Publication Bias Analyses

<table>
<thead>
<tr>
<th>Comparison Condition</th>
<th>Rank Correlation</th>
<th>Regression Test</th>
<th>Trimmed Studies</th>
<th>$\bar{d}'$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Therapy vs. TAU</td>
<td>$z = 1.36$</td>
<td>bias = 2.93</td>
<td>3</td>
<td>.16</td>
</tr>
</tbody>
</table>

Note. $\bar{d}'$ = adjusted aggregate effect size
In sum, the publication bias analyses did not present clear evidence of publication bias. The funnel plots appeared asymmetrical; however the rank correlation and regression tests were not statistically significant. The adjusted effect sizes in the trim-and-fill analyses were smaller than the original effect sizes. However, the change in effect size was not substantial.

**Discussion**

**Treatment Compared to Control**

Results of these analyses provide evidence for the efficacy of a range of interventions for PPD. Similar to previous meta-analyses (Cuijpers, Brannmark & van Straten, 2008; Dennis & Hodnett, 2009), psychological and psychosocial interventions in randomized trials were found to be effective when compared to a control group ($d = 0.76$). These interventions include evidence-based therapies (e.g. CBT, IPT) in individual and group format as well as support and psychosocial counseling led by health visitors. When considering the effect size in terms of distribution overlap, the results suggest that individuals receiving active treatments were 77.52% more likely to experience a positive outcome than those individuals in the control condition. Contrary to previous research, this meta-analysis showed no difference between psychological interventions to a control group in non-randomized studies. Given that there were only three studies and each of them had varying experimental designs (e.g. prevention study prior to treatment, minimally trained counselors, and manualized group treatment) these results should be interpreted with caution. Consistent with previous research (Bledsoe & Grote, 2006), pre-post studies showed psychological interventions in a group format having a large effect at post treatment. Three of the six pre-post studies examined IPT, which is a relatively new approach, in either an individual or group setting for PPD treatment. Currently there is only one IPT group randomized study and two IPT randomized studies in the PPD outcome literature. Results from
these pilot studies indicate that IPT is a useful therapeutic approach to improving PPD symptoms in mothers.

Previous research has also shown the effectiveness of biological interventions such as pharmacotherapy and hormone therapy for improving mood. Findings from this meta-analysis suggest that individuals in randomized trials who received medication or estrogen treatment performed better ($d = 0.72$) than individuals in a placebo condition. Since there were only a few antidepressant and hormone therapy studies, I decided to combine them in order to increase power. When considering the effect size in terms of distribution overlap, the results suggest that individuals receiving active treatments were 76.36% more likely to experience a positive outcome than those individuals in the placebo condition. This finding is consistent with Dennis, Ross and Herxheimer’s meta-analysis (1999) that indicated a larger outcome for hormonal interventions than for the placebo condition. Given that there is no pharmacological meta-analysis, this is the first meta-analysis that supports past literature reviews that have found open-label medication trials to be effective in treating PPD. Future trials may conduct single blind or double blind designs in order to limit subjective bias from participants and administrators. When non-randomized designs were examined, there was no difference between individuals in Nomifensine drug group to the control group. There were no non-randomized estrogen studies. In addition, pharmacological and hormone therapy also had a large effect in pre-post studies ($d = 4.30$), which is consistent with past research (Bledsoe & Grote, 2006). Within this comparison there was significantly high heterogeneity due to one influential study influencing the aggregate effect size. Akohas et al. (2001) was the only pre-post hormone patch study, and when removed accounted for half of the variance and dropped the effect size to $d = 1.38$. It is important to interpret this conclusion with caution given that influential studies can distort
aggregate effect size and estimates of heterogeneity, and make it difficult to detect moderators. In the future, it would be important to separate pharmacological interventions from hormone therapy studies in order to make generalizable conclusions to a specific treatment.

Also consistent with a previous meta-analysis (e.g., Daley et al., 2009), one randomized study showed a moderate effect that favored combination of exercise and social support interventions when compared to a control condition. In non-randomized trials, one study examined the difference between a combination of therapy and medication to a control group. Results showed no difference between these conditions. With only one study in each of these experimental designs this result should be interpreted with caution until more studies are published.

**Treatment Compared to TAU**

Randomized studies that compared therapeutic interventions to TAU reported a smaller effect ($\bar{d} = 0.31$). It is known from other studies that TAU typically results in smaller effects than those of control groups (Cuipers, Andersoon et al., 2008). In this meta-analysis there was a broad range of psychological interventions including evidenced-based protocols, counseling administered by nurses to peer, and partner support, indicating the diverse therapeutic interventions for treating PPD. Similar to the earliest meta-analysis (Ray & Hodnett, 1998), non-randomized trials that compared psychological interventions to TAU had a moderate effect favoring psychological treatments.

There were several randomized studies that examined alternative biological treatments. In the present meta-analysis, exercise fared better than TAU ($\bar{d} = 0.44$), which is comparable to Daley et al. (2009) meta-analysis that reported a large effect for exercise compared to a control condition. This result shows that physical activity is a safe and inexpensive alternative to more
traditional interventions like medications and therapy. In addition, this is the first meta-analysis that examined combination treatments with TAU. A moderate effect from three studies showed individuals who received two interventions fared better than those who were in a TAU group. Given that the three treatments differed greatly from each other, there was significant between-study variance that could not be accounted for by these moderators.

Follow-up results after 4 to 244 weeks post treatment showed a small to moderate effect favoring either therapy and exercise interventions to TAU in randomized studies. As the literature expands, future reviews should include effect sizes at available follow-up time points in order to assess the longevity of the effects of treatment.

**Treatment Compared to Another Condition**

Two of the comparisons comparing one PPD treatment to another showed no difference between treatments and homogeneity was accepted (e.g. alternative interventions vs. other treatments, and combination treatments vs. alternative treatment). This result is surprising given that some of these comparisons had significant variability within the comparison. For instance, the combination treatments comparison had two or more divergent interventions. Due to the great variability in some of these comparisons and so few studies it is difficult to make any conclusive statements about their effectiveness.

When non-randomized designs were examined, there were no differences between two of the comparisons (i.e. combination vs. medication, medication vs. therapy). Power was low with only a few studies which made it difficult to make meaningful comparisons and interpretations.

The fact that alternative biological treatments in a randomized trial when compared to each other or to a support group did not show significant difference contradicts past research that indicated superior effects favoring alternative interventions including exercise when compared to
psychological interventions (Cuijpers, Brannmark et al., 2008). In the previous meta-analysis, there was relatively high heterogeneity because there were three vastly different interventions (pram walking, medication, systematic care) being compared; therefore, these results should be interpreted with caution. Given that there were only four studies in my randomized comparison and no non-randomized and no pre-post studies that looked at these alternative biological treatments, it is evident that this is a new area of research that needs to be explored in the future.

This meta-analysis included results from one randomized study (Sharp et al., 2010) that had a small effect favoring antidepressants when compared to individuals in a non-directive counseling group. However, there was one non-randomized study (Pearlstein et al., 2006) that observed no differences between PPD individuals that were in an antidepressant and IPT condition. These results are consistent with past studies that showed mixed findings (Appleby et al., 1997; Misri et al., 2004). There are several differences between these two studies: duration (4 weeks vs. 12 weeks) and research designs (randomized vs. non-randomized), which could account for the differences. Nevertheless, a common concern in both of these studies is the reservations women had to be in the antidepressant group given the stigma and side effects. In the non-randomized study, only two individuals chose to take antidepressants, while in the other study, individuals were given a two-arm randomized trial where mothers could switch interventions after 4 weeks if their first assigned treatment was not working. Although I only calculated the effect size prior to the switch, this additional information could have changed the attitudes and perspectives of the women. The choice to allow PPD women to experience both conditions may reflect the understanding that women continue to have reservations about taking medications when they are breastfeeding. Due to the controversial nature of
psychopharmacology after childbirth, psychological interventions continue to represent the first line of treatment for postpartum women.

Moderator and Multivariate Analyses

There was some heterogeneity in these comparisons, indicating that there were some systematic differences between studies. Although a majority of the comparisons had low to moderate heterogeneity, I conducted a moderator analysis to account for variance between studies. Moderator analyses showed that attrition, administration of treatment, and proportion of single to married females were not significant moderators in predicting effect sizes for most comparisons.

Power was also an issue in the multivariate analyses. I compared effect sizes for depression outcomes to other measures to see if these PPD treatments were more effective in dealing with the outcomes they were designed to treat. For most of the comparisons, there were no significant differences. However, when psychological treatments were compared to a control, depression measures had a larger effect than other types of measures. Finally, there was no consistent evidence that publication bias threatened the validity of these results.

Limitations

The present meta-analysis has several limitations. First, given that PPD is a relatively new field, the number of studies included in each research design is small. Moderator analyses were likewise limited by the small number of studies included. The mixing of pharmacological and other biological studies may also be a limitation, but this was necessitated by the fact that, currently, there are a limited number of studies that use biological interventions. Therefore, it is difficult to assess which types of interventions are truly effective when power is low. As the field develops, future research is needed to establish separate effects with distinct biological
treatments. Research should continue to address the safety of pharmacological treatments for PPD as well as inform mothers of these treatments in order to de-stigmatize the potential side effects. Second, future research should pay attention to sample size, statistical power, retention of patients, and differences in treatment appropriateness and outcome. Additional approaches, such as length of treatment and dosage of medication need to be tested for the substantial number of patients who enter treatment and do not recover. Third, the number of studies that included follow-up data is too small. Future research should include long-term outcomes in their research design in order to assess whether the benefits of treatment for PPD are maintained over time.

In addition to study limitations, all of these studies focused on mothers who were diagnosed with non-psychotic PPD, which limits these findings to a specific population. There are very few studies that examine PPD psychosis and even fewer treatment studies. Therefore, it is difficult to make any conclusions about which biological or non-biological interventions are effective for PPD psychosis. Lastly, most of the studies reported marital status which provides demographic information about the mother. However, there was little information given about cultural diversity, social economic status, and other relevant characteristics about the mothers. These factors should be considered and possibly incorporated in future PPD research so that treatments are targeted to certain populations.

**Conclusion**

Due to the negative effects PPD has on mothers, infants and families, it is imperative that health professionals inform women about the symptoms and treatments that are available for PPD. Given that PPD outcome treatments is a relatively new field, providing current information to the public about the various treatments, formats, and settings that are accessible and available to mothers is critical to effectively treating these symptoms quickly. This meta-
analysis has shown psychological interventions, pharmacology, and exercise treatments improve PPD symptoms when compared to a control or TAU. However, there is an ongoing debate about whether pharmacological treatments should be used because of the potential side effects it has on infant development. Better understanding on this controversy needs to be explored by policy makers, researchers, clinicians, and patients before we can make any conclusions. As the literature grows, we can use meta-analysis and related techniques to gain a full view on this public health issue.
References


*Articles used in the meta-analysis have an asterisk.*


Dennis, C.L., Ross, L., & Herxheimer, A H. (1999). Oestrogens and progestogens for preventing and treating postnatal depression. *Cochrane Database of Systematic Reviews (Online), (3),* CD001690.


Postpartum Treatment Coding Manual

General Instructions

1. Note that each code below is numbered. When coding, please highlight empirical evidence in support of the code in the study report, and mark it with the coding reference number.
2. You should guess a code when a plausible guess is possible. However, if insufficient evidence exists in the study report to make a plausible guess, or does not provide the information then fill in the coding blanks with ".99" to indicate unknown.

Standardized Codes

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
<th>Code</th>
</tr>
</thead>
<tbody>
<tr>
<td>-.99</td>
<td>UNKNOWN/info not given</td>
<td></td>
</tr>
<tr>
<td>-.55</td>
<td>not a medication tx (dosage)</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>no comparisons for pre/post study</td>
<td></td>
</tr>
<tr>
<td>-.77</td>
<td>for placebo (length of med tx)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Code #</th>
<th>Code Description</th>
<th>Code</th>
</tr>
</thead>
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<tr>
<td><strong>STUDY LEVEL CODES</strong></td>
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<td></td>
</tr>
<tr>
<td>1</td>
<td>Study Identification Number</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Comparison Number</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Measure Number</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Year of Study</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td><strong>Study Design</strong></td>
<td></td>
</tr>
<tr>
<td>1.</td>
<td>Two-group comparison: Clients were randomly assigned, random numbering, random allocation, alternating to treatment and comparison conditions. <em>Note</em>: The key point is whether or not the clients were randomly allocated to a condition.</td>
<td>Make this a drop down list</td>
</tr>
<tr>
<td>2.</td>
<td>Two-group comparison: Clients were <strong>not</strong> randomly assigned to treatment and comparison conditions. <em>Note</em>: Clients may have self-selected into a particular kind of treatment.</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>One-group comparison: Pre/post design. <em>Note</em>: only treatment group</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td><strong>Method of recruitment</strong></td>
<td></td>
</tr>
<tr>
<td>1.</td>
<td>Midwife</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Self</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Doctor/ health professionals/counselor (e.g. pediatricians)</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>By hospital unit</td>
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</tr>
<tr>
<td><strong>5.</strong> Advertisement</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>6.</strong> Combination of referrals</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>7.</strong> Other</td>
<td>-.99 Unsure/Not Reported</td>
<td></td>
</tr>
<tr>
<td><strong>7.</strong> Initial assessment of PPD (<em>Note:</em> This code is defined by the number of weeks after giving birth they were assessed for PPD. If the timing is before birth, code 0. If they provide age of child, then use this information, if not then include info about inclusion criteria. If they give a range, calculate the average.) i.e. 12 months = 52 weeks 16 months = 64 weeks (16X4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>8.</strong> PPD diagnostically defined- using a standardized inventory or interview (<em>Note:</em> This code is determined by the method in which mothers were recruited for the study) 1. Clinical interview by researcher or physician (using a diagnostic interview e.g. DISC, DSM, ICD-10) 2. Self-report measures (EPDS, BDI, HAM-D, PDSS) 3. Both, completed a screening measure and clinical interview 4. Screening questionnaire through phone 5. Other</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>9.</strong> Proportion of Single Mothers in Sample (including divorced, separated, living alone) (<em>Note:</em> This number should be a decimal (two places) of total sample)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>10.</strong> Proportion of Married/Cohabiting Mothers in Sample (<em>Note:</em> This number should be a decimal (two places) of total sample)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>COMPARISON LEVEL CODES</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>11.</strong> Treatment Type (<em>Note:</em> this code has been operationalized by the studies, not by the raters) 1. Psychotherapy (e.g. CBT, IPT, Psychodynamic) 2. Support Group (e.g. peer, partner) 3. Non-directive counseling 4. Counseling by health visitor (support health visitor, nurse, nursing student) 5. Omega-3 fatty acids 6. Hormones (e.g. 17Beta-oestradiol, Norethisterone) 7. SSRIs (e.g., Sertraline, Paroxetine, Fluoxetine, Citalopram, Fluvoxamine) 8. Tri-cyclics (e.g. imipramine and nortriptyline)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
9. Norepinephrine-dopa RIs (Nomifensine, Bupropion SR)
10. Other antidepressant (e.g., Nefazodone)
11. Bright light therapy
12. Physical Therapy (e.g., pram walking, massage, yoga)
13. Repetitive TMS
14. Specialized psychiatric day hospital
15. Mother-Baby Intervention (e.g., massages, M-ITG)
16. Community group services
17. Combination of Omega/ Supportive Therapy
18. Combination of exercise/ social support
19. Combination of Drug/therapy
20. Combination peer support/ TAU
21. Combination of massage/support group
22. Combination of placebo/therapy

Format of Treatment in treatment condition. Note:
Biological treatment includes drug, hormone therapy and vitamin supplements.
1. Individual (Client is seen in a one-on-one situation with a therapist)
2. Group (Client is seen in a group setting where there is more than one client per therapist)
3. Home visits
4. Phone therapy, telecare
5. Couples therapy
6. Biological Treatment Condition (e.g., medication, hormone therapy)
7. Medication Placebo
8. Combination Biological/ Individual Therapy
9. Combination Biological/ Group Therapy
10. Combined placebo/ Individual Therapy
11. Combined placebo/ Group Therapy
12. Exercise

How was treatment administered
1. Nurse/ midwife
2. Psychologist/ therapist
3. Psychiatrist
4. Self
5. Health workers
6. Nursing students
7. No person administered treatment
8. Other
- .99 - Unreported/unknown

Comparison Type
Note: The comparison condition in this meta-analysis is defined
as the condition to which either manualized therapy, non-directive counseling, medication, hormonal therapy, exercise is compared. Code 0 for pre/post study.

1. No Treatment or Wait-List or non-intervention (Client is not receiving any type of treatment, education or support)
2. Control group (non-depressed mothers receiving treatment)
3. Usual Care, Routine Care, or Treatment-as-Usual (treatment is genuine therapy but not under the control or direction of researcher)
4. Drug Treatment
5. Placebo (imitation of stimulus medicine)
6. Alternative Therapy Treatment (CBT, interpersonal, psychoeducation, psychodynamic)
7. Alternative Biological Treatment (Treatment condition is compared to hormone, dim light, acupuncture, massage, omega fatty acids)
8. Alternative counseling (supportive counseling, home visits, health visitor)
9. Combined treatment (Treatment condition is compared to a combination of therapy and medication treatment)
10. Combined treatment (exercise plus therapy/social support)
11. Combined treatment (placebo plus support/therapy)
0 - pre/post

<table>
<thead>
<tr>
<th>Format of Treatment in the Comparison Condition. Note: Biological treatment includes drug, hormone therapy and vitamin supplements. Code 0 for pre/post study.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Individual (Client is seen in a one-on-one situation with a therapist)</td>
</tr>
<tr>
<td>2. Group (Client is seen in a group setting where there is more than one client per therapist)</td>
</tr>
<tr>
<td>3. Home visits</td>
</tr>
<tr>
<td>4. Phone therapy, telecare</td>
</tr>
<tr>
<td>5. No treatment/control condition</td>
</tr>
<tr>
<td>6. Biological Treatment Condition (e.g. medication, hormone therapy)</td>
</tr>
<tr>
<td>7. Medication Placebo</td>
</tr>
<tr>
<td>8. Combination Biological/ Individual Therapy</td>
</tr>
<tr>
<td>9. Combination Biological/ Group Therapy</td>
</tr>
<tr>
<td>10. Combined placebo/ Individual Therapy</td>
</tr>
<tr>
<td>11. Combined placebo/ Group Therapy</td>
</tr>
<tr>
<td>- .99 - Unknown</td>
</tr>
<tr>
<td>0 - pre/post</td>
</tr>
</tbody>
</table>
| 16 | **How was comparison treatment administered.** Code 0 for pre/post study.
| 1. Nurse
| 2. Psychologist/ therapist
| 3. Psychiatrist
| 4. Self
| 5. Health workers
| 6. Nursing students
| 7. No person administered treatment
| 8. Other
| 9. multiple (i.e. therapist and psychiatrist)
| -99 - Unknown
| 0 - pre/post |
| 17 | **Number of subjects assigned to the treatment condition.**
*Note:* For a randomized experiment, count all those subjects assigned to the treatment condition even if they were later dropped, unless the dropped subject was later found to not meet inclusion criteria. For a nonrandomized study, count all those subjects assigned to start treatment.
| 18 | **Number of subjects assigned to the comparison condition.**
*Note:* For a randomized experiment, count all those subjects assigned to the comparison condition even if they were later dropped, unless the dropped subject was later found to not meet inclusion criteria. For a nonrandomized study, count all those subjects assigned to start treatment. Note: Code 0 for pre/post study.
| 19 | **Duration of Treatment in Number of Weeks in Treatment Condition**
*Note:* Record the length of each treatment, in weeks. If the length of each treatment was variable, record the average length of each treatment.
| 20 | **Duration of Treatment in Number of Weeks in Comparison Condition**
*Note:* Record the length of each treatment, in weeks. If the length of each treatment was variable, record the average length of each treatment. Note: Code 0 for pre/post study.
| 21 | **Duration of Treatment in Number of Sessions in Treatment Condition**
*Note:* Record the length of each treatment, in sessions. If the length of each treatment was variable, record the average length of each treatment.
| 22 | **Duration of Treatment in Number of Sessions in Comparison Condition**
*Note:* Record the length of each treatment, in sessions. If the length of each treatment was variable, record the average length of each treatment. Note:
| 23 | **Length of Sessions in the Treatment Condition.** *Note:* Record the length of each session, **in minutes**. If the length of the sessions was variable, record the average length of each session. Take an average of the range of time. |
| 24 | **Length of Sessions in the Comparison Condition.** *Note:* Record the length of each session, **in minutes**. If the length of the sessions was variable, record the average length of each session. Take an average of the range of time. *Note:* Code 0 for pre/post study. |
| 25 | **Dosage in the Treatment Condition**  
*Note:* Record the exact number in mg of the dosage prescribed to the participants in the treatment condition. If there is a range of dosages that differs for everyone, take an average. If there is the same “highest” dosage for everyone, record that number. If it’s not a medication treatment, such as therapy, code -.55. |
| 26 | **Dosage in the Comparison Condition**  
*Note:* Record the exact number in mg of the dosage prescribed to the participants in the comparison condition. If there is a range of dosages that differs for everyone, take an average. If there is the same “highest” dosage for everyone, record that number. If it’s not a medication treatment, such as therapy, code -.55. Code 0 for pre/post study. |
| 27 | **Length of time in Days That the Participant was on the Medication in Treatment Condition** *Note:* Record the length of time in exact number of days that the participant was on the medication in the Treatment Condition.  
*Code*: -.55 for no medication such as therapy, code -.77 for placebo.  
i.e. 30 days=1 month |
| 28 | **Length of time in Days That the Participant was on the Medication in Comparison Condition** *Note:* Record the length of time in exact number of days that the participant was on the medication in the Comparison Condition.  
*Code*: -.55 for no medication such as therapy, code -.77 for placebo. Code 0 for pre/post study. |
<p>| 29 | <strong>Attrition Number in Treatment Condition</strong> <em>Note:</em> Record the exact number of participants who drop out prior to any conclusion of treatment (does not include follow-up) in the |</p>
<table>
<thead>
<tr>
<th>Measure Name</th>
<th>MEASURE LEVEL CODES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attrition Number in Comparison Condition</td>
<td>Note: Record the exact number of participants who drop out prior to any conclusion of treatment (does not include follow-up) in the comparison condition (Treatment attrition). Note: Code 0 for pre/post study.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Measure Name</th>
<th>EFFECT SIZE LEVEL CODES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Measure Rater</td>
<td>Drop down list of all the measures used in each study</td>
</tr>
<tr>
<td>1. Self</td>
<td></td>
</tr>
<tr>
<td>2. Clinician</td>
<td></td>
</tr>
<tr>
<td>3. Partner</td>
<td></td>
</tr>
<tr>
<td>4. Other</td>
<td></td>
</tr>
</tbody>
</table>

**Timing of Assessment.** (Was the assessment a pretreatment condition, post treatment or follow-up?)

1. Pretreatment (Assessment immediately before treatment takes place)
2. Post Treatment (Assessment immediately after the finish of therapy or the first assessment after tx ended; i.e. within one month of treatment)
3. Follow-up (Assessment takes place after an amount of time since treatment has passed; i.e. 6 months)
4. Other (Assessment takes place during treatment)

**Timing of Follow-up.**

*Note: If the timing of the assessment is pre or post then code as 0. Otherwise, indicate the follow-up timing as the number of weeks from the point of ending treatment. i.e. 4 weeks=1 month*
Appendix B

Articles to be Coded in this Meta-Analysis
Postpartum Depression: Comparisons & Measures List

**ALL Randomized Studies**

**Therapy VS Therapy**


PPD women, RA

Comparison:

4. Support Group vs Control

Measures:

1 - BDI - Beck Depression Inventory – Pre
2 - BDI - Beck Depression Inventory - Post
3 - PSS - Perceived Stress Scale - Pre
4 - PSS - Perceived Stress Scale - Post
5 - ISEL - Interpersonal Support Evaluation List – Pre
6 - ISEL - Interpersonal Support Evaluation List - Post
7 - SEI - Self - Esteem Inventory - Pre
8 - SEI - Self - Esteem Inventory - Post

Timing of Assessment:

6. Pre
7. Post test

Note: To calculate Post test, we added baseline mean to D (difference), and used pre-test SD. e.g. 15.73 + (-6.6)=9.13


PPD women, RA

Comparison:

12. Interpersonal psychotherapy (IPT) vs. Mother Infant Support Group (M-ITG)
13. Interpersonal psychotherapy (IPT) vs. Control
14. Mother Infant Support Group (M-ITG) vs. Control

Measures:

1 - BDI - Beck Depression Inventory - Pre
2 - BDI - Beck Depression Inventory - Post
3 - CES-D - Center for Epidemiological Studies Depression Scale - Pre
4 - CES-D - Center for Epidemiological Studies Depression Scale - Post
5 - PSI - Child Domain Total - Pre
6 - PSI - Child Domain Total - Post
7 - PSI - Child Adaptability - Pre
8 - PSI - Child Adaptability - Post
9 - PSI - Child Acceptability - Pre
10 - PSI - Child Acceptability - Post
11 - PSI - Child Demandingness - Pre
12 - PSI - Child Demandingness - Post
13 - PSI - Child Mood - Pre
14 - PSI - Child Mood - Post
15 - PSI - Child Distractibility/Hyperactivity - Pre
16 - PSI - Child Distractibility/Hyperactivity - Post
17 - PSI - Child Reinforces - Pre
18 - PSI - Child Reinforces - Post
19 - PCERA - Parent Child Early Relational Assessment - Factor 1 - Pre
20 - PCERA - Parent Child Early Relational Assessment - Factor 1 - Post
21 - PCERA- factor 2 - Pre
22 - PCERA- factor 2 - Post
23 - PCERA- factor 3 - Pre
24 - PCERA- factor 3 - Post
25 - PCERA- factor 4 - Pre
26 - PCERA- factor 4 - Post
27 - PCERA- factor 5 - Pre
28 - PCERA- factor 5 - Post
29 - PCERA- factor 6 - Pre
30 - PCERA- factor 6 - Post
31 - PCERA- factor 7 - Pre
32 - PCERA- factor 7 - Post
33 - PCERA- factor 8 - Pre
34 - PCERA- factor 8 - Post

Timing of Assessment:
  10. pre
  11. post


PPD women, RA
Comparison:
  5. Cognitive Behavior Therapy (CBT) vs. Routine Primary Care
  7. Cognitive Behavior Therapy (CBT) vs. Psychodynamic Therapy
  8. Psychodynamic Therapy vs. Routine Primary Care
10. Non-Directive Counseling vs. Routine Primary Care

Measures
1. EPDS - Edinburgh Postnatal Depression Scale - Post
2. EPDS - Edinburgh Postnatal Depression Scale – FU 9 mon
3. EPDS - Edinburgh Postnatal Depression Scale – FU 18 mon
4. EPDS - Edinburgh Postnatal Depression Scale – FU 5 yr
5. SCID - Structured Clinical Interview for DSM III Diagnosis - Post
6. SCID - Structured Clinical Interview for DSM III Diagnosis – FU 9 mon
7. SCID - Structured Clinical Interview for DSM III Diagnosis – FU 18 mon
8. SCID - Structured Clinical Interview for DSM III Diagnosis – FU 5 yr
9. Maternal management problems checklist - Post
10. Murray et al 1996a global rating scales using video tape - Post
11. BSQ - Behavioural Screening Questionnaire (infant emotional and behavioral problems) – FU 18 mon
12. Ainsworth Strange situation procedure (assessed infant attachment) – FU 18 mon
13. Mental Development Index of the Bayley Scales (assessed infant cognitive development) – FU 18 mon
14. Rutter A^2 Scale (assessed child behavior problems, parent report) – FU 5 yr
15. PBCL - Pre-school Behavioral Checklist (teacher report, child behavior problems) – FU 5 yr
16. McCarthy Scale of Children’s abilities (child cognitive development) – FU 5 yr

Timing of Assessment
2. Post (4 months)
12. FU – 9 mon
3. FU- 18 mon
3. FU- 5 years

Note: measures 5-10 and 12, use #0201 where success is # without depression and failure is total-success.
For measure 11, 13-16, use #0601 where you enter the p-values and sample sizes, for only 1, 4, 6 comparison group – all are against control group.


PPD women, RA
Comparison:
1. Telephone Peer Support plus Treatment as Usual (TAU) vs Treatment as Usual (TAU)

Measures:
1. EPDS - Edinburgh Postnatal Depression Scale - Post
2. Rosenberg Self-esteem Scale (SES) - Pre
3. Rosenberg Self-esteem Scale (SES) - Post
4. Child Care stress Checklist - Pre
5. Child Care stress Checklist - Post
6. UCLA Loneliness Scale - Pre
7 - UCLA Loneliness Scale – Post
Timing of Assessment:
1. Pre
2. post tx (8 weeks)
Note: For EPDS, post, complete it using chi-sq success and failures, #0201


PPD Women, RA
Comparison:
1. Psycho-educational group (PEG) vs. Routine Primary Care (RPC)
Measures:
1 - EPDS - Edinburgh Postnatal Depression Scale - Pre
2 - EPDS - Edinburgh Postnatal Depression Scale - Post
3 - EPDS - Edinburgh Postnatal Depression Scale - FU
4 - Duke - UNC Social Support Questionnaire - Pre
5 - Duke - UNC Social Support Questionnaire - Post
6 - Duke - UNC Social Support Questionnaire - FU
7 - DAS - Dyadic (Marital) Adjustment Scale - Pre
8 - DAS - Dyadic (Marital) Adjustment Scale - Post
9 - DAS - Dyadic (Marital) Adjustment Scale - FU
10 - WCCR - Ways of Coping Checklist Revised - Pre
11 - WCCR - Ways of Coping Checklist Revised - Post
12 - WCCR - Ways of Coping Checklist Revised – FU
Note: for measures 4-12 that are not significant, use #0603 with ES being 0.

Timing of Assessment:
1. pre
2. post (8 weeks)
3. FU (6 months)
Note: No effects of time, group, or interactions between time and group (pg. 407).


PPD women, RA comparison:
1 - Nurse Intervention vs. Control Group (Treatment as usual)
Measures:
1 - BDI - Beck Depression Inventory - Pre
2 - BDI - Beck Depression Inventory - Post
3 - DMC - Dyadic Mutuality Code - Pre
4 - DMC - Dyadic Mutuality Code - Post
Timing of Assessment:
1 - pre
2 - post


**Similar Treatment Comparison**

comparison:
1 - Mother-baby Intervention vs. Telephone Support

Measures:
1 - Emotional Availability Scales (EAS) - maternal sensitivity - Pre
2 - Emotional Availability Scales (EAS) - maternal sensitivity - Post
3 - Emotional Availability Scales (EAS) - maternal sensitivity – FU 6 mon
4 - Emotional Availability Scales (EAS) - maternal structuring - Pre
5 - Emotional Availability Scales (EAS) - maternal structuring - Post
6 - Emotional Availability Scales (EAS) - maternal structuring – FU 6 mon
7 - Emotional Availability Scales (EAS) - maternal nonintrusiveness - Pre
8 - Emotional Availability Scales (EAS) - maternal nonintrusiveness - Post
9 - Emotional Availability Scales (EAS) - maternal nonintrusiveness – FU 6 mon
10 - Emotional Availability Scales (EAS) - maternal nonhostility - Pre
11 - Emotional Availability Scales (EAS) - maternal nonhostility - Post
12 - Emotional Availability Scales (EAS) - maternal nonhostility – FU 6 mon
13 - Emotional Availability Scales (EAS) - child responsiveness - Pre
14 - Emotional Availability Scales (EAS) - child responsiveness - Post
15 - Emotional Availability Scales (EAS) - child responsiveness – FU 6 mon
16 - Emotional Availability Scales (EAS) - child involvement - Pre
17 - Emotional Availability Scales (EAS) - child involvement - Post
18 - Emotional Availability Scales (EAS) - child involvement – FU 6 mon
19 - Beck Depression Index score (BDI) - Pre
20 - Beck Depression Index score (BDI) - Post
21 - Beck Depression Index score (BDI) – FU 6 mon
22 - Attachment Q-sort (AQS) attachment security – FU 6 mon
23 - Infant Toddler Social and Emotional Assessment (ITSEA) externalizing – FU 6 mon
24 - Infant Toddler Social and Emotional Assessment (ITSEA) internalizing – FU 6 mon
25 - Infant Toddler Social and Emotional Assessment (ITSEA) dysregulation – FU 6 mon
26 - Infant Toddler Social and Emotional Assessment (ITSEA) competence – FU 6 mon
27 - Quality of Maternal Interactive behavior - FU- 49 mon
28 - Attachment Story Completion Task, adapted version (attachment security to mother) - FU-49 mon
29 - Puppet Interview (self-esteem) – FU 49 mon
30 - California Child Q-set (ego resiliency) - FU 49 mon
31 - Peabody Picture Vocabulary Test (verbal intelligence) - FU 49 mon
32 - Preschool Social Behavior Questionnaire (PSBQ) (prosocial behaviour) - FU 49 mon
33 - Stress Response Scale (school adjustment), teacher report - FU 49 mon
34 - Child Behavior Checklist (CBCL/1.5-5) - mother rated, internalising problems - FU 49 mon
35 - Caregiver-Teacher Report Form (C-TRF) teacher, internalising problems - FU 49 mon
36 - Child Behavior Checklist (CBCL/1.5-5) - mother rated, externalising problems - FU 49 mon
37 - Caregiver-Teacher Report Form (C-TRF) teacher, externalising problems - FU 49 mon

Timing of Assessment:
1 - pre
2 - post test
3 - FU: 6 month
4- FU- 49 months (study 75)


RA, PPD ** Camile move this to RA section, after you code it.

Comparison:
1. Group Treatment (based on social/emotional/partner support, education, CBT, networking, communication, practice homework tasks) vs. Control

Measures:
1 - BDI - Beck Depression Inventory – Post
2 - EPDS - Edinburgh Postnatal Depression Scale – Post
3 - POMS depression - Profile of Mood States – Post
4 - POMS tension – Post
5 - POMS confusion – Post
6 - POMS fatigue – Post

Timing of Assessment:
2 - post (10-weeks)

Note: Use p-values on pg. 857


RA, PPD ** Camile move this to RA section, after you code it.

Comparison:
1. Cognitive Behavior Therapy Group vs. Group Counseling
2. Cognitive Behavior Therapy Group vs. Individual Counseling
3. Cognitive Behavior Therapy Group vs. Routine Primary Care
4. Group Counseling vs. Individual Counseling
5. Group Counseling vs. Routine Primary Care
6. Individual Counseling vs. Routine Primary Care

Measures:
1. BAI - Beck Anxiety Inventory - Pre
2. SPS - Social Provisions Scale - Pre
3. BDI - Beck Depression Inventory - Pre
4. BAI - Beck Anxiety Inventory - Post
5. SPS - Social Provisions Scale - Post
6. BDI - Beck Depression Inventory - Post
7. BDI - Beck Depression Inventory - FU

Timing of Assessment: [hard to code since they only have graphs]
1. pre
2. post
3. FU

NOTE: Convert all SE into S, then use M and SD for ES.


PPD women, RA using cluster levels

Comparison:
1. CBA vs. PCA (person-centered)
2. CBA vs. Health Visitor TAU
3. PCA vs. Health Visitor TAU

Measures: (starts from pg. 56-66)
1. EPDS - Edinburgh Postnatal Depression Scale - FU 6 mon
2. SF-12 PCS, physical component summary - FU 6 mon
3. SF-12 MCS, mental component summary - FU 6 mon
4. SF-6D - FU 6 mon
5. CORE-OM - well-being - FU 6 mon
6. CORE-OM – risk - FU 6 mon
7. CORE-OM – symptoms - FU 6 mon
8. CORE-OM – functioning - FU 6 mon
9. CORE-OM - total score - FU 6 mon
10. State Trait Anxiety Inventory -state - FU 6 mon
11. State Trait Anxiety Inventory – trait - FU 6 mon
12. PSI - (Parent Stress Inventory) parenting distress - FU 6 mon
13. PSI PCDI (parent–child dysfunctional interaction) - FU 6 mon
14. PSI difficult child - FU 6 mon
15. PSI total stress - FU 6 mon

Timing of Assessment:
1. FU - 6 month


PPD women, RA

Comparison:

1. Interpersonal Psychotherapy (IPT) Group vs. TAU

Measures
1. EPDS - Edinburgh Postnatal Depression Scale - Pre
2. BDI - Beck Depression Inventory - Pre
3. HAM-D - Hamilton Depression Rating Scale - Pre
4. DAS - Dyadic Adjustment Scale - Pre
5. ISEL - Interpersonal Support Evaluation List - Pre
6. MAI - Maternal Attachment Inventory - Pre
7. EPDS - Edinburgh Postnatal Depression Scale - Post
8. BDI - Beck Depression Inventory - Post
9. HAM-D - Hamilton Depression Rating Scale - Post
10. DAS - Dyadic Adjustment Scale - Post
11. ISEL - Interpersonal Support Evaluation List - Post
12. MAI - Maternal Attachment Inventory - Post
13. EPDS - Edinburgh Postnatal Depression Scale – FU
14. BDI - Beck Depression Inventory – FU
15. DAS - Dyadic Adjustment Scale – FU
16. ISEL - Interpersonal Support Evaluation List – FU
17. MAI - Maternal Attachment Inventory – FU

Timing of Assessment
1. Pre
2. Post tx (8 weeks)
3. FU – 3 mon


Note: Will not use Forman study because it splits the IPT group into two groups (recovered and not recovered)

RA, PPD [part of O’Hara 2000 study]

Comparison:
1 - Interpersonal Psychotherapy (IPT) vs. Control
2 - Interpersonal Psychotherapy (IPT) vs. nondepressed group (part of Forman, 2007 study)

Measures:
1 - HRSD - Hamilton Rating Scale for Depression - Pre
2 - BDI - Beck Depression Inventory - Pre
3 - SAS - Social Adjustment Scale - Pre
4 - PostPartum Adjustment Scale - Pre
5 - DAS - Dyadic Adjustment Scale - Pre
6 - HRSD - Hamilton Rating Scale for Depression - Post
7 - BDI - Beck Depression Inventory - Post
8 - SAS - Social Adjustment Scale - Post
9 - PostPartum Adjustment Scale - Post
10 - DAS - Dyadic Adjustment Scale - Post
11 - Maternal responsiveness (Ainsworth Global ratings & Kochanska’s coding system) (part of Forman, 2007 study) - Pre
12 - PSI - Child Domain Total (part of Forman, 2007 study) - Pre
13 - PSI - Parent Domain Total (part of Forman, 2007 study) - Pre
14 - Observed infant negative emotion (part of Forman, 2007 study) - Pre
15 - Observed infant positive emotion (part of Forman, 2007 study) - Pre
16 - Infant Behavior Questionnaire: Infant Negative Emotion (part of Forman, 2007 study) - Pre
17 - Infant Behavior Questionnaire: Infant Positive Emotion (part of Forman, 2007 study) - Pre
18 - Maternal responsiveness (Ainsworth Global ratings & Kochanska’s coding system) (part of Forman, 2007 study) - Post
19 - PSI - Child Domain Total (part of Forman, 2007 study) - Post
20 - PSI - Parent Domain Total (part of Forman, 2007 study) - Post
21 - Observed infant negative emotion (part of Forman, 2007 study) - Post
22 - Observed infant positive emotion (part of Forman, 2007 study) - Post
23 - Infant Behavior Questionnaire: Infant Negative Emotion (part of Forman, 2007 study) - Post
24 - Infant Behavior Questionnaire: Infant Positive Emotion (part of Forman, 2007 study) - Post
25 - Waters’ Attachment Q-set (child’s attachment security) (part of Forman, 2007 study) - FU
26 - Child Behavior Checklist Internalizing Problems (part of Forman, 2007 study) - FU
27 - Child Behavior Questionnaire Extrav/surgency (child’s temperament) (part of Forman, 2007 study) - FU
28 - Child Behavior Questionnaire - Negative Affectivity (child’s temperament) (part of Forman, 2007 study) - FU

Timing of Assessment:
1. pre
2. post tx (12 weeks, 9 months: part of Forman 2007 study)
3. FU (18 months) (part of Forman, 2007 study)


RA, PPD

Comparison:
1 - Nurses trained in Cognitive Behavior Therapy vs. Routine Primary Care
Measures:
1 - EPDS - Edinburgh Postnatal Depression Scale - Pre
2 - MADRS - Montgomery-Asberg Depression Rating Scale - Pre
3 - EPDS - Edinburgh Postnatal Depression Scale - Post
4 - MADRS - Montgomery-Asberg Depression Rating Scale - Post
5 - EPDS - Edinburgh Postnatal Depression Scale - FU
6 - MADRS - Montgomery-Asberg Depression Rating Scale - FU

Timing of Assessment:
1. pre
2. post
3. FU - 6 months


PPD women, RA
Comparison:
1 - Multicomponent intervention (drug psycho ed) vs. Treatment as Usual (TAU)

Measure:
1 - EPDS - Edinburgh Postnatal Depression Scale - Pre
2 - SF-36 short form- mental health - Pre
3 - SF-36 short form- emotional role - Pre
4 - SF-36 short form- social function - Pre
5 - SF-36 short form- vitality - Pre
6 - EPDS - Edinburgh Postnatal Depression Scale - Post
7 - SF-36 short form- mental health - Post
8 - SF-36 short form- emotional role - Post
9 - SF-36 short form- social function - Post
10 - SF-36 short form- vitality - Post
11 - EPDS - Edinburgh Postnatal Depression Scale - FU
12 - SF-36 short form- mental health - FU
13 - SF-36 short form- emotional role - FU
14 - SF-36 short form- social function - FU
15 - SF-36 short form- vitality – FU

Timing of Assessment
1. Pre (baseline)
2. post tx (3 months, 1 month after tx)
3. FU (@6 months, but 3 mon after tx)

Notes: *pg. 1631, “women in this group received medical appts for weeks 2 and 4 and subsequently every month for the first 6 months.” But 8 weeks of group therapy

RA, PPD ** Camile move this to RA section, after you code it.
Comparison:
  1. Gruen Group Therapy vs. Control
Measures:
  1 - BDI - Beck Depression Inventory – Pre
  2 - BDI - Beck Depression Inventory - Post
Timing of Assessment:
  1. pre
  2. post (10 weeks)

Meds VS Meds
PPD women, RA
Not in English

RA, PPD

Similar Treatment Comparison
Comparison:
  1. omega-3 fatty acid 0.5g/day vs. omega-3 fatty acid 1.4g/day
  2. omega-3 fatty acid 0.5g/day vs. omega-3 fatty acid 2.8g/day
  3. omega-3 fatty acid 1.4g/day vs. omega-3 fatty acid 2.8g/day
** remember to convert g to mg!

Measures:
  1 - EPDS - Edinburgh Postnatal Depression Scale - Pre
  2 - HRSD - Hamilton Rating Scale for Depression - Pre
  3 - EPDS - Edinburgh Postnatal Depression Scale - Post
  4 - HRSD - Hamilton Rating Scale for Depression - Post

Timing of Assessment:
  1. pre
  2. post (8 weeks)

Note: All not significant (pg. 34), use #0603, to code ES.

PPD women, RA
Comparison:
1. 17Beta-oestradiol (transdermal oestrogen) vs placebo

Measures:
1. EPDS - Edinburgh Postnatal Depression Scale - Pre
2. SADS - Schedule for Affective Disorders and Schizophrenia - Pre
3. EPDS - Edinburgh Postnatal Depression Scale - Post
4. SADS - Schedule for Affective Disorders and Schizophrenia - Post

Timing of Assessment:
1. pre (baseline scores)
2. post (6 months)

Note: EPDS post ES were measured by ruler from chart to get M and solving for SE to get SD, SADS use p-value of .02 for #0601.


Not strict PPD population, RA

Comparison:
1. Norethisterone (synthetic progestogen) vs. placebo

Measures:
1. MADRS - Montgomery-Asberg Depression Rating Scale - Pre
2. EPDS - Edinburgh Postnatal Depression Scale - Pre
3. MADRS - Montgomery-Asberg Depression Rating Scale - Post
4. EPDS - Edinburgh Postnatal Depression Scale - Post
5. MADRS - Montgomery-Asberg Depression Rating Scale - FU
6. EPDS - Edinburgh Postnatal Depression Scale - FU

Timing of Assessment:
1. pre
2. post tx (6 week)
3. FU - 3 month

Note: no length of tx, only a one-time intervention, 48 hours after delivery, not strict PPD population.


RA, pregnant mothers

Comparison:
1. Omega-3 Fatty acids vs Placebo

Measures:
1. HAM-D - Hamilton Depression Rating Scale - Pre
2. EPDS - Edinburgh Postnatal Depression Scale - Pre
Timing of Assessment:
1. pre
2. post (8 weeks)

Note: pregnant mothers, not PPD mothers

PPD women, RA

Similar Treatment Comparison

Comparison:
1. Sertraline vs Nortriptyline

Measures:
1 - HRSD - 17-item Hamilton Rating Scale for Depression - Post
2 - GAS - Global Assessment Scale- Functioning - Post
3 - HRSD - 17-item Hamilton Rating Scale for Depression - FU
4 - GAS - Global Assessment Scale- Functioning - FU

Timing of Assessment
2. post tx (8 weeks), pg. 358
3. FU (20-24 weeks)

Note: time to response, is not a primary outcome interest

PPD women, RA

Comparison:
1. Paroextine vs Placebo

Measures:
1 - IDS-SR - Inventory of Depressive Symptomatology–Self-Report - Pre
2. HAMD - 17-item Hamilton Rating Scale for Depression - Pre
3. CGI-S - Clinical Global Impressions- Severity of Illness - Pre
4. IDS-SR - Inventory of Depressive Symptomatology–Self-Report - Post
5. HAMD - 17-item Hamilton Rating Scale for Depression - Post
6. CGI-S - Clinical Global Impressions- Severity of Illness - Post

Timing of Assessment:
1. pre
2. post

**Alternative**

PPD, women, RA
Comparison:
1. exercise and social support vs. control

Measures:
1. EPDS - Edinburgh Postnatal Depression Score - Pre
2. DASS - Depression Anxiety Stress Scale - Pre
3. GHQ-12 - General Health Questionnaire - Pre
4. SSI - Social Support Interview - Pre
5. EPDS - Edinburgh Postnatal Depression Score - Post
6. DASS - Depression Anxiety Stress Scale - Post
7. GHQ-12 - General Health Questionnaire - Post
8. SSI - Social Support Interview - Post

Timing of Assessment:
1. pre
2. post tx (12 weeks)

Note: pg. 134, just over half of the mothers were taking medication and only a limited number were receiving counseling.


PPD women, RA
Comparison:
1. Pram-walking group vs Social Support group

Measures:
1. EPDS - Edinburgh Postnatal Depression Scale - Pre
2. VO2max - maximum volume of oxygen consumption - Pre
3. SSI - Social Support Interview - Pre
4. EPDS - Edinburgh Postnatal Depression Scale - Post
5. VO2max - maximum volume of oxygen consumption - Post
6. SSI - Social Support Interview - Post

Timing of Assessment:
1. pre
2. post tx (12 weeks)
Note: pg. 183, half of the participants also received counseling and were taking antidepressants


PPD women, RA
Comparison:
1. bright light vs. dim red light

Measures:
1. SIGH-SAD - 29-item Structured Interview Guide for the Hamilton Depression Rating Scale-Seasonal Affective Disorder Version - Pre
2. EPDS - Edinburgh Postnatal Depression Scale - Pre
3. CGI - Clinical Global Improvement - Pre
4. CGI-S - Clinical Global Impressions - Severity of Illness SPAQ - Seasonal Pattern Assessment Questionnaire - Pre
5. SIGH-SAD - 29-item Structured Interview Guide for the Hamilton Depression Rating Scale-Seasonal Affective Disorder Version - Post
6. EPDS - Edinburgh Postnatal Depression Scale - Post
7. CGI - Clinical Global Improvement - Post
8. CGI-S - Clinical Global Impressions - Severity of Illness SPAQ - Seasonal Pattern Assessment Questionnaire - Post

Timing of Assessment:
1. pre
2. post tx (week 6)

Note: not treatment study


PPD women, RA
Comparison:
1. Home Exercise program vs Treatment as Usual (TAU)

Timing of Assessment:
1. pre
2. post tx (3 months)
3. FU (6 months)

Measures:
1. EPDS - Edinburgh Postnatal Depression Scale - Pre [from 2009]
2. HAM-D - Hamilton Rating Scale for Depression - Pre [from 2009]
3. EPDS - Edinburgh Postnatal Depression Scale - Post
4. HAM-D - Hamilton Depression Rating Scale - Post
5. MFI-20 - Multidimensional Fatigue inventory - Physical Fatigue - Pre
6. MFI-20 Mental Fatigue - Pre
7. MFI-20 General Fatigue - Pre
8. MFI-20 Reduced Motivation - Pre
9. MFI-20 Reduced Activity - Pre
10. MFI-20 - Multidimensional Fatigue inventory - Physical Fatigue - Post
11. MFI-20 Mental Fatigue - Post
12. MFI-20 General Fatigue - Post
13. MFI-20 Reduced Motivation - Post
14. MFI-20 Reduced Activity - Post
15. MFI-20 - Multidimensional Fatigue inventory - Physical Fatigue - FU
16. MFI-20 Mental Fatigue - FU
17. MFI-20 General Fatigue - FU
18. MFI-20 Reduced Motivation - FU
19. MFI-20 Reduced Activity - FU

Timing of Assessment:
1. pre
2. post
3. FU - 3 months

Note: For measures 3-4, use means from graph (pg. 195), and pretest SD scores from (pg. 194)
For measures 10-19, calculate SD by using CI and then use #0402 and enter change mean.
On 8/25/11, I emailed Dr. DaCosta and Dritsa for SD for EPDS and HAMD post and FU
measures.

Depression, Maternal Attitudes and Behavior in New Mothers. Journal of Child Psychology and
pg. 687, recruits PPD and nondepressed people to be in the same groups?
Note: not treatment study, prevention

effects on depressed adolescent mothers. Adolescence, 31, 903 - 911.**Move to RA.

Comparison:
1. Massage Therapy vs. Relaxation Therapy

Measures:
1. POMS - Profile of Mood States - Post
2. State Anxiety Scale - Post
3. Behavior Observation Scale – state - Post
4. Behavior Observation Scale – affect - Post
5 - Behavior Observation Scale – activity - Post
6 - Behavior Observation Scale – vocalization - Post
7 - Behavior Observation Scale – anxiety - Post
8 - Behavior Observation Scale – cooperation - Post
9 - Behavior Observation Scale – fidgetiness - Post

Timing of Assessment:
2 - post at 5 weeks
Note: In ES, only code for measures 3-9, using significant or non-significant #0602 or 0603.


RA, PPD (pg. 62)
Comparison:
   1. Exercise and Social Support Group vs. Treatment as Usual (TAU)

Measures:
1 - EPDS - Edinburgh Postnatal Depression Scale – Pre
2 - EPDS - Edinburgh Postnatal Depression Scale - Post

Timing of Assessment:
   1. pre
   2. post (5 months)


PPD Women, RA
Comparison:
   1. Counseling by health visitor vs. Control

Measures:
1 - Goldberg’s standardized psychiatric interview (total score) - Pre
2 - EPDS - Edinburgh Postnatal Depression Scale – Pre
3 - Goldberg’s standardized psychiatric interview (observed depression) - Pre
4 - Goldberg’s standardized psychiatric interview - Post
5 - EPDS - Edinburgh Postnatal Depression Scale – Post
6 - Goldberg’s standardized psychiatric interview (observed depression) – Post
7 – DSM depressive criteria - Post

Timing of Assessment:
   1. pre (time 1)
   2. post (time 2)
Note: will use p-values to calculate ES, #0601
Note: Not a PPD population


PPD women, RA

**Similar Treatment Comparison**

Comparison:
1. Partner Support Group vs. Control (All therapy sessions alone)

Measures:
1. EPDS - Edinburgh Postnatal Depression Scale – Pre
2. Kellner Symptom Questionnaire - Pre
3. DAS – Dyadic Adjustment Scale – Pre
4. PBI – Parental Bonding Instrument – Pre
5. DAS – Dyadic Adjustment Scale, partner report – Pre
6. GHQ-12 - General Health Questionnaire, partner report - Pre
7. PBI – Parental Bonding Instrument, partner report - Pre
8. EPDS - Edinburgh Postnatal Depression Scale – Post
9. Kellner Symptom Questionnaire – Post
10. DAS – Dyadic Adjustment Scale – Post
11. PBI – Parental Bonding Instrument – Post
12. DAS – Dyadic Adjustment Scale, partner report – Post
13. GHQ-12 - General Health Questionnaire, partner report - Post
14. PBI – Parental Bonding Instrument, partner report - Post
15. GHQ-12 - General Health Questionnaire, partner report - Post

Timing of Assessment:
1. Pre
2. Post tx (visit 6)
3. FU (visit 7, 1 month)


RA, PPD women

**EDIT:**

Comparison 1: RA, but Comp 2 &3: NON RA comparison:
1. baby massage vs. support group
2. baby massage vs. control group (non depressed women)
3. support group vs. control group

Measures:
1. EPDS - Edinburgh Postnatal Depression Scale - Pre
2. SSAI - Spielberger State Anxiety Inventory - Pre
3 - ICQ - Infant Characteristics Questionnaire - Pre
4 - ICQ - Maternal Sensitivity in Interaction - Pre
5 - ICQ - Infant Performance in Interaction - Pre
6 - ICQ - Overall Interaction - Pre
7 - EPDS - Edinburgh Postnatal Depression Scale - Post
8 - SSAI - Spielberger State Anxiety Inventory - Post
9 - ICQ - Infant Characteristics Questionnaire - Post
10 - ICQ - Maternal Sensitivity in Interaction - Post
11 - ICQ - Infant Performance in Interaction - Post
12 - ICQ - Overall Interaction - Post
13 - EPDS - Edinburgh Postnatal Depression Scale - FU
14 - SSAI - Spielberger State Anxiety Inventory - FU
15 - ICQ - Infant Characteristics Questionnaire - FU
16 - ICQ - Maternal Sensitivity in Interaction - FU
17 - ICQ - Infant Performance in Interaction - FU
18 - ICQ - Overall Interaction - FU

Timing of Assessment:
1 - pre
2 - post
3 - FU one year


PPD women, RA comparison:
1 - Infant Massage plus support group vs. Support Group

Measures:
1 - EPDS - Edinburgh Postnatal Depression Scale - Post

Timing of Assessment:
2 – post

Note: Emailed V. Glover for M and SD for study on 8/25/11. Calculate post tx, using p-value from pg. 204.


women, RA ( prevention study, reduce PPD sx)


RA, PPD
Comparison:
1. Home Visits by Nurses vs. Treatment as Usual (TAU)

Measures:
1 - EPDS - Edinburgh Postnatal Depression Scale – Post
2 – Quality of Life physical - Post
3 – Quality of Life psychological - Post
4 – Quality of Life social - Post
5 – Quality of Life environmental - Post
6 – Quality of Life global - Post

Timing of Assessment:
1. pre (time 1)
2. post (time 3)

Note: Tried to email Dr. Tamaki on 8/25/11, however it is an incorrect email error. Because of this, only used post measurements.


PPD women, RA
Comparison:
1. Non-directive Counseling (Nurse) vs. Treatment as Usual (TAU)

Measures:
1 - MADRS - Montgomery-Asberg Depression Rating Scale - Pre
2 - DSM - 3 Criteria for Depressive Symptoms - Pre
3 - MADRS - Montgomery-Asberg Depression Rating Scale - Post
4 - DSM - 3 Criteria for Depressive Symptoms - Post

Timing of Assessment:
1. pre (time 1)
2. post (time 2)

Note: use 0201, for measure 2 and 4 using info on pg. 212. For measures 1 & 3, use SD: 11.1.

**Therapy VS Meds**


PPD women, RA
Comparison:
1. Fluoxetine plus 1 session CBT vs. Fluoxetine plus 6 session CBT
2. Fluoxetine plus 1 session CBT vs. Placebo plus 1 session CBT
3. Fluoxetine plus 1 session CBT vs. Placebo plus 6 sessions CBT
4. Fluoxetine plus 6 session CBT vs. Placebo plus 1 session CBT
5. Fluoxetine plus 6 session CBT vs. Placebo plus 6 sessions CBT
6. Placebo plus 1 session CBT vs. Placebo plus 6 sessions CBT

Measures:
1. CIS - Clinical Interview Schedule (for antidepressant trials) - Pre
2. EPDS - Edinburgh Postnatal Depression Scale - Pre
3. HAM-D - Hamilton Depression Rating Scale - Pre
4. CIS - Clinical Interview Schedule (for antidepressant trials) - Post
5. EPDS - Edinburgh Postnatal Depression Scale - Post
6. HAM-D - Hamilton Depression Rating Scale - Post

Timing of Assessment:
1. pre
2. post tx (12 weeks)

Note: Does not include mg amount for fluoxetine and placebo. Sent an email to Louis Appleby on 8/25/11 to ask about dosage. For ES, use intent to treat analysis in [].


PPD women, RA

Comparison:
1. Paroxetine vs. Paroxetine and Cognitive Behavior Therapy

Measures:
1. HAM-D - Hamilton Depression Rating Scale - Pre
2. HAM-A - Hamilton Rating Scale for Anxiety - Pre
3. EPDS - Edinburgh Postnatal Depression Scale - Pre
4. YBOCS - Yale-Brown Obsessive Compulsive Scale - Pre
5. CGI-S - Clinical Global Impressions- Severity of Illness - Pre
6. HAM-D - Hamilton Depression Rating Scale - Post
7. HAM-A - Hamilton Rating Scale for Anxiety - Post
8. EPDS - Edinburgh Postnatal Depression Scale - Post
9. YBOCS - Yale-Brown Obsessive Compulsive Scale - Post
10. CGI-S - Clinical Global Impressions- Severity of Illness - Post
11. PSI - Parenting Stress Index total stress scores [from Misri, 2006] - Pre
12. PSI - Parenting Stress Index total stress scores [from Misri, 2006] – Post
13. PSI - Parenting Stress Index Parent Domain scores [from Misri, 2006] - Pre
14. PSI - Parenting Stress Index Parent Domain scores [from Misri, 2006] - Post
15. PSI - Parenting Stress Index Child Domain scores [from Misri, 2006] - Pre
16. PSI - Parenting Stress Index Child Domain scores [from Misri, 2006] - Post

Note: For measures 11-16, for pretest, use Means from graphs, and SD from table 1. For post test, not significant, #0603.

Timing of Assessment:
1. pre (baseline)
2. post (final)

PPD women with anxiety, RA
Comparison:
1. Paroxetine vs. Paroxetine plus Cognitive Behavior Therapy
Measures:
1 - HAM-D - Hamilton Rating Scale for Depression - Pre
2 - HAM-A - Hamilton Rating Scale for Anxiety - Pre
3 - EPDS - Edinburgh Postnatal Depression Scale - Pre
4 - YBOCS - Yale-Brown Obsessive Compulsive Scale - Pre
5 - CGI – Severity of Illness - Pre
6 - HAM-D - Hamilton Rating Scale for Depression - Post
7 - HAM-A - Hamilton Rating Scale for Anxiety - Post
8 - EPDS - Edinburgh Postnatal Depression Scale - Post
9 - YBOCS - Yale-Brown Obsessive Compulsive Scale - Post
10 - CGI - CGI – Severity of Illness - Post
Timing of Assessment:
3. pre (baseline)
4. post (final)


PPD women, RA
Comparison:
1. Exercise vs. Treatment as Usual (TAU)
Measures:
1 - Godin Leisure-Time Exercise Questionnaire - mild intensity exercise - Pre
2 - Godin Leisure-Time Exercise Questionnaire - moderate intensity exercise - Pre
3 - Godin Leisure-Time Exercise Questionnaire - vigorous intensity exercise - Pre
4 - Self-efficacy for Exercise Questionnaire - Pre
5 - EPDS - Edinburgh Postnatal Depression Scale - Pre
6 - Godin Leisure-Time Exercise Questionnaire - mild intensity exercise - Post
7 - Godin Leisure-Time Exercise Questionnaire - moderate intensity exercise - Post
8 - Godin Leisure-Time Exercise Questionnaire - vigorous intensity exercise - Post
9 - Self-efficacy for Exercise Questionnaire - Post
10 - EPDS - Edinburgh Postnatal Depression Scale - Post
Timing of Assessment:
1. pre
2. post

Note: About 57% of women were taking antidepressants, and 39% were receiving psychological support.


Note: Not a psychotherapy study, drug portion of study was reported in a 2008 study. Note: Results are reported, however unsure if the groups were randomly assigned by status or not.

Comparisons
1 - Omega plus Supportive therapy vs. Placebo plus Supportive Therapy

Measures
1 - HAM-D - Hamilton Rating Scale for Depression - Pre
2 - EPDS - Edinburgh Postnatal Depression Scale - Pre
3 - HAM-D - Hamilton Rating Scale for Depression - Post
4 - EPDS - Edinburgh Postnatal Depression Scale - Post

Timing of Assessment
1 - Pre
2 – Post

Note: For attrition, used stats from g. 146 to calculate.


RA comparison:
1 - SSRI vs. Non-Directive Counseling [clients who felt that nondirective was not working, was allowed to try SSRI]

measures:
1 - EPDS - Edinburgh Postnatal Depression Scale - Pre
2 - CIS- R - Pre
3 - Short Form Health Survey -12 mental component - Pre
4 - Short Form Health Survey -12 physical component – Pre
5 - EuroQol-5D (EQ-5D, measures health outcome, utility) - Pre
6 - EuroQol-5D (EQ-5D, measures health outcome, VAS) - Pre
7 - Maternal adjustment and maternal attitudes (MAMA) – Pre
8 - GRIMS - The Golombok Rust Inventory of Marital State (marital discord) - Pre
9 - EPDS - Edinburgh Postnatal Depression Scale - Post
10 - Short Form Health Survey -12 mental component- Post
11 - Short Form Health Survey -12 physical component – Post
12 - EuroQol-5D (EQ-5D, measures health outcome, utility) - Post
13 - EuroQol-5D (EQ-5D, measures health outcome, VAS) - Post
14 - Maternal adjustment and maternal attitudes (MAMA) – Post
15 - GRIMS - The Golombok Rust Inventory of Marital State (marital discord) – Post
4—EPDS—Edinburgh Postnatal Depression Scale—FU
6—Short Form Health Survey—12 mental component—FU
8—Short Form Health Survey—12 physical component—FU
10—EuroQol 5D (EQ-5D, measures health outcome, utility)—FU
12—EuroQol 5D (EQ-5D, measures health outcome, VAS)—FU
14—Maternal adjustment and maternal attitudes (MAMA)—FU
16—GRIMS—The Golombok Rust Inventory of Marital State (marital discord)—FU

timing of assessment:
1 - pre
2 - post: 4 weeks
3 – FU: 18 weeks

NOTE; Only used data up to 4 weeks because it was a two-arm randomized trial.

ALL Not Randomized Studies

Therapy VS Therapy


Treatment vs Treatment

Comparison:
1. Individual Therapy (psychodynamic principles) vs. Group Therapy (gestalt, psychodynamic principles, or art therapy) - found on pg 210

Measures:
1 - EPDS - Edinburgh Postnatal Depression Scale - Pre
2 - GRIMS - The Golombok Rust Inventory of Marital State - Pre
3 - PSE - Parenting self-efficacy - Pre
4 - PSI - Parenting Stress Index - Pre
5 - EPDS - Edinburgh Postnatal Depression Scale – Post
6 - GRIMS - The Golombok Rust Inventory of Marital State – Post
7 - PSE - Parenting self-efficacy – Post
8 - PSI - Parenting Stress Index – Post

Timing of Assessment:
1. pre
2. FU (6 months)

Note: median scores, use Mann-Whitney U-test as p-values. Also 19 patient were also taking anti depressants (pg. 217)


Comparison:
1. Counseling vs. Control
Measures:
1. AKUADS - Aga Khan University Anxiety and Depression Scale – Pre
2. AKUADS - Aga Khan University Anxiety and Depression Scale - Post

Timing of Assessment:
1. pre (when identified)
2. post (after 8th week)


Comparison:
1. Specialized psychiatric day hospital vs. Routine Primary Care

Measures:
1. EPDS - Edinburgh Postnatal Depression Scale – Pre
2. CIS - Clinical Interview Schedule – Pre
3. Anxiety Subscale of the Hospital Anxiety and Depression Scale – Pre
4. DAS - Dyadic Adjustment Scale – Pre
5. WLFLQ–M - Work Leisure and Family Life Questionnaire–Modified – Pre
6. EPDS - Edinburgh Postnatal Depression Scale - Post
7. CIS - Clinical Interview Schedule - Post
8. Anxiety Subscale of the Hospital Anxiety and Depression Scale - Post
9. DAS - Dyadic Adjustment Scale - Post
10. WLFLQ–M - Work Leisure and Family Life Questionnaire–Modified - Post

Timing of Assessment:
1. pre
2. post (6 months)

Note: make a note of meds use for both groups, pg. 146


PPD women, not RA

Comparison:
1. Nomifensine vs. Control (Nondepressed)

Measures:
1. HRSD - Hamilton Rating Scale for Depression – Post

Assessment:
1. Pre
2. Post

PPD, NONRA [use second part of study] b/c the prevention part is RA, but the PPD intervention is selected from the given groups and is NONR.

Comparison:
23. Home visit Intervention vs. Control

Measures:
1 - BDI - Beck Depression Inventory – Pre
2 - BDI - Beck Depression Inventory - Post
3 - HRSD - 17-item Hamilton Rating Scale for Depression - Pre
4 - HRSD - 17-item Hamilton Rating Scale for Depression - Post
5 - EPDS - Edinburgh Postnatal Depression Scale – Pre
6 - EPDS - Edinburgh Postnatal Depression Scale - Post

Timing of assessment:
9. pre
10. post

Note: received a prevention intervention before coming to this study


Comparison:
1. Mother-infant therapy group (similar to IPT) vs. Control (wait list group)

Measures:
1 - BDI - Beck Depression Inventory - Post
2 - PSI - Child Adaptability - Post
3 - PSI - Child Acceptability - Post
4 - PSI - Child Demandingness - Post
5 - PSI - Child Mood - Post
6 - PSI - Child Distractibility/Hyperactivity - Post
7 - PSI - Child Reinforces - Post
8 - PSI - Child Domain Total - Post
9 - PSI - Parent Depression - Post
10 - PSI - Parent Attachment - Post
11 - PSI - Parent Restricted Role - Post
12 - PSI - Parent Sense of Competence - Post
13 - PSI - Parent Social Isolation - Post
14 - PSI - Spousal Relationship - Post
15 - PSI - Parent Health - Post
16 - PSI - Parent Life Stress - Post
17 - PSI - Parent Domain Total - Post
18 - PSI - Total Stress - Post
19 - PCERA - Parent-Child Early Relational Assessment - Factor 1 - Post
20 - PCERA- factor 2 - Post
21 - PCERA- factor 3 - Post
22 - PCERA- factor 4 - Post
23 - PCERA- factor 5 - Post
24 - PCERA- factor 6 - Post
25 - PCERA- factor 7 - Post
26 - PCERA- factor 8 - Post
27 - Mental Development Index of the Bayley Scales (assessed infant cognitive development) - Post

Timing of Assessment:
1. post (pg. 529, using p, since M/SD is marginal)


Non RA, PPD women comparison:
1 - Nurse Counseling vs. Treatment as Usual

Measures:
1 - EPDS - Edinburgh Postnatal Depression Scale - Pre
2 - EPDS - Edinburgh Postnatal Depression Scale – Post
3 - EPDS - Edinburgh Postnatal Depression Scale – FU 6 mon
4 - EPDS - Edinburgh Postnatal Depression Scale – FU 12 mon
5 - PSI - Child Adaptability – FU 12 mon
6 - PSI - Child Acceptability – FU 12 mon
7 - PSI - Child Demandingness – FU 12 mon
8 - PSI - Child Mood – FU 12 mon
9 - PSI - Child Distractibility/Hyperactivity – FU 12 mon
10 - PSI - Reinforces Child – FU 12 mon
11 - PSI - Child Domain Total – FU 12 mon
12 - PSI - Parent Depression – FU 12 mon
13 - PSI - Parent Attachment – FU 12 mon
14 - PSI - Parent Restricted Role – FU 12 mon
15 - PSI - Parent Sense of Competence – FU 12 mon
16 - PSI - Parent Social Isolation – FU 12 mon
17 - PSI - Spousal Relationship – FU 12 mon
18 - PSI - Parent Health – FU 12 mon
19 - PSI - Total Stress – FU 12 mon
20 - PSI - Parent Life Stress – FU 12 mon
21 - PSI – Parent Domain Total - FU 12 mon

Timing of Assessment:
1 – Pre : baseline
2 - Post: 3 months
3 - FU: 6 months
3 - FU: 12 months

Note: for measures 1-3 use CI to calculate SD with excel sheet. For measure 4, use p-value. For remaining measure, use table 3.
Therapy VS Meds


Non RA, PPD
1 – Treatment (CBT, support counseling, medications) vs. wait-list control

Measures:
1 - EPDS - Edinburgh Postnatal Depression Scale – Post
2 - State Trait Anxiety Inventory – Post
3 - GHQ - General Health Questionnaire – Post

Timing of Assessment:
2 - post

Note: pg. 214, “half of subjects were already taking antidepressants medication”. Results did not specify how many clients were in each specific condition without overlapping it with other conditions. Since results are confusing, was only able to product post-test results comparing any-type of tx to wait list. Use p-values on pg. 214.


Comparison:
1. Interpersonal Psychotherapy (IPT) vs. Sertaline (12 weeks)
2. Interpersonal Psychotherapy (IPT) vs. Interpersonal Psychotherapy (IPT) plus Sertraline
3. Sertaline vs. Interpersonal Psychotherapy (IPT) plus Sertraline

Measures:
1 - HRSD - Hamilton Rating Scale for Depression – Pre
2 - BDI - Beck Depression Inventory – Pre
3 - EPDS - Edinburgh Postnatal Depression Scale – Pre
4 - HRSD - Hamilton Rating Scale for Depression - Post
5 - BDI - Beck Depression Inventory - Post
6 - EPDS - Edinburgh Postnatal Depression Scale - Post

Timing of Assessment:
1. pre
2. post

**ALL Pre and Post Studies**

**make a separate ES file for pre/post ES i.e. “Sarah 2 pre-post”**

When calculating FU ES, you compare FU with pre M and SD.

Therapy VS Therapy

Treatment:
1. 17beta-estradiol

Measures:
1. MADRS - Montgomery-Asberg Depression Rating Scale - Post

Timing of Assessment:
1. pre & post (8 weeks)


Treatment: In-Home Cognitive-Behavior Therapy

Measures:
1. PRIME-MD Primary Care Evaluation of Mental Disorders - Post
2. BDI - Beck Depression Inventory - Post
3. MAQ - Maternal Attitudes Questionnaire – Post

Timing of Assessment:
1. pre & post (calculate: post-pre)

Note. Pg. 8, there are 2 pregnant mothers in the study.


PPD Women, pre-post

Treatment:
1. 9-week cognitive-behavioral group

Measures
1. EPDS - Edinburgh Postnatal Depression Scale - Post
2. HADS-A - Post
3. HADS-D – Post
4. EPDS - Edinburgh Postnatal Depression Scale – FU 6 wk
5. HADS-A - FU 6 wk
6. HADS-D - FU 6 wk
7. EPDS - Edinburgh Postnatal Depression Scale – FU 3 mon
8. HADS-A - FU 3 mon
9. HADS-D - FU 3 mon

Timing of Assessment
1. pre & post
2. FU – 6 week
   2. FU- 3 mon

Treatment:
1 - relational developmental therapy group (psychodynamic therapy)

Measures:
1 - GAS - Global Assessment Scale- Functioning – Post

Timing of Assessment:
1. pre & post (calculate: post-pre)


Treatment:
1 - group therapy

Measures:
1 - BDI - Beck Depression Inventory – Post

Timing of Assessment:
1. Pre & Post (calculate: post-pre)


Treatment:
1 - IPT group

Measures:
1 - EPDS - Edinburgh Postnatal Depression Scale - Post
2 - HRSD - 21-item Hamilton Rating Scale for Depression - Post
3 - DAS - Dyadic Adjustment Scale - Post
4 - IIP - Inventory of Interpersonal Problems - Post
5 - EPDS - Edinburgh Postnatal Depression Scale - FU
6 - HRSD - 21-item Hamilton Rating Scale for Depression - FU
7 - DAS - Dyadic Adjustment Scale - FU
8 - IIP - Inventory of Interpersonal Problems - FU

Timing of Assessment:
1. Pre & post
2. FU - 6 months (pg. 127, figure FU from baseline using p-value)


Treatment:
1 - IPT group

Measures:
1 - BDI - Beck Depression Inventory - Post
2 - EPDS - Edinburgh Postnatal Depression Scale - Post
3 - SAS - Social Adjustment Scale - Post
4 - BDI - Beck Depression Inventory - FU
5 - EPDS - Edinburgh Postnatal Depression Scale - FU
6 - SAS - Social Adjustment Scale - FU

Timing of Assessment:
1. Pre & post (8 weeks)
2. FU (3 months)

Note: 66.6% were on medications, pg. 35


Treatment:
1 - modified IPT

Measures:
1 - HRSD - Hamilton Rating Scale for Depression - Post
2 - BDI - Beck Depression Inventory - Post
3 - EPDS - Edinburgh Postnatal Depression Scale - Post
4 - SAS - Social Adjustment Scale total - Post
5 - SAS - Social Adjustment Scale marital - Post

Timing of Assessment:
1. Pre (intake) & post (termination, 12 weeks)

**Meds VS Meds**


Treatment:
1 - Bupropion SR

Measures:
1 - HAM-D - Hamilton Depression Rating Scale - Post
2 - CGI - Clinical Global Improvement - Post
3 - Kellner Symptom Questionnaire Depression - Post

Timing of Assessment:
1. Pre & post

Note: use p-values


Treatment:
1 - sertraline
Measures:
1 - SIGH-D - Structured interview guide for the Hamilton Depression Rating Scale for Depression - Post
Timing of Assessment:
   pre & post


Treatment:
   1 - Fluvoxamine

Measures:
1 - HRSD - 21-item Hamilton Rating Scale for Depression – Post

Timing of Assessment:
1. Pre & post (use p-values)


Treatment:
   1 - Nefazodone

Measures:
1 - HAM-A - Hamilton Anxiety Rating Scale - Post
2 - HAM-D - Hamilton Depression Rating Scale - Post

Timing of Assessment:
1. pre & post (use p-values)

Alternative


Treatment:
   1 - Repetitive TMS

Measures:
1 - HRSD - 24-item Hamilton Rating Scale for Depression - Post
2 - EPDS - Edinburgh Postnatal Depression Scale - Post
3 - IDS-SR - Inventory of Depressive Symptomatology–Self-Report - Post
4 - CGI-S - Clinical Global Impressions- Severity of Illness - Post
5 - HRSD - 24-item Hamilton Rating Scale for Depression - FU
6 - EPDS - Edinburgh Postnatal Depression Scale - FU
7 - IDS-SR - Inventory of Depressive Symptomatology–Self-Report - FU
8 - CGI-S - Clinical Global Impressions- Severity of Illness - FU
Timing of Assessment:
1. pre & post (4 week)
2. FU (6 months)
Note: only study, will compute ES, but nothing to compare it with


Treatment:
1 - telecare by nursing students

Measures:
1 - BDI - Beck Depression Inventory – Post

Timing of Assessment:
1. Pre & post (after 10 weeks)
Appendix C

Measure Level Codes
Overall Measure ID #

1. Depression

BDI - Beck Depression Inventory -7  
CES-D - Center for Epidemiological Studies Depression Scale -19  
CIS - Clinical Interview Schedule of Mental Health -24  
DSM - 3 Criteria for Depressive Symptoms -39  
EPDS - Edinburgh Postnatal Depression Scale -47  
HADS-D -58  
HAM-D - Hamilton Depression Rating Scale -60  
HRSD - 17-item Hamilton Rating Scale for Depression -61  
HRSD - 21-item Hamilton Rating Scale for Depression -62  
HRSD - 24-item Hamilton Rating Scale for Depression -63  
HRSD - Hamilton Rating Scale for Depression -64  
IDS-SR - Inventory of Depressive Symptomatology–Self-Report -71  
Kellner Symptom Questionnaire Depression -84  
MADRS - Montgomery-Asberg Depression Rating Scale -85  
PostPartum Adjustment Scale -117  
POMS Depression (profile mood of states) -114  
PRIME-MD Primary Care Evaluation of Mental Disorders -119  
SIGH-D - Structured interview guide for the Hamilton Depression Rating Scale for Depression -155  
SIGH-SAD - 29-item Structured Interview Guide for the Hamilton Depression Rating Scale-Seasonal Affective Disorder Version -156  
148 - SCID - Structured Clinical Interview for DSM III Diagnosis

2. Anxiety

BAI - Beck Anxiety Inventory -6  
HADS-A -57  
HAM-A - Hamilton Rating Scale for Anxiety -59  
Kellner Symptom Questionnaire Anxiety -83  
SSAI - Spielberger State Anxiety Inventory - 169  
State Trait Anxiety Inventory -175  
State Trait Anxiety Inventory – State -190  
State Trait Anxiety Inventory – Trait -191  
State Anxiety Scale -172  
WCCR - Ways of Coping Checklist Revised (cope w/ stress/anxiety) -178  
YBOCS - Yale-Brown Obsessive Compulsive Scale -180/181  
WLFLQ–M - Work Leisure and Family Life Questionnaire–Modified (adjustment to the baby) -179
3. Depression and Anxiety/ Mood

AKUADS - Aga Khan University Anxiety and Depression Scale -2
Anxiety Subscale of the Hospital Anxiety and Depression Scale -3
DASS - Depression Anxiety Stress Scale -27
Goldberg’s standardized psychiatric interview (measures anxiety, depression, irritability, etc) -54
Kellner Symptom Questionnaire -82
POMS - Profile of Mood State -112
POMS confusion (profile of mood states) -113
POMS fatigue -115
POMS tension - Profile of Mood States -116
Rosenberg Self-esteem Scale (SES) -196
SEI - Self - Esteem Inventory -149
CORE-OM - well-being (similar to SCL-90, measures sx distress) -185
CORE-OM – risk -186
CORE-OM – symptoms -187
CORE-OM – functioning -188
Quality of Life psychological -206
146 - SADS - Schedule for Affective Disorders and Schizophrenia

4. Physiological / Health

EQ-5D Utility -202
EQ-5D VAS -203
GHQ - General Health Questionnaire, partner report -49
GHQ-12 - General Health Questionnaire -50
Godin Leisure-Time Exercise Questionnaire - mild intensity exercise -51
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