THE DEVELOPMENT, TREATMENT, AND PREVENTION OF PERINATAL MOOD AND ANXIETY DISORDERS

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A DISSERTATION

in

Psychology

Presented to the Faculties of the University of Pennsylvania in

Partial Fulfillment of the Requirements for the Degree of Doctor of Philosophy

2013

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ACKNOWLEDGEMENTS

I imagine that there are many similarities between writing a dissertation and having a child. Foremost among these is the need for social support along the way – both instrumental and emotional! I have been lucky to have an amazing support network throughout graduate school.

The quality of my work has been immeasurably improved by the strong academic support I have received. First and foremost I am indebted to my advisor, Dr. Jacques P. Barber, for noticing a graduate applicant whose research interests had nothing to do with psychotherapy process research and saying, "I'm interested in your ideas." Thank you for accommodating my night-owl schedule and being available to answer questions from across the world. Dr. Angela Duckworth was an inspiring and involved committee chair. Thank you for introducing me to calendars and making me stick to mine. Dr. Robert J. DeRubeis could always be counted on for challenging philosophical and statistical questions that pushed me to take my work to the next level. Fortuitously, Dr. C. Neill Epperson came to the University of Pennsylvania at exactly the point in my academic career when I needed her expertise. Thank you for taking me under your wing!

I am also profoundly grateful for the support I have received from the clinical program and from my clinical supervisors throughout graduate school. Thank you for pushing me to be a true clinical scientist – one whose practice is informed by research and whose research is informed by practice. Thank you to Dr. Dianne Chambless and Dr. Melissa Hunt for being wonderful mentors. Thank you to Dr. Alan Goldstein, Dr. Jodi

Rosenfeld, Dr. Linda Welsh, Dr. Jon Grayson, Harold Kirby, Dr. Nicholas Forand, Dr. Judith Coché, and Dr. Lindsay Sortor for being excellent supervisors.

My fellow graduate students provided the bulk of the emotional support – as well as some key practical advice along the way. Thank you especially to Eli Tsukayama, whose willingness to exchange statistical knowledge for food was of great benefit during the dissertation process. Thank you to my officemates, Emily Gentes, Lauren Hallion, and Dahlia Mukherjee, for telling me to get back to work or out of the office – whichever was necessary at the time. The graduate students who paved the way before me gave me hope that I really would finish the dissertation someday – thank you especially to Hilary Dingfelder, Jay Fournier, Julian Lim, Jared Minkel, Acacia Parks, Stephen Schueller, Christian Webb, and Alyson Zalta,. My ability to spend long hours in my office was considerably supported by the many friends who would stop by for gossip and chocolate throughout the day. Thanks to Pavel Atanasov, Nick Hindy, Noah Snyder-Mackler, Marc Coutanche, Marie Forgeard, Anna Rudo-Hutt, Irena Ilieva, Adrienne Scutellaro, Moriah Brier, Betty Kim, Allison Nahmias, Ann Marie Roepke, Alejandro Adler, Zachary Cohen, Gwen Lawson, Lorenzo Lorenzo-Luaces, Eliora Porter, and Kelly Allred. Remember to keep the candy bowl full, you never know when someone will need it!

Thank you to my family for your willingness to support me in all of my endeavors – no matter how incomprehensible they seemed. Thank you to my father, Stuart Sockol, for keeping my office full of strange dollar-store finds. Thank you to my mother, Janet Basta, and my stepfather, Frank Basta, for doing my laundry when my dissertation got in the way of ADLs. Thanks to my siblings, Edward Sockol, Chelsea Basta, Mackenzie

Basta, and Nicole Basta, for always being there when I needed a distraction. I would also like to take this opportunity to remind you that, as the eldest, I have perpetual dibs on shotgun. Thank you to my grandmothers, Frances Flood, Mary Ann Hynd, and Libby Sockol, and to my grandfather Frank Basta, for being wonderful role models.

Finally, I want to thank all of the women whose participation has made this research possible. I am continually surprised by the willingness of total strangers to spend a considerable amount of time telling someone on the other side of a computer screen about their experiences. Participants in my studies have gone above and beyond, contacting me to ask insightful questions about my research and passing on pictures of their beautiful babies. It would not have been possible without them!

ABSTRACT

THE DEVELOPMENT, TREATMENT, AND PREVENTION OF PERINATAL MOOD AND ANXIETY DISORDERS

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Mood and anxiety disorders are common during pregnancy and the postpartum period. The goal of this dissertation was to investigate factors specific to the perinatal period related to the development, treatment, and prevention of depression and anxiety. In Chapter 1, we investigated the role of a risk factor specific to the perinatal period: maternal attitudes. We developed a measure of this construct and used this measure to assess the relationship between these attitudes and symptoms of depression and anxiety among first-time pregnant and postpartum mothers. Dysfunctional maternal attitudes predicted symptoms of depression and anxiety, and these attitudes had incremental predictive validity over general cognitive biases and interpersonal risk factors. In Chapters 2 and 3, we conducted meta-analyses assessing the efficacy of interventions for depression among perinatal populations and investigated whether characteristics of study design and interventions were associated with systematic differences in effect sizes. In Chapter 2, we included 27 studies assessing the efficacy of treatments for depression during pregnancy and the first year postpartum. We found that interventions resulted in significant reductions in depressive symptoms from pre-treatment to post-treatment, and symptom levels at post-treatment were below cutoff levels indicative of clinically significant symptoms. At post-treatment, intervention groups demonstrated significantly

greater reductions in depressive symptoms compared to control groups. In Chapter 3, we included 37 studies assessing the efficacy of preventive interventions for postpartum depression. We found that depressive symptoms at six months postpartum were significantly lower in intervention conditions as compared to control conditions, and there was a significant reduction in the prevalence of depressive episodes in treatment conditions compared to control conditions. These studies further our understanding of the processes that place women at risk for emotional distress in the context of pregnancy and the postpartum period and suggest that a wide range of interventions are effective for treating and preventing depression in this population.

TABLE OF CONTENTS

LIST OF TABLES

Chapter 1

Chapter 2

Chapter 3

LIST OF FIGURES

Chapter 2

Chapter 3

General Introduction

Perinatal depression and anxiety are common psychological disorders with important public health implications. Approximately 10-15% of women experience a depressive episode during pregnancy or the first year postpartum (Bennett, Einarson, Taddio, Koren & Einarson, 2004; Joseffson, Berg, Nordin & Sydsjo, 2001). Anxiety disorders are also common during pregnancy (Lee, Lam, Lau, Cong, Chui, & Fong, 2007) and the first year postpartum (Stuart, Couser, Schilder, O'Hara, & Gorman, 1998; Wenzel, Haugen, Jackson, & Brendle, 2005). While clinicians previously believed that pregnancy was protective against mental illness, we now know that the risk of mental illness is at least comparable between childbearing and non-childbearing women (O'Hara, Zekoski, Philipps, & Wright, 1990), and there is some evidence that women are at increased risk for psychopathology during the perinatal period (Eberhard-Gran, Eskild, Tambs, Samuelsen, & Opjordsmoen, 2002; Eberhard-Gran, Tambs, Opjordsmoen, Skrondal, & Eskild, 2003).

The context in which women with perinatal depression and anxiety experience their symptoms is important to consider in order to fully understand these disorders. The distress experienced by women with these disorders is often exacerbated by feelings of guilt and isolation that accompany women's perceptions that their experiences deviate from cultural norms and expectations (Mauthner, 1999). Clinicians may not identify women experiencing these disorders due to beliefs that pregnancy is protective against mental illness (Cohen et al., 2006). Difficulty distinguishing between normal responses to the stresses of pregnancy and parenting, the "baby blues," and psychopathology can also result in a failure by women and their physicians to identify psychological disorders

during this time. Even when women at-risk for psychological difficulties are identified during pregnancy and the early postpartum period, the majority do not receive treatment for their symptoms (Horowitz & Cousins, 2006).

In addition to the distress experienced by women who experience perinatal depression and anxiety, these disorders confer additional risk on the developing fetus and child. Depression during pregnancy is associated with increased risk for preterm birth, low birth weight, intrauterine growth restriction, and preeclampsia (Grote, Bridge, Gavin, Melville, Iyengar, & Katon, 2010; Kim, Sockol, Sammel, Kelly, Moseley, & Epperson, 2012). Prenatal anxiety is also associated with poor birth outcomes (Littleton, Breitkopf, & Berenson, 2007). Children of depressed and anxious mothers are at increased risk for a wide range of problems. Maternal depression during the first year postpartum is associated with long-term behavioral problems, emotional difficulties, and impaired cognitive development, particularly among boys and children of low socioeconomic status (Grace, Evindar, & Stewart, 2003). Maternal anxiety is also associated with increased risk for behavioral and emotional difficulties (O'Conner, Heron, Glover, & the ALSPAC Study Team, 2002).

Identification of risk factors for perinatal depression and anxiety can help guide researchers and clinicians in identifying women at-risk for these disorders and suggest potential targets for intervention and prevention. Epidemiological research has identified many risk factors for these disorders. A personal or familial history of depression or an anxiety disorder is among the strongest predictors that a woman will experience depression or anxiety during pregnancy or the first year postpartum (C. Beck, 2001;

O'Hara & Swain, 1996; Robertson, Grace, Wallington, & Stewart, 2004). Women who are members of racial/ethnic minorities, single women, and women of low socioeconomic status are at increased risk for depression and anxiety (C. Beck, 2001; Littleton, Breitkopf, & Berenson, 2007; O'Hara & Swain, 1996; Robertson, Grace, Wallington, & Stewart, 2004). Interpersonal stressors, including a lack of social support and low marital satisfaction, are also associated with increased risk (C. Beck, 2001; O'Hara & Swain, 1996; Robertson, Grace, Wallington, & Stewart, 2004). Psychological characteristics associated with increased risk for these disorders include perfectionism and a negative attributional style (C. Beck, 2001; O'Hara & Swain, 1996; Robertson, Grace, Wallington, & Stewart, 2004).

While these risk factors can help clinicians and researchers identify women at-risk for distress during the transition to parenthood, it is important to note that many of these risk factors are challenging targets for intervention. An important goal for research in this area is the identification of risk factors that can serve as targets for prevention and intervention. Beck's cognitive model provides an approach to conceptualizing these disorders that suggests risk factors that may be amenable to intervention. According to this model, the relationship between an individual's experience and his emotional response is mediated by cognitive processes (A. Beck, 1967; 1976; 1985). Maladaptive emotional responses, such as depression and anxiety, result from systematic biases in cognition. Importantly, these biases can be targeted through psychological interventions, particularly cognitive-behavioral therapies (Appleby, Warner, Whitton, & Faragher, 1997; Cooper, Murray, Wilson, & Romaniuk, 2003).

It is also important for research to identify risk factors that may be specific to perinatal psychopathology. There is evidence that a subset of women who are particularly vulnerable to depression and anxiety during the perinatal period are not otherwise at risk for these disorders (Bloch, Schmidt, Danaceau, Murphy, Nieman, & Rubinow, 2000; Cooper & Murray, 1995). Beliefs and attitudes related to motherhood may function as a specific cognitive vulnerability to depression and anxiety in the context of pregnancy and the transition to parenthood. There is evidence that maternal attitudes are associated with poor psychological adjustment for pregnant and postpartum women (Sockol, 2008; Warner, Appleby, Whitton, & Faragher, 1997). However, research in this area has been limited by conceptual and psychometric problems with measures commonly used to assess maternal attitudes (Sockol, 2008).

Identification of women at risk for perinatal depression and anxiety may help clinicians and researchers identify women who would benefit from preventive interventions. Research has investigated the efficacy of a wide range of interventions to reduce the prevalence of psychopathology during the perinatal period. The perinatal period may be a particularly opportune time to initiate preventive interventions, as pregnancy and the early postpartum period are times of increased healthcare utilization and access.

While research suggests that the prevalence of depression and anxiety can be reduced through preventive interventions, high-risk individuals who receive these interventions often develop psychological disorders (Cuijpers, van Straten, Andersson, & Van Oppen, 2008; Zalta, 2011). Given the prevalence and consequences of these

disorders, the identification of effective treatments has important public health implications. Concerns unique to the perinatal period may influence the efficacy of treatments for perinatal women (Kim, O'Reardon, & Epperson, 2010). Due to concerns about fetal exposure to antidepressants, concerns about breastfeeding, and the need for higher doses of medication during pregnancy, medications may be prescribed below therapeutic levels (Bennett, Einarson, Taddio, Koren, & Einarson, 2004; Dawes & Chowienczyk, 2001; Epperson, Anderson, & McDougle, 1997; Epperson, Jatlow, Czarkowski, & Anderson, 2003; Hostetter, Stowe, & Strader, 2000; Wisner, Perel, & Wheeler, 1993). Biological and psychosocial changes that occur in the context of pregnancy and parenting, including sleep deprivation, disruptions to the hormonal milieu, alterations to HPA axis functioning, and changes to interpersonal relationships, introduce challenges that may affect the efficacy of both pharmacological and psychotherapeutic interventions (Dennis & Ross, 2005; Kammerer, Taylor, & Glover, 2006). There are also concerns related to the acceptability of interventions for perinatal psychopathology: the majority of women indicate a preference for psychological interventions to medication during both pregnancy and the postpartum period, and the overall acceptability of pharmacotherapy among these groups is low (Chabrol, Teissedre, Armitage, Danel & Walburg, 2004; Kim et al., 2011). Thus the identification of efficacious interventions, particularly psychological interventions, for this population is an important and growing area of research.

The goals of the studies included in this dissertation are to further our understanding of the development, treatment, and prevention of mental illness during

pregnancy and the first year postpartum. In Chapter 1, we developed a measure for assessing maternal attitudes, a potential risk factor for perinatal depression and anxiety. We then used this measure to assess the predictive validity of maternal attitudes in relation to symptoms of depression and anxiety among first-time mothers, and to investigate the relationship between maternal attitudes and other known risk factors for these disorders. In Chapter 2, we conducted a quantitative review of the literature on the treatment of depression during pregnancy and the first year postpartum. We used meta-analysis to assess the overall effectiveness of interventions for perinatal depression by assessing changes in depressive symptoms over time and the differences between treatment and control conditions in randomized and quasi-randomized trials of interventions for these disorders. We also assessed whether characteristics of studies and interventions were associated with systematic differences in effect sizes. Finally, in Chapter 3, we conducted a quantitative review of the literature on the prevention of postpartum depression. We used meta-analysis to assess whether preventive interventions are associated with decreased levels of depressive symptoms and reduced incidence of depressive episodes during the first six months postpartum. As in Chapter 2, we also assessed whether characteristics of studies and interventions were associated with systematic differences in effect sizes. Overall, these studies further our understanding of the mechanisms by which perinatal depression and anxiety disorders develop and the most effective ways to treat and prevent them.

References

Appleby, L., Warner, R., Whitton, A., & Faragher, B. (1997). A controlled study of fluoxetine and cognitive behavioural counselling in the treatment of postnatal depression. *British Medical Journal, 314,* 932-936.

Beck, A. (1967). *Depression: Clinical, Experimental & Theoretical Aspects.* Philadelphia, PA: University of Pennsylvania Press.

Beck A. (1976). *Cognitive Therapy and the Emotional Disorders.* New York: Penguin.

Beck, A. (1985). *Anxiety Disorders and Phobias: A Cognitive Perspective.* New York: Basic Books.

Beck, C. (2001). Predictors of postpartum depression: An update. *Nursing Research, 50,* 275-285.

Bennett, H., Einarson, A., Taddio, A., Koren, G. & Einarson, T. (2004). Prevalence of depression during pregnancy: Systematic review. *Obstetrics & Gynecology, 103,* 698-709.

Bloch, M., Schmidt, P., Danaceau, M., Murphy, J., Nieman, L. & Rubonow, D. (2000). Effects of gonadal steroids in women with a history of postpartum depression. *American Journal of Psychiatry, 157,* 924-930.

Chabrol, H., Teissedre, F., Armitage, J., Danel, M., & Walburg, V. (2004). Acceptability of psychotherapy and antidepressants for postnatal depression among newly delivered mothers. *Journal of Reproductive and Infant Psychology, 22,* 5-12.

Cohen, L. S., Altshuler, L. L., Harlow, B. L., Nonacs, R., Newport, J., Viguera, A. C., Suri, R., Burt, V. K., Hendrick, V., Reminick, A. M., Loughead, A., Vitonis, A.

F., & Stowe, Z. N. (2006). Relapse of major depression during pregnancy in women who maintain or discontinue antidepressant treatment. *Journal of the American Medical Association, 295.*

Cooper, P. J., & Murray, L. (1995). Course and recurrence of postpartum depression: Evidence for the specificity of the diagnostic concept. *British Journal of Psychiatry, 166,* 191-195.

Cooper, P. J., Murray, L., Wilson, A., & Romaniuk, H. (2003). Controlled trial of the short- and long-term effect of psychological treatment of post-partum depression 1: Impact on maternal mood. *British Journal of Psychiatry, 182*(5), 412-419.

Cuijpers, P., van Straten, A., Andersson, G., & van Oppen, P. (2008). Psychotherapy for depression in adults: A meta-analysis of comparative outcome studies. *Journal of Consulting and Clinical Psychology*, 76, 909-922.

Dawes, M., & Chowienczyk, P.J. (2001). Pharmacokinetics in pregnancy. *Best Practice & Research Clinical Obstetrics & Gynaecology, 15*, 819-826.

Dennis, C.L., & Ross, L. (2005). Relationships among infant sleep patterns, maternal fatigue, and development of depressive symptomatology. Birth, 32, 187-193.

Eberhard-Gran, M., Eskild, A., Tambs, K., Samuelsen, S. O., & Opjordsmoen, S. (2002). Depression in postpartum and non-postpartum women: Prevalence and risk factors. *Acta Psychiatrica Scandinavia*, *106*(6), 426-433.

Eberhard-Gran, M., Tambs, K., Opjordsmoen, S., Skrondal, A., & Eskild, A. (2003). A comparison of anxiety and depressive symptomatology in postpartum and non-

ostpartum mothers. *Social Psychiatry and Psychiatric Epidemiology, 38*(10), 551-556.

Epperson, C.N., Anderson, G.M., & McDougle, C.J. (1997). Sertraline and breast-feeding. *New England Journal of Medicine, 336,* 1189-1190.

Epperson, C.N., Jatlow, P.I., Czarkowski, K., & Anderson, G.M. (2003). Maternal fluoxetine treatment in the postpartum period: Effects on platelet serotonin and plasma drug levels in breastfeeding mother-infant pairs. *Pediatrics, 112,* e425.

Grace, S., Evindar, A. & Stewart, D. (2003). The effect of postpartum depression on child cognitive development and behavior: A review and critical analysis of the literature. *Archives of Women's Mental Health,* 263-274.

Grote, N. K., Bridge, J. A., Gavin, A. R., Melville, J. L., Iyengar, S., & Katon, W. J. (2010). A meta-analysis of depression during pregnancy and the risk of preterm birth, low birth weight, and intrauterine growth restriction. *Archives of General Psychiatry, 67*(10), 1012-1024.

Horowitz, J. A., & Cousins, A. (2006). Postpartum depression treatment rates for at-risk women. *Nursing Research, 55*(2), S23-S27.

Hostetter, A., Stowe, Z.N., Strader, J.R., McLaughlin, E., & Llewellyn, A. (2000). Dose of selective serotonin uptake inhibitors across pregnancy: Clinical implications. *Depression & Anxiety, 11*, 51-57.

Joseffson, A., Berg, G., Nordin, C. & Sydsjo, G. (2001). Prevalence of depressive symptoms in late pregnancy and postpartum. *Acta Obstetrica et Gynecologica Scandinavica,* 80, 251-255.

Kammerer, M., Taylor, A., Glover, V. (2006). The HPA axis and perinatal depression: A hypothesis. *Archives of Women's Mental Health, 9,* 187-196.

Kim, D.R., O'Reardon, J.P., & Epperson, C.N. (2010). Guidelines for the management of depression during pregnancy. *Current Psychiatry Reports, 12,* 279-281.

Kim, D.R., Sockol, L., Barber, J.P., Moseley, M., Lamprou, L., O'Reardon, J.P., & Epperson, C.N. (2011). A survey of patient acceptability of repetitive Transcranial Magnetic Stimulation (TMS) during pregnancy. *Journal of Affective Disorders, 129,* 385-390.

Kim, D. R., Sockol, L. E., Sammel, M., Kelly, C., Moseley, M., & Epperson, C. N. (2012). Elevated risk of preeclampsia in pregnant women with depression. Unpublished manuscript. University of Pennsylvania. Philadelphia, PA.

Lee, A., Lam, S., Lau, S., Chong, C., Chui, H., & Fong, D. (2007). Prevalence, course, and risk factors for antenatal anxiety and depression. *Obstetrics & Gynecology, 110,* 1102-1112.

Littleton, H., Breitkopf, C., & Berenson, A. (2007). Correlates of anxiety symptoms during pregnancy and association with perinatal outcomes: A meta-analysis. *American Journal of Obstetrics & Gynecology, 196,*424-432.

Mauthner, N. S. (1999). Feeling low and feeling really bad about feeling low: Women's experiences of motherhood and postpartum depression. *Canadian Psychology, 40*(2), 143-161.

O'Conner, T., Heron, J., Glover, V., & the ALSPAC Study Team. (2002). Antenatal anxiety predicts child behavioral/emotional problems independently of postnatal

depression. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41, 1470-7.

O'Hara, M. W., & Swain, A. (1996). Rates and risk of postpartum depression: A meta-analysis. *International Review of Psychiatry, 8,* 37-54.

O'Hara, M., Zekoski, E., Philipps, L. & Wright, E. (1990). Controlled prospective study of postpartum mood disorders: Comparison of childbearing and nonchildbearing women. *Journal of Abnormal Psychology, 99,* 3-15.

Robertson, E., Grace, S., Wallington, T., & Stewart, D. (2004). Antenatal risk factors for postpartum depression: a synthesis of recent literature. *General Hospital Psychiatry, 26,* 289-295.

Sockol, L. E. (2008). Predicting depressive symptoms during pregnancy: Integrating cognitive, coping and social perspectives. (Unpublished master's thesis). University of Pennsylvania, Philadelphia, PA.

Stuart, S., Couser, G., Schilder, K., O'Hara, M.W., & Gorman, L.L. (1998). Postpartum anxiety and depression: onset and comorbidity in a community sample. *Journal of Nervous and Mental Disease, 186,* 420-424.

Warner, R., Appleby, L., Whitton, A & Faragher, B. (1997). Attitudes toward motherhood in postnatal depression: Development of the maternal attitudes questionnaire. *Journal of Psychosomatic Research, 43,* 351-358.

Wenzel, A., Haugen, E. N., Jackson, L. C., & Brendle, J. R. (2005). Anxiety disorders at eight weeks postpartum. *Journal of Anxiety Disorders, 19,* 295-311.

Wisner, K.L., Perel, J.M., & Wheeler, S.B. (1993). Tricyclic dose requirements across pregnancy. *American Journal of Psychiatry, 150,* 1541-1542.

Zalta, A. K. (2011). A meta-analysis of anxiety symptom prevention with cognitive-behavioral interventions. *Journal of Anxiety Disorders, 25,* 749-760.

Chapter 1:

The Relationship Between Maternal Attitudes and Symptoms of Depression and Anxiety Among Pregnant and Postpartum First-Time Mothers

Abstract

Two studies examined the role of attitudes toward motherhood in relation to symptoms of depression and anxiety among first-time mothers during pregnancy and the early postpartum period. In the first study, a measure of maternal attitudes, the Attitudes Toward Motherhood Scale (AToM) was developed and validated in a sample of first-time mothers. The AToM was found to have good internal reliability and convergent validity with general cognitive biases and an existing measure of maternal attitudes. Exploratory and confirmatory factor analyses determined that the measure comprises three correlated factors representing beliefs about others' judgments, beliefs about maternal responsibility, and maternal role idealization. In the second study, we used the AToM to assess the relationship between maternal attitudes and other psychological variables among pregnant and postpartum first-time mothers. The factor structure of the measure was confirmed and found to be invariant across pregnant and postpartum subjects. Dysfunctional maternal attitudes predicted symptoms of depression and anxiety, and these attitudes had incremental predictive validity over general cognitive biases and interpersonal risk factors. Dysfunctional maternal attitudes were related to neuroticism but not to other personality factors. Overall, the results of these studies suggest that attitudes toward motherhood are related to psychological distress among first-time mothers during the transition to parenthood and may provide a useful means of identifying women who may benefit from intervention during pregnancy and the early postpartum period.

The Relationship Between Maternal Attitudes and Symptoms of Depression and Anxiety Among Pregnant and Postpartum First-Time Mothers

Emotional distress during the perinatal period is one of the most common complications of childbearing. Approximately 10-15% of women experience a depressive episode during pregnancy or the first year postpartum (Bennett, Einarson, Taddio, Koren & Einarson, 2004; Joseffson, Berg, Nordin & Sydsjo, 2001). High levels of anxiety are also common during pregnancy (Lee, Lam, Lau, Cong, Chui, & Fong, 2007) and the first year postpartum (Stuart, Couser, Schilder, O'Hara, & Gorman, 1998; Wenzel, Haugen, Jackson, & Brendle, 2005).

Depression and anxiety during the perinatal period are associated with adverse fetal and child outcomes. Depression during pregnancy is associated with increased risk for preterm birth, low birth weight, and preeclampsia (Grote, Bridge, Gavin, Melville, Iyengar, & Katon, 2010; Kim, Sockol, Sammel, Kelly, Moseley, & Epperson, 2012). Postpartum depression is a risk factor for a range of adverse child outcomes, including behavioral problems and impaired cognitive development (Grace, Evindar, & Stewart, 2003). Prenatal anxiety is also associated with poor birth outcomes (Littleton, Breitkopf, & Berenson, 2007), and maternal anxiety is a risk factor for behavioral and emotional maladjustment in children, even controlling for the effects of depressive symptoms (O'Conner, Heron, Glover, & the ALSPAC Study Team, 2002). Given the prevalence and potential consequences of perinatal depression and anxiety, research that helps clinicians and researchers effectively identify women at-risk for these disorders and develop effective interventions is vitally necessary.

Epidemiological research has identified a wide range of risk factors for perinatal depression and anxiety. A personal or familial history of major depressive episodes is among the most potent predictors of perinatal depressive symptoms (Beck, 2001; O'Hara & Swain, 1996; Robertson, Grace, Wallington, & Stewart, 2004). Women of low socioeconomic status, ethnic/racial minorities, and single women are also at higher risk for perinatal depression and anxiety (Beck, 2001; Littleton, Breitkopf, & Berenson, 2007; O'Hara & Swain, 1996; Robertson, Grace, Wallington, & Stewart, 2004). While these risk factors can help clinicians and researchers identify women at-risk for distress during the transition to parenthood, many of these risk factors are challenging to modify. An important goal for research in this area is the identification of risk factors that can serve as targets for prevention and intervention.

Beck's cognitive model provides a conceptual framework that may guide us in the identification of vulnerability factors for depression and anxiety that could be targeted for intervention. According to this model, cognitive biases confer a vulnerability to symptoms of depression and anxiety in the context of potentially stressful life events (Beck, 1967; 1976; 1985). According to this model, the relationship between life events and emotional experiences is mediated by cognitive processes. Maladaptive emotional responses, such as depression and anxiety, result from biases in these cognitive processes. In depressed individuals, these biases are commonly characterized by a negative view of the self, world, and future (Beck, 1967; 1976). Among individuals with anxiety disorders, cognitions are frequently characterized by heightened perceptions of threat and danger (Beck, 1985). Previous research has demonstrated that negative cognitive biases are

associated with depression during pregnancy and the postpartum (Cutrona, 1983; Grazioli & Terry, 2000; Hull & Mendolia, 1991; O'Hara, Rehm, & Campbell, 1982). While this construct has received relatively less attention in relation to perinatal anxiety, cognitive biases are also associated with symptoms of anxiety among this population (Littleton, Breitkopf, & Berenson, 2007). Importantly, these biases can be targeted through psychological interventions, particularly cognitive-behavioral therapies (Appleby, Warner, Whitton, & Faragher, 1997; Cooper, Murray, Wilson, & Romaniuk, 2003).

While much research on cognitive vulnerability to depression and anxiety has focused on general negative biases, there is also evidence that specific types of cognitions may interact with particular stressors to produce maladaptive emotional responses. According to this "event congruency hypothesis," an individual's characteristic cognitive style may leave them differentially vulnerable to distress in the context of negative events that are congruent with the important components of their maladaptive schemas (Francis-Raniere, Alloy, & Abramson, 2006; Segal, Shaw, Vella, & Katz, 1992). Most research in this area has investigated the role of self-criticism and dependency, two particular styles of negative cognition, in interaction with life events that are achievement- or interpersonally-oriented. Overall, the results of longitudinal research in this area suggest that individuals who have a self-critical cognitive style are particularly vulnerable to depressive episodes following achievement-oriented stressors, while individuals with a dependent cognitive style are particularly vulnerable to depressive episodes following interpersonally-oriented stressors (Hammen, Marks, Mayos, & deMayo, 1985; Hammen, Ellicott, Gitlin, & Jamison, 1989; Francis-Raniere, Alloy, & Abramson, 2006).

Interestingly, research among perinatal populations suggests that self-criticism is a risk factor for depressive symptoms, while dependency is not associated with increased risk for depression (Besser & Priel, 2003; Besser, Priel, Flett, & Wiznitzer, 2007).

There is also evidence that the specific content of maladaptive cognitions may confer risk for psychopathology in the context of relevant stressors. For example, Schmidt, Lerew, and Jackson (1997) studied whether anxiety sensitivity, a hypothesized cognitive risk factor for panic disorder, predicted the occurrence of panic attacks among young adults enrolled in a five-week basic training program at the Air Force Academy. They found that anxiety sensitivity predicted the probability that recruits would experience a panic attack during this period, even controlling for a history of previous panic attacks. Hillman and Garber (1995) found that cognitions related to academic competence and academic self-control predicted negative affect and depressive symptoms among elementary-school children whose grades were lower than expected. Beliefs specifically related to academic competence and self-control had incremental predictive validity beyond the students' general attributional style. These studies suggest that it may be possible to identify specific maladaptive beliefs that are conceptually related to potential stressors that place an individual at-risk for negative emotional responses.

With regard to perinatal depression and anxiety, beliefs and attitudes about motherhood have the potential to function as a specific cognitive vulnerability. While cognitive biases are a general risk factor for depression and anxiety, attitudes toward motherhood may also mediate the relationship between the specific stressors women

experience during pregnancy and early parenthood and their emotional responses to these events. As such, maternal attitudes would represent an additional and more specific risk factor for symptoms of depression and anxiety among perinatal populations.

There is evidence supporting an association between negative attitudes towards motherhood and depression during pregnancy and the first year postpartum. Women's expectations of motherhood and attitudes toward role conflict predict subsequent depressive symptoms during pregnancy (Warner, Appleby, Whitton, & Faragher, 1997). Women's attitudes toward performance-oriented elements of motherhood are predictive of later depressive symptoms (Grazioli & Terry, 2000). Women's attitudes towards motherhood have also been found to mediate the relationship between parental stress and depressive symptoms (Church, Brechman-Toussaint, & Hine, 2005). Dysfunctional maternal attitudes predict concurrent levels of depressive symptoms during pregnancy and the early postpartum period, and changes in maternal attitudes from pregnancy through six weeks postpartum predict depressive symptoms at six weeks postpartum, even controlling for prior depressive symptoms (Sockol, 2008).

While maternal attitudes appear promising as a means of identifying women at-risk for perinatal depression and anxiety and as a target for intervention, research in this area has been limited by the lack of an adequate measure of this construct. Several measures designed for use by women during pregnancy emphasize women's *expectations* of parenthood (Belsky, 1985; Harwood, 2004; Kalmuss, Davidson, & Cushman, 1992). However, given that pregnancy and parenthood are inherently periods of increased stress, negative expectations may not only be accurate, but may serve a protective function.

Women tend to have overly negative expectations of parenthood, but these negative expectations do not predict psychological maladjustment – rather, women whose actual experiences are more negative than their expectations are at increased risk for postpartum depressive symptoms (Harwood, 2004). Other measures of maternal attitudes likewise confound attitudes toward motherhood with women's *experiences* of pregnancy and parenting (DiPietro, Ghera, Costigan, & Hawkins, 2004; Kumar, Robson, & Smith, 1984; Warner, Appleby, Whitton, & Faragher, 1997). The cognitive models of depression and anxiety emphasize the role of individuals' beliefs in the interpretation of the events they experience, thus it is important that assessments of cognitive biases are careful to distinguish between appraisals of life events and the events themselves.

In addition to conceptual limitations of existing measures of maternal attitudes, our previous research has identified psychometric problems with a commonly used measure of this construct. Specifically, in a previous study of the role of maternal attitudes in predicting perinatal depressive symptoms, we found that the Maternal Attitudes Questionnaire (Warner, Appleby, Whitton, & Faragher, 1997) had poor internal reliability, particularly among subjects who were pregnant with their first child (Sockol, 2008). Examination of the content of the measure reveals items, such as "Having a baby has made me as happy as I expected," that may be confusing or inappropriate for primiparous pregnant women. Given these limitations, the development of a valid and reliable measure of maternal attitudes is necessary for further research assessing their role as a potential risk factor for perinatal depression and anxiety.

The overarching goal of the present research was to develop a measure of maternal attitudes appropriate for use with first-time mothers, both pregnant and postpartum, and to use this measure to assess the relationship between maternal attitudes and emotional distress. We hypothesized that dysfunctional maternal attitudes would predict symptoms of depression and anxiety and have incremental predictive validity over and beyond general cognitive biases and interpersonal risk factors.

Study 1

The goal of Study 1 was to validate a self-report measure of maternal attitudes in a sample of first-time mothers. We conducted exploratory and confirmatory factor analyses to assess the psychometric properties of the measure. We expected that attitudes toward motherhood would comprise several factors reflecting specific types of beliefs related to motherhood. We tested the convergent validity of the Attitudes Toward Motherhood Scale (AToM) with a measure of general cognitive biases (the Dysfunctional Attitudes Scale, DAS; Weissman & Beck, 1978) and an existing but flawed measure of maternal attitudes (the Maternal Attitudes Questionnaire, MAQ; Warner, Appleby, Whitton, & Faragher, 1997). We expected that maternal attitudes as measured by the AToM and MAQ would correlate strongly with one another and that participants' scores on the AToM would also be strongly related to general cognitive biases. Furthermore, we expected the AToM to have superior reliability to the MAQ, particularly among pregnant participants.

Method

Participants and procedures.

Participants for this study were recruited online via social media through sites such as Facebook, Twitter, and online forums for women who are pregnant or parenting (e.g., CafeMom). Specifically, an invitation to participate in a "study of the way women think about motherhood" and a link to an online survey was posted on these sites in the spring of 2011. The survey site included an online consent form followed by eligibility questions, a series of self-report questionnaires and, finally, questions about demographic variables. Participants were compensated via a raffle for $150.

Two hundred thirty-four women initiated participation in the study. Women were eligible to participate if they were between the ages of 18 and 45, resided in the United States, and were either pregnant with their first child (between 13 and 40 weeks gestational age) or had given birth to their first child within the previous 6 months. Three subjects were excluded because they were under age 18, 14 subjects were excluded because they were not pregnant or within six months of giving birth to their first child, and 7 subjects were excluded because they did not reside in the United States. Of the 210 women who were eligible to participate, 65% (n = 136) women completed at least one measure and 50% (n = 104) completed all study measures. We compared women who dropped out at each stage of the study to women who completed the measures; there were no differences between dropouts and completers on any measure.

Demographic characteristics of the sample are presented in Table 1. About 59% of subjects were currently pregnant with their first child (n = 80) and 41% (n = 56) had given birth to their child within the past 6 months. For pregnant subjects, the mean gestational age was 26.1 weeks (SD = 8.3 weeks, range 13-40 weeks). The mean age of

postpartum subjects' children was 12.5 weeks (*SD* = 8.2 weeks, range 1-24 weeks). The sample was predominantly married (65%) and Caucasian (78%) and represented a wide range of socioeconomic backgrounds.

Measures.

Development of the Attitudes Toward Motherhood Scale. We began by generating a pool of 62 items reflecting attitudes toward motherhood. Some items were derived by modifying measures of general cognitive biases to reflect content specific to motherhood (e.g., "Making mistakes caring for my baby is fine because I can learn from them" was modified from the DAS item "Making mistakes is fine because I can learn from them," Weissman & Beck, 1978). We also modified items from a measure of women's expectations of motherhood to reflect beliefs, rather than expectations (e.g., "I should not have difficulty becoming comfortable caring for my baby" was modified from the Parenting Expectations Measure item "I will not have difficulty becoming comfortable caring for my baby," Harwood, 2004). Additional items were derived from a manual of cognitive-behavioral therapy for postpartum depression which listed common maladaptive beliefs expressed by these women (e.g., "Now that I am a mother, my past lifestyle and activities should not be important," Olioff, 1991) and from interviews with women who were pregnant or mothers of children under the age of two years (e.g., "I feel guilty about wanting to do the things I did before I became pregnant"). The set of items proposed for inclusion in the measure was reviewed by a small group of graduate students in clinical psychology who are familiar with the cognitive models of depression and anxiety disorders. Their comments regarding item clarity and wording and additional

suggested items were used to modify the item pool. Finally, the measure was piloted online with a small group of women recruited separately from those in the present study. These women were asked to provide qualitative feedback after completing the measure; these responses were used to refine the measure and to generate additional items.

Dysfunctional Attitudes Scale. General cognitive biases were assessed with the short form of the Dysfunctional Attitude Scale (DAS), a 40-question self-report measure designed to assess the various assumptions and beliefs posited by Beck (1967, 1976) to underlie psychological maladjustment (Weissman & Beck, 1978). Subjects were asked to rate, on a 7-point Likert scale, the degree to which they agree with statements of beliefs or attitudes (e.g., "If I do not do well all the time, people will not respect me"). Higher scores reflect more maladaptive cognitions. Cronbach's alpha in the sample was 0.91 and was comparable for the pregnant ($\alpha = 0.90$) and postpartum ($\alpha = 0.90$) samples.

Maternal Attitudes Questionnaire. Participants also completed an existing measure of maternal attitudes, the Maternal Attitudes Questionnaire (MAQ; Warner et al., 1997). This is a 14-question self-report measure that assesses cognitions in three domains: expectations of motherhood, expectations of the self as a mother, and role conflict (e.g., "I think my baby is very demanding"). Higher scores are indicative of more maladaptive cognitions. As in our previous research, internal reliability for the measure was low ($\alpha = 0.63$), especially among pregnant participants ($\alpha = 0.57$, postpartum sample $\alpha = 0.64$).

Results and Discussion

Of the original 62 items considered for inclusion in the AToM, we initially retained 50% of items representing items with the greatest variance. We then conducted

an exploratory factor analysis on the remaining 31 items. We used several means of assessing the optimal number of factors: Cattell's (1966) scree test, Bartlett's chi-square test (Geweke & Singleton, 1980), parallel analysis (Horn, 1965), and the minimum average partial criterion (Velicer, 1976). These tests all suggested the optimal factor solution would contain 2-4 factors, with a modal solution of 3 factors. We assessed the 2-, 3-, and 4-factor structures for conceptual clarity of extracted factors and the best approximation of simple structure. A three-factor structure with promax rotation satisfied these criteria. An oblique factor rotation was used because the factors were assumed to correlate with one another. Factor analysis was also conducted using an orthogonal (varimax) rotation; the three-factor structure was replicated with this rotation (results not shown).

The initial factor solution produced a 3-factor structure with 6-8 items loading on each factor, for a total of 21 items. Each of these factors was then examined for redundancy and item clarity. To reduce subject burden, we eliminated items with the lowest factor loadings. Items with factor loadings < 0.60 were eliminated. See Table 2 for the 12 retained items. The first factor contains four items that reflect beliefs related to others' judgments, the second factor contains four items reflecting beliefs related to maternal responsibility, and the third factor contains four items reflecting beliefs related to maternal role idealization. Cronbach's alpha in the full sample was 0.81 and was comparable for the pregnant ($\alpha = 0.82$) and postpartum ($\alpha = 0.79$) samples.

To test the integrity of the final three-factor solution, we used confirmatory factor analysis to assess the overall fit of the three-factor model and to compare the three-factor

model to a one-factor model. The three-factor model demonstrated good fit to the data , $\chi^2(51) = 83.3$, $p < 0.01$, SRMR = 0.08, CFI = 0.95, RMSEA = 0.07, $p > 0.05$. Comparison of the three-factor solution to a one-factor solution indicated that the three-factor solution was a significantly better fit to the data, $\chi^2(3) = 233.81$, $p < 0.001$.

Descriptive statistics for the study measures and intercorrelations among the measures are presented in Table 3. We assessed the convergent validity of the AToM using the DAS and MAQ. Due to the low reliability of the MAQ, we corrected the correlations among the measures for attenuation according to Block's (1963) method. The AToM was significantly correlated with both general cognitive biases ($r = 0.50$ after correction for attenuation) and maternal attitudes as assessed by the MAQ ($r = 0.43$ after correction for attenuation), with magnitudes in the medium range. The MAQ was also significantly correlated with general cognitive biases ($r = 0.34$ after correction for attenuation). Meng, Rosenthal, and Rubin's (1992) approach for comparing the magnitude of correlation coefficients was used to assess the difference between these correlations; there were no significant differences in the magnitude of the strength of the relationships among the three variables.

We assessed the convergent validity of the three subscales of the AToM using the same approach. Factor 1 of the AToM, comprising beliefs related to others' judgments, was moderately associated with Factor 3 of the AToM (comprising beliefs related to maternal role idealization) and with the MAQ, and was strongly associated with general cognitive biases. Factor 2 of the AToM, comprising beliefs related to maternal responsibility, was not associated with Factor 1 of the AToM, general cognitive biases, or

the MAQ, but was strongly associated with Factor 3 of the AToM. Factor 3 of the AToM, comprising beliefs related to maternal role idealization, was moderately associated with Factor 1 of the AToM, general cognitive biases, and the MAQ, and was strongly associated with Factor 2 of the AToM.

Results of these analyses support our hypothesis that dysfunctional maternal attitudes, as assessed using the AToM, are associated with general cognitive biases. We also demonstrated that our measure has good convergent validity with an existing, but problematic, measure of maternal attitudes. We did not find that dysfunctional maternal attitudes, as assessed by the AToM and MAQ, were related more strongly to one another than to general cognitive biases.

The pattern of correlations observed among the subscales of the AToM and the MAQ and DAS suggest that beliefs related to maternal responsibility represent a distinct facet of attitudes toward motherhood that, while related to maternal role idealization, are distinct from general patterns of negative cognitive biases and other elements of maternal attitudes. Beliefs related to others' judgments appear most strongly related to general cognitive biases, while beliefs related to maternal role idealization are only moderately related to these general cognitive biases. The patterns of correlations observed among the subscales of the measure suggest that, while the maternal attitudes assessed by the AToM are related to general cognitive biases, they represent a separate construct.

Study 2

The goal of Study 2 was to use the AToM to assess the relationship between maternal attitudes and psychological variables among first-time mothers during the

transition to parenthood. We hypothesized that dysfunctional maternal attitudes would predict symptoms of depression and anxiety among pregnant and postpartum first-time mothers and that these attitudes would have incremental predictive validity over general cognitive biases and interpersonal risk factors for depression and anxiety. We also assessed the discriminant validity of the measure by assessing the relationship between maternal attitudes and a broad range of psychological symptoms, as assessed by the Brief Symptom Inventory (Derogatis & Spencer, 1982), and personality factors, as assessed by the Big Five Inventory (John, Naumann, & Soto, 2008). We predicted that dysfunctional maternal attitudes would be most strongly related to psychological symptoms that are closely related to depression and anxiety (e.g., obsessiveness) and less strongly related to other psychological symptoms (e.g., psychoticism and paranoia). We predicted that dysfunctional maternal attitudes would be strongly related to neuroticism, but would be less strongly associated with other personality factors.

Method

Participants and procedures.

Participants for this study were recruited in the spring of 2012 through the same social media sites as for Study 1. The survey site included an online consent form followed by eligibility questions, a series of self-report questionnaires and, finally, questions about demographic variables. Participants were compensated by a raffle for $150.

Three hundred and eighty-three women initiated participation in the study. Women were eligible to participate if they were between the ages of 18 and 45, resided in

the United States, and were either pregnant with their first child (between 13 and 40 weeks gestational age) or had given birth to their first child within the previous 6 months. Four subjects were excluded because they were under age 18 or over age 45, 29 subjects were excluded because they were not pregnant or within six months of giving birth to their first child, and 11 subjects were excluded because they did not reside in the United States. Of the 339 women who were eligible to participate, 85% (n = 288) completed at least one measure and 62% (n = 211) completed all study measures. We compared subjects who completed each measure to subjects who dropped out at each stage of the study. Subjects who dropped out prior to completing the DAS had significantly higher AToM scores than subjects who completed the DAS; subjects who dropped out prior to completing the DYAD had significantly higher AToM and STAI scores than those who completed the DYAD, and subjects who dropped out prior to completing the MDPSS had significantly higher AToM and DYAD scores than those who completed the MDPSS. Overall, the results of these analyses suggest that subjects at higher risk for psychological difficulties were more likely to drop out of the study prior to completion of all measures.

Demographic characteristics of the sample are presented in Table 1. About 43% of subjects were currently pregnant with their first child (n = 145), and 57% (n = 195) had given birth to their child within the previous 6 month period. For pregnant subjects, the mean gestational age was 24.1 weeks (SD = 7.2 weeks, range 12-39 weeks). The mean age of postpartum subjects' children was 13.9 weeks (SD = 7.0 weeks, range 1-24 weeks). The sample was predominantly married (79.5%) and Caucasian (94%). The sample was highly educated and relatively affluent: 44.5% of subjects had a

graduate/professional degree, 29.8% of subjects reported annual household incomes greater than $100,000, and 51.6% of subjects reported that they were employed full-time.

Measures.

Cognitive risk factors. Attitudes toward motherhood were assessed using the Attitudes Towards Motherhood Scale (AToM), described in Study 1. Cronbach's alpha for the scale was 0.86. General cognitive biases were assessed using the Dysfunctional Attitudes Scale (DAS); to reduce subject burden we utilized the 17-item version (de Graaf, Roelofs, & Huibers, 2009). Cronbach's alpha for the scale was 0.92.

Interpersonal risk factors. Subjects who were married or in a committed relationship completed the Dyadic Adjustment Scale (DYAD; Spanier, 1976), a 32-item measure of relationship satisfaction. Cronbach's alpha for the scale was 0.93. All subjects completed the Multidimensionalal Scale of Perceived Social Support (MDPSS; Zimet, Dahlem, Zimet, & Farley, 1988), which assesses satisfaction with perceived social support from partners, family, and friends. Cronbach's alpha for the scale was 0.94. As subjects' scores on the MDPSS were non-normally distributed, the variable was square-root transformed prior to analyses.

Psychological symptoms. Depressive symptoms were assessed using the Edinburgh Post-Natal Depression Scale (EPDS; Cox, Holden, & Sagovsky, 1987). This 10-item measure was developed for use by pregnant and postpartum women; the scale takes into account normative experiences of perinatal women that correspond with diagnostic criteria for depression (e.g., weight change and fatigue) that can bias other measures of depressive symptoms. Scores greater than or equal to 12 are indicative of a possible depressive episode

(Cox et al., 1996). Cronbach's alpha for the scale was 0.87. Syptoms of anxiety were assessed using the State-Trait Anxiety Inventory (STAI; Spielberger, 1983), a 40-item measure that assesses current symptoms of anxiety and subjects' global tendency toward trait anxiety. Cronbach's alpha for both the state and trait subscales of the STAI was 0.94. As the two subscales of the STAI were highly correlated (r = 0.80), a composite STAI score was calculated and used as the outcome variable for all analyses; Cronbach's alpha for the composite scale was 0.96.

Global symptoms of psychological distress were assessed using the Brief Symptom Inventory (BSI; Derogatis & Spencer, 1982), a 53-item measure of psychological symptoms that reflects nine domains of problems (somatization, obsessiveness, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism). Cronbach's alpha for the subscales of the BSI ranged from 0.71 to 0.87. Because the distribution of scores on the BSI subscales was non-normal, the subscales were coded into dichotomous variables for all analyses. Subjects who rated any item on a subscale as "quite a bit" or "extremely" distressing received a score of 1, while subjects who rated all items as "moderately" distressing or lower received a score of 0.

Personality. Personality was assessed using the Big Five Inventory (BFI; John, Naumann, & Soto, 2008), a 44-item measure that asesses five domains of personality structure (openness to experience, conscientiousness, extraversion, agreeableness, and neuroticism). Cronbach's alpha for the subscales of the BFI ranged from 0.76 to 0.88.

Results and Discussion

Descriptive statistics for the primary study measures and intercorrelations among the measures are presented in Table 4. As expected, cognitive risk factors, interpersonal risk factors, and psychological symptoms were moderately to highly correlated. The AToM was most highly correlated with the DAS (r = 0.50), followed by symptoms of depression (r = 0.41) and anxiety (r = 0.41). Meng, Rosenthal, and Rubin's (1992) approach for comparing the magnitude of correlation coefficients was used to assess the difference between these correlations; there was a trend for the AToM to be more strongly associated with the DAS than with the EPDS (p = 0.06) and the STAI (p = 0.06). The correlations between the AToM and measures of interpersonal risk, including the DYAD (r = -0.15) and the MDPSS (r = 0.20) were significantly smaller than the correlations between the AToM and the DAS and psychological symptoms (all p values < 0.001).

In contrast to the results of Study 1, each of the three subscales of the AToM was moderately to strongly correlated with the other subscales. Factor 2 of the AToM was also moderately associated with general cognitive biases in this sample, although the magnitude of this association was smaller than for Factors 1 and 3. All three factors of the AToM correlated moderately to highly with general cognitive biases and symptoms of depression and anxiety. Factors 1 and 2 of the AToM were weakly correlated with inadequate social support, and Factor 1 of the AToM was weakly associated with low marital satisfaction. Factors 2 and 3 of the AToM were not associated with marital satisfaction, and Factor 3 was not associated with inadequate social support.

As expected, psychological symptoms were strongly correlated with one another, Marital satisfaction and social support were moderately correlated with one another.

Factor structure of the AToM.

To assess the stability of the factor structure of the AToM identified in Study 1, confirmatory factor analysis was use to assess the fit of the original three-factor model and to compare this model to a one-factor model. The three-factor model was a marginally acceptable fit to the data , $\chi^2(51) = 202.8$, $p < 0.001$, SRMR = 0.10, CFI = 0.89, RMSEA = 0.10, $p < 0.001$. Comparison of the three-factor solution to a one-factor solution indicated that the three-factor solution was a significantly better fit to the data, $\chi^2(3) = 346.8$, $p < 0.001$.

We also assessed whether the factor structure of the AToM was comparable for pregnant and postpartum subjects. We first specified a model in which the factor loadings and factor covariances were allowed to vary freely between the two groups. We then specified a model in which factor loadings were constrained to be equal between the two groups. There was not a significant difference in the fit of the constrained model $\chi^2(12) = 16.48$, $p > 0.05$, and the change in the CFI was less than 0.01 (ΔCFI = 0.003), which indicates that the constrained model has comparable model fit (Cheung & Rensvold, 2002). We then specified a model in which both the factor loadings and the factor correlations were constrained to be equal between the two groups. There was not a significant difference in the fit of the constrained model, $\chi^2(3) = 6.82$, $p > 0.05$, and the change in the CFI was less than 0.01 (ΔCFI = 0.003), which indicates that the constrained model has comparable model fit (Cheung & Rensvold, 2002). Results of these analyses

suggest that the factor structure of the AToM and the correlations among the factors are comparable for pregnant and postpartum subjects.

Convergent and predictive validity of the AToM.

We conducted a series of multiple regression models to assess the convergent and predictive validity of the AToM (see Table 5). In Model 1, we assessed the convergent validity of maternal attitudes (as assessed by the AToM) with general cognitive biases (as assessed by the DAS). After controlling for demographic variables, dysfunctional maternal attitudes significantly predicted general cognitive biases ($\beta = 0.50$). The predictive validity of maternal attitudes was assessed using depressive symptoms (Model 2, as assessed by the EPDS) and anxiety symptoms (Model 3, as assessed by the STAI composite) as outcome measures. After controlling for demographic variables, dysfunctional maternal attitudes were a significant predictor of both depressive symptoms ($\beta = 0.43$) and anxiety symptoms ($\beta = 0.43$).

We then conducted a series of multiple regression models to assess the convergent and predictive validity of the subscales of the AToM (see Table 5). In Model 4, we assessed the convergent validity of the three subscales with general cognitive biases (as assessed by the DAS). After controlling for demographic variables, only beliefs related to others' judgments (AToM Factor 1) predicted general cognitive biases. The predictive validity of the three subscales was assessed using depressive symptoms (Model 5, as assessed by the EPDS) and anxiety symptoms (Model 6, as assessed by the STAI composite) as outcome measures. After controlling for demographic variables, beliefs related to others' judgments (AToM Factor 1) and beliefs related to maternal

responsibility (AToM Factor 2) were significantly associated with depressive symptoms. Only beliefs related to others' judgments (AToM Factor 1) were significantly associated with anxiety symptoms.

A series of multiple regressions were conducted to assess the incremental predictive validity of maternal attitudes as compared to general cognitive biases and interpersonal risk factors (marital satisfaction, as assessed by the DYAD, and inadequate social support, as assessed by the MDPSS, see Table 6). We first conducted hierarchical multiple regressions in which demographic variables were entered in Step 1, cognitive biases were added in Step 2, and maternal attitudes were added in Step 3. Separate regressions were conducted for depressive symptoms (Model 1) and anxiety symptoms (Model 4). After controlling for demographic variables and general cognitive biases, the AToM was a significant predictor of both depressive symptoms ($\beta = 0.24$) and anxiety symptoms ($\beta = 0.18$). We then conducted hierarchical multiple regressions in which demographic variables were entered in Step 1, interpersonal risk factors were entered in Step 2, cognitive biases were entered in Step 3, and maternal attitudes were entered in step 4. Separate regressions were conducted for depressive symptoms (Model 2) and anxiety symptoms (Model 5). After controlling for demographic variables, interpersonal risk factors, and cognitive biases, dysfunctional maternal attitudes were significant predictors of both depressive symptoms ($\beta = 0.15$) and anxiety symptoms ($\beta = 0.18$). In order to assess the relative contributions of interpersonal and cognitive risk factors, we then conducted hierarchical multiple regressions in which demographic variables were entered in Step 1, maternal attitudes were entered in Step 2, cognitive biases were entered

in Step 3, and interpersonal risk factors were entered in Step 4. Separate regressions were conducted for depressive symptoms (Model 5) and anxiety symptoms (Model 6). Both inadequate social support and marital satisfaction were significant predictors of symptoms of depression (β_{MDPSS} = 0.23, β_{DYAD} = -0.17) and anxiety (β_{MDPSS} = 0.27, β_{DYAD} = -0.19), after controlling for maternal attitudes and general cognitive biases.

We also conducted a series of multiple regressions were conducted to assess the incremental predictive validity of the three subscales as compared to general cognitive biases and interpersonal risk factors (marital satisfaction, as assessed by the DYAD, and inadequate social support, as assessed by the MDPSS) (see Table 7). We first conducted hierarchical multiple regressions in which demographic variables were entered in Step 1, cognitive biases were added in Step 2, and the three subscales of the AToM were added in Step 3. Separate regressions were conducted for depressive symptoms (Model 1) and anxiety symptoms (Model 4). After controlling for demographic variables and general cognitive biases, beliefs related to others' judgments (AToM Factor 1) and beliefs related to maternal responsibility (AToM Factor 2) were significantly associated with symptoms of depression. Only beliefs related to others' judgments (AToM Factor 1) were significantly associated with symptoms of anxiety.

We then conducted hierarchical multiple regressions in which demographic variables were entered in Step 1, interpersonal risk factors were entered in Step 2, cognitive biases were entered in Step 3, and maternal attitudes were entered in Step 4. Separate regressions were conducted for depressive symptoms (Model 2) and anxiety symptoms (Model 5). After controlling for demographic variables, interpersonal risk

factors, and cognitive biases, each of the subscales of the AToM was significantly associated with symptoms of depression, while only beliefs related to others' judgments (AToM Factor 1) were significantly associated with symptoms of anxiety.

In order to assess the relative contributions of interpersonal and cognitive risk factors, we then conducted hierarchical multiple regressions in which demographic variables were entered in Step 1, maternal attitudes were entered in Step 2, cognitive biases were entered in Step 3, and interpersonal risk factors were entered in Step 4. Separate regressions were conducted for depressive symptoms (Model 5) and anxiety symptoms (Model 6). Both inadequate social support and marital satisfaction were significant predictors of symptoms of depression and anxiety after controlling for demographic variables and cognitive biases. This suggests that both interpersonal and cognitive factors are uniquely associated with perinatal distress.

Discriminant validity.

To assess the discriminant validity of the AToM, a series of multiple regressions was conducted to assess the relationship between the AToM and psychological symptoms, as assessed by the BSI, and personality factors, as assessed by the BFI. The total AToM score and each subscale of the AToM were assessed as outcomes in separate regressions, and demographic variables and each of the subscales of the measure (BSI or BFI) were entered into the model simultaneously. Of the nine subscales of the BSI, only obsessiveness and hostility were significantly associated with overall dysfunctional maternal attitudes after controlling for demographic variables and the other subscales of the BSI (see Table 8). After controlling for demographic variables and the other subscales

of the BSI, obsessiveness was significantly associated with all three subscales of the AToM (see Table 8). Hostility was significantly associated with beliefs related to others' judgments (AToM Factor 1) and beliefs related to maternal responsibility (AToM Factor 2). Paranoia was significantly associated with beliefs related to maternal responsibility (AToM Factor 2).

Of the five personality factors assessed by the BFI, only neuroticism was significantly associated with overall dysfunctional maternal attitudes after controlling for demographic variables and other personality factors (see Table 9). After controlling for demographic variables and the other subscales of the BFI, neuroticism and extraversion were associated with beliefs related to others' judgments (AToM Factor 1); no other subscales of the BFI were related to other subscales of the AToM (see Table 9).

General Discussion

The results of these studies suggest that dysfunctional attitudes toward motherhood are a specific predictor of symptoms of depression and anxiety during the transition to parenthood, even when known risk factors are controlled. In Study 1, we developed a measure of maternal attitudes that is appropriate for use among first-time mothers. We demonstrated that the measure has good convergent validity with general cognitive biases and an existing but flawed measure of maternal attitudes. In Study 2, we used this measure to assess the relationship between maternal attitudes and psychological symptoms among pregnant and postpartum first-time mothers. Dysfunctional maternal attitudes were strongly predictive of both depression and anxiety and demonstrated incremental predictive validity over and beyond general cognitive biases and

interpersonal risk factors for these symptoms. Dysfunctional maternal attitudes were associated with neuroticism, the dimension of personality that is most strongly associated with depression and anxiety (Kotov, Gamez, Schmidt & Watson, 2010), but were not associated with other personality factors.

Our findings are consistent with the results of other studies assessing risk factors for perinatal distress. Reviews of research in this area have consistently found that both cognitive and interpersonal factors have moderate to large associations with depressive symptoms (Beck, 2001; O'Hara & Swain, 1996; Robertson et al., 2004). We found that both cognitive and interpersonal risk factors have unique predictive validity for symptoms of depression and anxiety, even when other risk factors are controlled for. The results of these studies build upon this previous literature by demonstrating that a risk factor specific to the perinatal period, maternal attitudes, has incremental predictive validity above and beyond these established risk factors.

The results of this study are consistent with Beck's (1967) cognitive model of psychopathology, which posits that psychological symptoms occur when maladaptive beliefs are activated in the context of a relevant stressor. Given the inherently stressful nature of pregnancy and new motherhood, this model would predict that women with maladaptive beliefs about motherhood would be at increased risk for depression and anxiety. While general maladaptive beliefs may also be activated by stressful events during this time, specific beliefs about motherhood may be most strongly activated by the particular stressors of pregnancy and parenting. For example, consider the following subject from the current study: This woman is currently 16 weeks pregnant with her first

child. Her EPDS score of 5 is in the nondepressed range, and her DAS score of 33 is in the 16th percentile for our sample. However, her AToM score of 56 places her in the 95th percentile for our sample. This subject reported that she "almost always" believes "If I love my baby, I should want to be with him/her all the time." This subject's generally low level of overall cognitive biases may be protective against depression in the context of other life stressors. However, it is likely that during the postpartum period she will have the experience of wanting to spend some time away from her baby. Because of her specific attitudes toward motherhood, she may interpret this desire to mean that she does not love her child enough, and may then interpret this belief to mean that she is a bad mother. These beliefs may then lead to symptoms of depression, including feelings of sadness, guilt and worthlessness.

A major limitation of the current studies is their cross-sectional design. As risk factors and psychological symptoms were assessed simultaneously, it is impossible to establish whether dysfunctional maternal attitudes contributed causally to the development of these symptoms or whether they simply reflect the presence of depression and anxiety. Future research is necessary to establish whether these maladaptive beliefs precede the development of symptoms. To address this concern, a follow-up to the current study is planned in which the pregnant subjects will be contacted at 12 weeks postpartum. This will allow us to assess whether maternal attitudes during pregnancy predict changes in symptoms of depression and anxiety during the postpartum period.

Another limitation of the current studies was the homogeneity of our subjects. Participants in these studies were more likely to be white, highly-educated, affluent and married. The lack of sociodemographic diversity is a demonstrated problem in healthcare research (Woodall, Morgan, Sloan & Howard 2010). This is particularly relevant to the current research, as several studies have suggested that different factors may be predictive of depression and anxiety among ethnic minorities, women of low socioeconomic status, and women without a partner (e.g. Halbreich, 2005; Logsdon & Usui, 2001; Seguin, Potvin, St. Denis & Loiselle, 1995; Surkan, Peterson, Hughes & Gottlieb, 2006). We did not find that demographic characteristics were associated with psychological symptoms in our sample, but our ability to detect potential differences may have been limited by the relatively small numbers of single women, racial/ethnic minorities, and women of low socioeconomic status who participated in these studies. Further efforts to increase the diversity of participants in this research are necessary in order to determine whether maternal attitudes are related to demographic characteristics and whether the role of maternal attitudes may differ among women of different backgrounds.

The association between dysfunctional maternal attitudes and perinatal distress may provide clinicians with a means of detecting women at-risk for perinatal depression and anxiety. There is some evidence that there is a subgroup of women who are particularly vulnerable to perinatal depressive episodes (Bloch, Schmidt, Danaceau, Murphy, Nieman, & Rubinow, 2000; Cooper & Murray, 1995). By identifying women whose beliefs about motherhood may put them at risk for psychological distress during

pregnancy and the postpartum period, clinicians may be able to intervene early to prevent symptoms from occurring or reduce their severity or duration. A wide range of preventive interventions have been found to effectively reduce the prevalence of depressive episodes during the postpartum period (Sockol, 2012); women with maladaptive beliefs about motherhood may be likely to benefit from these interventions.

Our findings may also prove useful for clinicians and researchers interested in developing interventions for perinatal depression and anxiety, particularly cognitive-behavioral interventions. There is some evidence that cognitive-behavioral therapy may not be as effective for perinatal depression as interpersonal psychotherapy (Sockol, Epperson, & Barber, 2011). However, researchers have developed a manualized version of interpersonal psychotherapy specific to perinatal depression that takes into account common interpersonal challenges that women face during the transition to parenthood (O'Hara, Stuart, Gorman, & Wenzel, 2000). Our findings provide evidence for themes that may characterize depressed women's beliefs about motherhood. While cognitive-behavioral therapy is inherently sensitive to individuals' particular cognitive biases, developing specific interventions for perinatal populations that incorporate common cognitive distortions could lead to improved efficacy of cognitive-behavioral interventions for this population.

Overall, the results of these studies suggest that dysfunctional maternal attitudes are strongly associated with psychological symptoms during the perinatal period. While dysfunctional maternal attitudes are strongly associated with general cognitive biases, they have incremental predictive validity over these more general beliefs. Moreover,

dysfunctional maternal attitudes continue to predict symptoms of depression and anxiety even controlling for interpersonal factors. These findings are consistent with Beck's cognitive model of psychopathology. Results of these studies suggest that maladaptive attitudes toward motherhood are a specific risk factor for perinatal depression and anxiety that may be used by clinicians and researchers to identify women at-risk for these disorders and as targets for intervention and prevention. Although further research is necessary to establish the causal role of these attitudes in the development of psychological symptoms and to assess whether these beliefs play a similar role among more diverse populations, these findings indicate that maternal attitudes play an important role in perinatal depression and anxiety.

References

Appleby, L., Warner, R., Whitton, A., & Faragher, B. (1997). A controlled study of fluoxetine and cognitive behavioural counselling in the treatment of postnatal depression. *British Medical Journal, 314*, 932-936.

Beck, A. (1967). *Depression: Clinical, Experimental & Theoretical Aspects.* Philadelphia, PA: University of Pennsylvania Press.

Beck A. (1976). *Cognitive Therapy and the Emotional Disorders.* New York: Penguin.

Beck, A. (1985). *Anxiety Disorders and Phobias: A Cognitive Perspective.* New York: Basic Books.

Beck, C. (2001). Predictors of postpartum depression: An update. *Nursing Research, 50,* 275-285.

Belsky, J. (1985). Exploring individual differences in marital change across the transition to parenthood: The role of violated expectations. *Journal of Marriage and the Family, 47*, 1037-1044.

Bennett, H., Einarson, A., Taddio, A., Koren, G. & Einarson, T. (2004). Prevalence of depression during pregnancy: Systematic review. *Obstetrics & Gynecology, 103*, 698-709.

Besser, A. & Priel, B. (2003). Trait vulnerability and coping strategies in the transition to motherhood. *Current Psychology: Developmental, Learning, Personality, Social, 22*, 57-72.

Besser, A., Priel, B., Flett, G. & Wiznitzer, A. (2007). Linear and nonlinear models of vulnerability to depression: Personality and postpartum depression in a high-risk population. *Individual Differences Research, 5,* 1-29.

Bloch, M., Schmidt, P., Danaceau, M., Murphy, J., Nieman, L. & Rubonow, D. (2000). Effects of gonadal steroids in women with a history of postpartum depression. *American Journal of Psychiatry, 157,* 924-930.

Block, J. (1963) Recognizing attenuation effects in the strategy of research. *Psychological Bulletin, 62*(3), 214-216.

Cattel, R. B. (1966). The scree test for the number of factors. *Multivariate Behavioral Research, 1,* 245-276.

Cheung, G. W., & Rensvold, R. B. (2002). Evaluating goodness-of-fit indexes for testing measurement invariance. *Structural Equation Modeling, 9*(2), 233-255.

Church, N., Brechman-Toussaint, M., & Hine, D. (2005). Do dysfunctional cognitions mediate the relationship between risk factors and postnatal depression symptomatology? *Journal of Affective Disorders, 87,* 65-72.

Cooper, P. J., & Murray, L. (1995). Course and recurrence of postpartum depression: Evidence for the specificity of the diagnostic concept. *British Journal of Psychiatry, 166,* 191-195.

Cooper, P. J., Murray, L., Wilson, A., & Romaniuk, H. (2003). Controlled trial of the short- and long-term effect of psychological treatment of post-partum depression 1: Impact on maternal mood. *British Journal of Psychiatry, 182*(5), 412-419.

Cox, J., Chapman, G., Murray, D. & Jones, P. (1996). Validation of the Edinburgh Post-Natal Depression Scale (EPDS) in non-postnatal women. *Journal of Affective Disorders, 39,* 185-189.

Cox, J., Holden, J. & Sagovsky, R. (1987). Detection of postnatal depression: Develoment of the 10-item Edinburgh Postnatal Depression Scale. *British Journal of Psychiatry, 150,* 782-786.

Cutrona, C. (1983). Causal attributions and perinatal depression. *Journal of Abnormal Psychology, 92,*161-172.

de Graaf, L. E., Roelofs, J., & Huibers, M. J. H. (2009). Measuring dysfunctional attitudes in the general population: The Dysfunctional Attitudes Scale (Form A) Revised. *Cognitive Therapy and Research, 33*(4), 345-355.

Derogatis, L. & Spencer, P. (1982). *The Brief Symptom Inventory (BSI): Administration, and Procedures Manual-I.* Baltimore, MD: Clinical Psychometric Research.

DiPietro, J.A., Ghera, M., Costigan, K. A., & Hawkins, M. (2004). Measuring the ups and downs of pregnancy stress. *Journal of Psychosomatic Obstetrics and Gynecology, 25,* 189-201.

Francis-Raniere, E. L., Alloy, L. B., & Abramson, L. Y. (2006). Depressive personality styles and bipolar spectrum disorders: Prospective tests of the event congruency hypothesis. *Bipolar Disorders, 8,* 382-399.

Geweke, J. F., & Singleton, K. J. (1980). Interpreting the likelihood ratio statistic in factor models when sample size is small. *Journal of the American Statistical Association*133-137.

Grace, S., Evindar, A. & Stewart, D. (2003). The effect of postpartum depression on child cognitive development and behavior: A review and critical analysis of the literature. *Archives of Women's Mental Health,* 263-274.

Grazioli, R. & Terry, D. (2000). The role of cognitive vulnerability and stress in the prediction of postpartum depressive symptomatology. *British Journal of Clinical Psychology, 39,* 329-347.

Grote, N. K., Bridge, J. A., Gavin, A. R., Melville, J. L., Iyengar, S., & Katon, W. J. (2010). A meta-analysis of depression during pregnancy and the risk of preterm birth, low birth weight, and intrauterine growth restriction. *Archives of General Psychiatry, 67*(10), 1012-1024.

Halbreich, U. (2005). Postpartum disorders: Multiple interacting underlying mechanisms and risk factors. *Journal of Affective Disorders, 88*, 1-7.

Hammen, C., Ellicott, A., Gitlin, M., & Jamison, K. (1989). Sociotropy/autonomy and vulnerability to specific life events in patients with unipolar depression and bipolar disorders. *Journal of Abnormal Psychology, 98*(2), 154-160.

Hammen C., Marks, T., de Mayo, R. & Mayol, A. (1985). Self-schemas and risk for depression: A prospective study. *Journal of Personality and Social Psychology, 49*, 1147-1159.

Harwood, K. (2004). Women's parenting expectations and their influence on adjustment to parenthood. Unpublished doctoral dissertation, University of Western Australia.

Hilsman, R. & Garber, J. (1995). A test of the cognitive diathesis-stress model of depression in children: Academic stressors, attributional style, perceived competence, and control. *Journal of Personality and Social Psychology, 69*(2), 370-380.

Horn, J. L. (1965). A rationale and test for the number of factors in factor analysis. *Psychometrika, 30,* 179-185.

Hull, J. & Mendolia, M. (1991). Modeling the relations of attributional style, expectancies and depression. *Journal of Personality and Social Psychology, 61,* 85-97.

John, O. P., Naumann, L. P., & Soto, C. J. (2008). Paradigm shift to the integrative big-five trait taxonomy: History, measurement, and conceptual issues. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of Personality: Theory and Research.* (pp. 114-158). New York, NY: Guilford Press.

Joseffson, A., Berg, G., Nordin, C. & Sydsjo, G. (2001). Prevalence of depressive symptoms in late pregnancy and postpartum. *Acta Obstetrica et Gynecologica Scandinavica,* 80, 251-255.

Kalmuss, D., Davidson, A., & Cushman, L. (1992). Parenting expectations, experiences, and adjustment to parenthood: A test of the violated expectations framework. *Journal of Marriage and the Family, 54,* 516-526.

Kim, D. R., Sockol, L. E., Sammel, M., Kelly, C., Moseley, M., & Epperson, C. N. (2012). Elevated risk of preeclampsia in pregnant women with depression. Unpublished manuscript. University of Pennsylvania. Philadelphia, PA.

Kotov, R., Gamez, W., Schmidt, F., & Watson. D. (2010). Linking "big" personality traits to anxiety, depressive, and substance use disorders: A meta-analysis. *Psychological Bulletin, 136*(5), 768-821.

Kumar, R., Robson, K. M., & Smith, A. M. R. (1984). Development of a self-administered questionnaire to measure maternal adjustment and maternal attitudes during pregnancy and after delivery. *Journal of Psychosomatic Research, 28,* 43-51.

Lee, A., Lam, S., Lau, S., Chong, C., Chui, H., & Fong, D. (2007). Prevalence, course, and risk factors for antenatal anxiety and depression. *Obstetrics & Gynecology, 110,* 1102-1112.

Littleton, H., Breitkopf, C., & Berenson, A. (2007). Correlates of anxiety symptoms during pregnancy and association with perinatal outcomes: A meta-analysis. *American Journal of Obstetrics & Gynecology, 196,*424-432.

Logsdon, M. & Usui, W. (2001). Psychosocial predictors of postpartum depression in diverse groups of women. *Western Journal of Nursing Research, 23,* 563-574.

Meng, X-L., Rosenthal, R., & Rubin, D. B. (1992). Comparing correlated correlation coefficients. *Psychological Bulletin, 111*(1), 172-175.

O'Conner, T., Heron, J., Glover, V., & the ALSPAC Study Team. (2002). Antenatal anxiety predicts child behavioral/emotional problems independently of postnatal depression. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41, 1470-7.

O'Hara, M. W., Rehm, L. & Campbell, S. (1982). Predicting depressive symptomatology: Cognitive-behavioral models and postpartum depression.

O'Hara, M.W., Stuart, S., Gorman, L., & Wenzel, A (2000). Efficacy of interpersonal psychotherapy for postpartum depression. *Archives of General Psychiatry, 57,* 1039-1045,

O'Hara, M. W., & Swain, A. (1996). Rates and risk of postpartum depression: A meta-analysis. *International Review of Psychiatry, 8,* 37-54.

Olioff, M. (1991). The application of cognitive therapy to postpartum depression. *The Challenge of Cognitive Therapy: Applications to Non-Traditional Populations,* Eds. T. Vallis, J. Howes, & P. Miller. New York: Plenum.

Robertson, E., Grace, S., Wallington, T., & Stewart, D. (2004). Antenatal risk factors for postpartum depression: a synthesis of recent literature. *General Hospital Psychiatry, 26,* 289-295.

Schmidt, N. B., Lerew, D. R., & Jackson, R. J. (1997). Prospective evaluation of anxiety sensitivity in the pathogenesis of panic: Replication and extension. *Journal of Abnormal Psychology, 108*(3), 532-537.

Segal, Z. V., Shaw, B. F., Vella, D. D., & Katz, R. (1992). Cognitive and life stress predictors of relapse in remitted unipolar depressed patients: Test of the congruency hypothesis. *Journal of Abnormal Psycholgy, 101*(1), 26-36.

Seguin, L., Potvin, L., St. Denis, M. & Loiselle, J. (1995). Chronic stressors, social support and depression during pregnancy. *Obstetrics & Gynecology, 85,* 583-589.

Sockol, L. E. (2008). Predicting depressive symptoms during pregnancy: Integrating cognitive, coping and social perspectives. (Unpublished master's thesis). University of Pennsylvania, Philadelphia, PA.

Sockol, L. E. (2012). Preventing postpartum depression: A meta-analytic review. Unpublished manuscript. University of Pennsylvania, Philadelphia, PA.

Sockol, L.E., Epperson, C. N., & Barber, J. P. (2011). A meta-analysis of treatments for perinatal depression. *Clinical Psychology Review, 31,* 839-849.

Spanier, G. (1976). Measuring dyadic adjustment: New scales for assessing the quality of marriage and similar dyads. *Journal of Marriage and the Family, 38,* 15-28.

Spielberger, C. (1983). *Manual for the State-Trait Anxiety Inventory (STAI)*. Palo Alto, Calif, Consulting Psychologists Press.

Stuart, S., Couser, G., Schilder, K., O'Hara, M.W., & Gorman, L.L. (1998). Postpartum anxiety and depression: onset and comorbidity in a community sample. *Journal of Nervous and Mental Disease, 186,* 420-424.

Surkan, P., Peterson, K., Hughes, M. & Gottlieb, B. (2006). The role of social networks and support in postpartum women's depression: A multiethnic urban sample. *Maternal and Child Health Journal, 10,* 375-383.

Velicer, W. F. (1976). Determining the number of components from the matrix of partial correlations. *Psychometrika, 41,* 321-327.

Warner, R., Appleby, L., Whitton, A & Faragher, B. (1997). Attitudes toward motherhood in postnatal depression: Development of the maternal attitudes questionnaire. *Journal of Psychosomatic Research, 43,* 351-358.

Weissman, A. & Beck, A. (1978). Development and validation of the Dysfunctional Attitude Scale: A preliminary investigation. Paper presented at the meeting of the American Educational Research Association, Toronto.

Wenzel, A., Haugen, E. N., Jackson, L. C., & Brendle, J. R. (2005). Anxiety disorders at eight weeks postpartum. *Journal of Anxiety Disorders, 19,* 295-311.

Woodall, A., Morgan, C., Sloan, C., & Howard, L. (2010). Barriers to participation in mental health research: Are there specific gender, ethnicity, and age related barriers? *BMC Psychiatry, 10,* 103.

Zimet, G., Dahlem, N., Zimet, S. & Farley, G. (1988). The multidimensional scale of perceived social support. *Journal of Personality Assessment, 52,* 30-41.

Table 1

Demographic Characteristics of Samples in Studies 1 and 2

	Study 1	Study 2
Age, *M (SD)*	28.3(4.7)	29.2 (4.8)
Gestational Age (weeks), *M (SD)*	26.1 (8.3)	24.1 (7.2)
Infant Age (weeks), *M (SD)*	12.5 (8.2)	13.9 (7.0)
Relationship Status		
Married	65%	80%
In a relationship, living together	13%	13%
In a relationship, not living together	2%	1%
Other	2%	6%
Race/Ethnicity		
Asian/Pacific Islander	3%	1%
Black/African-American	1%	4%
Caucasian	78%	94%
Latina	2%	5%
Annual Household Income		
< $25,000	16%	11%
$25,000-$49,999	18%	21%
$50,000-$74,999	18%	21%
$75,000-$99,999	12%	17%
> $100,000	18%	30%
Employment Status		
Full-Time	30%	52%
Part-Time	15%	16%
Unemployed	29%	23%
Highest Level of Education		
Did Not Complete High School	1%	0%
High School Diploma/GED	3%	7%
Some College	19%	13%
Associate's Degree/Trade School	4%	6%
Bachelor's Degree	32%	29%
Graduate or Professional Degree	24%	44%

Table 2

Common Factor Analysis of Attitudes Toward Motherhood Scale with Promax Rotation in Study 1

Factor	Promax loading	Item-total *r*
Beliefs Related to Others' Judgments		
If I make a mistake, people will think I am a bad mother.	0.85	0.42
People will probably think less of me if I make parenting mistakes.	0.85	0.42
If my baby is crying, people will think I cannot care for him/her properly.	0.83	0.32
Seeking help with my baby from other people makes me feel incompetent.	0.68	0.37
Beliefs Related to Maternal Responsibility		
If I love my baby, I should want to be with him/her all the time.	0.85	0.51
I should feel more devoted to my baby.	0.76	0.33
I am the only person who can keep my baby safe.	0.68	0.47
Good mothers always put their baby's needs first.	0.66	0.46
Beliefs Related to Maternal Role Idealization		
It is wrong to feel disappointed by motherhood.	0.88	0.61
It is wrong to have mixed feelings about my baby.	0.84	0.65
Negative feelings towards my baby are wrong.	0.75	0.45
If I fail at motherhood, then I am a failure as a person.	0.69	0.51

Table 3

Summary Statistics and Intercorrelations Among Maternal Attitudes and General Cognitive Biases in Study 1

	n	Range	*M*(*SD*)	AToM Total	AToM Factor 1	AToM Factor 2	AToM Factor 3	MAQ	DAS
AToM Total	136	16-58	37.22 (8.32)	(.81)	.62***	.74***	.83***	.31**	.43**
Others' Judgments (AToM Factor 1)	136	4-21	11.34 (3.84)	.76***	(.82)	.10	.24**	.30**	.53***
Maternal Responsibility (AToM Factor 2)	136	5-24	14.24 (3.61)	.96***	.13	(.74)	.55***	.16	.13
Role Idealization (AToM Factor 3)	136	4-23	11.65 (3.97)	1.03***	.30**	.71***	(.80)	.21*	.27**
Maternal Attitudes (MAQ)	111	0-15	4.34 (2.77)	.43**	.42**	.23	.30*	(.63)	.27**
Cognitive Biases (DAS)	104	64-178	119.23 (24.21)	.50**	.62***	.16	.32**	.36**	(.90)

* $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

Note. Uncorrected correlations are displayed above the diagonal. Correlations below the diagonal have been corrected for attenuation. Internal reliability coefficients are displayed in parentheses on the diagonal.

Table 4

Descriptive Statistics and Correlations Among Primary Study Measures in Study 2

	n	Range	*M*(*SD*)	AToM Total	AToM Factor 1	Atom Factor 2	Atom Factor 3	DAS	EPDS	STAI	DYAD	MDPSS
Maternal Attitudes (AToM Total)	288	12-67	38.8 (10.6)	(.86)	.73***	.82***	.87***	.50***	.41***	.41***	-.15***	.20**
Others' Judgments (AToM Factor 1)	293	4-22	11.5 (3.9)	.88***	(.80)	.37***	.45***	.57***	.41***	.48***	-.18*	.19**
Maternal Responsibility (AToM Factor 2)	290	4-24	13.8 (4.3)	1.03***	.48***	(.73)	.62***	.31***	.33***	.30***	-.11	.19**
Role Idealization (AToM Factor 3)	292	4-24	13.5 (4.8)	1.04***	.56***	.81***	(.81)	.37***	.25***	.26***	-.09	.12
Cognitive Biases (DAS)	237	17-94	49.2 (16.6)	.57***	.67***	.38***	.43***	(.91)	.49***	.58***	-.33***	.16*
Depressive Symptoms (EPDS)	278	0-25	8.3 (4.8)	.47***	.49***	.41***	.30***	.55***	(.87)	.79***	-.36***	.30***
Anxiety Symptoms (STAI)	240	40-155	73.3 (21.3)	.45***	.55***	.36***	.29***	.62***	.86***	(.96)	-.43***	.44***
Marital Satisfaction (DYAD)	211	73-145	118.8 (14.5)	-.17***	-.21*	-.13	-.10	-.36***	-.40***	-.46***	(.93)	-.21**
Inadequate Social Support (MDPSS)	229	12-84	70.5 (13.9)	.22***	.22**	.23**	.17*	.17*	.33***	.42***	-.22**	(.95)

* $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

[1] Descriptive statistics are presented for the non-transformed MDPSS. As subjects' scores on the MDPSS were not normally distributed, correlations were calculated using the transformed variable.

Note. Uncorrected correlations are displayed above the diagonal. Correlations below the diagonal have been corrected for attenuation. Internal reliability coefficients are displayed in parentheses on the diagonal.

Table 5

Multiple Regressions Assessing the Convergent and Predictive Validity of Maternal Attitudes (AToM) in Study 2

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	General Cognitive Biases (DAS)	Depressive Symptoms (EPDS)	Anxiety Symptoms (STAI)	General Cognitive Biases (DAS)	Depressive Symptoms (EPDS)	Anxiety Symptoms (STAI)
	β	β	β	β	β	β
Age	0.12	0.04	0.04	0.10	0.02	0.04
Pregnant vs. Postpartum	0.11	-0.13	-.06	0.11	-0.10	-0.05
Married vs. Nonmarried	-0.01	-0.01	0.10	0.04	0.03	0.15*
White vs. Nonwhite	0.07	0.05	0.08	0.04	0.02	0.05
Maternal Attitudes (AToM Total)	0.50***	0.43***	0.43***			
Others' Judgments (AToM1)				0.50***	0.42***	0.45***
Maternal Responsibility (AToM2)				0.11	0.25**	0.15
Role Idealization (AToM3)				0.02	-0.11	-0.04
R^2	0.27***	0.20***	0.21***	0.35***	0.27***	0.28***

* $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

Table 6

Hierarchical Multiple Regressions Assessing the Incremental Predictive Validity of Maternal Attitudes (AToM Total) in Study 2

Outcome	Depressive Symptoms (EPDS)			Anxiety Symptoms (STAI)		
	Model 1[a]	Model 2[b]	Model 3[c]	Model 4[a]	Model 5[b]	Model 6[c]
	β	*β*	*β*	*β*	*β*	*β*
Step 1[a/b/c]						
Age	-0.05	0.06	0.06	-0.07	0.01	0.01
Pregnant vs. Postpartum	-0.07	-0.11	-0.11	-0.02	-0.02	-0.02
Married vs. Nonmarried	0.02	0.03	0.03	0.13	0.14	0.14
White vs. Nonwhite	0.09	-0.03	-0.03	0.11	0.01	0.01
Step 2[b]/4[c]						
Lack of Social Support (MDPSS)		0.26**	0.23***		0.33***	0.27***
Marital Satisfaction (DYAD)		-0.29***	-0.17*		-0.28***	-0.19**
Step 2[a]/3[b/c]						
Cognitive Biases (DAS)	0.50***	0.42***	0.40***	0.58***	0.47***	0.47***
Step 2[c]/3[a]/4[b]						
Maternal Attitudes (AToM)	0.24**	0.15*	0.36***	0.18**	0.18*	0.41***
Step 1 R^2	0.02	0.02	0.02	0.04	0.02	0.02
Step 2 ΔR^2	0.24***	0.16***	0.13***	0.32***	0.22***	0.16***
Step 3 ΔR^2	0.04**	0.15***	0.12***	0.02*	0.20***	0.16***
Step 4 ΔR^2		0.02*	0.08***		0.02*	0.12***

* $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

[a] In models 1 and 4, demographic variables were entered in step 1, cognitive biases (DAS) were added in step 2, and maternal attitudes (AToM) were added in step 3.

[b] In models 2 and 5, demographic variables were entered in step 1, interpersonal risk factors (MDPSS and DYAD) were added in step 2, cognitive biases (DAS) were added in step 3, and maternal attitudes (AToM) were added in step 4.

[c] In models 3 and 6, demographic variables were entered in step 1, maternal attitudes (AToM) were entered in step 2, cognitive biases (DAS) were added in step 3, and interpersonal risk factors (MDPSS and DYAD) were entered in step 4.

Table 7

Hierarchical Multiple Regressions Assessing the Incremental Predictive Validity of Maternal Attitudes (AToM) in Study 2

	Depressive Symptoms (EPDS)			Anxiety Symptoms (STAI)		
	Model 1[a]	Model 2[b]	Model 3[c]	Model 4[a]	Model 5[b]	Model 6[c]
	β	*β*	*β*	*β*	*β*	*β*
Step 1[a/b/c]						
Age	-0.05	0.06	0.06	-0.07	0.01	0.01
Pregnant vs. Postpartum	-0.07	-0.11	-0.11	-0.02	-0.02	-0.02
Married vs. Nonmarried	0.02	0.03	0.03	0.13	0.14	0.14
White vs. Nonwhite	0.09	-0.03	-0.03	0.11	0.00	0.00
Step 2[b]/4[c]						
Lack of Social Support (MDPSS)		0.26**	0.16*		0.33***	0.25***
Marital Satisfaction (DYAD)		-0.29***	-0.16*		-0.28***	-0.19**
Step 2[a]/3[b/c]						
Cognitive Biases (DAS)	0.50***	0.42***	0.32***	0.58***	0.47***	0.38***
Step 2[c]/3[a]/4[b]						
Others' Judgments (AToM1)	0.26***	0.26**	0.45***	0.22**	0.26**	0.49***
Maternal Responsibility (AToM2)	0.20*	0.20*	0.31**	0.13	0.12	0.22*
Role Idealization (AToM3)	-0.12	-0.20*	-0.27**	-0.08	-0.09	-0.17
Step 1 R^2	0.04	0.02	0.02	0.04	0.02	0.02
Step 2 ΔR^2	0.24***	0.16***	0.25***	0.32***	0.22***	0.28***
Step 3 ΔR^2	0.07***	0.15***	0.07***	0.04**	0.20***	0.09***
Step 4 ΔR^2		0.06**	0.025**		0.05**	0.10***

* $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

[a] In models 1 and 4, demographic variables were entered in step 1, cognitive biases (DAS) were added in step 2, and maternal attitudes (AToM) were added in step 3.

[b] In models 2 and 5, demographic variables were entered in step 1, interpersonal risk factors (MDPSS and DYAD) were added in step 2, cognitive biases (DAS) were added in step 3, and maternal attitudes (AToM) were added in step 4.

[c] In models 3 and 6, demographic variables were entered in step 1, maternal attitudes (AToM) were entered in step 2, cognitive biases (DAS) were added in step 3, and interpersonal risk factors (MDPSS and DYAD) were entered in step 4.

Table 8

Multiple Regressions Predicting Maternal Attitudes (AToM Total and Subscales) from Psychological Symptoms (BSI Subscales) in Study 2

	Maternal Attitudes (AToM Total)	Others' Judgments (AToM Factor 1)	Maternal Responsibility (AToM Factor 2)	Role Idealization (AToM Factor 3)
	β	β	β	β
Age	-0.13	-0.03	-0.16*	-0.13
Pregnant vs. Postpartum	0.05	0.04	-0.06	0.11
Married vs. Nonmarried	0.05	-0.10	0.09	0.11
White vs. Nonwhite	0.04	0.06	0.07	-0.01
Somatization	-0.03	0.01	0.01	-0.08
Obsessiveness	0.26**	0.20*	0.22**	0.21*
Interpersonal Sensitivity	-0.07	-0.07	-0.05	-0.04
Depression	0.07	0.14	0.05	0.01
Anxiety	-0.03	0.00	-0.07	-0.01
Hostility	0.22**	0.26**	0.20*	0.09
Phobic Anxiety	0.15	0.08	0.14	0.14
Paranoia	-0.13	-0.04	-0.21**	-0.11
Psychoticism	0.09	0.02	0.06	0.14
R^2	0.26***	0.21***	0.22***	0.18***

* $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

Table 9

Multiple Regressions Predicting Maternal Attitudes (AToM Total and Subscales) from Personality Factors (BFI Subscales) in Study 2

	Maternal Attitudes (AToM Total)	Others' Judgments (AToM Factor 1)	Maternal Responsibility (AToM Factor 2)	Role Idealization (AToM Factor 3)
	β	β	β	β
Age	-0.20**	-0.10	-0.21**	-0.17*
Pregnant vs. Postpartum	0.05	-0.04	-0.03	0.10
Married vs. Nonmarried	0.06	-0.04	0.09	0.10
White vs. Nonwhite	0.05	0.07	0.09	-0.02
Openness	-0.10	-0.01	-0.14	-0.11
Conscientiousness	0.02	0.01	-0.02	0.07
Extraversion	-0.05	-0.07	-0.04	-0.03
Agreeableness	-0.11	-0.17*	-0.04	-0.07
Neuroticism	0.20*	0.26**	0.13	0.12
R^2	0.14**	0.16***	0.13**	0.09†

† $p < 0.10$ * $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

Chapter 2:

A Meta-Analysis of Treatments for Perinatal Depression

This chapter originally appeared as:

Sockol, L.E., Epperson, C. N., & Barber, J. P. (2011). A meta-analysis of treatments for perinatal depression. *Clinical Psychology Review, 31,* 839-849.

Abstract

This meta-analysis assessed the efficacy of pharmacologic and psychological interventions for the treatment of perinatal depression. A systematic review identified 27 studies, including open trials (n = 9), quasi-randomized trials (n = 2), and randomized controlled trials (n = 16) assessing change from pretreatment to posttreatment or comparing these interventions to a control group. Uncontrolled and controlled effect sizes were assessed in separate meta-analyses. There was significant improvement in depressive symptoms from pretreatment to posttreatment, with an uncontrolled overall effect size (Hedges' g) of 1.61 after removal of outliers and correction for publication bias. Symptom levels at posttreatment were below cutoff levels indicative of clinically significant symptoms. At posttreatment, intervention groups demonstrated significantly greater reductions in depressive symptoms compared to control groups, with an overall controlled effect size (Hedges' g) of 0.65 after removal of outliers. Individual psychotherapy was superior to group psychotherapy with regard to changes in symptoms from pretreatment to posttreatment. Interventions including an interpersonal therapy component were found to have greater effect sizes, compared to control conditions, than interventions including a cognitive-behavioral component. The implications of the findings for clinical practice and future research are discussed.

A Meta-Analysis of Treatments for Perinatal Depression

Perinatal depression is one of the most common complications of childbearing. Approximately 10 to 15% of women experience a clinically significant major depressive episode during pregnancy or the early postpartum period (Bennett, Einarson, Taddio, Koren, & Einarson, 2004b; Epperson, 1999; Gavin, Gayner, Lohr, Meltzer-Brody, Gartlehner, & Swinson, 2005; O'Hara & Swain, 1996). These prevalence estimates predominantly reflect rates of depressive symptoms in developed countries; there is evidence that rates of depression vary more widely in non-developed countries (Halbreich & Karkun, 2006). In addition to the distress and impairment experienced by depressed women, depression during this time period is associated with further adverse outcomes for both mother and child. Women who experience perinatal depressive episodes are at increased risk for subsequent episodes of both postpartum and non-postpartum depression (Cooper & Murray, 1995). Prenatal depression is associated with increased risk for negative birth outcomes, including preterm labor, low birthweight, and intrauterine growth restriction (Grote, Bridge, Gavin, Melville, Iyengar, & Katon, 2010). Maternal depression during the postpartum period is also a risk factor for a range of adverse child outcomes, including behavioral problems and impaired cognitive development (Grace, Evindar, & Stewart, 2003).

Given the prevalence of perinatal depression and the adverse effects this disorder has on women and their children, the identification of effective treatments for this disorder has important public health implications. Although there is a great deal of evidence for the efficacy of both antidepressant medication and psychological interventions for depression (see Joffe, Sokolov & Streiner, 1996 and Cuijpers, van

Straten, Andersson, & van Oppen, 2008 for meta-analyses of the efficacy of antidepressant medication and psychotherapy for depression, respectively), concerns unique to the perinatal period may influence the efficacy of these treatments for this population (Kim, O'Reardon, & Epperson, 2010). For example, in an attempt to limit fetal exposure, antidepressants may be prescribed below therapeutic dosage levels (Bennett, Einarson, Taddio, Koren, & Einarson, 2004a). This problem is complicated further by the fact that most women actually require higher doses of antidepressant medication during pregnancy (Dawes & Chowienczyk, 2001; Hostetter, Stowe, & Strader, 2000; Wisner, Perel, & Wheeler, 1993). Concerns regarding the effects of infant exposure to antidepressant medication via breastmilk may also lead clinicians to prescribe inadequate doses of these medications during the postpartum period (Epperson, Anderson, & McDougle, 1997; Epperson, Jatlow, Czarkowski, & Anderson, 2003). Biological and psychosocial changes that occur in the context of pregnancy and parenting, including sleep deprivation, disruptions to the hormonal milieu, alterations to HPA axis functioning, and changes to interpersonal relationships, introduce challenges that may affect the efficacy of both pharmacological and psychotherapeutic interventions (Dennis & Ross, 2005; Kammerer, Taylor, & Glover, 2006). The efficacy of psychological interventions for depression may also be reduced among women who have had previous pregnancy losses, complications, or traumatic deliveries, as they may experience post-traumatic stress disorder or other comorbid anxiety disorders (Forray, Mayes, Magriples, & Epperson, 2009). There are also differences in the acceptability of interventions, particularly among women who are pregnant or breastfeeding: the majority of women indicate a preference for psychological interventions to antidepressant

medication during both pregnancy and the postpartum period, and the overall acceptability of antidepressant medication among these groups is low (Chabrol, Teissedre, Armitage, Danel & Walburg, 2004; Kim et al., 2011). Thus the identification of efficacious interventions, particularly psychological interventions, for this population is an important and growing area of research.

Two meta-analytic reviews of psychological treatments for postpartum depression have found these interventions to be superior to routine care or control conditions. A Cochrane review of psychological and psychosocial interventions for postpartum depression found that, compared to routine care, these interventions were associated with a 30% reduction in relative risk for depressive symptomatology (Dennis & Hodnett, 2007). Cuijpers, Brännmark, and van Straten (2008) conducted a meta-analysis of 17 studies in which a psychological intervention initiated during the postpartum period was compared to a control or active treatment condition. They reported that psychological interventions were superior to control conditions, with an overall effect size in the moderate range.

In the only published meta-analysis to include interventions for both antenatal and postpartum depression, Bledsoe and Grote (2006) evaluated the efficacy of 16 psychological and pharmacological interventions and found that depressive symptoms decreased significantly from pre- to posttreatment. They did not assess the effect of treatments compared to control conditions. While their findings provide preliminary evidence for the efficacy of these interventions, a major limitation of their meta-analysis is the difficulty of interpreting effect sizes representing changes in symptoms from pretreatment to posttreatment. As there is evidence that, for many women, depressive

symptoms remit naturally over the course of the postpartum period, it is not possible to determine whether these effect sizes reflect the effects of the interventions or simply natural decreases in symptom levels over time (Heron, O'Connor, Evans, Golding, & Glover, 2004). The authors also did not report analyses of homogeneity, tests for outliers or publication bias, did not specify whether the analysis was conducted using fixed or random effects models, and included multiple effect sizes for three studies in which more than one active intervention was assessed. As these methodological issues may substantially impact estimation of effect sizes, these results should be interpreted with caution.

The present meta-analysis addresses several of the limitations of the above studies. Unlike Cuijpers, Brännmark, and van Straten (2008), we included pharmacological interventions in addition to psychological interventions, and included interventions initiated during pregnancy. As many studies of interventions for this population are either open trials or do not include a no-treatment control condition, we elected not to restrict these analyses to studies in which interventions were compared to a control condition. However, to address the possibility that effect sizes calculated from these studies may reflect natural symptom remission over time, we also compared active treatments to control conditions in studies where it was possible to do so. We have also included several new studies of treatments for perinatal depression that have been published since these earlier meta-analyses were conducted. The goal of the current meta-analysis was to assess the efficacy of psychological and pharmacological interventions for perinatal depression, defined as the period encompassing pregnancy and the first 12 months postpartum. Both the overall effect of these interventions on depressive

symptoms over time and the relative efficacy of interventions compared to control conditions were assessed. We also conducted exploratory moderator analyses assessing elements of both study design and interventions as potential moderators of the magnitude of effect size when significant heterogeneity of effect sizes was observed.

Method

Search Procedures and Selection of Studies

Relevant studies were identified through searches of databases through September 2010, including PubMed and PsycInfo, using the following terms as descriptors: *postpartum depression, pregnancy* AND *depression, therapy, drug therapy, cognitive behavior therapy, interpersonal psychotherapy, psychodynamic therapy, treatment,* and *treatment outcome.* The reference lists of existing meta-analyses, reviews, chapters, and retrieved articles were inspected for further studies. Clinical trial databases (including the Cochrane Pregnancy and Childbirth Group, Cochrane Depression, Anxiety and Neurosis Group, and the International Standard Randomised Controlled Trial Number Register) were also reviewed for eligible studies.

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

(a) Used a prospective pretreatment-posttreatment, quasi-randomized trial or randomized controlled trial design.

(b) Assessed the impact of antidepressant treatment or specified/manualized psychological intervention for perinatal depression. Hormonal pharmacological interventions, such as estrogen therapy, were excluded. Nonspecific psychosocial interventions, such as peer support groups, were excluded. Interventions that did not

explicitly target depressive symptoms, such as smoking cessation programs, were also excluded.

(c) Subjects were limited to women with unipolar depression (defined by diagnostic criteria or symptom severity) during pregnancy or the postpartum period (defined as the 12 months following the birth of a child).

(d) Reported outcomes for depressive symptoms using a validated self-report or clinician-administered measure.

(e) Reported sufficient outcomes to allow for the calculation of either uncontrolled or controlled effect sizes.

A flow chart depicting the search process and exclusion of studies is presented in Figure 1. After removal of duplicates, the search procedure yielded 1447 studies. The 152 studies whose abstracts indicated evaluation of an intervention for antenatal or postnatal depression were obtained and reviewed for inclusion. Of these 152 studies, 122 were excluded for the following reasons: described an intervention without reporting results of an evaluation (n = 10), study design was not a prospective pretest-posttest, quasi-randomized or randomized controlled trial (e.g., retrospective chart reviews, n = 14), prevention studies that included women without elevated depressive symptoms or a diagnosis of depression (n = 30), no pharmacological or psychological intervention (e.g., exercise, hormonal, and social support interventions, n = 47), population was not restricted to women during the perinatal period or with unipolar depression (n = 7), studies that reported only qualitative data (n = 3) or insufficient data for the calculation of effect sizes (n = 8), and secondary sources for included studies that did not report outcomes relevant to the analyses (n = 3). The remaining 30 articles, representing 27

studies, were eligible for inclusion in the meta-analysis. When a secondary source was available for a given study, the primary source was used to calculate the effect size unless reported data was insufficient. Sufficient outcome measures for calculation of effect sizes representing change from pretreatment to posttreatment were reported in 25 studies, and 14 studies reported sufficient outcome measures for calculation of effect sizes representing the difference between treatment and control conditions at posttreatment.

Coding of Studies

All studies were coded for: intervention type (antidepressant medication vs. psychotherapy vs. combined), study design (open trial vs. quasi-randomized trial vs. randomized controlled trial), type of control group (treatment as usual vs. enhanced treatment as usual vs. waiting list vs. active), population (antepartum vs. postpartum), outcome measure, whether the study required a clinician-verified diagnosis of depression for inclusion, treatment length (weeks), and percent attrition. Studies including a psychological intervention were also coded for therapeutic orientation, whether therapy was conducted individually or in a group format, and the location in which therapy was administered (clinic vs. home vs. school). As a majority of studies included the Edinburgh Postnatal Depression Scale (EPDS) as an outcome measure, this measure was used to calculate effect sizes for all studies reporting EPDS outcomes. For studies that did not include the EPDS as an outcome measure, or for which effect sizes could not be calculated using the reported EPDS values, the primary outcome measure was used.

Effect sizes and moderators were coded by the first author. Three of the variables included in the moderator analyses were also coded by a second rater, who was trained in the coding scheme and utilized a written coding manual. Observed agreement was 27/27

for each variable, or 100%. As all of the variables that were coded for use in the moderator analyses were objective variables that were explicitly specified in the studies and required minimal judgment on the part of the coder, this reliability check was considered adequate.

Analyses

Two separate analyses were conducted. The first analysis assessed the change in depressive symptoms from pretreatment to posttreatment using the standardized mean gain score for all treatment groups. To differentiate these analyses from those comparing treatment to control conditions, these within group effect sizes will be referred to as "uncontrolled effect sizes" (Feske & Chambless, 1995). To ensure the independence of included effect sizes, a single effect size was calculated on the basis of the overall mean and standard deviation of the total group of treated subjects in studies that included more than one active treatment. Uncontrolled effect sizes were calculated by dividing the mean change from pretreatment to posttreatment by the pooled standard deviation of the difference score, corrected for upward bias using Hedges' g (Hedges, 1981):

$$g = c_m \left[\frac{M_{Post} - M_{Pre}}{SD_{Diff}/\sqrt{(2(1-r))}} \right]$$

where the pooled standard deviation is defined as

$$SD_{Diff} = \sqrt{SD_{Pre}^2 + SD_{Post}^2} - 2rSD_{Pre}SD_{Post}$$

and

$$c_m = 1 - \frac{3}{4(n-1)}$$

Uncontrolled effect sizes were calculated so that positive effect sizes represented a

decrease in depressive symptoms from pretreatment to posttreatment.

None of the studies included in the meta-analysis reported the pretest-posttest correlation for the sample or data that would allow this value to be calculated. Following the recommendations of Lipsey and Wilson (2001), the test-retest reliability of the measures was used as a proxy for the pretest-posttest correlation. These values were estimated from published validation studies of each measure; when multiple test-retest reliabilities were available for a single measure, r was computed as the weighted mean of the reliabilities. As it is likely that these values are inflated estimates of the pretest-posttest correlation, the overall analyses were also conducted using values of 0.3, 0.5, and 0.8 as estimates of low, medium, and high correlations, respectively. There were no substantive differences between these effect sizes, suggesting that using the test-retest correlations as a proxy for this value would not impact the results of the analyses.

The second analysis compared the efficacy of active treatments to control conditions using the standardized mean group difference. To differentiate these effect sizes from those defined previously, these between group effect sizes will be referred to as "controlled effect sizes" (Feske & Chambless, 1995). Means and standard deviations for the total group of treated subjects were calculated for all studies in which multiple active treatments were compared to a control group in order to ensure the independence of effect sizes. Controlled effect sizes were calculated by dividing the difference between treatment and control means by the pooled standard deviation, corrected for upward bias using Hedges' g (Hedges, 1981):

$$g = c_m \left[\frac{M_T - M_C}{SD_P} \right]$$

where the pooled standard deviation is defined as

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

and c_m is defined as described above. Controlled effect sizes were calculated so that positive effects represented lower scores in the intervention group compared to the control group.

The heterogeneity of effect sizes was examined using the *Q* statistic and the I^2 index. Significant *Q* statistics indicate that the observed range of effect sizes is significantly larger than would be expected based on within-study variance. While a significant *Q* statistic indicates heterogeneous effect sizes, nonsignificant *Q* statistics should be interpreted with caution, as heterogeneous effect sizes may yield a nonsignificant *Q* value due to low power. The I^2 value indicates the proportion of variance in effect sizes accounted for by between-study variance. The index has a range from 0 to 100; Higgins and colleagues (2003) suggest that 25, 50 and 75% I^2 values indicate low, medium and high levels of hetereogeneity, respectively.

When analyses indicated significant heterogeneity among effect sizes, exploratory analyses were conducted to assess for moderators of effect size. Categorical moderators were assessed using an analysis of variance (ANOVA) of mixed-effects models for each variable hypothesized to influence the effect size. Meta-regression analyses were conducted to assess the effects of continuous moderators. Two types of moderators were included in the analyses. The first were variables that reflected elements of the research design of included studies; significant findings of moderation would indicate that differences in effect sizes could be attributed to methodological variability among studies

(e.g., whether studies required a clinician-verified diagnosis for inclusion). The second were variables related to characteristics of interventions; for example, pharmacotherapy versus psychotherapy and differences between psychological interventions of different therapeutic orientations. For studies assessing psychotherapeutic interventions, we also assessed whether characteristics of the intervention (including the mode of administration, location the intervention was delivered, and therapeutic orientation) were related to effect size.

Calculations of weighted mean effect sizes, heterogeneity, and moderators were conducted using Comprehensive Meta-Analysis, version 2.2.046 (Borenstein, Hedges, Higgins, & Rothstein, 2005). A decision was made to estimate overall effect sizes using random effects models, as it was presumed that the included studies represent a distribution of true intervention effects. Fixed effect models assume that variability in effect sizes is due to random error within studies, and that there is a common true effect size across all studies. The overall effect size represents the estimate of the true effect size for the population of studies, but is not generalizable beyond the sample of included studies. In contrast, random effects models assume that variability in effect sizes is due to both random error within studies and systematic variability between studies – the true effect size is allowed to vary across studies. The overall effect size represents the estimated average of the true effect sizes, and results can be generalized to studies not included in the analysis. Considerable heterogeneity of effect sizes was expected given the differences in study design, interventions, and samples across the included studies. As the *Q* statistic is underpowered in cases of small sample size, random effects models were estimated regardless of the observed heterogeneity.

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

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

Results

Study Characteristics

Table 1 displays characteristics of the 27 studies included in the analyses. Of the included studies, 9 were open trials (33%), 2 were quasi-randomized trials (7%), and 16 were randomized controlled trials (59%). Nineteen studies assessed psychological interventions (70%), four assessed pharmacological interventions (15%), and four assessed interventions including psychological and pharmacological components (15%). Most studies targeted postpartum depression (*n* = 22, 81%), four targeted antenatal depression (15%), and one study included subjects across the perinatal period. Length of treatment ranged from 6 to 16 weeks, with an average of 10 weeks. Interpersonal psychotherapy (IPT) was the most common psychological intervention (*n* = 11, 41%), followed by cognitive-behavioral (CBT) interventions (*n* = 9, 33%); other interventions included non-directive counseling (*n* = 3), a Mother-Infant Therapy Group (*n* = 1), a CBT-oriented psychoeducational group (*n* = 1), manualized supportive psychotherapy (*n* = 2), and psychodynamic therapy (*n* = 2). Nine studies assessed group interventions (33%), 4 included home-based interventions (15%), and one study included a school-based intervention.

Table 2 presents characteristics of the included studies indicative of their methodological quality. Given the range of designs that were included in the analyses, methodological quality was not quantified or used in the weighting of effect sizes. Of the 27 included studies, 19 included intent-to-treat analyses. As the average attrition rate was 21%, it is likely that completer analyses represent biased outcomes. In 8 studies, subjects were not excluded if they were currently receiving additional treatment for depression, all of which were studies of psychological interventions. In some trials, rates of concurrent

antidepressant use were quite high (e.g., 54% across both intervention and control groups, Klier, Muzik, Rosenblum, & Lenz, 2001). While some studies specifically assessed the potential effect of concurrent antidepressant use on outcome (e.g., Honey, Bennett, & Morgan, 2002), the inclusion of subjects receiving adjunctive pharmacological treatment in trials assessing psychological interventions is a significant limitation of the research base for these interventions. Among the 23 studies that included a psychological intervention, most studies provided information regarding therapist characteristics and use of therapy manuals. Fifteen studies provided information about therapist training, 13 indicated that therapists received regular ongoing supervision, and 9 studies assessed for adherence to the treatment model. Among the 8 studies that included a pharmacological intervention, only 3 included a placebo condition in which both patients and clinicians were blind to medication status.

The included studies vary widely in the demographic characteristics and variability of their samples. Most studies were conducted in the United States (*n* = 13, 48%), six studies were conducted in Australia (22%), three studies in the United Kingdom (11%), and the remaining studies were conducted in Austria (*n* = 1), Canada (*n* = 1), France (*n* = 1), and Sweden (*n* = 2). Fourteen studies (52%) reported at least some information regarding the race, ethnicity, or national origin of subjects. In the 10 studies (37%) that reported demographic information that provided sufficient data regarding the racial composition of their samples, the percentage of subjects who identified as racial/ethnic minorities ranged from 0% (Klier et al., 2001) to 100% (Miller et al., 2008), with a mean of 45.6% and standard deviation of 32.7%. Twenty-three studies (85%) reported information regarding the marital status of subjects. The percentage of single

subjects in these studies ranged from 0% (O'Hara et al., 2000, who required women to be married of living with a partner for 6 months or more to be eligible to participate) to 72.7% (Miller et al., 2008). Twenty studies (74%) reported information regarding parity or the number of children in subjects' households. Of the 14 studies that reported the percentage of primiparous subjects, this value ranged from 25% (O'Hara et al., 2000) to 85.1% (Wiklund, Mohlkert, & Edman, 2010), with a mean of 55.8% and standard deviation of 17.7%.

Uncontrolled Effect Sizes

Table 3 presents the results of the random effects model for uncontrolled effect sizes, representing results from 25 studies. These values should be interpreted with caution, as they reflect within-study change and cannot differentiate between reductions in symptoms that occurred as a result of the intervention versus the passage of time. All studies demonstrated significant positive effects, indicating improvement over pretreatment scores, with Hedges' *g* ranging from 0.78 to 4.39. Two studies had SAMD values greater than 2.58. Visual inspection of the scree plot of the rank-ordered SAMD scores suggested that the value for the study by Grote and colleagues (2009) was consistent with the overall distribution of SAMD scores, while the study by Appleby and colleagues (1997) was discrepant. This study was excluded from subsequent analyses; the average effect size excluding this outlier was 1.54 (95% CI 1.34-1.73, $p < 0.001$).

The *Q* statistic indicated that there was significant heterogeneity among the effect sizes ($p < 0.001$). The I^2 value indicated a high level of heterogeneity, with 86% of the variance in effect sizes attributable to between-study variance. The fail-safe value was 9102, far exceeding the tolerance level for an unlikely number of non-significant studies

(130). The funnel plot was slightly asymmetric; trim-and-fill procedures suggested that two studies with effect sizes to the right (more strongly positive) of the mean were missing. The corrected average effect size was 1.61 (95% CI 1.40-1.81). This adjusted value suggests that if the included studies do reflect a publication bias, it is in the direction of underestimating the true effect size of the interventions.

As the magnitude of uncontrolled effect sizes is difficult to interpret, a separate meta-analysis was conducted to determine the average level of depressive symptoms at posttreatment. A random effects model was used to calculate the overall mean for the 15 studies that used the EPDS as an outcome measure. The average score at posttreatment was 8.62 (95% CI 7.66-9.58), which is below the commonly used cutoff of 11-13 considered indicative of clinically significant depressive symptoms (Cox, Chapman, Murray, & Jones, 1996). A second random effects model was used to calculate the overall mean for the 5 studies that used the Hamilton Depression Rating Scale as an outcome measure. The average score at posttreatment was 6.12 (95% CI 2.90-9.33), which is also below the cutoff of 7 commonly considered indicative of symptom remission (Frank et al., 1991).

Moderator Analyses: Uncontrolled Effect Sizes

As both the *Q* statistic and I^2 index indicated significant heterogeneity of effect sizes, exploratory analyses of potential moderators were conducted. These analyses assessed whether effect sizes differed on the basis of characteristics of the included studies and interventions. As the reporting of sample characteristics was inconsistent across studies, none of these variables were assessed as potential moderators. Subgroups including only one study were excluded from moderator analyses.

Study characteristics. Four characteristics of the included studies were assessed as potential moderators: study design, type of sample, whether a clinician-verified diagnosis of a depressive disorder was required for inclusion in the study, and outcome measure (see Table 4). No significant differences in the average effect size were found among the three types of study designs, between studies assessing interventions for antenatal depression versus postpartum depression, or between studies that did and did not require a clinician-verified diagnosis for inclusion. Studies for which the effect size was calculated using the BDI had significantly smaller effect sizes (g = 1.10, n = 2) than studies for which the effect size was calculated using the EPDS (g = 1.62, n = 16) or HDRS (g = 1.65, n = 5).

Intervention variables. Two characteristics of the interventions were assessed for potential moderation (see Table 4). Studies including three types of interventions were compared: pharmacological, psychological, and combined (pharmacological + psychological). There were no significant differences in effect sizes among the three major types of interventions. Meta-regression analysis was used to assess the relationship between length of treatment and effect size. There was a trend for a positive association of length of treatment with effect size; however, this result did not reach significance (slope = 0.03, p = 0.07).

For studies that included a psychological intervention, three characteristics of the intervention were assessed for moderation: method of administration (individual vs. group), location of administration (clinic vs. home), and therapeutic orientation. Studies in which therapy was administered individually had significantly larger effect sizes (g = 1.79, n = 12) than those utilizing a group therapy format (g = 1.23, n = 7). There was a

trend (p = 0.08) toward home-administered treatments (g = 2.33, n = 2) having larger effect sizes than clinic-based treatments (g = 1.64, n = 16).

Four analyses were conducted to assess whether inclusion of two well-established therapeutic interventions, cognitive-behavioral therapy (CBT) or interpersonal psychotherapy (IPT), was a moderator of effect size. First, studies were categorized as either including or not including each approach. There was a trend for studies that included one of these interventions having larger effect sizes (g = 1.61, n = 18) than studies that included other psychological interventions (g = 1.11, n = 2). There were not significant differences between studies that included CBT compared to those that did not (including IPT interventions) or between studies that included IPT compared to those that did not (including CBT interventions). Finally, the effect sizes of studies including CBT and IPT were compared to one another. No study included both a CBT and an IPT intervention. There was not a significant difference in the average effect size of the two interventions.

Controlled Effect Sizes

Table 5 presents the results of the random effects model for controlled effect sizes, representing results from 14 studies. All effect sizes were positive, indicating superiority of treatment to control conditions, with Hedges' g ranging from 0.31 to 2.33. Two studies had SAMD values greater than 2.58. Visual inspection of the scree plot of the rank-ordered SAMD scores indicated that the value for the study by Milgrom and colleagues (2005) was consistent with the overall distribution of SAMD scores, while the study by Chabrol and colleagues (2002) was discrepant. This study was excluded from subsequent analyses. The average effect size, excluding the outlier, was 0.65 (95% CI

0.45-0.86, $p < 0.001$). Cohen's *U3* metric provides an intuitive metric through which to interpret the magnitude of this effect size; this value indicates that 74% of subjects in treatment conditions could be expected to report levels of depressive symptoms lower than the mean of the control group (Lipsey & Wilson, 2001).

The *Q* statistic indicated that there was significant heterogeneity among the effect sizes ($p < 0.05$). The I^2 value indicated a medium level of heterogeneity, with 43% of the variance in effect sizes attributable to between-study variance. The fail-safe N was 229, which substantially exceeds the tolerance level for an unlikely number of non-significant studies (75). The funnel plot was symmetric and the trim-and-fill procedures suggested no missing studies.

Moderator Analyses: Controlled Effect Sizes

As both the *Q* statistic and I^2 value indicated heterogeneity among effect sizes, exploratory analyses of potential moderators were conducted to assess whether effect sizes differed on the basis of study or intervention characteristics. Subgroups including only one study were excluded from moderator analyses.

Study characteristics. As for uncontrolled effect sizes, target population, diagnostic status, and outcome measure were assessed as potential moderators (see Table 6). In addition, the average effect size for studies utilizing different control groups (treatment as usual vs. enhanced treatment as usual vs. waiting list control) was assessed. The only significant moderator of effect size was the target population of the study; interventions for antenatal depression had significantly larger effect sizes (g =1.18, n = 2) than those for postpartum depression (g = 0.57, n = 11).

Intervention variables. As only one study assessing pharmacological treatment

was included in this analysis, it was not possible to compare the average effect size for pharmacological vs. psychological interventions. Length of treatment was assessed for moderation using meta-regression analysis; treatment length was not significantly associated with effect size (slope = 0.05, p = 0.12).

For the studies assessing psychological interventions, three potential moderators were assessed (see Table 6). There were no significant differences among average effect sizes with respect to the method of administration of therapy (individual vs. group vs. combined). There was a trend for studies that included clinic-based interventions to have larger effect sizes (g = 0.73, n = 9) than studies that included home-based interventions (g = 0.38, n = 2). Studies that included an IPT intervention were compared to those that included a CBT intervention. As only one study included an intervention representing different therapeutic orientation, analyses comparing IPT and CBT separately to all other treatments combined were not conducted. Studies that included an IPT intervention had significantly larger effect sizes (g = 0.96, n = 5) than those that included a CBT intervention (g = 0.40, n = 6).

Discussion

The results of these analyses provide evidence for the efficacy of a range of interventions for perinatal depression. All studied interventions demonstrated symptomatic improvement from pretreatment to posttreatment, with posttreatment means for both the EPDS and HDRS below cutoffs for clinically significant depressive symptoms. All interventions also demonstrated superiority to control conditions, with an overall effect size in the moderate range. The overall effect size (g = 0.65) is comparable to that found in a meta-analysis of the efficacy of psychological treatments for adult

depression, which reported an average posttreatment effect size of 0.67 (Cuijpers, Smit, Bohlmeijer, Hollon, & Andersson, 2010).

Our finding indicating the superiority of IPT to CBT for the treatment of perinatal depression has important implications for both clinicians and researchers. This is consistent with the findings of Bledsoe and Grote (2006), who reported that IPT interventions were associated with greater decreases in symptoms from pretreatment to posttreatment; our study is the first to find that IPT results in a greater reduction in symptoms compared to control conditions, as well. It is possible that this finding reflects a true difference in the efficacy of a specific form of psychotherapy for this population. In describing their adaptations of IPT for postpartum depression, Stuart and O'Hara (1995) noted that the focus on interpersonal problem areas, particularly role transitions and interpersonal disputes, may be particularly well-suited to the problems women experience during the perinatal period, such as disruptions in their interpersonal relationships. While a possible interpretation of this finding is that IPT is a more efficacious intervention for depression among this population, our findings may also have resulted from characteristics of the included studies unrelated to the interventions themselves. Studies assessing an IPT intervention were more likely to report utilization of a therapy manual; the implementation of manualized IPT for this population is likely facilitated by the ready availability of a treatment manual containing specific adaptations for postpartum depression (O'Hara, Stuart, Gorman, & Wenzel, 2000). It is unclear whether studies of CBT interventions did not utilize a specific therapy manual or whether these studies simply followed widely accepted and available manuals for CBT for depression (e.g., Beck, Rush, Shaw, & Emery, 1987). Studies assessing a CBT intervention were also

more likely to include other interventions, either as an explicit element of the intervention itself (e.g., Honey, Bennett, & Morgan, 2002¸ whose intervention included educational, cognitive-behavioral, and relaxation components), or because a single effect size was calculated for studies including multiple active treatments for the purpose of our analyses (e.g., Cooper, Murray, Wilson, & Romaniuk, 2003, who included CBT, psychodynamic, and nondirective counseling interventions). Given the possibility that other aspects of study design may have been confounded with the therapeutic orientation of the interventions included in the different studies, our results regarding the superiority of IPT to CBT should be interpreted with caution. Further research evaluating the efficacy of well-defined cognitive-behavioral interventions for this population, particularly trials in which faithfully administered CBT and IPT protocols are compared directly to one another, is necessary to establish whether IPT is truly a more effective intervention for perinatal depression. Such studies would be greatly beneficial given the low acceptability of antidepressant medication in this population and preference for non-pharmacologic treatments, including psychotherapy (Kim et al., 2011).

Another important moderator identified in these analyses is the superiority of individually-administered therapeutic interventions to those conducted in a group format. Uncontrolled effect sizes were larger for studies in which therapy was conducted on an individual basis; a comparable pattern was observed for controlled effect sizes, although this difference did not reach significance due to the smaller number of included studies. There has been great interest in the potential use of group treatments for perinatal depression, and some have suggested that the format may be particularly well-suited for this population because it provides an opportunity for normalizing women's experiences

and decreasing social isolation (Mulcahy, Reay, Wilkinson, & Owen, 2009). The results of these analyses suggest that these benefits may not be sufficient to lead to a reduction in depressive symptoms comparable to that which can be achieved through individual psychotherapy.

Our results suggest that further assessment of home-based interventions is necessary in order to determine the efficacy of these programs. There was a trend for depressive symptoms to decrease more over time in studies that incorporated home-based interventions; however, there was also a trend for greater effects in studies that incorporated clinic-based interventions with respect to the superiority of treated groups to control conditions. Both of these results should be interpreted with caution, as only two studies of home-based interventions were included in these moderator analyses. Further research, ideally trials in which comparable interventions administered either in a clinic or home setting can be directly compared, is necessary to determine whether the location in which therapy is conducted effects the efficacy of these interventions.

There was a trend for longer treatments to have larger uncontrolled effect sizes, but treatment length was not associated with controlled effect sizes. As uncontrolled effect sizes do not distinguish between the effects of interventions and decreases in symptoms over time, this raises the concern that the apparent effects of treatment may simply reflect a natural decrease in symptoms over time. There is evidence that, for most women, high levels of depressive symptoms naturally remit over the perinatal period (Heron, O'Connor, Evans, Golding, & Glover, 2004). One study included in these analyses found that three active treatment groups were only superior to routine primary care at 4.5 months postpartum; by 9 months postpartum the depressive symptoms of the

control group had declined to levels comparable to those observed in the treated groups, and differences among the groups remained nonsignificant through five years postpartum (Cooper, Murray, Wilson & Romaniuk, 2003). However, other included studies found that treatment groups continued to be superior to control groups at follow-up times ranging from 3-6 months posttreatment (Grote et al., 2009; Honey, Bennett & Morgan, 2002; Mulcahy, Reay, Wilkinson & Owen, 2009).

Unfortunately, the number of studies that have included follow-up assessments of treated subjects is too small for a meta-analysis of long-term outcomes. Five studies in which an intervention was compared to a control group reported follow-up outcomes, with the timing of follow-up assessments ranging from 3 months to 5 years posttreatment. An initial analysis suggested that the treated group remained superior at the first posttreatment follow-up assessment (which ranged from 3 to 6 months posttreatment), with a positive effect size in the moderate range. However, the SAMD values indicated that two studies were significant outliers, and the fail-safe N for this analysis indicated that the number of studies with null results necessary to reduce the effect size to zero was below the tolerance limit. Further assessment of long-term outcomes for subjects in controlled trials of interventions for perinatal depression are necessary to assess whether the benefits of treatment for perinatal depression are maintained over time.

The present meta-analysis has several limitations. Reflecting the status of the field, the number of included studies is relatively small, particularly for the analysis of controlled effect sizes. Moderator analyses were likewise limited by the small number of included studies. The quality of studies assessing interventions among this population is somewhat limited (see also Cuijpers, Brännmark, & van Straten, 2008; Dennis, 2004). Of

particular concern is the fact that 8 studies of psychological interventions did not exclude subjects who were receiving pharmacological treatment, and rates of concurrent antidepressant use were quite high in some of these studies. As this raises the possibility that the purported effects of psychotherapy in these studies could have been the result of pharmacological interventions, future research is necessary both to establish the separate effects of psychotherapy and pharmacotherapy (by excluding subjects receiving concurrent treatment from research) and to explicitly assess the efficacy of combined psychological and pharmacological treatments. Compared with psychological interventions, there has been relatively little assessment of the efficacy of antidepressant medication in this population. Of the seven studies that included antidepressant treatment, three were open trials and two compared a combined treatment to either psychological or pharmacological monotherapy. Given the ethical concerns regarding the use of no-treatment control groups in treatment studies with this population, further studies in which pharmacological treatments are directly compared to psychological or combined interventions are necessary to address the relative efficacy of these interventions. Barber (2009) also suggested that there is room for large scale, relatively well controlled, naturalistic studies for examining the efficacy of psychotherapy, as the field does not have the resources to conduct all the RCTs that need to be conducted. Finally, because psychotherapies are large packages, research that focuses on specific interventions from a package could be used to determine which specific interventions are particularly useful (e.g., Barber et al., 1996, DeRubeis & Feeley, 1990; Webb, DeRubeis, & Barber, 2010).

In summary, these meta-analyses demonstrated that a range of interventions are effective in the reduction of perinatal depressive symptoms. Reductions in symptoms

from prettest to posttreatment are large, and symptom levels at posttreatment are below cutoffs for clinically significant symptoms. These interventions reliably lead to moderate reductions in depressive symptoms compared to control groups. Interestingly, there was initial evidence that IPT may be more effective than CBT, although further research is necessary to establish whether this can be attributed to methodological differences between studies assessing the two forms of psychotherapy. Relatively few studies of antidepressant medication for this population have been conducted compared to psychological interventions, and overall most interventions have not been assessed in comparison to control or other active treatment conditions. Given the prevalence of perinatal depression and the negative outcomes associated with depressive symptoms during this period, the identification of effective and acceptable treatments for this population is vitally necessary. Although more research is needed to confirm and extend the results of these meta-analyses, these results suggest a range of interventions for further investigation as treatments for this disorder.

References

References marked with an asterisk indicate studies included in the meta-analyses. References marked with two asterisks indicate secondary sources and were only used when insufficient outcome data was presented in the primary article.

*Appleby, L., Warner, R., Whitton, A., & Faragher, B. (1997). A controlled study of fluoxetine and cognitive-behavioural counselling in the treatment of postnatal depression. *British Medical Journal, 314,* 932-936.

Barber, J.P. (2009). Towards a working through of some core conflicts in psychotherapy research. *Psychotherapy Research, 19,* 1-12.

Barber, J.P., & Crits-Christoph, P. (1996). Development of an adherence/competence scale for dynamic therapy: Preliminary findings. *Psychotherapy Research, 6,* 81-94.

Beck, A.T., Rush, A.J., Shaw, B.F., Emery, G. (1979). *Cognitive Therapy of Depression.* New York: Guilford Press.

Beal, D.J., Corey, D.M., & Dunlap, W.P. (2002). On the bias of Huffcutt and Arthur's (1995) procedure for identifying outliers in the meta-analysis of correlations. *Journal of Applied Psychology, 87,* 583-589.

Bennett, H.A., Einarson, A., Taddio, A., Koren, G., & Einarson, T.R. (2004a). Depression during pregnancy: Overview of clinical factors. *Clinical Drug Investigation, 24,* 157-179.

Bennett, H.A., Einarson, A., Taddio, A., Koren, G., & Einarson, T.R. (2004b). Prevalence of depression during pregnancy: Systematic review. *Obstetrics & Gynecology, 103,* 698-709.

Bledsoe, S.E., & Grote, N.K. (2006). Treating depression during pregnancy and the postpartum: A preliminary meta-analysis. *Research on Social Work Practice, 16*(2), 109-120.

Borenstein, M., Hedges, L., Higgins, J., & Rothstein, H. (2005). Comprehensive Meta-analysis Version 2, Biostat, Englewood NJ.

Chabrol, H., Teissedre, F., Armitage, J., Danel, M., & Walburg, V. (2004). Acceptability of psychotherapy and antidepressants for postnatal depression among newly delivered mothers. *Journal of Reproductive and Infant Psychology, 22,* 5-12.

*Chabrol, H., Teissedre, F., Saint-Jean, M., Teisseyre, N., Rogé, B., & Mullet, E. (2002). Prevention and treatment of post-partum depression: A controlled randomized study on women at risk. *Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences, 32*(6), 1039-1047.

*Clark, R., Tluczek, A., & Wenzel, A. (2003). Psychotherapy for postpartum depression: A preliminary report. *American Journal of Orthopsychiatry, 73,* 441-454.

*Cohen, L.S., Viguera, A.C., Bouffard, S.M., Nonacs, R.M., Morabito, C., Collins, M.H., & Ablon, J.S. (2001). Venlafaxine in the treatment of postpartum depression. *Journal of Clinical Psychiatry, 62*(8), 592-596.

Collins, N.L., Dunkel-Schetter, C., Lobel, M., & Scrimshaw, S.C. (1993). Social support in pregnancy: Psychosocial correlates of birth outcomes and postpartum depression. *Journal of Personality and Social Psychology, 65,* 1243-1258.

Cooper, P.J. & Murray, L. (1995). Course and recurrence of postpartum depression: Evidence for the specificity of the diagnostic concept. *British Journal of Psychiatry, 166,* 191-195.

**Cooper, P.J., & Murray, L. (1997). The impact of psychological treatments of postpartum depression on maternal mood and infant development. In L. Murray & P. Cooper (Eds.) *Postpartum Depression and Child Development.* New York: Guilford Press. 201-220.

*Cooper, P.J., Murray, L., Wilson, A., & Romaniuk, H. (2003). Controlled trial of the short- and long-term effect of psychological treatment of post-partum depression 1: Impact on maternal mood. *British Journal of Psychiatry, 182*(5), 412-419.

Cox, J.L., Chapman, G., Murray, D., & Jones, P. (1996). Validation of the Edinburgh Post-Natal Depression Scale (EPDS) in non-postnatal women. *Journal of Affective Disorders, 39,* 185-189.

*Craig, E., Judd, F., & Hodgins, G. (2005). Therapeutic group programme for women with postnatal depression in rural victoria: A pilot study. *Australasian Psychiatry, 13,* 291-295.

Cuijpers, P., Brännmark, J.G., & van Straten, A. (2008). Psychological treatment of postpartum depression: A meta-analysis. *Journal of Clinical Psychology, 64,* 103-118.

Cuijpers, P., Smit, F., Bohlmeijer, E., Hollon, S.D., & Andersson, G. (2010). Efficacy of cognitive-behavioural therapy and other psychological treatments for adult depression: Meta-analytic study of publication bias. *British Journal of Psychiatry, 196,* 173-178.

Cuijpers, P., van Straten, A., Andersson, G., & van Oppen, P. (2008). Psychotherapy for depression in adults: A meta-analysis of comparative outcome studies. *Journal of Consulting and Clinical Psychology,* 76, 909-922.

Cuijpers, P., Smit, F., & van Straten, A. (2007). Psychological treatments of subthreshold depression: A meta-analytic review. *Acta Psychiatrica Scandinavica, 115,* 434–441.

Dawes, M., & Chowienczyk, P.J. (2001). Pharmacokinetics in pregnancy. *Best Practice & Research Clinical Obstetrics & Gynaecology, 15,* 819-826.

Dennis, C.L., & Hodnett, E.D. (2007). Psychosocial and psychological interventions for treating postpartum depression. *Cochrane Database of Systematic Reviews.*

Dennis, C.L. (2004). Treatment of postpartum depression, part 2: A critical review of nonbiological interventions. *Journal of Clinical Psychiatry, 65,* 1252-1265.

Dennis, C.L., & Ross, L. (2005). Relationships among infant sleep patterns, maternal fatigue, and development of depressive symptomatology. *Birth, 32,* 187-193.

Dennis, C.L., Ross, L.E., & Grigoriadis, S. (2007). Psychosocial and psychological interventions for treating antenatal depression. *Cochrane Database of Systematic Reviews.*

DeRubeis, R.J., & Feeley, M. (1990). Determinants of change in cognitive therapy for depression. *Cognitive Therapy and Research, 14,* 469-482.

Duval, S., & Tweedie, R. (2000). Trim and fill: a simple funnel-plot-based method of testing and adjusting for publication bias in meta-analysis. *Biometrics, 56,* 455-463.

Epperson, C.N. (1999) Postpartum major depression: Detection and treatment. *American*

Family Physician, 59, 2247-2254.

Epperson, C.N., Anderson, G.M., & McDougle, C.J. (1997). Sertraline and breast-feeding. *New England Journal of Medicine, 336,* 1189-1190.

Epperson, C.N., Jatlow, P.I., Czarkowski, K., & Anderson, G.M. (2003). Maternal fluoxetine treatment in the postpartum period: Effects on platelet serotonin and plasma drug levels in breastfeeding mother-infant pairs. *Pediatrics, 112,* e425.

Feske, U., & Chambless, D.L. (1995). Cognitive behavioral versus exposure only treatment for social phobia: A meta-analysis. *Behavior Therapy, 26,* 695-720.

Forray, A., Mayes, L.C., Magriples, U., & Epperson, C.N. (2009). Prevalence of post-traumatic stress disorder in pregnant women with prior pregnancy complications. *Journal of Maternal and Fetal Neonatal Medicine, 22,* 522-527.

Frank, E., Prien, R.F., Jarrett, R.B., Keller, M.B., Kupfer, D.J., Lavori, P.W., Rush, A.J., & Weissman, M.M. (1991). Conceptualization and rationale for consensus definitions of terms in major depressive disorder: Remission, recovery, relapse, and recurrence. *Archives of General Psychiatry, 48,* 851-855.

*Freeman, M.P., Hibbeln, J.R,, Wisner, K.L., Watchman, M., & Gelenberg, A.J. (2006). An open trial of omega-3 fatty acids for depression in pregnancy. *Acta Neuropsychiatrica, 18*(1), 21-24.

Gavin, N.I., Gayner, B.N., Lohr, K.N., Meltzer-Brody, S., Gartlehner, G., & Swinson, T. (2005). Perinatal depression: A systematic review of prevalence and incidence. *Obstetrics & Gynecology, 106*(5), 1071-1083.

**Goodman, S.H., Broth, M.R., Hall, C.M., & Stowe, Z.N. (2008). Treatment of postpartum depression in mothers: Secondary benefits to the infants. *Infamt Mental Health Journal, 29,* 492-513.

Grace, S.L., Evindar, A., & Stewart, D.E. (2003). The effect of postpartum depression on child cognitive development and behavior: A review and critical analysis of the literature. *Archives of Women's Mental Health,* 263-274.

Grote, N.K., Bridge, J.A., Gavin, A.R., Melville, J.L., Iyengar, S., & Katon, W.J. (2010). A meta-analysis of depression during pregnancy and the risk of preterm birth, low birth weight, and intrauterine growth restriction. *Archives of General Psychiatry, 67*(10), 1012-1024.

*Grote, N.K., Swartz, H.A., Geibel, S.L., Zuckoff, A., Houck, P.R., & Frank. E. (2009). A randomized controlled trial of culturally relevant, brief interpersonal psychotherapy for perinatal depression. *Psychiatric Services, 60,* 313-321.

Halbreich, U., & Karkun, S. (2006). Cross-cultural and social diversity of prevalence of postpartum depression and depressive symptoms. *Journal of Affective Disorders, 91,* 97-111.

Hedges, L.V. (1981). Distribution theory for Glass's estimator of effect size and related estimators. *Journal of Educational Statistics, 6,* 107-128.

Heron, J., O'Connor, T.G., Evans, J., Golding, J., & Glover, V. (2004). The course of anxiety and depression through pregnancy and the postpartum in a community sample. *Journal of Affective Disorders, 80,* 65-73.

Higgins, J.P., Thompson, S.G., Deeks, J.J., & Altman, D.G. (2003). Measuring inconsistency in meta-analyses. *British Medical Journal, 327,* 557-560.

Hoffman, S., & Hatch, M.C. (1996). Stress, social support and pregnancy outcome: A reassessment based on recent research. *Pediatric and Perinatal Epidemiology, 10,* 380-405.

*Honey, K.L., Bennett, P., & Morgan, M. (2002). A brief psycho-educational group intervention for postnatal depression. *British Journal of Clinical Psychology, 41,* 405-409.

Hostetter, A., Stowe, Z.N., Strader, J.R., McLaughlin, E., & Llewellyn, A. (2000). Dose of selective serotonin uptake inhibitors across pregnancy: Clinical implications. *Depression & Anxiety, 11*, 51-57.

Huffcutt, A.I., & Arthur, W., Jr. (1995). Development of a new outlier statistic for meta-analytic data. *Journal of Applied Psychology, 80*, 327-334.

Joffe, R., Sokolov, S., & Streiner, D. (1996). Antidepressant treatment of depression: A meta-analysis. *Canadian Journal of Psychiatry, 41*, 613-616.

Kammerer, M., Taylor, A., Glover, V. (2006). The HPA axis and perinatal depression: A hypothesis. *Archives of Women's Mental Health, 9,* 187-196.

Kim, D.R., O'Reardon, J.P., & Epperson, C.N. (2010). Guidelines for the management of depression during pregnancy. *Current Psychiatry Reports, 12,* 279-281.

Kim, D.R., Sockol, L., Barber, J.P., Moseley, M., Lamprou, L., O'Reardon, J.P., & Epperson, C.N. (2011). A survey of patient acceptability of repetitive Transcranial Magnetic Stimulation (TMS) during pregnancy. *Journal of Affective Disorders, 129*, 385-390.

*Klier, C.M., Muzik, M., Rosenblum, K.L., & Lenz, G. (2001). Interpersonal psychotherapy adapted for the group setting in the treatment of postpartum

depression. *Journal of Psychotherapy Practice and Research, 10*, 124-131.

Lipsey, M.W., & Wilson, D. (2001). *Practical Meta-Analysis*. Applied Social Research Methods Series (Vol. 49). Thousand Oaks, CA: SAGE Publications, Inc.

*Meager, I., & Milgrom, J. (1996). Group treatment for postpartum depression: A pilot study. *Australian and New Zealand Journal of Psychiatry, 30*(6), 852-860.

*Milgrom, J., Negri, L.M., Gemmill, A.W., McNeil, M., & Martin, P.R. (2005). A randomized controlled trial of psychological interventions for postnatal depression. *British Journal of Clinical Psychology, 44*(4), 529-542.

*Miller, L., Gur, M., Shanok, A., & Weissman, M. (2008). Interpersonal psychotherapy with pregnant adolescents: Two pilot studies. *Journal of Child Psychology and Psychiatry, 49*(7), 733-742.

*Misri, S., Reebye, P., Corral, M., & Mills, L. (2004). The use of paroxetine and cognitive-behavioral therapy in postpartum depression and anxiety: A randomized controlled trial. *Journal of Clinical Psychiatry, 65*(9), 1236-1241.

*Mulcahy, R., Reay, R.E., Wilkinson, R.B., & Owen, C. (2009). A randomised controlled trial for the effectiveness of group interpersonal psychotherapy for postnatal depression. *Archives of Women's Mental Health, 13*, 125-139.

**Murray, L., Cooper, P.J., Wilson, A., & Romaniuk, H. (2003). Controlled trial of the short- and long-term effect of psychological treatment of post-partum depression 2: Impact on the mother-child relationship and child outcome. *British Journal of Psychiatry, 182*, 420-427.

*O'Hara, M.W., Stuart, S., Gorman, L.L., & Wenzel, A. (2000). Efficacy of interpersonal psychotherapy for postpartum depression. *Archives of General Psychiatry, 57,* 1039-1045.

O'Hara, M.W., & Swain, A.M.. (1996). Rates and risk of postpartum depression: A meta-analysis. *International Review of Psychiatry, 8,* 37-54.

*Pearlstein, T.B., Zlotnick, C., Battle, C.L., Stuart, S., O'Hara, M.W., Price, A.B., Grause, M.A., & Howard, M. (2006). Patient choice of treatment for postpartum depression: A pilot study. *Archives of Women's Mental Health,* 303-308.

*Prendergast, J., & Austin, M.P. (2001). Early childhood nurse-delivered cognitive behavioural counselling for post-natal depression. *Australasian Psychiatry, 9*(3), 255-259.

*Reay, R., Fisher, Y., Robertson, M., Adams, E. & Owen, C. (2006). Group interpersonal psychotherapy for postnatal depression: A pilot study. *Archives of Women's Mental Health, 9*, 31-39.

Rosenthal, R. (1979). The "file drawer problem" and tolerance for null results. *Psychological Bulletin, 86*, 638-641.

Rosenthal, R. (1991). *Meta-analytic procedures for social research*. Newbury Park, CA: Sage.

*Spinelli, M.G. (1997). Interpersonal psychotherapy for depressed antepartum women: A pilot study. *American Journal of Psychiatry, 154,* 1028-1030.

*Spinelli, M.G., & Endicott, J. (2003). Controlled clinical trial of interpersonal psychotherapy versus parenting education program for depressed pregnant women. *The American Journal of Psychiatry, 160*(3), 555-562.

*Stowe, Z.N., Casarella, J., Landrey, J., & Nemeroff, C.B. (1995). Sertraline in the treatment of women with postpartum major depression. *Depression, 3,* 49-55.

*Stuart, S., & O'Hara, M.W. (1995). Interpersonal therapy for postpartum depression: A treatent program. *Journal of Psychotherapy Practice and Research, 4*, 18-29.

*Suri, R., Burt, V.K., & Altshuler, L.L. (2005). Nefazodone for the treatment of postpartum depression. *Archives of Women's Mental Health, 8*(1), 55-56.

Webb, C.A., DeRubeis, R.J., & Barber, J.P. (2010). Therapist adherence/competence and treatment outcome: A meta-analytic review. *Journal of Consulting and Clinical Psychology,78,* 200-211.

*Wickberg, B., & Hwang, C.P. (1996). Counselling of postnatal depression; A controlled study on a population based Swedish sample. *Journal of Affective Disorders, 39*, 209-216.

*Wiklund, I., Mohlkert, P., & Edman, G. (2010). Evaluation of a brief cognitive intervention in patients with signs of postnatal depression: A randomized controlled trial. *Acta Obstetricia et Gynecologica, 89,* 1100-1104.

Wisner, K.L., Perel, J.M., & Wheeler, S.B. (1993). Tricyclic dose requirements across pregnancy. *American Journal of Psychiatry, 150,* 1541-1542.

*Yonkers, K.A., Lin, H., Howell, H.B., Health, A.C., & Cohen, L.S. (2008). Pharmacologic treatment of postpartum women with new-onset major depressive disorder: A randomized controlled trial with paroxetine. *Journal of Clinical Psychiatry, 69,* 659-665.

Table 1

Characteristics of Studies Assessing Interventions for Perinatal Depression

Study	Country	N	Study Design	Control Type	Population	Intervention	Dx	Measure	Tx Length	% Attrition	Treatment	Format	Admin
Appleby et al. (1997)	UK	87	RCT		POST	COMB	Y	EPDS	12	30	CBT	IND	CLIN
Chabrol et al. (2002)	FR	48	RCT	TAU	POST	THER	Y	EPDS	12	0	CBT	IND	HOME
Clark et al. (2003)	US	39	QRT	WL	POST	THER	Y	BDI	12	10	M-ITG + IPT	COMB	CLIN
Cohen et al. (2001)	US	15	OT		POST	MED	Y	HDRS	8	33	Venlafaxine		
Cooper et al. (2003)	UK	193	RCT	TAU	POST	THER	Y	EPDS	10	17	CBT + NDC + PDT	IND	HOME
Craig et al. (2005)	AUS	16	OT		POST	THER	N	EPDS	9	13	CBT	GRP	CLIN
Freeman et al. (2008)	US	59	RCT		MIXED	COMB	Y	EPDS	8	34	SUPP + Omega-3 Fatty Acids	IND	CLIN
Grote et al. (2009)	US	53	RCT	TAU+	ANTE	THER	Y	EPDS	NS	13	IPT	IND	CLIN
Honey et al. (2002)	UK	45	RCT	TAU	POST	THER	N	EPDS	8	9	PEG	GRP	CLIN
Klier et al. (2001)	AUST	17	OT		POST	THER	Y	EPDS	12	35	IPT	GRP	CLIN
Meager & Milgrom (1996)	AUS	20	RCT	WL	POST	THER	N	EPDS	10	40	CBT	GRP	CLIN
Milgrom et al. (2005)	AUS	120	RCT	TAU	POST	THER	Y	BDI	12	37	CBT + NDC	COMB	CLIN
Miller et al. (2008)	US	11	OT		ANTE	THER	Y	EPDS	12	0	IPT	GRP	SCHOOL
Misri et al. (2004)	CAN	35	RCT		POST	COMB	Y	EPDS	12	9	CBT	IND	CLIN
Mulcahy et al. (2009)	AUS	50	RCT	TAU	POST	THER	Y	EPDS	8	15	IPT	GRP	CLIN
O'Hara et al. (2000)	US	99	RCT	WL	POST	THER	Y	HDRS	12	18	IPT	IND	CLIN
Pearlstein et al. (2006)	US	23	QRT		POST	COMB	Y	EPDS	12	22	IPT	IND	CLIN
Prendergast & Austin (2001)	AUS	37	RCT	TAU+	POST	THER	Y	EPDS	6	0	CBT	IND	HOME

Study	Country	N	Study Design	Control Type	Population	Intervention	Dx	Measure	Tx Length	% Attrition	Treatment	Format	Admin
Reay et al. (2006)	AUS	18	OT		POST	THER	Y	EPDS	8	6	IPT	GRP	CLIN
Spinelli (1997)	US	13	OT		ANTE	THER	Y	EPDS	16	31	IPT	IND	CLIN
Spinelli & Endicott (2003)	US	50	RCT	ACT	ANTE	THER	Y	EPDS	16	24	IPT	IND	CLIN
Stowe et al. (1995)	US	26	OT		POST	MED	Y	BDI	8	19	Sertraline		
Stuart & O'Hara (1995)	US	12	OT		POST	THER	Y	HDRS	NS	42	IPT	IND	CLIN
Suri, Burt, & Altshuler (2005)	US	4	OT		POST	MED	Y	HDRS	8	25	Nefazodone		
Wickberg & Hwang (1996)	SWE	48	RCT	TAU	POST	THER	N	MADRS	6	15	NDC	IND	COMB
Wiklund, Mohlkert, & Edman (2010)	SWE	66	RCT	TAU	POST	THER	N	EPDS	7	0	CBT	IND	CLIN
Yonkers et al. (2008)	US	70	RCT	PLA	POST	MED	Y	HDRS	8	56	Paroxetine		

Note. Dx = required clinician-administered diagnostic assessment, Tx Length = treatment length in weeks, Admin = Location of therapy administration, AUS = Australia, AUST = Austria, CAN = Canada, FR = France, SWE = Sweden, UK = United Kingdom, US = United States, OT = open trial, QRT = quasi-randomized trial, RCT = randomized controlled trial, TAU = treatment as usual, TAU+ = enhanced treatment as usual, WL = waiting list, ACT = active control, PLA = placebo, ANTE = antepartum, POST = postpartum, MIXED = antepartum + postpartum, MED = antidepressant medication, COMB = combined antidepressant medication + psychotherapy, THER = psychotherapy, BDI = Beck Depression Inventory, EPDS = Edinburgh Postnatal Depression Scale, HDRS = Hamilton Depression Rating Scale, MADRS = Montgomery-Asberg Depression Rating Scale, CBT = cognitive-behavioral therapy, IPT = interpersonal psychotherapy, M-ITG = mother-infant therapy group, NDC = nondirective counseling, PEG = psychoeducational group, PDT = psychodynamic therapy, SUPP = manualized supportive psychotherapy, COMB = combined individually + group administered, IND = individually-administered, GRP = group-administered, CLIN = clinic-based intervention, COMB = combined clinic- + home-based intervention, HOME = home-based intervention, SCHOOL = school-based intervention

Table 2

Methodological Quality of Studies Assessing Interventions for Perinatal Depression

					RCTs	Psychological Interventions					Pharmacologic Interventions	
Study	ITT	Char Sample	Concurr Tx	Blind Assess	Random	Spec Ther	Manual	Trng	Super	Adher	Blind Clinic	Blind Pt
Open Trials												
Cohen et al. (2001)	+	+	NS	NS							–	–
Craig, Judd, & Hodgins (2005)	–	–	NS	NS		+	+	+	–	–		
Klier et al. (2001)	+	+	Y	–		+	+	+	–	–		
Miller et al. (2008)	+	+	N	+		+	+	–	–	–		
Reay et al. (2006 US)	+	+	Y	+		+	+	+	+	+		
Spinelli, (1997)	+	+	NS	NS		–	–	–	–	–		
Stowe et al. (1995)	–	+	NS	–							–	–
Stuart & O'Hara (1995)	–	+	Y	NS		–	–	–	–	–		
Suri, Burt, & Altshuler (2005)	–	–	NS	+							–	–
Quasi-Randomized Trials												
Clark, Tluczek, & Wenzel (2003)	–	+	Y	NS		+	+	+	+	–		
Pearlstein et al. (2006)	–	+	N	NS		+	–	+	+	–	–	–
Randomized Controlled Trials												
Appleby et al. (1997)	+	+	NS	+	+	+	–		–	+	+	+
Chabrol et al. (2002)	+	+	N	–	–	+	+		+	+		

					RCTs	Psychological Interventions					Pharmacologic Interventions	
Study	ITT	Char Sample	Concurr Tx	Blind Assess	Random	Spec Ther	Manual	Trng	Super	Adher	Blind Clinic	Blind Pt
Cooper et al. (2003)	+	+	NS	+	−	+	+		+	+		
Freeman et al. (2008)	+	+	N	NS	−	−	+		−	−	+	+
Grote et al. (2009)	+	+	N	NS	+	+	+		+	+		
Honey et al. (2002)	+	+	Y	NA	+	+	−		−	−		
Meager & Milgrom (1996)	−	+	Y	NA	−	+	−		−	−		
Milgrom et al. (2005)	+	+	N	NA	−	+	+		+	+		
Misri et al. (2004)	+	+	N	−	+	+	+		−	−	−	−
Mulcahy et al. (2009)	+	+	Y	+	+	+	+		+	+		
O'Hara et al. (2000)	+	+	NS	−	+	+	+		+	+		
Prendergast & Austin (2001)	+	+	Y	NS	+	+	+		+	+		
Spinelli, & Endicott (2003)	+	+	N	NS	+	+	+		+	+		
Wickberg & Hwang (1996)	−	+	N	+	+	+	−		+	+		
Wiklund, Mohlkert, & Edman (2010)	+	+	Y	NA	+	+	−		−	−		
Yonkers et al. (2008)	+	+	N	+	+						+	+

Note. ITT = report intent-to-treat analyses, Char Sample = specify characteristics of sample, Concurr. Tx = subjects allowed to receive concurrent antidepressant or psychological treatment, Blind Assess. = clinician-administered diagnostic measures conducted by independent evaluator blind to treatment condition, Random. = specification of method of randomization, Spec. Ther. = specify therapist characteristics, Manual = specify use of therapy manual, Trng = describe therapist training, Super. = describe therapist supervision, Adher. = indicate therapy was assessed for adherence to model, Blind Clinic. = clinician blind to treatment status, Blind Pt. = patient blind to treatment status, + = yes, − = no, NA = not applicable, NS = not specified, Y = yes, N = no

Table 3

Random Weighted Uncontrolled Effect Sizes from Studies Assessing Interventions for Perinatal Depression

Study	*n*	Hedges' *g*	SAMD		
Appleby et al. (1997)	87	4.39***	10.70[1]		
Chabrol et al. (2002)	18	1.92***	0.46		
Clark, Tluczek, & Wenzel (2003)	24	1.03***	-1.30		
Cohen et al. (2001)	15	1.68***	0.03		
Craig, Judd, & Hodgins (2005)	14	1.44***	-0.33		
Freeman et al. (2008)	51	1.29***	-1.12		
Grote et al. (2009)	25	3.00***	2.82		
Honey et al. (2002)	23	0.78***	-1.76		
Klier et al. (2001)	17	1.35***	-0.52		
Meager & Milgrom (1996)	6	0.95*	-0.59		
Miller et al. (2008)	11	0.91***	-0.98		
Misri et al. (2004)	35	1.72***	0.17		
Mulcahy et al. (2009)	23	1.56***	-0.20		
O'Hara et al. (2000)	48	2.05***	1.15		
Pearlstein et al. (2006)	23	2.66***	2.02		
Prendergast & Austin (2001)	17	2.60***	1.61		
Reay et al. (2006)	18	1.61***	-0.09		
Spinelli, & Endicott (2003)	21	1.20***	-0.88		
Spinelli, (1997)	13	1.28***	-0.55		
Stowe et al. (1995)	19	1.20***	-0.83		
Stuart & O'Hara (1995)	6	1.39***	-0.23		
Suri, Burt, & Altshuler (2005)	3	1.67***	0.00		
Wickberg & Hwang (1996)	20	0.83***	-1.53		
Wicklund, Mohlkert, & Edman (2010)	66	2.03***	-0.40		
Yonkers et al. (2008)	17	1.42***	0.92		
	k	Hedges' *g*	95% CI	*Q*(*df*)	I^2
Total (all studies)	25	1.66***	1.41-1.91	280.99(24)***	91.46
Total (outlier excluded)	24	1.54***	1.34-1.73	159.22(23)***	85.56
Total (trim-and-fill correction)		1.61	1.40-1.81	199.09	

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

[1] Outlier excluded from subsequent analyses.

Table 4

Analyses of Moderation for Uncontrolled Effect Sizes

Moderator	*n*	Hedges' *g*	95% CI	*Q*(*df*)	*p*
Study Design				1.86(2)	0.39
Open Trial	9	1.39***	1.22-1.56		
Quasi-Randomized Trial	2	1.81*	0.22-3.41		
RCT	13	1.62***	1.32-1.92		
Population				0.003(1)	0.96
Antepartum	4	1.57***	0.76-2.38		
Postpartum	19	1.55***	1.33-1.77		
Clinician-Verified Diagnosis				1.82(1)	0.18
Yes	19	1.61***	1.40-1.82		
No	5	1.22***	0.69-1.75		
Measure				15.44(2)	0.000***
BDI	2	1.10***	0.94-1.27		
EPDS	16	1.62***	1.34-1.89		
HDRS	5	1.65***	1.35-1.95		
Intervention Type				1.09(2)	0.58
Combination	3	1.83***	1.17-2.49		
Medication	4	1.46***	1.22-1.69		
Therapy	17	1.51***	1.22-1.79		
Therapy Type				6.93(1)	0.008**
Group	7	1.23***	0.95-1.51		
Individual	12	1.79***	1.48-2.10		
Therapy Location				3.16(1)	0.08
Clinic	16	1.64***	1.36-1.92		
Home	2	2.33***	1.63-3.03		
CBT/IPT vs. Other Psychological				3.72(1)	0.05*
CBT/IPT	18	1.61***	1.34-1.89		
Other	2	1.11***	0.67-1.54		
CBT vs. Other Psychological				0.14(1)	0.71
CBT	7	1.63***	1.16-2.10		
Other	13	1.52***	1.21-1.83		
IPT vs. Other Psychological				0.23(1)	0.64
IPT	11	1.61***	1.25-1.97		
Other	9	1.49***	1.13-1.85		
CBT vs. IPT				0.003(1)	0.95
CBT	7	1.63***	1.16-2.10		
IPT	11	1.61***	1.25-1.97		

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 5

Random Weighted Controlled Effect Sizes from Studies Comparing Interventions for Perinatal Depression to Control Conditions

Study	*n*	Hedges' *g*	SAMD		
Chabrol et al. (2002)	48	2.33***	5.38[1]		
Clark, Tluczek & Wenzel (2003)	35	0.46	-0.89		
Cooper et al. (2003)	184	0.39*	-2.42		
Grote et al. (2009)	53	1.35***	2.14		
Honey, Bennett & Morgan (2002)	45	0.36	-1.35		
Meager & Milgrom (1996)	12	0.97	0.31		
Milgrom et al. (2005)	192	0.31	-2.95		
Mulcahy et al. (2009)	50	0.63*	-0.47		
O'Hara et al. (2000)	99	1.19***	2.13		
Prendergast & Austin (2001)	37	0.31	-1.35		
Spinelli & Endicott (2003)	38	0.96**	0.57		
Wickberg & Hwang (1996)	41	0.81*	0.14		
Wiklund, Mohlkert, & Edman (2010)	33	0.51*	-0.73		
Yonkers et al. (2008)	31	0.60	-0.44		
	k	Hedges' *g*	95% CI	*Q(df)*	I^2
Total	14	0.76***	0.50-1.03	40.75(13)***	68.10
Total (excluding outlier)	13	0.65***	0.45-0.86	21.12(12)*	43.19

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

[1] Outlier excluded from subsequent analyses.

Table 6

Analyses of Moderation for Controlled Effect Sizes

Moderator	*n*	Hedges' *g*	95% CI	*Q*(*df*)	*p*
Control Type				3.72(2)	0.16
TAU	6	0.45***	0.27-0.63		
TAU+	2	0.84	-2.04		
Wait List	3	0.93***	0.45-1.41		
Population				6.24(1)	0.01*
Antepartum	2	1.18***	0.74-1.62		
Postpartum	11	0.57***	0.38-0.75		
Clinician-Verified Diagnosis				0.003(1)	0.96
Yes	10	0.65***	0.40-0.90		
No	3	0.66***	0.30-1.02		
Measure				3.87(2)	0.14
BDI	2	0.35*	0.02-0.68		
EPDS	8	0.63***	0.38-0.87		
HDRS	2	0.97***	0.41-1.53		
Therapy Type				3.41(2)	0.18
Combined	2	0.35*	0.02-0.68		
Group	3	0.55**	0.17-0.93		
Individual	7	0.78***	0.46-1.09		
Therapy Location				2.96(1)	0.09
Clinic	9	0.73***	0.45-1.00		
Home	2	0.38*	0.09-0.67		
CBT vs. IPT				8.81(1)	0.003**
CBT	6	0.40***	0.21-0.59		
IPT	5	0.96***	0.64-1.28		

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

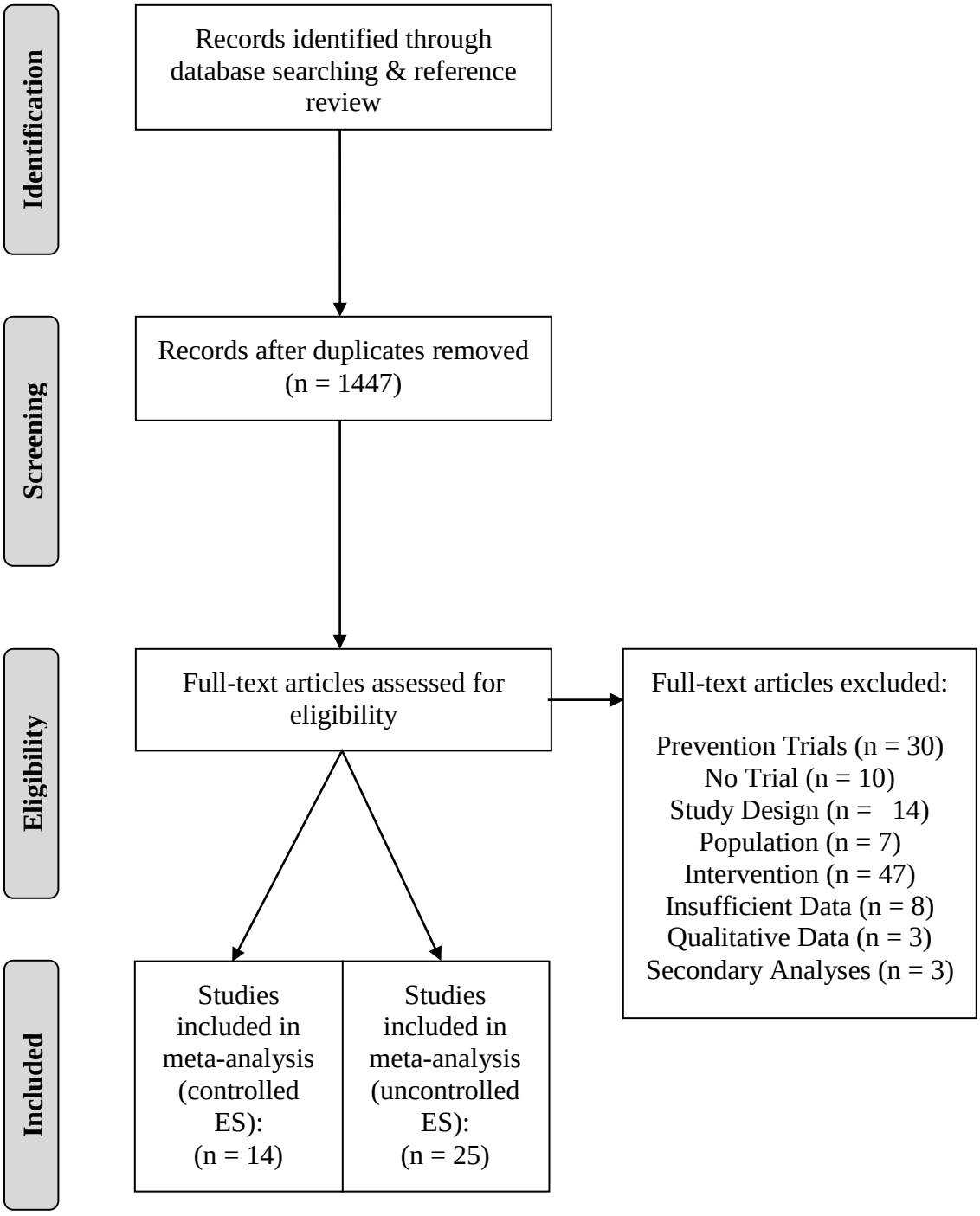

Figure 1. Flow chart illustrating the identification of included studies

Chapter 3:

Preventing Postpartum Depression: A Meta-Analytic Review

Abstract

This meta-analysis assessed the efficacy of a wide range of preventive interventions designed to reduce the severity of postpartum depressive symptoms or decrease the prevalence of postpartum depressive episodes. A systematic review identified 37 randomized or quasi-randomized controlled trials in which an intervention was compared to a control condition. Differences between treatment and control conditions in the level of depressive symptoms and prevalence of depressive episodes at 6 months postpartum were assessed in separate analyses. Depressive symptoms were significantly lower at post-treatment in intervention conditions as compared to control conditions, with an overall effect size in the small range after exclusion of outliers (Hedges' $g = 0.18$). There was a 27% reduction in the prevalence of depressive episodes in intervention conditions compared to control conditions at 6 months postpartum, $OR = 0.73$, after removal of outliers and correction for publication bias. Later timing of postpartum assessments was associated with smaller differences between intervention and control conditions in both analyses. Among studies that assessed depressive symptoms using the EPDS, higher levels of depressive symptoms at pre-treatment were associated with smaller differences in depressive symptoms between treatment and control conditions at 6 months postpartum. No other moderators were identified in either analysis. These findings suggest that interventions designed to prevent postpartum depression effectively reduce levels of postpartum depressive symptoms and decrease risk for postpartum depressive episodes.

Preventing Postpartum Depression: A Meta-Analytic Review

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

Postpartum depression is a specific mental disorder for which preventive interventions could yield dramatic benefits. Depression is one of the most common complications of childbearing; a meta-analytic review found that approximately 13% of women will experience a major depressive episode during the first postpartum year (O'Hara & Swain, 1996). According to the World Health Organization, depression is the leading cause of disability worldwide (WHO, 2012). Children of mothers with postpartum depression are at increased risk for long-term cognitive impairment, emotional difficulties, and behavioral problems (Grace, Evindar, & Stuart, 2003).

The context in which postpartum depression occurs provides unique opportunities for preventive interventions. Women with fewer financial resources may have greater access to healthcare during pregnancy than during other points in the lifespan; for example, in the United States, women are eligible for Medicaid during pregnancy and the

first 60 days postpartum (Centers for Medicare and Medicaid Services, 2012). More generally, pregnancy is a time of increased healthcare utilization, which provides opportunities for screening and intervention. Research has identified demographic groups at high risk for postpartum depression, such as minority women and women of low socioeconomic status, which may be used to target women at increased risk for the disorder (Beck, 1996; O'Hara & Swain, 1996). Finally, there is some evidence that preventive interventions may be more acceptable, particularly among African-American women, than treatment for depression (Crockett, Zlotnick, Davis, Payne & Washington, 2008).

A wide range of interventions for preventing postpartum depression have been assessed in randomized controlled trials. Many preventive interventions have modified treatments demonstrated to be effective for postpartum depression. For example, psychotherapy – particularly cognitive-behavioral and interpersonal psychotherapy – and antidepressant medication have all been shown to be effective in the treatment of postpartum depression (Sockol, Epperson, & Barber, 2011). Some studies have assessed whether implementation of these interventions before the onset of a depressive episode can effectively prevent the disorder (e.g., Austin et al., 2008; Wisner, Perel, Peindl, Hanusa, Findling & Rapport, 2001; Zlotnick, Capezza, & Parker, 2011). Non-therapeutic social support and educational interventions have also been assessed as preventive interventions (e.g., Dennis et al., 2009). Other research has investigated whether modifications to standard postpartum care, such as having women attend their first postpartum checkup at 1 week instead of 6 weeks postpartum, can reduce the incidence of depression after childbirth (Gunn, Lumley, Chondros & Young, 1998). Alternative

approaches to treatment, notably dietary supplements and hormonal interventions, have also been assessed as potential preventive interventions for postpartum depression (e.g., Lawrie, Hofmeyr, De Jager, Berk, Paiker & Viljoen, 1998; Llorente, Jensen, Voigt, Fraley, Berretta & Heird, 2003). Given the wide range of approaches that have been utilized in prevention research, a comprehensive review of the research in this area is needed to provide clinicians and researchers with important information regarding the absolute and relative efficacy of these interventions.

While a great number of reviews of the literature on the prevention of postpartum depression have been published, most of these reviews are qualitative in nature (e.g., Boath, Bradley, & Henshaw, 2005; Dennis, 2004a; Dennis, 2004b). Several quantitative systematic reviews have attempted to synthesize prior findings in this area. Lumley, Austin, and Mitchell (2004) reviewed studies initiated during pregnancy and the postpartum period; their meta-analysis found that only indicated postnatal interventions were associated with decreased risk for postpartum depression. This meta-analysis did not assess possible moderators of effect sizes. In another quantitative review, Dennis (2005) conducted a meta-analysis of 15 psychological and psychosocial interventions for preventing postpartum depression. These analyses found that prevention programs did not significantly reduce risk for postpartum depression. However, analyses of moderators suggested that interventions were more effective when they targeted women at increased risk, when they included a postnatal component, and when they were administered individually. In their review of hormonal interventions for preventing and treating postpartum depression, Dennis, Ross, and Herxheimer (2009) identified only one study in which hormones were utilized as a preventive intervention. Similarly, a review of

antidepressant prevention of postnatal depression identified only two studies in which medication was utilized for prevention, rather than treatment, of postpartum depression (Howard, Hoffbrand, Henshaw, Boath, & Bradley, 2009). A protocol for a review of dietary supplements for preventing postpartum depression has been published, but the review has yet to be conducted (Miller, Murray, Beckmann, Kent, & Macfarlane, 2011).

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

The present meta-analysis addresses several limitations of the above studies. We included a wide range of interventions, which allows for the direct comparison of the efficacy of different approaches. We included interventions other than antidepressant medication and psychotherapy, as there is evidence that women may prefer alternative treatments during pregnancy and the postpartum period (Uebelacker, Epstein-Lubow, Gaudiano, Tremont, Battle, & Miller, 2010). In order to assess whether these alternative

interventions are as effective as empirically supported treatments, we elected to include as wide a range of preventive interventions as was possible. We limited our analyses to those in which postpartum depression was assessed within the first 6 months postpartum. We assessed characteristics of included studies and interventions as potential moderators of effect size. We also included several studies that have been published since earlier meta-analyses were conducted. The goal of the current meta-analysis was to assess the efficacy of a range of preventive interventions for postpartum depression. We assessed both the level of depressive symptoms in treatment conditions compared to control conditions and the difference in the prevalence of depressive episodes at six months postpartum.

Method

Search Procedures and Selection of Studies

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

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

(a) Study design included intervention and control group(s). Both randomized and quasi-randomized controlled trials were eligible for inclusion.

(b) Authors specified that the goal of the intervention was to reduce postpartum depressive symptoms and/or the prevalence of postpartum major depressive episodes. Interventions that did not explicitly target depressive symptoms, such as smoking cessation programs, were excluded, even if authors reported outcome data for depressive symptoms and/or major depressive episodes. Interventions in which maternal depression was not the primary outcome of interest, such as studies of infant development, were excluded. Interventions designed to treat postpartum depression were excluded. Interventions were classified as treatment studies if all subjects met criteria for a major depressive episode at pre-treatment or if all subjects had depressive symptoms above a cutoff indicative of clinically significant depressive symptoms at pre-treatment.

(c) Intervention was initiated during pregnancy or within 4 weeks of childbirth.

(d) Reported outcomes for depressive symptoms and/or prevalence of depressive episodes between 1 and 6 months postpartum using a validated self-report or clinician-administered measure.

(e) Reported sufficient outcomes to allow for the calculation effect size(s).

A flow chart summarizing the search process and exclusion of studies is presented in Figure 1. After removal of duplicates, the search procedure yielded 797 studies, of which 117 studies were obtained and reviewed for inclusion. Of these 117 studies, 80 were excluded for the following reasons: 17 studies were excluded because the target outcome of the intervention was not depressive symptoms or depression diagnosis, 16 were excluded because they were not randomized or quasi-randomized controlled trials, 14 studies were excluded because they did not report outcome data or reported

insufficient data for the calculation of effect sizes, 11 were excluded because the intervention was initiated after 4 weeks postpartum, 5 were excluded because they did not include a postpartum assessment between 1 and 6 months postpartum, 4 were excluded because they were treatment studies in which subjects were selected on the basis of depressive symptoms and/or diagnosis, and 1 was excluded because the measure of depressive symptoms was not validated. Secondary manuscripts were identified for 12 studies; all original manuscripts provided sufficient information for coding and calculation of effect sizes so these were not utilized. The remaining 37 articles were eligible for inclusion in the meta-analysis. Twenty-four studies reported sufficient outcome measures for calculation of effect sizes representing the difference in depressive symptoms between treatment and control conditions at 6 months postpartum, and 28 studies reported sufficient outcome measures for calculation of effect sizes representing the difference in prevalence of depressive episodes at 6 months postpartum.

Coding of Studies

All studies were coded for intervention type (dietary supplement vs. educational vs. hormonal vs. medication vs. modified care vs. therapy vs. social support). Interventions were classified as educational when the intervention consisted of providing information, either verbal or written, regarding postpartum depression and accessing treatment without actively engaging participants in activities designed to change behavior or mood. Interventions were coded as therapy when they were clinician-led and participants were engaged in activities with a goal of modifying behavior, cognition, or mood. Interventions in which participants were provided with nonspecific support were coded as social support interventions. For moderator analyses, interventions were also

coded as biological interventions (dietary supplement, hormonal, and medication) or psychosocial interventions (educational, modified care, therapy, and social support) and as established treatments for postpartum depression (cognitive-behavioral therapy, interpersonal psychotherapy, and antidepressant medication) and non-established treatments for postpartum depression (dietary supplements, educational interventions, hormonal interventions, modified care, other psychotherapies, and social support).

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

treatment across treatment and control conditions.

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

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

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

Analyses

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

$$g = c_m \left[\frac{M_T - M_C}{SD_P} \right]$$

where the pooled standard deviation is defined as

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

and c_m is defined as

$$c_m = 1 - \frac{3}{4(n-1)}$$

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

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

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

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

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

models for each variable hypothesized to influence the effect size. Meta-regression analyses were conducted to assess the effects of continuous moderators.

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

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

maximize the variance available to assess the role of moderators.

Calculations of weighted mean effect sizes, heterogeneity, and moderators were conducted using Comprehensive Meta-Analysis, version 2.2.046 (Borenstein, Hedges, Higgins, & Rothstein, 2005). We estimated overall effect sizes using random effects models, based on the assumption that the included studies represent a distribution of true intervention effects. Considerable heterogeneity of effect sizes was expected given the differences in interventions and samples across the included studies. As the *Q* statistic is underpowered in cases of small sample size (Lipsey & Wilson, 2001), random effects models were estimated regardless of the observed heterogeneity.

Results

Characteristics of Included Studies

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

Characteristics of therapy interventions. Eighteen studies assessed therapeutic

interventions. One study assessed training in guided relaxation provided via videotape; this study was excluded from moderator analyses of therapy characteristics due to differences in the method of administration of the intervention. The remaining studies assessed cognitive-behavioral (n = 10), interpersonally-oriented (n = 5), and eclectic (n = 2) interventions. Studies included both group therapy (n = 10) and individually-administered therapy (n = 7). The average number of therapy sessions was 5.9 (SD = 3.0).

Methodological Quality

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

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

Methodological quality of pharmacological interventions. Five studies

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

Postpartum Depressive Symptoms

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

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

treatment conditions, compared to 1.57 in control conditions.

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

Moderator Analyses: Postpartum Depressive Symptoms

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

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

Intervention variables. Three characteristics of interventions for studies assessing psychotherapeutic interventions were assessed as potential moderators: therapeutic orientation, method of administration, and number of sessions. There were not enough studies representing other types of interventions to assess moderators for these interventions. No categorical characteristics of psychotherapeutic interventions were significant moderators of effect size. There was a trend for studies with more therapy sessions to have smaller effect sizes; slope = -0.04, $p = 0.06$.

Postpartum Depression Diagnosis

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

Results of tests for publication bias indicated potential bias in the included studies. The fail-safe *N* value was 147, which exceeds the tolerance limit of 140. The funnel plot was asymmetric (see Figure 3), and the trim-and-fill correction suggested 5

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

Moderator Analyses: Postpartum Depression Diagnosis

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

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

Discussion

Results of these meta-analyses suggest that a wide range of interventions may be

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

For both depressive symptoms and depression diagnosis, a later assessment was associated with a smaller difference between intervention and control conditions. This is consistent with the results of a meta-analysis of treatments for postpartum depression, which found that treatment length was associated with smaller effect sizes (Sockol, Epperson, & Barber, 2011). Moreover, it is consistent with evidence that postpartum depression tends to naturally remit over time (Heron et al., 2004). Given that the natural course of postpartum depression is for symptom severity to decrease over time, it is unsurprising that preventive interventions appear to be most efficacious when they are assessed early during the postpartum period. However, this should not be taken as an indication that preventive interventions are unnecessary. Given the adverse impact of depression on depressed women and their children, even a self-limiting depressive episode may be extremely distressing and increase the risk for long-term negative

outcomes.

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

Interestingly, we found that intervention type was not related to the effectiveness of treatments for either reducing depressive symptoms or preventing depressive episodes. A lack of social support is an established risk factor for postpartum depression (Beck, 1996). It may be that nonspecific social contact and support is sufficient for reducing risk for depression among this population and that the specific active elements of treatment are less important. However, further research assessing the efficacy of less well-studied interventions is necessary to determine whether our failure to identify moderators simply results from a lack of sufficient evidence. Given the small number of studies representing antidepressant medication and non-traditional interventions, particularly dietary supplements and hormonal interventions, further research is necessary to establish whether these approaches are truly equally efficacious.

One limitation of this meta-analysis was the use of uncontrolled effect sizes. This

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

While the number of studies included in these meta-analyses is comparable to other meta-analyses of preventive interventions (e.g., Cuijpers et al., 2008; Zalta, 2011), moderator analyses assessed small subgroups of studies. Because of this, moderator analyses should be interpreted with caution. This is particularly true for the analyses of intervention type. There were relatively few studies assessing antidepressant medication, dietary supplements, educational interventions, hormonal interventions, and social support programs. More research assessing the efficacy of these interventions is necessary in order to establish whether there are systematic differences between types of interventions. Similarly, psychotherapy was the only type of intervention for which enough studies were present to assess for potential moderation of specific aspects of the intervention. Further evaluation of other types of interventions would allow for similar questions to be asked of these interventions; for example, whether phone-based social

support programs have comparable efficacy to in-person support groups.

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

In summary, these analyses suggest that a wide range of interventions are effective in the prevention of postpartum depression. At six months postpartum, these interventions are associated with a 27% reduction in the prevalence of depressive episodes and a reduction in levels of depressive symptoms compared to control conditions. Effect sizes were larger in studies that assessed depression earlier in the postpartum period; this is consistent with natural remission of depressive symptoms over the course of the postpartum period. There were no differences between types of interventions, and different types of psychotherapeutic interventions appeared to have comparable efficacy. There were few studies assessing antidepressant medication and other non-therapeutic interventions; more research is necessary to assess whether these interventions are effective and to establish whether characteristics of other intervention

types are related to efficacy. Although more research is needed to confirm and extend the results of these meta-analyses, these results suggest that a wide range of interventions should be targeted for further investigation as preventive interventions for this disorder.

References

References marked with an asterisk (*) indicate studies included in the meta-analyses.

*Armstrong, K. L., Fraser, J. A., Dadds, M. R., & Morris, J. (1999). A randomized, controlled trial of nurse home visiting to vulnerable families with newborns. *Journal of Paediatric Child Health, 35*, 237-244.

*Austin, M. P., Frilingos, M., Lumley, J., Hadzi-Pavlovic, D., Roncolato, W., Acland, S., Saint, K., Segal, N., & Parker, G. (2008). Brief antenatal cognitive behavior therapy group intervention for the prevention of postnatal depression and anxiety: A randomized controlled trial. *Journal of Affective Disorders, 105*, 35-44.

Beal, D.J., Corey, D.M., & Dunlap, W.P. (2002). On the bias of Huffcutt and Arthur's (1995) procedure for identifying outliers in the meta-analysis of correlations. *Journal of Applied Psychology, 87*, 583-589.

Beck, C. T. (1996). Predictors of postpartum depression: An update. *Nursing Research, 50*, 275-285.

Boath, E., Bradley, E., & Henshaw, C. (2005). The prevention of postnatal depression: A narrative systematic review. *Journal of Psychosocial Obstetrics & Gynaecology, 26*, 185-192.

Borenstein, M., Hedges, L., Higgins, J., & Rothstein, H. (2005). Comprehensive Meta-analysis Version 2, Biostat, Englewood NJ.

*Brugha, T. S., Wheatley, S., Taub, N. A., Culverwell, A., Friedman, T., Kirwan, P., Jones, D. R., & Shapiro, D. A. (2000). Pragmatic randomized trial of antenatal

intervention to prevent post-natal depression by reducing psychosocial risk factors. *Psychological Medicine, 30,* 1273-1281.

Centers for Medicare and Medicaid Services (2012). Pregnant women. Retrieved from http://www.medicaid.gov/Medicaid-CHIP-Program-Information/By-Population/Pregnant-Women/Pregnant-Women.html

*Chabrol, H., Teissedre, F., Saint-Jean, M., Teisseyre, N., Roge, B., & Mullet, E. (2002). Prevention and treatment of post-partum depression: A controlled randomized trial on women at risk. *Psychological Medicine, 32,* 1039-1047.

Cox, J., Chapman, G., Murray, D. & Jones, P. (1996). Validation of the Edinburgh Post-Natal Depression Scale (EPDS) in non-postnatal women. *Journal of Affective Disorders, 39,* 185-189.

Crockett, K., Zlotnick, C., Davis, M., Payne, N., & Washington, R. (2008). A depression preventive intervention for rural low-income African-American pregnant women at risk for postpartum depression. *Archives of Women's Mental Health, 11,* 319-325.

Cuijpers, P., van Straten, A., Andersson, G., & van Oppen, P. (2008). Psychotherapy for depression in adults: A meta-analysis of comparative outcome studies. *Journal of Consulting and Clinical Psychology,* 76, 909-922.

Dennis, C. L. (2004a). Preventing postpartum depression part I: A review of biological interventions. *Canadian Journal of Psychiatry, 49,* 467-475.

Dennis, C. L. (2004b). Preventing postpartum depression part I: A critical review of nonbiological interventions. *Canadian Journal of Psychiatry, 49,* 526-538.

Dennis, C. L. (2005). Psychosocial and psychological interventions for prevention of postnatal depression: Systematic review. *British Medical Journal, 331*.

*Dennis, C. L., Hodnett, E., Kenton, L., Weston, J., Zupancic, J., Stewart, D. E., & Kiss. A. (2009). Effect of peer support on prevention of postnatal depression among high-risk women: Multisite randomized controlled trial. *British Medical Journal, 338.* .

Dennis, C. L., Ross, L., & Herxheimer, A. (2009) . Oestrogens and progestins for preventing and treating postpartum depression. *Cochrane Database of Systematic Reviews.*

Dozois, D. J. & Dobson, K. S. (2002). Depression. In *Handbook of Assessment and Treatment Planning for Psychological Disorders,* M. M. Antony & D. H. Barlow, Eds. New York, NY: Guilford Press. 259-299.

Duval, S., & Tweedie, R. (2000). Trim and fill: a simple funnel-plot-based method of testing and adjusting for publication bias in meta-analysis. *Biometrics, 56,* 455-463.

*Elliott, S. A., Leverton, T. J., Sanjack, M., Turner, H., Cowmeadow, P., Hopkins, J., & Bushnell, D. (2000). Promoting mental health after childbirth: A controlled trial of primary prevention of postnatal depression. *British Journal of Clinical Psychology, 39,* 223-241.

*Gao, L., Chan, S. W., & Sun, K. (2012). Effects of an interpersonal-psychotherapy-oriented childbirth education programme for Chinese first-time childbearing women at 3-molnth follow up: Randomized controlled trial. *International Journal of Nursing Studies, 49,* 274-281.

Gavin, N.I., Gayner, B.N., Lohr, K.N., Meltzer-Brody, S., Gartlehner, G., & Swinson, T. (2005). Perinatal depression: A systematic review of prevalence and incidence. *Obstetrics & Gynecology*, *106*(5), 1071-1083.

*Gorman, L. L. (1997). *Prevention of postpartum adjustment difficulties.* (Unpublished doctoral dissertation). University of Iowa, Iowa City.

Grace, S.L., Evindar, A., & Stewart, D.E. (2003). The effect of postpartum depression on child cognitive development and behavior: A review and critical analysis of the literature. *Archives of Women's Mental Health,* 263-274.

*Gunn, J., Lumley, J., Chondros, P., & Young, D. (1998). Does an early postnatal check-up improve maternal health: Results from a randomized trial in Australian general practice. *British Journal of Obstetrics & Gynecolgy*, *105,* 991-997.

*Hagan, R., Evans, S. F., & Pope, S. (2004). Preventing postnatal depression in mothers of very preterm infants: A randomized controlled trial. *British Journal of Obstetrics & Gynecology*, *111,* 641-647.

*Hayes, B. A., Muller, R., & Bradley, B. S. (2001). Perinatal depression: A randomized controlled trial of an antenatal educational intervention for primiparas. *Birth, 28,* 28-35.

Hedges, L.V. (1981). Distribution theory for Glass's estimator of effect size and related estimators. *Journal of Educational Statistics, 6,* 107-128.

Heron, J., O'Connor, T.G., Evans, J., Golding, J., & Glover, V. (2004). The course of anxiety and depression through pregnancy and the postpartum in a community sample. *Journal of Affective Disorders, 80,* 65-73.

Higgins, J.P., Thompson, S.G., Deeks, J.J., & Altman, D.G. (2003). Measuring inconsistency in meta-analyses. *British Medical Journal, 327,* 557-560.

*Ho, S., Heh, S., Jevitt, C. M., Huang, L., Fu, Y., & Wang, L. (2009). Effectiveness of a discharge education program in reducing the severity of postpartum depression: A randomized controlled evaluation study. *Patient Education and Counseling, 77,* 68-71.

Howard, L., Hoffbrand, S. E., Henshaw, C., Boath, L., & Bradley, E. (2009). Antidepressant prevention of postnatal depression. *Cochrane Database of Systematic Reviews.*

Huffcutt, A.I., & Arthur, W., Jr. (1995). Development of a new outlier statistic for meta-analytic data. *Journal of Applied Psychology, 80,* 327-334.

*Kozinszky, Z., Dudas, R. B., Devosa, I., Csatordai, S., Toth, E., Szabo, D., Sikovanyecz, J., Barabas, K., & Pal, A. (2012). Can a brief antepartum preventive group intervention help reduce postpartum depressive symptomatology? *Psychotherapy and Psychosomatics, 81,* 98-107.

*Lara, M. A., Navarro, C., & Navarrete, L. (2010). Outcome results of a psycho-educational intervention in pregnancy to prevent PPD: A randomized control trial. *Journal of Affective Disorders, 122,* 109-117.

*Lawrie, T. A., Hofmeyr, G. J., De Jager, M., Berk, M., Paiker, J., & Viljoen, E. (1998). A double-blind randomize placebo controlled trial of postnatal norethisterone enanthate: The effect on postnatal depression and serum hormones. *British Journal of Obstetrics & Gynecology, 105,* 1082-1090.

*Le, H., Perry, D. F., & Stuart, E. A. (2011). Randomized controlled trial of a preventive intervention for perinatal depression in high-risk Latinas. *Journal of Consulting and Clinical Psychology, 79,* 135-141.

Lipsey, M.W., & Wilson, D. (2001). *Practical Meta-Analysis*. Applied Social Research Methods Series (Vol. 49). Thousand Oaks, CA: SAGE Publications, Inc.

*Llorente, A. M., Jensen, C. L., Voigt, R. G., Fraley, J. K., Berretta, M. C., & Heird, W. C. (2003). Effect of maternal docosahexaenoic acid supplementation on postpartum depression and information processing. *American Journal of Obstetrics & Gynecology, 188,* 1348-1353.

*Logsdon, M. C., Birkimer, J. C., Simpson, T., & Looney, S. (2003). Postpartum depression and social support in adolescents. *Journal of Obstetric, Gynecologic, & Neonatal Nursing, 34,* 46-54.

Lumley, J., Austin, M. P., & Mitchell, C. (2004). Intervening to reduce depression after birth: A systematic review of the randomized trials. *International Journal of Technology Assessment in Health Care, 20,* 128-144.

* Marks, M. N., Siddle, K., & Warwick, C. (2003). Can we prevent postnatal depression? A randomized controlled trial to assess the effect of continuity of midwifery care on rates of postnatal depression in high-risk women. *Journal of Maternal-Fetal and Neonatal Medicine, 13,* 119-127.

*Meeker, C. H. (1985). *A preventive intervention for postpartum depression in primiparous women.* (Unpublished doctoral dissertation). University of Kansas, Lawrence, Kansas.

*Milgrom, J., Schembri, C., Ericksen, J., Ross, J., & Gemmill, A. W. (2011). Towards parenthood: An antenatal intervention to reduce depression, anxiety and parenting difficulties. *Journal of Affective Disorders, 130*, 385-394.

Miller, B. J., Murray, L., Beckmann, M. M., Kent, T., & Macfarlane, B. (2011). Dietary supplements for preventing postnatal depression (Protocol). *Cochrane Database of Systematic Reviews, 5.*

* Mokhber, N., Namjoo, M., Tara, F., Boskabadi, H., Rayman, M P., Ghayour-Mobarhan, M., Sahebkar, A., Majdi, M. R., Tavallaie, S., Azimi-Nezhad, M., Shakeri, M. T., Nematy, M.,Oladi, M., Mohammadi, M., & Ferns, G. (2011). Effect of supplementation with selenium on postpartum depression: a randomized double-blind placebo-controlled trial. *Journal of Maternal-Fetal and Neonatal Medicine, 24,* 104-108.

Mrazek, P. J., & Haggerty, R. J. (Eds.) (1994). *Reducing risks for mental disorders: Frontiers for preventive intervention research.* Washington, D.C.: National Academy Press.

* Muñoz, R. F., Le, H., Ippen, C. G., Diaz, M. A., Urizar, G. G., Soto, J., Mendelson, T., Delucchi, K., & Lieberman, A. F. (2007). Prevention of postpartum depression in low-income women: Development of the mamás y bebés/ mothers and babies course. *Cognitive and Behavioral Practice, 14*, 70-83.

Muñoz, R. F., Beardslee, W. R., & Leykin, Y. (2012). Major depression can be prevented. *American Psychologist, 67,* 285-295.

* Nalepka, C. D., & Coblentz, M. J. (1995). Incidence of postpartum depression following antepartal intervention. *Journal of Perinatal Depressiopn, 4*, 29-37.

National Research Council & Institute of Medicine. (2009). *Preventing mental, emotional, and behavioral disorders among young people: Progress and possibilities* (M. E. O'Connell, T. Boat, & K. E. Warner, Eds.). Washington, DC: National Academies Press.

* Ngai, F. W., Chan, S. W., & Ip, W. Y. (2009). The effects of a childbirth psychoeducation program on learned resourcefulness, maternal role competence and perinatal depression: A quasi-experiment. *International Journal of Nursing Studies, 46,* 1298-1306.

O'Hara, M.W., & Swain, A.M.. (1996). Rates and risk of postpartum depression: A meta-analysis. *International Review of Psychiatry, 8,* 37-54.

* Rees. (1995). Effect of relaxation with guided imagery on anxiety depression and self esteem in primiparas. *Journal of Holistic Nursing, 13,* 255-267.

Rosenthal, R. (1991). *Meta-analytic procedures for social research*. Newbury Park, CA: Sage.

*Shields, N., & Reid, M. (1997). Impact of midwife-managed care in the postnatal period: An exploration of psychosocial outcomes. *Journal of Reproductive & Infant Psycholgy, 15,* 91-109.

* Silverstein, M., Feinberg, E., Cabral, H., Sauder, S., Egbert, L., Schainker, E., Kamholz, K., Hegel, M., & Beardslee, W. (2011). Problem-solving education to prevent depression among low-income mothers of preterm infants: a randomized controlled pilot trial. *Archives of Women's Mental Health, 14,* 317-324.

* Small, R., Lumley, J., Donohue, L., Potter, A., & Waldonstrom, U. (2000). Randomized controlled trial of midwife led debriefing to reduce maternal depression after operative childbirth. *British Medical Journal, 321,* 1043-1047.

Sockol, L.E., Epperson, C. N., & Barber, J. P. (2011). A meta-analysis of treatments for perinatal depression. *Clinical Psychology Review, 31,* 839-849.

* Stamp, G. E., Williams, A. S., & Crowther, C. (1995). Evaluation of antenatal and postnatal support to overcome postpartum depression: A randomized, controlled trial. *Birth, 22,* 138-143.

Uebelacker, L. A., Epstein-Lubow, G., Gaudiano, B. A., Tremont, G., Battle, C. L., & Miller, I. W. (2010). Hatha yoga for depression: Critical review of the evidence for efficacy, plausible mechanisms of action, and directions for future research. *Journal of Psychiatric Practice, 16,* 22-33.

* Webster, J., Linnane, J., Roberts, J., Starrenburg, S., Hinson, J., & Dibley, J. (2003). Identify, Educate, and Alert trial: An intervention to reduce postnatal depression. *British Journal of Obstetrics & Gynecology, 110,* 842-846.

* Wisner, K. L., Perel, J. M., Peindl, K. S., Hanusa, B. H., Findling, R. L., & Rapport, D. (2001). Prevention of recurrent postpartum depression: A randomized clinical trial. *Journal of Clinical Psychiatry, 62,* 82-86.

* Wisner, K. L., Perel, J. M., Peindl, K. S., Hanusa, B. H., Piontek, C. M., & Findling, R. L. (2004). Prevention of postpartum depression: A pilot randomized clinical trial. The *American Journal of Psychiatry, 161,* 1290-1292.

* Wolman, W. L., Chalmers, B., Hofmeyr, G. J., & Nikodem, V. C. (1993). Postpartum depression and companionship in the clinical birth environment: A randomized controlled study. *American Journal of Obstetrics & Gynecology, 168*, 1388-1393.

World Health Organization. (2012). Depression. Retrieved from http://www.who.int/mental_health/management/depression/definition/en/index.html

Zalta, A. K. (2011). A meta-analysis of anxiety symptom prevention with cognitive-behavioral interventions. *Journal of Anxiety Disorders, 25*, 749-760.

* Zayas, L. H., McKee, M. D., & Jankowski, K. R. B. (2004). Adapting psychosocial intervention research to urban primary care environments: A case example. *Annals of Family Medicine, 2*, 504-508.

* Zlotnick, C., Johnson, S. L., Miller, I. W., Pearlstein, T., & Howard, M. (2001). Postpartum depression in women receiving public assistance: Pilot study of an interpersonal-therapy oriented group intervention. *American Journal of Psychiatry, 158*, 638-640.

* Zlotnick, C., Miller, I. W., Pearlstein, T., Howard, M., & Sweeney, P. (2006). A preventive intervention for pregnant women on public assistance at risk for postpartum depression. *American Journal of Psychiatry, 163*, 1443-1445.

* Zlotnick, C., Capezza, N. M., & Parker, D. (2011). An interpersonally based intervention for low-income pregnant women with intimate partner violence: a pilot study. *Archives of Women's Mental Health, 14,* 55-65.

Table 1

Characteristics of Included Studies

Study	Country	Intervention Type	Control Type	Intervention Timing	Prevention Type	Symptom Measure	Diagnosis Criteria	Postpartum Assessment Timing (Weeks)	Psychotherapy Orientation	Psychotherapy Administration	Number of Sessions
Armstrong et al. (1999)	AUS	Modified Care	TAU	POST	Selected	EPDS	EPDS > 12	6			
Austin et al. (2008)	AUS	Therapy	EDUC	PREG	Selected/ Indicated		MINI	16	CBT	Group	6
Brugha et al. (2000)	UK	Therapy	TAU	PREG	Selected/ Indicated		SADS	12	CBT	Group	6
Chabrol et al. (2002)	FR	Therapy	TAU	POST	Indicated	EPDS	EPDS > 11	4-6	CBT	Individual	1
Dennis et al. (2009)	CAN	Social Support	TAU	POST	Indicated	EPDS	EPDS > 12	24			
Elliott et al. (2000)	UK	Social Support	TAU	PREG	Selected		PSE	12			
Gao, Chan, & Sun (2012)	CHINA	Therapy	EDUC	PREG	Universal	EPDS		12	IPT	Group	2
Gorman (1997)	USA	Therapy	TAU	PREG	Selected/ Indicated	EPDS	SCID	24	IPT	Individual	5
Gunn et al. (1998)	AUS	Modified Care	TAU	POST	Universal	EPDS	EPDS ≥ 13	24			
Hagan, Evans, & Pope (2004)	AUS	Therapy	EDUC	POST	Selected		SADS	24	CBT	Group	6
Hayes, Muller, & Bradley (2001)	AUS	Educational	TAU	PREG	Universal	POMS		16-24			
Ho et al. (2009)	CHINA	Educational	TAU	POST	Universal	EPDS		12			
Kozinszky et al. (2012)	HUN	Therapy	EDUC	PREG	Universal		LQ ≥ 12	6-8	Eclectic	Group	4
Lara, Navarro, & Navarrete (2010)	MEX	Therapy	EDUC	PREG	Selected/ Indicated		SCID	16-24	Eclectic	Group	8
Lawrie et al. (1998)	S AFR	Hormonal	PLA	POST	Universal	EPDS	EPDS ≥ 12	12			
Le, Perry, & Stuart (2011)	USA	Therapy	TAU	PREG	Selected/ Indicated	BDI-II	BDI-II ≥ 20	16	CBT	Group	8

Study	Country	Intervention Type	Control Type	Intervention Timing	Prevention Type	Symptom Measure	Diagnosis Criteria	Postpartum Assessment Timing (Weeks)	Psychotherapy Orientation	Psychotherapy Administration	Number of Sessions
Llorente et al. (2003)	USA	Dietary Supplement	PLA	POST	Universal	BDI-II		16			
Logsdon et al. (2003)	USA	Social Support	TAU	PREG	Selected	CES-D		6			
Marks, Siddle, & Warwick (2003)	UK	Modified Care	TAU	PREG	Selected/ Indicated	EPDS	SCID	12			
Meeker (1985)	USA	Social Support	TAU	PREG	Universal	BDI-II		7			
Milgrom et al. (2011)	AUS	Therapy	TAU	PREG	Selected/ Indicated		BDI-II ≥ 14	12	CBT	Individual (Phone)	8
Mokhber et al. (2011)	IRAN	Dietary Supplement	PLA	PREG	Universal	EPDS		8			
Muñoz et al. (2007)	USA	Therapy	TAU	PREG	Selected/ Indicated	EPDS	MMS	24	CBT	Group	12
Nalepka & Coblentz (1995)	USA	Social Support	EDUC	PREG	Universal		EPDS ≥ 10	12			
Ngai, Chan, & Ip (2009)	CHINA	Therapy	EDUC	PREG	Universal	EPDS		24	CBT	Group	6
Rees (1995)	USA	Therapy	ACT	POST	Universal	CES-D		4	Guided Relaxation	Individual (Home)	N/A
Shields & Reid (1997)	UK	Modified Care	TAU	PREG	Universal	9 Item EPDS	EPDS > 13	7			
Silverstein et al. (2011)	USA	Therapy	TAU	POST	Selected		QIDS ≥ 11	24	CBT	Individual	4
Small et al. (2000)	AUS	Modified Care	TAU	POST	Selected	EPDS	EPDS ≥ 13	24			
Stamp, Williams, & Crowther (1995)	AUS	Social Support	TAU	PREG	Selected	EPDS	EPDS > 12	24			
Webster et al. (2003)	AUS	Educational	TAU	PREG	Selected		EPDS > 12	16			
Wisner et al. (2001)	USA	ADM Nortriptyline	PLA	POST	Selected		RDC	17			
Wisner et al. (2004)	USA	ADM Sertraline	PLA	POST	Selected		DSM-IV	17			
Wolman et al. (1993)	S AFR	Modified Care	TAU	BIRTH	Universal	PITT	PITT ≥ 35	6			
Zayas, McKee, & Jankowski (2004)	USA	Therapy	TAU	PREG	Indicated	BDI-II		12	CBT	Individual	12

Study	Country	Intervention Type	Control Type	Intervention Timing	Prevention Type	Symptom Measure	Diagnosis Criteria	Postpartum Assessment Timing (Weeks)	Psychotherapy Orientation	Psychotherapy Administration	Number of Sessions
Zlotnick, Capezza, & Parker (2011)	USA	Therapy	TAU	PREG	Selected	EPDS	LIFE	12	IPT	Individual	4
Zlotnick et al. (2001)	USA	Therapy	TAU	PREG	Selected/ Indicated		SCID	12	IPT	Group	4
Zlotnick et al. (2006)	USA	Therapy	TAU	PREG	Selected	BDI-II	LIFE	12	IPT	Group	4

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

Table 2

Methodological Quality of Included Studies

						Therapy					Pharmacological	
Study	ITT	Char Sample	Excl Curr MDE	Blind Assess	Spec Random	Spec Ther	Manual	Training	Super	Adher	Blind Clin	Blind Pt
Armstrong et al. (1999)	-	+	-	+	+							
Austin et al. (2008)	+	+	-	+	+	+	+	+	-	-		
Brugha et al. (2000)	+	+	-	+	+	+	+	+	+	-		
Chabrol et al. (2002)	+	+	-	N/A	+	+	+	+	+	+		
Dennis et al. (2009)	+	+	-	N/A	+							
Elliott et al. (2000)	+	-	-	+	QR							
Gao, Chan, & Sun (2012)	+	+	-	N/A	+	+	-	+	+	-		
Gorman (1997)	-	+	-	+	+	-	+	-	-	+		
Gunn et al. (1998)	+	+	-	N/A	+							
Hagan, Evans, & Pope (2004)	-	+	+	+	+	+	-	+	+	-		
Hayes, Muller, & Bradley (2001)	-	+	+	-	+							
Ho et al. (2009)	-	+	-	N/A	+							
Kozinszky et al. (2012)	+	+	-	+	+	+	-	+	-	+		

Study	ITT	Char Sample	Excl Curr MDE	Blind Assess	Spec Random	Therapy					Pharmacological	
						Spec Ther	Manual	Training	Super	Adher	Blind Clin	Blind Pt
Lara, Navarro, & Navarrete (2010)	+	+	+	-	+	+	+	+	+	+		
Lawrie et al. (1998)	+	+	-	+	+						+	+
Le, Perry, & Stuart (2011)	+	+	+	-	+	+	+	+	+	+		
Llorente et al. (2003)	-	+	-	N/A	+						-	+
Logsdon et al. (2003)	-	+	-	N/A	+							
Marks, Siddle, & Warwick (2003)	+	+	-	-	+							
Meeker (1985)	+	+	-	N/A	-							
Milgrom et al. (2011)	+	+	-	N/A	+	+	+	-	+	+		
Mokhber et al. (2011)	-	+	+	N/A	-						+	+
Muñoz et al. (2007)	-	+	+	-	+	+	+	+	+	+		
Nalepka & Coblentz (1995)	-	+	-	N/A	+							
Ngai, Chan, & Ip (2009)	+	+	-	N/A	QR	+	-	+	-	-		
Rees (1995)	+	+	-	N/A	-	N/A	N/A	N/A	N/A	N/A		
Shields & Reid (1997)	-	+	-	N/A	-							
Silverstein et al. (2011)	+	+	-	+	+	+	+	+	+	+		
Small et al. (2000)	+	+	-	N/A	+							

						Therapy					Pharmacological	
Study	ITT	Char Sample	Excl Curr MDE	Blind Assess	Spec Random	Spec Ther	Manual	Training	Super	Adher	Blind Clin	Blind Pt
Stamp, Williams, & Crowther (1995)	+	+	-	N/A	+							
Webster et al. (2003)	+	+	-	N/A	+							
Wisner et al. (2001)	+	-	+	+	+						+	+
Wisner et al. (2004)	+	+	+	+	+						+	+
Wolman et al. (1993)	-	+	-	+	+							
Zayas, McKee, & Jankowski (2004)	-	+	-	N/A	-	+	-	+	+	-		
Zlotnick, Capezza, & Power (2011)	+	+	+	-	+	+	+	+	+	-		
Zlotnick et al. (2001)	-	+	+	-	-	-	-	-	-	-		
Zlotnick et al. (2006)	-	+	+	-	+	+	-	+	+	-		

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

Table 3

Random Weighted Effect Sizes (Hedges' g) Comparing Depressive Symptoms Between Treatment and Control Conditions at 6 Months Postpartum

Study	*n*	Hedges' *g*	95% CI	SAMD	
Armstrong et al. (1999)	181	0.44**	0.14-0.73	1.97	
Chabrol et al. (2002)	211	0.42**	0.15-0.70	2.05	
Dennis et al. (2009)	600	0.13	-0.03-0.29	-0.17	
Gao, Chan, & Sun (2012)	194	0.34*	0.06-0.62	1.39	
Gorman (1997)	30	0.02	-0.68-0.72	-0.33	
Gunn et al. (1998)	475	0.02	-0.16-0.20	-1.38	
Hayes, Muller, & Bradley (2001)	188	0.1	-0.18-0.39	-0.27	
Ho et al. (2009)	168	0.39*	0.09-0.70	1.61	
Lawrie et al. (1998)	168	-0.12	-0.42-0.19	-1.67	
Le, Perry, & Stuart (2011)	174	-0.09	-0.38-0.21	-1.52	
Llorente et al. (2003)	89	-0.15	-0.56-0.26	-1.38	
Logsdon et al. (2003)	109	-0.2	-0.65-0.25	-1.76	
Marks, Siddle, & Warwick (2003)	85	0	-0.42-0.42	-0.65	
Mokhber et al. (2011)	85	0.39	-0.03-0.82	1.15	
Munoz et al. (2007)	41	0.24	-0.36-0.84	0.30	
Ngai, Chan, & Ip (2009)	184	0.42**	0.13-0.71	1.89	
Rees (1995)	60	0.61*	0.10-1.12	1.78	
Shields & Reed (1997)	788	0.18**	0.04-0.32	0.60	
Small et al. (2000) [1]	917	-0.08	-0.21-0.05	-3.55	
Wolman et al. (1993) [1]	149	12.10***	10.69-13.51	70.69	
Zayas, McKee, & Jankowski (2004)	57	0.07	-0.44-0.59	-0.25	
Zlotnick, Capezza, & Parker (2011)	35	0.32	-0.21-0.85	0.86	
Zlotnick et al. (2001)	86	0.44	-0.22-1.10	-0.22	
Zlotnick et al. (2006)	54	0.09	-0.33-0.51	0.64	
	k	Hedges' *g*	95% CI	*Q*(*df*)	I^2
Total (all studies)	24	0.37***	0.15-0.60	321.40(23)***	92.84
Total (outliers excluded)	22	0.18***	0.09-0.27	33.32(21)*	36.98

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

[1] Outlier excluded from subsequent analyses.

Table 4

Analyses of Moderation for Depressive Symptoms at 6 Months Postpartum

Moderator	*N*	Hedges' *g*	95% CI	*Q*(*df*)	*p*
Intervention Type				2.73(4)	0.60
Dietary Supplement	2	0.12	-0.42-0.65		
Educational	2	0.24	-0.04-0.53		
Modified Care	4	0.16	-0.01-0.33		
Therapy	11	0.27***	0.14-0.40		
Social Support	2	0.04	-0.25-0.33		
Intervention Type				1.06(1)	0.30
Biological	3	0.02	-0.30-0.35		
Psychosocial	19	0.20***	0.11-0.29		
Intervention Type				1.56(1)	0.21
EST	10	0.25***	0.12-0.38		
Non-EST	12	0.14*	0.03-0.25		
Control Group Type				4.89(2)	0.09
Educational	2	0.38***	0.18-0.58		
Placebo	3	0.02	-0.30-0.35		
TAU	16	0.16***	0.07-0.25		
Intervention Timing				0.06(1)	0.81
Pregnancy	14	0.18***	0.09-0.26		
Postpartum	8	0.20*	0.03-0.36		
Type of Prevention				2.17(3)	0.54
Indicated	3	0.22*	0.01-0.44		
Selected	4	0.18	-0.10-0.47		
Selected/Indicated	5	0.03	-0.11-0.23		
Universal	10	0.19**	0.07-0.32		
Measure				4.34(2)	0.11
BDI-II	5	0.00	-0.19-0.18		
CES-D	2	0.20	-0.60-.99		
EPDS	13	0.23***	0.12-0.34		
Exclude Current MDE				0.32(1)	0.58
No	15	0.19***	0.08-0.29		
Yes	7	0.13	-0.02-0.29		
Psychotherapy Orientation				0.06(1)	0.80
CBT	5	0.23	0.00-0.46		
IPT	5	0.27**	0.07-0.47		
Method of Psychotherapy Administration				0.39(1)	0.53
Group	6	0.23*	0.04-0.41		
Individual	4	0.31**	0.11-0.52		

† $p < 0.10$,* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 5

Random Weighted Effect Sizes (Odds Ratio) Comparing Prevalence of Depressive Episodes Between Treatment and Control Conditions at 6 Months Postpartum

Study	*n*	*OR*	95% CI	SAMD	
Armstrong et al. (1999)	181	0.24	0.09-0.65	-3.30	
Austin et al. (2008)	277	0.94	0.50-1.76	1.79	
Brugha et al. (2000)	190	0.49	0.12-2.02	-1.54	
Chabrol et al. (2002)	211	0.46	0.26-0.81	-1.95	
Dennis et al. (2009)	600	0.80	0.49-1.31	1.00	
Elliott et al. (2000)	99	0.38	0.15-0.94	-1.73	
Gorman (1997)	37	0.57	0.11-3.03	-0.43	
Gunn et al. (1998)	475	1.26	0.76-2.09	5.63	
Hagan, Evans, & Pope (2004)	192	1.02	0.47-2.23	2.04	
Kozinszky et al. (2012) [1]	1719	1.79	1.30-2.48	18.00	
Lara, Navarro, & Navarrete (2010)	116	0.36	0.13-1.01	-1.95	
Lawrie et al. (1998)	168	1.13	0.59-2.18	2.60	
Le, Perry, & Stuart (2011)	174	1.38	0.52-3.67	4.18	
Marks, Siddle, & Warwick (2003)	87	1.05	0.41-2.73	1.51	
Milgrom et al. (2011)	89	0.24	0.08-0.69	-2.31	
Munoz et al. (2007)	41	0.17	0.01-3.82	-1.68	
Nalepka & Coblentz (1995)	72	0.94	0.18-4.98	0.88	
Shields & Reid (1997)	788	0.66	0.47-0.94	-0.70	
Silverstein et al. (2011)	42	0.40	0.11-1.51	-1.01	
Small et al. (2000) [1]	917	1.26	0.88-1.80	7.39	
Stamp, Williams, & Crowther (1995)	121	1.62	0.54-4.89	4.79	
Webster et al. (2003)	369	0.80	0.50-1.28	0.81	
Wisner et al. (2001)	51	0.95	0.26-3.45	0.77	
Wisner et al. (2004)	22	0.08	0.01-0.90	-1.44	
Wolman et al. (1993)	149	0.02	0.00-0.40	-4.17	
Zlotnick, Capezza, & Parker (2011)	35	1.68	0.36-7.86	2.68	
Zlotnick et al. (2001)	86	0.06	0.00-1.08	-3.00	
Zlotnick et al. (2006)	54	0.17	0.03-0.88	-2.01	
	k	*OR*	95% CI	*Q*(*df*)	I^2
Total (all studies)	28	0.72*	0.56-0.94	74.83(27)***	63.92
Total (outliers excluded)	26	0.67**	0.52-0.85	45.95(25)**	45.60
Total (trim-and-fill correction)		0.73*	0.56-0.95	61.93	

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

[1] Outlier excluded from subsequent analyses.

Table 6

Analyses of Moderation for Depressive Episodes at 6 Months Postpartum

Moderator	*n*	*OR*	95% CI	*Q*(*df*)	*p*
Intervention Type				1.13(3)	0.77
Medication	2	0.34	0.03-3.85		
Modified Care	5	0.61	0.31-1.19		
Therapy	13	0.57**	0.38-0.84		
Social Support	4	0.77	0.46-1.31		
Intervention Type				0.02(1)	0.88
Biological	3	0.71	0.24-2.12		
Psychosocial	23	0.61**	0.50-0.84		
Intervention Type				0.82(1)	0.37
EST	14	0.58**	0.39-0.87		
Non-EST	12	0.73*	0.54-1.00		
Control Group Type				1.13(2)	0.57
Educational	4	0.82	0.53-1.25		
Placebo	3	0.71	0.24-2.12		
TAU	19	0.62**	0.46-0.83		
Intervention Timing				0.03(1)	0.87
Pregnancy	16	0.70	0.47-1.05		
Postpartum	9	0.67**	0.50-0.90		
Type of Prevention				1.05(3)	0.79
Indicated	2	0.62	0.36-1.07		
Selected	10	0.60*	0.38-0.97		
Selected/Indicated	9	0.60*	0.36-0.99		
Universal	5	0.84	0.48-1.46		
Criterion for Diagnosis				0.03(1)	0.87
Clinical	12	0.64*	0.42-0.99		
Cutoff	14	0.67*	0.50-0.92		
Exclude Current MDE				0.27(1)	0.60
No	17	0.68**	0.53-0.89		
Yes	9	0.56	0.29-1.10		
Psychotherapy Orientation				0.41(1)	0.52
CBT	8	0.63*	0.41-0.97		
IPT	4	0.40	0.11-1.51		
Method of Psychotherapy Administration				0.71(1)	0.40
Group	8	0.62	0.36-1.07		
Individual	5	0.46**	0.29-0.73		

† $p < 0.10$,* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

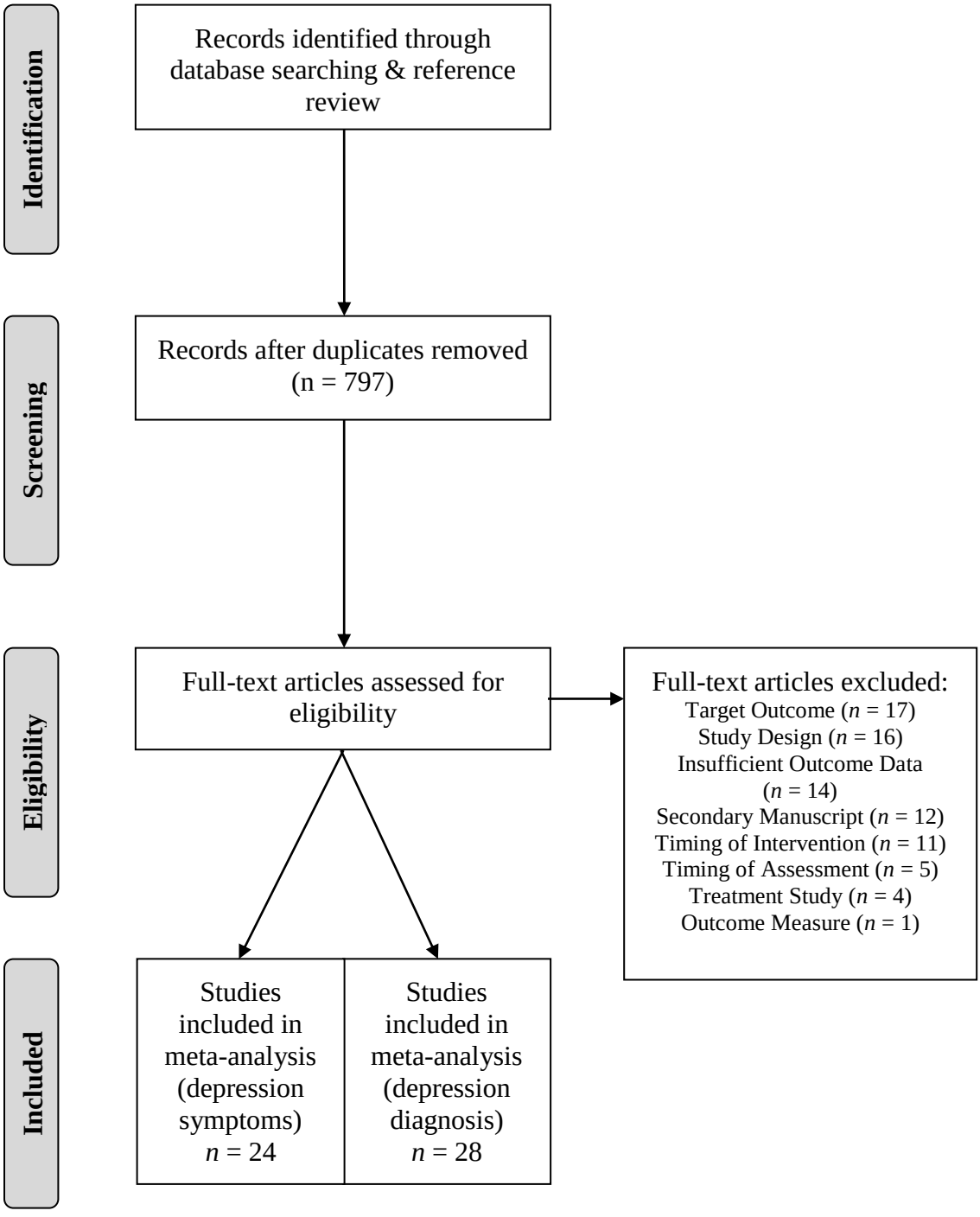

Figure 1. Flow chart illustrating identification of included studies.

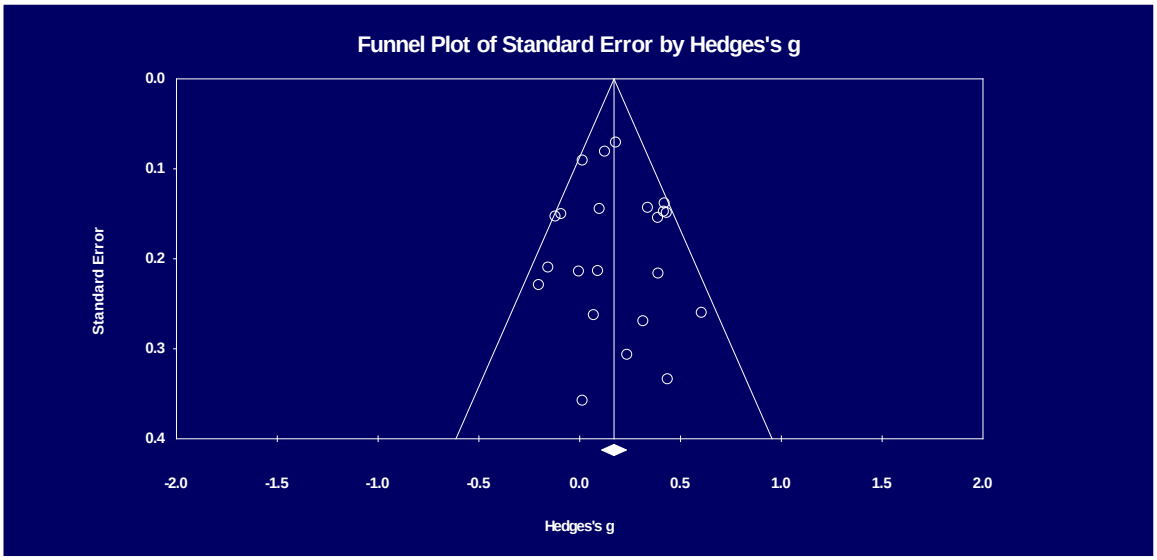

Figure 2. Funnel plot for studies assessing the difference between depressive symptoms between treatment and control conditions at 6 months postpartum. The asymmetric distribution of studies in the lower half of the funnel plot suggests that there are missing studies with negative effect sizes, in which control conditions would be superior to treatment conditions.

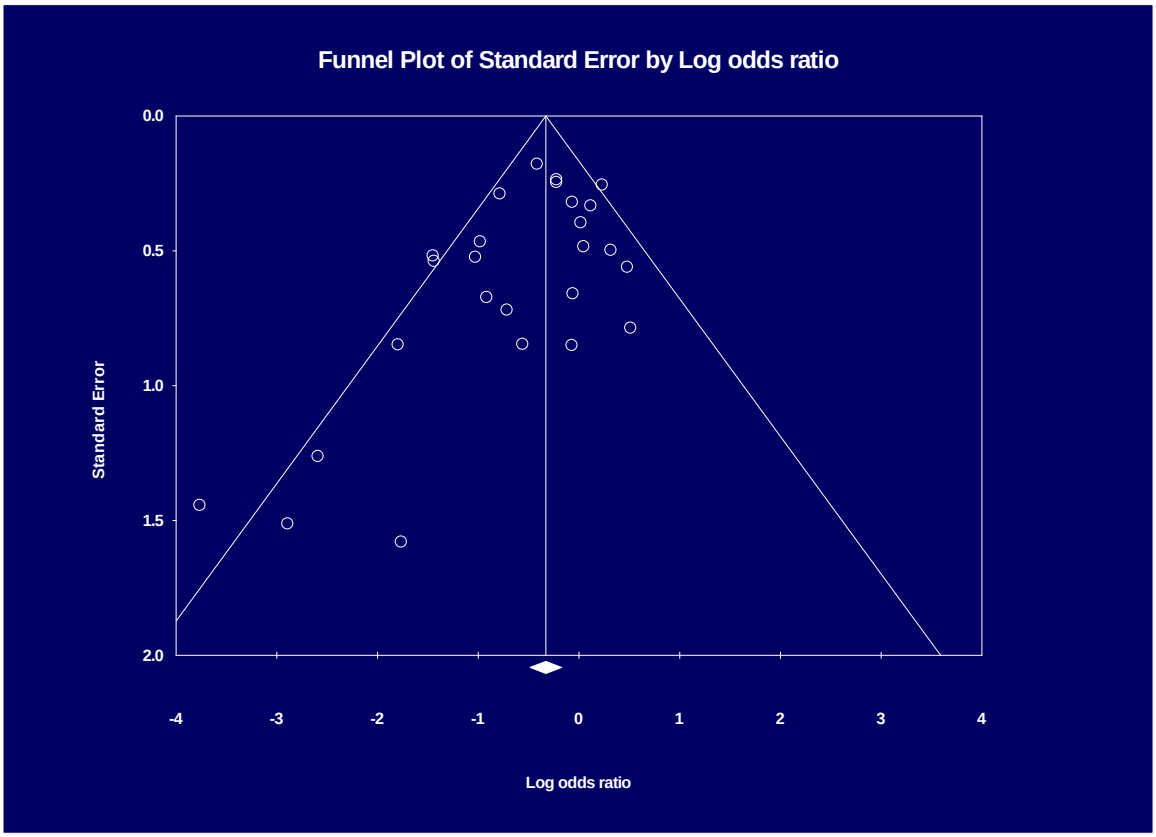

Figure 3. Funnel plot for studies assessing the difference in prevalence of depressive episodes between treatment and control conditions at 6 months postpartum. The asymmetric distribution of studies in the lower half of the funnel plot suggests that there are missing studies with odds ratios greater than 0, in which control conditions would be superior to treatment conditions.

General Discussion

Overall, the results of the studies included in this dissertation contribute to our understanding of perinatal mood and anxiety disorders. These studies build on previous research on risk factors and interventions for these disorders. The results of these studies provide important guidance for clinicians and researchers with interests in perinatal mental health.

The studies included in Chapter 1 help further our understanding of an important risk factor for perinatal depression and anxiety: maternal attitudes. Beck's cognitive model (1967, 1976, 1985) suggests that negative maternal attitudes could function as a specific vulnerability to perinatal depression and anxiety. Previous research into the role of maternal attitudes in the development of perinatal depression and anxiety had been limited by the need for a measure of maternal attitudes that was not confounded with women's expectations or experiences of motherhood and by the limited validity and reliability of existing measures, particularly among first-time mothers (Sockol, 2008). We demonstrated that our measure, the Attitudes Toward Motherhood scale (AToM), is reliable and has good convergent validity with general cognitive biases and an existing measure of maternal attitudes. This measure provides researchers and clinicians with an important tool for the assessment of this construct.

Development of the Attitudes Toward Motherhood scale enabled us to then use this measure to assess the relationship between maternal attitudes and symptoms of depression and anxiety among first-time pregnant and postpartum mothers. We found that dysfunctional maternal attitudes were strongly predictive of symptoms of depression and anxiety. This was true even after controlling for general cognitive biases, which suggests that maternal attitudes contribute uniquely to risk for perinatal distress above and beyond

general cognitive style. Furthermore, both dysfunctional maternal attitudes and general cognitive biases predicted symptoms of depression and anxiety after controlling for marital satisfaction and social support, which are known robust risk factors for these symptoms. Interpersonal risk factors also continued to predict symptoms of depression and anxiety when cognitive risk factors were controlled for, which suggests that both cognitive and interpersonal risk factors play an important role in the development of symptoms of depression and anxiety.

While the results of the studies included in Chapter 1 have interesting implications for clinicians and researchers interested in developing interventions for perinatal depression and anxiety, these findings are too preliminary to directly lead to changes in our approach to interventions for these disorders. The overarching goals for the studies included in Chapters 2 and 3 was to synthesize research that has already been conducted on treating and preventing postpartum depression in order to assess the overall efficacy of interventions that have been subjected to scientific study and to examine whether there might be characteristics of studies or interventions that are systematically associated with differences in efficacy.

The results of the meta-analyses included in Chapter 2 provide strong evidence for the efficacy of psychotherapy and antidepressant medications in the treatment of perinatal depression. These analyses demonstrate that these interventions are associated with significant decreases in depressive symptoms over time and that interventions lead to significantly greater reductions in depressive symptoms as compared to control conditions. Perhaps the most interesting finding from this study is that interpersonal psychotherapy was more effective than cognitive-behavioral therapy. This is somewhat

surprising, as the results of the studies included in Chapter 1 suggest that both interpersonal and cognitive risk factors are strongly associated with symptoms of depression – thus we might expect that interventions targeting either interpersonal or cognitive factors might be equally efficacious. One possible explanation for this finding is that there were notable methodological differences between studies assessing interpersonal psychotherapy compared to cognitive-behavioral therapy. The ambiguity of this finding highlights the need for further research in this area, and particularly suggests that a head-to-head comparison of interpersonal psychotherapy and cognitive-behavioral therapy in a methodologically rigorous trial would be an important contribution to research in this area.

The results of the meta-analyses included in Chapter 3 provide evidence that preventive interventions for postpartum depression result in significant reductions in depressive symptoms and the prevalence of depressive episodes, although the magnitude of effect is smaller than found for treatment studies in Chapter 2. At six months postpartum, subjects in treated conditions had significantly lower levels of depressive symptoms than subjects in control conditions. Subjects were also 27% less likely to experience a depressive episode during the first six months postpartum when they received an intervention. Interestingly, we did not find differences among the different types of interventions, and there was no difference in the efficacy of cognitive-behavioral therapy and interpersonal psychotherapy. This provides additional evidence that this result in Chapter 2 may reflect methodological differences between the included studies, rather than a true difference between these types of psychotherapy.

Limitations and Future Directions

The studies included in this dissertation represent promising first steps toward a more full understanding of these disorders. Further research is necessary to build upon the results of these studies and to help answer the questions that they raise.

One of the major limitations of Chapter 1 was the cross-sectional design of the included studies. While the results of these studies are consistent with a diathesis-stress model of depression and anxiety, a longitudinal design is necessary to assess whether dysfunctional maternal attitudes are truly a risk factor for symptoms of depression and anxiety, or whether these attitudes may simply be a reflection of the symptoms themselves. We are currently conducting a follow-up study to assess whether dysfunctional maternal attitudes during pregnancy are predictive of symptoms of depression and anxiety at 12 weeks postpartum.

Another limitation of the studies included in Chapter 1 was that we limited our sample to women who were pregnant with, or had recently given birth to, their first child. We decided to limit our sample due to differences that we had observed in the relationships among cognitive biases, maternal attitudes, and depressive symptoms between primiparous and multiparous subjects in our previous research (Sockol, 2008). As a previously utilized measure of maternal attitudes had proven particularly problematic for first-time mothers, we wanted to ensure that our measure was reliable and valid among this population. Replication of the results of this study with a sample of multiparous subjects is necessary to demonstrate that this measure is reliable and valid among all childbearing women.

As for all meta-analyses, the studies included in Chapters 2 and 3 are limited by the availability and quality of research studies assessing the efficacy of treatments and

preventive interventions for perinatal depression. While the absolute number of studies included in these meta-analyses was comparable to that of similar meta-analyses, certain sub-groups of interventions were represented by small numbers of studies. Results regarding interventions represented by small numbers of studies should be interpreted with caution. For example, in Chapter 2, we identified only four studies of antidepressant medication. Three of these studies were open trials, and none of these studies assessed the efficacy of antidepressants among depressed pregnant women. This was also true in Chapter 3, in which we only identified two randomized controlled trials of antidepressant medication for the prevention of postpartum depression, both of which were initiated in the immediate postpartum period. Clinicians are sometimes reluctant to prescribe antidepressant medication for women who are pregnant or breastfeeding and there is evidence that the use of selective serotonin reuptake inhibitors during pregnancy is associated with increased risk for congenital malformations (Alwan, Reefhuis, Rasmussen, Olney, & Friedman, 2007; Bar-Oz et al., 2007; Wurst, Poole, Ephross, & Olshan, 2010). However, there is also evidence for an increased risk of relapse among women who discontinue antidepressant treatment during pregnancy (Cohen et al., 2006). In order for clinicians and patients to make fully informed decisions about the risks and benefits of treatment, further research assessing the efficacy of antidepressant medications among this population – particularly in comparison to psychotherapeutic treatments, which may have lower risks or be more acceptable to patients – is necessary.

Perhaps more worrisome than the limited number of studies included in these meta-analyses, particularly for non-psychotherapeutic interventions, is evidence of publication bias in both treatment and prevention studies. In most of our analyses,

examination of funnel plots and Duval and Tweedie's (2000) trim-and-fill procedure suggested that we were missing studies with non-significant findings. For our main analyses, we were able to utilize statistical corrections that can estimate the overall effect size if these missing studies were included. However, this is not possible for moderator analyses. It is possible that there are systematic differences among studies that are published versus unpublished that might bias the findings of our meta-analyses. For example, consider our finding in Chapter 3 that there is no significant difference in the prevalence of postpartum depressive episodes at 6 months postpartum between the different types of interventions. This finding is based on 13 studies of psychotherapeutic interventions, 5 studies of modified medical care, 4 studies of social support, and 2 studies of antidepressant medication. If several unpublished studies represent randomized controlled trials of social support interventions that failed to find that they reduced the prevalence of postpartum depression during the postpartum period, we would expect to find a difference among intervention types if we were able to include these missing studies in our moderator analyses. Thus the evidence of publication bias found in these meta-analyses limits our confidence in our findings, particularly for moderator analyses.

The inclusion criteria we followed in the two meta-analyses have their own limitations. In Chapter 2, we decided to limit the included interventions to psychotherapeutic interventions and antidepressant medications. Because effective treatments for depression in adult populations have been identified (Cuijpers, van Straten, Andersson, & van Oppen, 2008; Joffe, Sokolov & Streiner, 1996), our goal for this meta-analysis was to assess the efficacy of established interventions among perinatal populations. As a result, we did not include other potential interventions, such as dietary

supplements or hormonal interventions. This allowed us to make more rigorous comparisons between treatments, but there is a risk that effective and acceptable treatments may have been excluded from our analyses. In Chapter 3, in contrast, we decided to include a much wider range of potential interventions. The strength of this approach is that we were able to identify a wide range of interventions that appear to be effective in reducing depressive symptoms and the prevalence of depressive episodes during the postpartum period. As women may be more receptive to complementary and alternative approaches to treatment, particularly during pregnancy, it is important for research to assess whether these approaches are efficacious (Battle, Uebelacker, Howard, & Castaneda, 2010). However, given the wide variations in intervention types, it was difficult to conduct direct comparisons of all of the interventions included in these analyses.

Conclusions

The cumulative results of these studies provide a hopeful message to clinicians, researchers, and mothers. In Chapter 1, we found that dysfunctional maternal attitudes, general cognitive biases, and interpersonal risk factors each have incremental predictive validity for symptoms of depression and anxiety. This provides evidence that emotional distress in this population is multi-factorial, and thus may be responsive to a wide range of approaches to intervention and prevention. The results of the meta-analyses in Chapters 2 and 3 provide further empirical support for this. Several types of antidepressant medications and psychotherapies were effective in the treatment of perinatal depression, and an even wider range of preventive interventions were found to effectively reduce depressive symptoms and the prevalence of depressive episodes during

the postpartum. The efficacy of such a wide range of interventions provides opportunities for selecting interventions that correspond with patients' preferences and access to care. While women often express a preference for psychotherapeutic interventions during this time period, there are also practical barriers to access to care that may lead some women to prefer pharmacologic or other interventions (Kim et al., 2011).

A more thorough understanding of the mechanisms that underlie the development of depression and anxiety during the perinatal period, and application of this understanding to the development of interventions to treat and prevent these disorders, is vitally important. These disorders are common and negatively affect not only the women who suffer from their symptoms, but also their developing children. While the context of pregnancy and the early postpartum period may confer additional risks, it also provides clinicians and researchers with opportunities – this is a time of increased access to and utilization of healthcare, and women may be particularly motivated to seek treatment by their desire to provide a healthy environment for their developing children. The studies included in this dissertation represent important steps toward an understanding of these disorders that can be used to help women achieve a healthy psychological adjustment during the transition to parenthood.

References

Alwan, S., Reefhuis, J., Rasmussen, S. A., Olney, R. S., & Friedman, J. M. (2007). Use of selective serotonin-reuptake inhibitors in pregnancy and the risk of birth defects. *New England Journal of Medicine, 356,* 2684-2692.

Bar-Oz et al., B., Einarson, T., Einarson, A., Boskovic, R., O'Brien, L., Malm, H., Berard, A., & Koren, G. (2007). Paroxetine and congenital malformations: Meta-analysis and consideration of potential confounding factors. *Clinical Therapeutics, 29*(5), 918-926.

Battle, C. L., Uebelacker, L. A., Howard, M., & Castaneda, M. (2010). Prenatal yoga and depression during pregnancy. *Birth, 37*(4), 353-354.

Beck, A. (1967). *Depression: Clinical, Experimental & Theoretical Aspects.* Philadelphia, PA: University of Pennsylvania Press.

Beck A. (1976). *Cognitive Therapy and the Emotional Disorders.* New York: Penguin.

Beck, A. (1985). *Anxiety Disorders and Phobias: A Cognitive Perspective.* New York: Basic Books.

Cohen, L. S., Altshuler, L. L., Harlow, B. L., Nonacs, R., Newport, J., Viguera, A. C., Suri, R., Burt, V. K., Hendrick, V., Reminick, A. M., Loughead, A., Vitonis, A. F., & Stowe, Z. N. (2006). Relapse of major depression during pregnancy in women who maintain or discontinue antidepressant treatment. *Journal of the American Medical Association, 295.*

Cuijpers, P., van Straten, A., Andersson, G., & van Oppen, P. (2008). Psychotherapy for depression in adults: A meta-analysis of comparative outcome studies. *Journal of Consulting and Clinical Psychology,* 76, 909-922.

Duval, S., & Tweedie, R. (2000). Trim and fill: a simple funnel-plot-based method of testing and adjusting for publication bias in meta-analysis. *Biometrics, 56,* 455-463.

Joffe, R., Sokolov, S., & Streiner, D. (1996). Antidepressant treatment of depression: A meta-analysis. *Canadian Journal of Psychiatry, 41,* 613-616.

Kim, D.R., Sockol, L., Barber, J.P., Moseley, M., Lamprou, L., O'Reardon, J.P., & Epperson, C.N. (2011). A survey of patient acceptability of repetitive Transcranial Magnetic Stimulation (TMS) during pregnancy. *Journal of Affective Disorders, 129,* 385-390.

Sockol, L. E. (2008). Predicting depressive symptoms during pregnancy: Integrating cognitive, coping and social perspectives. (Unpublished master's thesis.) University of Pennsylvania, Philadelphia, PA.

Wurst, K. E., Poole, C., Ephross, S. A., & Olshan, A. F. (2010). First trimester paroxetine use and the prevalence of congenital, specifically cardiac, defects: A meta-analysis of epidemiological studies. *Birth Defects Research Part A: Clinical and Molecular Teratology, 88*(3), 159-170.