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LOYOLA UNIVERSITY CHICAGO

THE IMPACT OF TREATING
MAJOR DEPRESSION DURING PREGNANCY
ON THE POSTPARTUM PHASE

A DISSERTATION SUBMITTED TO
THE FACULTY OF THE GRADUATE SCHOOL
IN CANDIDACY FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY

PROGRAM IN CLINICAL PSYCHOLOGY

BY

JAMIE SCALETTA KENT

CHICAGO, ILLINOIS

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ABSTRACT

Major depression during pregnancy is a risk factor for postpartum depression (PPD). Medically acceptable treatments for depression during pregnancy and postpartum are limited and many women are turning to complementary and alternative treatments. The current project examined whether treatment of major depression during pregnancy reduced the risk for PPD and explored predictors of PPD in this high-risk sample. One hundred twenty women were clinically assessed using the Hamilton Rating Scale for Depression (HRSD₁₇), the Beck Depression Inventory (BDI-II), and the Structured Clinical Interview for DSM-IV (SCID) at ten weeks, six months, and nine months postpartum following random assignment to one of three treatments delivered over eight weeks during pregnancy: active acupuncture, control acupuncture, and control prenatal massage. Mixed effects analyses revealed that responders to the acute phase of treatment during pregnancy had significantly lower postpartum BDI-II scores compared to non-responders. Results also revealed a significant treatment assignment by time interaction on postpartum HRSD₁₇ scores such that the active acupuncture group maintained improvement while the control groups worsened postpartum. Exploratory ROC analyses revealed the following predictors of PPD: discontinuing antidepressant medication, high intake HRSD₁₇ scores, high late pregnancy HRSD₁₇ scores, low expectations of treatment, and a history of childhood emotional abuse. Results suggest that successful treatment of depression during pregnancy may lower the risk of developing PPD.

CHAPTER ONE

INTRODUCTION

Over the past two decades increasing attention has been paid to the complex phenomenon of postpartum depression. While childbearing is a joyous event in the lives of women and their families, it is also a particularly vulnerable time for developing or exacerbating psychiatric illness. Virtually no life event rivals the hormonal, psychological and social changes associated with pregnancy and childbirth. In fact, postpartum depression is the most common complication of childbearing. It is estimated that among the nearly four million births in the United States annually, a half-million women will endure this disorder every year. Many of these women will be undiagnosed and untreated.

The consequences of untreated postpartum depression for women, their offspring and families can be devastating. Postpartum depression may have a deleterious effect on the woman's social and personal adjustment, the marital relationship, and the mother-infant interaction (Josefsson, Berg, Nordin, & Sydsjo, 2001). Additional consequences may include self-harm and suicide (Lindahl, Pearson, & Colpe, 2005). Given the magnitude of these consequences, it is imperative that health professionals are exhorted to recognize and treat this problem.

Despite its common occurrence and devastating consequences, postpartum depression has historically received minimal attention in modern clinical literature,

training, and practice. However, the detection and treatment of postpartum depression has been recently prioritized as a major public health concern. A United States House Resolution (H.Res.51) was introduced in 1999 proposing that the National Institutes of Health further study postpartum psychiatric illness (Lindahl et al., 2005). The resolution further encouraged health care providers to teach women about the prevalence, symptoms, and treatment of postpartum depression. Similarly, on the research front, the Agency for Healthcare Research and Quality recently reported that while much is known about the risks and vulnerabilities of the postpartum period, there is a significant lack of substantial high-quality research in this area (Wisner, Chambers, & Sit, 2006). Clearly, policy makers are encouraging greater surveillance of postpartum depression as well as better implementation of preventative and treatment methods for childbearing women.

Given the deleterious costs of postpartum depression to individuals and society, prevention and treatment are large concerns. Toward this end, and as called upon by governmental agencies, efforts must focus on better understanding this phenomenon. To date, the largest body of research has focused on individual characteristics that relate to increased risk for postpartum depression and predictors of postpartum depression. More recent work has begun to examine viable treatment options as well as measures to prevent initial onset of mood symptoms in the postpartum period. The present study is designed to extend this recent body of work by examining the role of treatment during pregnancy in helping to manage mood symptoms and reduce the likelihood of postpartum depression.

CHAPTER TWO

REVIEW OF THE RELATED LITERATURE

General Background

Clinicians since Hippocrates have noted an association between the postpartum period and mood disturbance. As early as 400 B.C. Hippocrates described puerperal fever in postpartum women and noted symptoms such as agitation, delirium, and mania (Demand, 1994). An 11th century author similarly pondered the postpartum period writing that “if the womb is too moist, the brain is filled with water, and the moisture running over to the eyes, compels them to involuntarily shed tears” (Leopold & Zoschnick, 1997). Thus, it has long been recognized that childbirth is associated with a considerable increase in psychological morbidity.

While historical references to postpartum depression were largely anecdotal and non-empirical, during the 1960’s researchers began to focus on the postpartum period as an area worth investigating. Converging evidence suggested that women were at increased risk for the development of depression during the postpartum period (Paffenbarger & McCabe, 1966; Pugh, Jerath, Schmidt, & Reed, 1963; Ryle, 1961). However, due to poorly defined or inadequate criteria used to assess and diagnose depression, there was little consensus on what constituted postpartum depression. While some measured seeking outpatient psychiatric help (Dalton, 1971), others used self-reported distress as their criterion of depression (Kane, Harmon, Keeler, & Ewing, 1968).

Finally, some did not specify the criteria used to make a depression diagnosis (Tod, 1964).

Extrapolating from the literature, it seems that within the broad spectrum of postpartum dysphoric mood states, three types of reactions began to emerge in the 1970's. These included (a) maternity blues, characterized by tearfulness, feelings of dysphoria, and emotional lability (Pitt, 1973); (b) postpartum depression, similar to an untreated clinical depression (Meares, Grimwade, & Wood, 1976); and (c) psychotic depression, often accompanied by delusions (Herzog & Detre, 1976). These working definitions of postpartum mood states greatly assisted in the differentiation between normal and abnormal reactions to childbirth.

As a result, a surge of studies in the 1980's began to investigate the prevalence and predictors of postpartum depression (Cutrona, 1983; O'Hara, Neunaber, & Zekoski, 1984; Watson, Elliott, Rugg, & Brough, 1984). Additionally, O'Hara (1987) more explicitly quantified postpartum mood disorders into three categories in ascending level of severity: blues, depression, and psychosis. Interest in mild and moderate levels of mood disturbance increased (O'Hara & Zekoski, 1988) as did the timing of the onset of the mood state (Handley, Dunn, Waldron, & Baker, 1980).

Based on the surge of studies in the 1980's and converging evidence that women were at risk for postpartum depression, this linkage entered official psychiatric nomenclature in 1994. At that time the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* added a postpartum onset specifier within the major depression diagnosis. DSM-IV defined major depression with postpartum onset as episodes of depression beginning within four weeks of giving birth (American Psychiatric

Association, 1994). This time frame corresponds to the rapid hormonal changes posited to contribute to vulnerability to depression. However, because psychosocial factors also play a major role in triggering postpartum depression, many researchers have used a working definition of the postpartum period as lasting up to six months post delivery (Miller, 2002). These discrepancies in the nosology of perinatal episodes have led to confusion in both clinical practice and research, leading to recommendations for revision in DSM-V to modify the postpartum onset specifier within the major depression diagnosis to begin within six months following birth (Jones, 2010).

Working Definitions

Postpartum mood disturbances, as classified today, range from postpartum blues to nonpsychotic major depression to depression with psychotic features. Postpartum blues are a transient state of heightened emotional reactivity that affects between 50% and 80% of new mothers (Miller, 2002; O'Hara & Segre, 2008). The blues are not typically considered a disorder necessitating professional intervention. Women with postpartum blues exhibit irritability, emotional lability, forgetfulness, confusion, and anxiety. Postpartum blues are short-lived with acute onset typically occurring between birth and two weeks postpartum and persisting from a few hours to a few days, with little evidence of long-term negative consequences (O'Hara & Segre, 2008). The propensity to develop postpartum blues is unrelated to psychiatric history, environmental stressors, breastfeeding, or parity (Miller, 2002). However, those factors may influence whether the blues lead to major depression.

Postpartum nonpsychotic depression constitutes a major depressive episode with clinical symptoms that onsets within four weeks of giving birth. Symptoms of

postpartum depression (PPD) may include irritability, anhedonia, sleep disturbance, fatigue, and anxiety. Although DSM requires symptoms to appear by four weeks postpartum, the literature has used this time frame loosely in defining PPD. While some studies assert that the majority of cases have an onset within six weeks postpartum (Stowe & Nemeroff, 1995), others report that symptoms of PPD may not appear until six months after delivery (Beck, 1998; Gold, 2002; Miller, 2002; Spinelli, 1998). As a result, most scientific literature has used more liberal criteria to diagnose PPD, and as a result of these discrepancies, initial recommendations for revisions to DSM-V propose extending the duration of the onset specifier to six months postpartum.

Finally, postpartum psychotic depression is the most severe and least common disorder. It is reported to occur in only 1 or 2 of every 1000 women (Kendell, Chalmers, & Platz, 1987). The psychosis, manifested by delusions, hallucinations, or both, typically occurs in the first three weeks of birth (Munk-Olsen, Laursen, Pedersen, Mors, & Mortensen, 2006). As compared with nonpostpartum mood episodes, there is more disorientation and lability in postpartum episodes as well as abnormal or obsessive thoughts. The symptoms constitute a psychiatric emergency and may warrant hospitalization.

Prevalence of Postpartum Depression

The current body of scientific literature exhibits a wide variance in reports of the prevalence of PPD. Rates of PPD have been reported to be between 7% and 15% in adult women (Gavin et al., 2005; O'Hara & Swain, 1996; Vesga-Lopez et al., 2008). The variance is due in part to the lack of clearly defined diagnostic criteria, including conflicting definitions of the onset and duration of the postpartum period. As described

above, DSM-IV requires the episode of major depression to begin within four weeks of birth, while ICD-10 permits designation of a postpartum depressive disorder within six weeks of delivery. In research, investigators' definitions have varied and lengths of time up to one year following birth have been used (Wisner, Perel, Peindl, & Hanusa, 2004).

Additionally, distinguishing between depressive symptoms and the normal sequelae of childbirth including changes in weight, sleep, and energy, is a further challenge complicating clinical diagnosis. Symptoms may be minimized by the professional or the mother as normal. Furthermore, physical causes which may contribute to depressive symptoms (including anemia, diabetes, and thyroid dysfunction) have not been consistently excluded in research studies on prevalence rates of PPD. Finally, the failure of mothers to report their symptoms has further confounded current prevalence rates. It has been estimated that only 20% of women report their symptoms to a professional (Gold, 2002) leaving many cases of PPD, up to 50%, unrecognized and even more cases untreated (Murray & Cooper, 1997).

Consequences of Postpartum Depression

The consequences of untreated postpartum depression have been well documented in the literature. PPD disrupts the relationship between the mother and the infant, which can have short and long-term adverse effects. PPD also has a dramatic impact on the mother's ability to function, enjoy relationships, cope with the stresses, or appreciate the joys of parenthood. Major depression creates suffering regardless of timing; however, depression during the postpartum is exacerbated by substantial demands on new mothers including infant care, household responsibilities, and often returning to work after a brief maternity leave (O'Hara, 2009). The deleterious effects of PPD on

offspring, the marital relationship, and on mothers are clinically significant and are further outlined below.

Effects on Offspring

The adverse impact of maternal mental illness on offspring has been researched at length (Forman et al., 2007; Goodman, 2007; Tronick & Reck, 2009; Weissman, Warner, Wickramartne, Moreau, & Olfson, 1997). These investigations have indicated that women suffering from PPD tend to be less responsive to their infants and report greater difficulty with infant feeding, crying, and overall fussiness than non-depressed mothers (Goodman & Brand, 2008). Similarly, studies have shown a positive correlation between maternal PPD and greater difficulty learning effective infant care taking and parenting skills (Fowles, 1996) as well as with interpreting infants' affective communication (Tronick & Reck, 2009). Furthermore, some studies have found a positive correlation between mothers with PPD and incidence of child abuse and neglect (Nonacs & Cohen, 1998; Spinelli, 1998). These behaviors have a well documented, adverse effect, on mother-infant attachment (Campbell, Cohn, & Meyers, 1995; Logsdon, Wisner, & Pinto-Foltz, 2006; Murray & Cooper, 1996, 1997; Teti, Gelfand, Messinger, & Isabella, 1995).

Several other studies have shown that depression may affect not only a mother's interaction with her infant, but also her perception of the infant. Depressed mothers have been shown to perceive their infants more negatively and as more difficult to care for than non-depressed controls view their infants (Fowles, 1996; Murray & Cooper, 1996). Murray (1992) concluded that if normal maternal communication is experimentally disrupted for even brief periods, infants as young as six weeks old respond with distress and avoidance. A recent study found that early maternal negative perceptions of the

infant predicted negative temperament and behavior problems eighteen months later (Forman et al., 2007). Therefore, the mother's negative perception of the infant in combination with her inability to respond effectively may lead to longer-term consequences for the child.

One of the earliest consequences of postnatal depression on infants is emotional disturbance. Offspring of mothers suffering from PPD tend to demonstrate depressive symptoms at an early age. Research indicates that infants of mothers with PPD exhibit more negative facial expressions and vocalizations than infants of non-depressed mothers (Beck, 1998; Cooper & Murray, 1997; Goodman & Brand, 2008; Murray & Cooper, 1996). For example, one study found that maternal PPD predicted a negative affective response from their infants in response to a still-face interaction with their mother at six months (Moore, Cohn, & Campbell, 2001). Furthermore, studies have found infants exhibit depressive behavior not only with their mother, but also with non-depressed adults (Fields et al., 1988). Finally, a meta-analysis examining the effects of PPD on child development also found that exposure to PPD had a significant effect on children's emotional development (Beck, 1998).

In addition to the adverse effects on infants' moods, research has also indicated that exposure to maternal depression may lead to more insidious, longer-lasting consequences. Most commonly, these consequences include emotional and behavioral problems in early childhood (Cogill, Caplan, Alexandra, Robson, & Kumar, 1986; Cutrona & Troutman, 1986; Goodman & Brand, 2008; Grace, Evindar, & Stewart, 2003; Josefsson et al., 2001; Milgrom, Westley, & Gemmill, 2004; Murray & Cooper, 1996, 1997; Sharp et al., 1995; Whiffen & Gotlib, 1989). Studies have found a positive

correlation between exposure to postnatal depression and externalizing behaviors in preschoolers and elementary school aged children (Luoma et al., 2001; Murray, Sinclair, Cooper, Ducournau, & Turner, 1999; Sinclair & Murray, 1998). Additionally, studies have found that persistent exposure to PPD predicts low social competence and low adaptive functioning (Luoma et al., 2001) as well as greater difficulty adjusting in the context of school (Sinclair & Murray, 1998). These findings suggest that, while maternal behavior varies with changing circumstances, exposure to postpartum depression may have an enduring influence on child psychological adjustment.

In addition to emotional and behavioral disturbance, research has also demonstrated cognitive delay in the children of postnatally depressed mothers on cognitive tests. A significant positive correlation between exposure to PPD and infant cognitive delay has been found at twelve months (Cooper & Murray, 1997) and eighteen months (Murray & Cooper, 1996) as compared to infants of nondepressed mothers. Similarly the same relationship between exposure to PPD and significant cognitive deficits has been found in preschool and school aged children (Cogill et al., 1986). While it is important to note that causal links cannot be inferred from these correlations, the association between maternal postpartum depression and children's lower cognitive performance, points to ways in which PPD might exert its impact.

Effects on Marital Relationship

Evidence also suggests that PPD may have a harmful effect on the quality of the marital relationship (Campbell, Cohn, Flanagan, Popper, & Meyers, 1992). Fishel (2004) states that men sometimes withdraw, feel blamed, or neglected during this highly charged, emotional time. Viinamaki, Niskanen, Pesonen, and Saarikoski (1997), found

that partners of women experiencing PPD reported their relationships deteriorated during pregnancy. These relationships had not improved when the same group was surveyed again two years later. In addition to feelings of confusion and anger, partners are also at risk for developing mood symptoms (Campbell et al., 1992; Paulson & Bazemore, 2010) because of the significant emotional burden the family unit endures as a result of the mother's struggle with PPD.

Effects on Mothers

Postpartum depression has a dramatic impact on the mother's ability to function, enjoy relationships, cope with the stresses, and appreciate the joys of parenthood. Women suffering from PPD may endure more serious sequelae if the syndrome goes untreated. The depressive episode can become a precursor of recurrent depression and the mother may be at risk for developing a chronic depressive disorder (Andrews-Fike, 1999; Nonacs & Cohen, 1998). In fact, Spinelli (1998) estimates women who have a history of PPD to be 300 times more likely to have another episode of PPD in subsequent pregnancies. Finally, if untreated, the mother may be at greater risk to engage in unhealthy behaviors such as excessive consumption of alcohol and smoking cigarettes (Ross & Dennis, 2009; Whitaker, Orzol, & Kahn, 2007).

In addition to psychological distress, the risk for suicidality is significantly elevated among depressed women during the postpartum period, and suicide has been found to be one of the leading causes of death in this depressed population (Appleby, Mortensen, & Faragher, 1998). While suicide deaths and attempts are lower during the postpartum than in the general population of women, when deaths do occur in the postpartum, suicides account for up to 20% (Lindahl et al., 2005). When a woman with

PPD becomes suicidal, she may also consider killing her infant and young children, not usually out of anger but stemming from a desire not to abandon her children (Spinelli, 2004). Self-harm ideation is more common than attempts or deaths, with thoughts of self-harm during the postpartum ranging from 5 to 14% (Lindahl et al., 2005). Therefore, the clinical significance of PPD and its impact on mothers is compelling. Based on the magnitude of the potential consequences of PPD, it is imperative researchers continue to understand the disorder and its causes.

Causes of Postpartum Depression

One of the most commonly posited theories of the cause of postpartum depression is hormone based. Many have theorized that the rapid decline in the levels of reproductive hormones that occurs after delivery overwhelmingly contributes to the development of depression in susceptible women. Furthermore, the biological changes underlying postpartum blues may lead to clinical depression in the context of genetic vulnerability, environmental stress, or insufficient social support. There is indirect evidence to support the notion that hormonal imbalance causes PPD. In one study, which simulated the postpartum state by administering hormones and rapidly withdrawing them, it was significantly more likely to produce depression in women with a history of postpartum depression than in women with no history of depression (Bloch et al., 2000). Therefore, women with a history of postpartum depression may be differentially sensitive to the effects on mood of hormone withdrawal. However, there is no direct evidence that hormonal imbalance causes PPD and although it is tempting to attribute PPD to hormonal decline, other psychosocial factors may predispose women to the condition. Thus, at this

time there is no clear cause of postpartum depression and there are likely many psychosocial contributory factors.

Predictors of Postpartum Depression

Beginning in the 1980's, research on the predictors of postpartum depression was initiated. The researchers found that a number of psychosocial and biological variables were significantly correlated with postpartum depression. Several meta-analyses have been conducted to determine the magnitude of the relationship between frequently cited predictor variables and postpartum depression (Beck, 1996; Beck, 2001; O'Hara & Swain, 1996; Robertson, Grace, Wallington, & Stewart, 2004). Based on the synthesis of these results, psychosocial variables consistently linked to PPD include poor social support, poor marital relationship, stressful life events, and low self-esteem. Perhaps the strongest predictors of the development of PPD were biologically based. A history of depression, including recurrent MDD or a past episode of PPD, independently predicted a new onset of PPD. Additionally, the strongest predictor of PPD, according to several studies, is depression during pregnancy (Beck 1996; O'Hara & Swain, 1996). While this suggests that hormones may play a role in the development of PPD, psychosocial variables also clearly impact the onset.

Social Factors

Several psychosocial variables have consistently been found to be related to PPD. These include low social support, poor marital relationship, stressful life events, and having low self-esteem. It is thought that increased support and self-esteem may serve as buffers against the stresses associated with the postpartum phase. Each of these variables is discussed in more detail below.

Social support. Low social support has consistently been linked to the development of PPD (Cutrona, 1984; O'Hara, 1986; Robertson et al., 2004). Each of the major meta-analytic studies concluded that poor social support is a significant predictor of PPD. Robertson and colleagues (2004) and O'Hara and Swain (1996) found moderate effect sizes, $d = -0.64$ and $d = -0.63$, respectively, between social support and PPD. Additionally, field observations have found that cultures with an apparently low prevalence of PPD are characterized by strong social support for new mothers such as help with child care, special foods, ritual baths, or return of the mother to her home of origin (Miller, 2002).

Poor marital relationship. Marital conflict has also been linked to the onset of PPD. Most commonly, marital dissatisfaction as measured by a lack of attachment to one's partner is positively correlated with PPD (Eberhard-Gran, Eskild, Tambs, Samuelson, & Opjordsmoen, 2002; O'Hara, 1986; Robertson et al., 2004). Meta-analyses of the relationship between the two has demonstrated a moderate effect size (Beck, 1996; Beck 2001; Robertson et al., 2004) at, $r = 0.35$, $r = 0.38$ to 0.39 , and $d = 0.39$, respectively. While marital discord alone does not fully account for the development of PPD, a supportive marital relationship may serve as a buffer against depression that can result subsequent to birth.

Stressful life events. Life stress has consistently emerged as a significant predictor of PPD (O'Hara et al., 1984; Paykel, Emms, Fletcher, & Rassaby, 1980; Righetti-Veltema, Conne-Perreard, Bousquet, & Manzano, 1998). The major meta-analytic studies found moderate effect sizes with stress due to life events. Robertson and colleagues (2004) and O'Hara and Swain (1996) reported a Cohen's d of 0.61 and 0.60

respectively. Similarly, Beck (1996, 2001) reported moderate effect sizes at $r = 0.40$ and $r = 0.36$ to 0.41 , respectively. Stressful life events during the year preceding childbirth have been detected as important predictors, especially financial and professional difficulties (O'Hara et al., 1984; Righetti-Veltema et al., 1998).

Self-esteem. Self-esteem has also emerged as a significant predictor of PPD. In Beck's (2001) meta-analysis of 84 studies examining PPD and its predictors, a moderate relationship between PPD and self-esteem, $r = 0.45$ to 0.47 , was found. It may be that high self-esteem serves as a buffer between the negative effects of stressful life events and the increased fragility of the ablest women during the postpartum period. Therefore, mothers with high self-esteem may have greater ability to withstand stressors that may contribute to the likelihood of developing postpartum depression.

Other factors. Several other variables have demonstrated inconclusive results in the literature. For example, the literature concerning the impact of obstetrical complications shows little agreement. While some have found that complications during pregnancy and delivery are a predictor of PPD (Campbell et al., 1992), other studies have found that the specialized care the woman receives may actually decrease the onset of PPD (Paykel et al., 1980). Likewise, some investigators have found a strong association between PPD and not breastfeeding (Eberhard-Grad et al., 2002; Misri, Sinclair, & Kuan, 1997); however, others have not found the same relationship (McCoy et al., 2008). Socioeconomic status has also been inconsistently linked to PPD, with some researchers reporting strong associations (Bernazzani, Saucier, David, & Borgeat, 1997; Righetti-Veltema et al., 1998) and others finding small effect sizes (Beck, 2001; Robertson et al., 2004). Additionally, inconclusive results have been reported with regard to unwanted

pregnancy as it emerges as a significant predictor in some studies (Beck, 2001), but not in others (O'Hara & Swain, 1996). Finally, other potential predictors for PPD cited as inconclusive in the literature include fewer years of education (Davis, Edwards, Mohay, & Wollin, 2003), a history of childhood sexual abuse (Buist & Barnett, 1995), and childcare stress (Honey, Bennett, & Morgan, 2003).

Depression History

In addition to social factors, depressive history is also a strong predictor of PPD. Research has indicated that baby blues, a history of MDD, a previous episode of PPD, and depression during pregnancy each independently predict PPD. Additionally, several researchers have identified depression during pregnancy to be the strongest predictor of PPD (Beck, 1996; O'Hara & Swain, 1996; Robertson et al., 2004).

Postpartum baby blues. Baby blues is a well-established risk factor for PPD (Bloch, Rotenberg, Koren, & Klein, 2005; Stowe & Nemeroff, 1995). While the blues do not require intervention, it is important to note that this reportedly normal syndrome may lead to later depression. Up to 20% of women with the blues will experience major depression in the first postpartum year (Campbell et al., 1992; O'Hara, 1987).

History of major depressive disorder. History of major depressive disorder has consistently been identified as a strong risk factor for PPD (Bloch et al., 2005; Stowe & Nemeroff, 1995). In fact, a past episode of depression is commonly one of the strongest predictors of PPD as women with a history of an affective disorder have a 1:3 chance of developing PPD (Appleby et al., 1998). Similarly, a previous episode of PPD has been found to put women at considerable risk for recurrent episodes following subsequent pregnancies (O'Hara, 1991). In fact, Spinelli (1998) estimates women with a history of

PPD to be 300 times more likely to have another episode of PPD in subsequent pregnancies.

Depression during pregnancy. Depression during pregnancy has been found to be a strong predictor of postpartum depression (Beck, 1996; Robertson et al., 2004; Terry, Mayocchi, & Hynes, 1996; Verkick, Pop, Van Son, & Van Heck, 2003). In the meta-analyses completed by Robertson and colleagues (2004), O'Hara and Swain (1996), and Beck (1996) the strongest predictor of postpartum depression was depression during pregnancy, $d = 0.75$, $d = 0.75$, and $r = 0.51$, respectively. Therefore, depression during pregnancy has consistently emerged as a strong predictor of PPD.

Treatment of Depression During Pregnancy

It is not clear whether the treatment of major depression during pregnancy and the early postpartum phase can protect women from the development of PPD. It is, however, an important empirical question considering the strong predictive relationship between depression during pregnancy and the onset of PPD. Treatment that both reduces maternal depression and offers protective advantages postpartum is advantageous not only for the woman herself, but also for the fetus, infant, and other family members. Empirical research on treatment during pregnancy during the past decade has focused largely on pharmacological, psychotherapeutic, and alternative therapies with few definitive results. Therefore, treatment options for depressed pregnant women are frequently limited.

Pharmacological Treatment

With the advent of effective and well-tolerated pharmacological treatments for major depression, a growing number of women have been treated with psychotropic medication during their reproductive years. In 2003, approximately 13% of women took

an antidepressant medication at some point during pregnancy, a rate that doubled since 1999 (Cooper, Willy, Pont, & Ray, 2007). The clinician faces certain challenges when treating a depressed woman during pregnancy. All psychotropic medications diffuse readily across the placenta and no psychotropic medication has been approved by the U.S. Food and Drug Administration for use during pregnancy, nor have any antidepressant drug efficacy trials been completed in depressed pregnant women (Yonkers, Smith, Gotman, & Belanger, 2009). Although data has accumulated suggesting that some medications may be used safely during pregnancy, current knowledge regarding the risks of prenatal exposure to psychotropic medications is incomplete. Clinical guidelines for the pharmacological treatment of depression during pregnancy recommend carefully weighing the risks to the women and fetus associated with no treatment relative to the risks of treatment (Yonkers et al., 2009).

Among antidepressant medications, selective serotonin reuptake inhibitors (SSRIs) are the most widely used in the general population (Mann, 2005) as well as among pregnant women (Reefhuis, Rasmussen, & Friedman, 2006). Fluoxetine, the first SSRI to be marketed, was introduced in 1988 and soon became the most frequently prescribed medication for depression worldwide (Morrison, Riggs, & Rurak, 2005). Other currently prescribed SSRIs include sertraline, paroxetine, citalopram, escitalopram and fluvoxamine.

Selective serotonin reuptake inhibitors (SSRIs). SSRIs are among the most commonly used antidepressant medications, with a prescription frequency of 2.3% in pregnant women (Reefhuis, Rasmussen, & Friedman, 2006). Potential adverse effects of maternal SSRI treatment on the developing embryo or fetus can be categorized into five

types: miscarriage; malformations; neonatal toxicity or withdrawal; prematurity or low birth weight; and functional abnormalities, including long term neurocognitive effects (Alwan & Friedman, 2009).

The current data on SSRI exposure does not support specific morphological teratogenic risks. The majority of studies have focused on cardiac malformations and have found no increased risk subsequent to in utero SSRI exposure (Alwan, Reefhuis, Rasmussen, Olney, & Friedman, 2007; Louik, Lin, Werler, Hernandez-Diaz, & Mitchell, 2007; Malm, Klaukka, Neuvonen, 2005; Yonkers et al., 2009). There has been some evidence that exposure to fluoxetine during the first trimester increased risk for minor cardiac malformations (Kallan & Otterblad Olausson, 2007; Kallan & Otterblad Olausson, 2006); however, other large projects have disputed these findings (Alwan et al., 2007; Louik et al., 2007). There has also been evidence of increased risk for cardiac malformations associated with the use of paroxetine in the first trimester. A 2007 meta-analysis found in utero exposure to paroxetine resulted in twice as many echocardiograms in the first year of life (Bar-Oz et al., 2007) and other researchers found a significant association between 1st trimester use of paroxetine and right ventricular outflow tract obstruction defects (Louik et al., 2007). According to Alwan and Friedman (2009), first trimester maternal paroxetine use may increase the risk for cardiac anomalies about 1.5-fold, with a risk of approximately 1%. Although maternal SSRI treatment early in pregnancy does not appear to increase the overall risk of birth defects greatly, SSRI treatment during the first trimester of pregnancy has been associated with an increased risk of cardiac defects. While overall risk of congenital malformation following early prenatal exposure appears low, further data are needed to ensure clinical confidence.

A number of studies have shown that maternal SSRI treatment late in pregnancy can impair neonatal adaptation. Several studies have indicated that third trimester exposure may increase risk for persistent pulmonary hypertension (PPHN), a rare but serious condition which can result in neonatal hypoxia (Chambers et al., 2006; Kallen & Olausson, 2008). Research has suggested a rate of absolute risk 3-6 per 1000 among infants subsequent to third trimester exposure (Kallen & Olausson, 2008). Other research findings indicate poor neonatal adaptation manifested by greater rates of respiratory distress following exposure to paroxetine in the 3rd trimester (Costei, Kozer, Ho, Ito, & Koren, 2002) or exposure to paroxetine, fluoxetine, or sertraline in the 2nd or 3rd trimester (Oberlander, Fitzgerald, Kostaras, Rurak, & Riggs, 2004). Finally, one study found that women who have endured longer periods of SSRI treatment, as opposed to shorter periods of treatment, were at higher risk of delivering an infant with respiratory complications (Oberlander, Warburton, Misri, Aghajanian, & Hertzman, 2008).

In addition to respiratory distress, a cluster of other symptoms associated with late pregnancy SSRI exposure has been consistently reported during the immediate days following birth. Reported findings among newborn infants whose mothers took an SSRI in late pregnancy include temperature instability, hypoglycemia, tachypnea, irritability, and seizures (Chambers, Johnson, Dick, Felix & Jones, 1996; Costei et al., 2002). Additionally, analyses of the Swedish Medical Registry found that 649 SSRI exposed infants (citalopram, paroxetine, fluoxetine or sertraline) as well as SNRI exposed infants (venlafaxine) exhibited greater risk for neonatal adaptation compared to all other infants in the database (Kallen, 2004). These risks included respiratory distress, hypoglycemia, lower Apgar scores, and convulsions. Some difficulty with neonatal adaptation is

apparent in about 30% of newborns whose mothers are treated with SSRIs late in pregnancy, though symptoms in newborns are transient and tend to resolve within two weeks following delivery (Chambers et al., 1996; Moses-Kolko et al., 2005; Oberlander et al., 2004; Oberlander et al., 2008).

The rate of preterm birth (<37 weeks gestation) in the United States is 12.7% and is a leading cause of perinatal mortality (Yonkers et al., 2009). There is substantial evidence that preterm birth is higher among women taking antidepressant medication during pregnancy (Chambers et al., 1996; Costei et al., 2002; Djulus et al., 2006; Kallen, 2004; Simon, Cunningham, & Davis, 2002). Additionally, mothers who use SSRIs late in pregnancy have on average a three-fold increased risk of having a premature baby (Alwan & Friedman, 2009). Further studies show that the effects of SSRIs on gestational age are dependent on the duration of in utero exposure and longer exposure leads to decreased gestational age (Oberlander et al., 2008).

Finally, there has been limited research examining the longer term effects of SSRI exposure in utero. One study found that children born to women treated with SSRIs during pregnancy were delayed in their psychomotor development and fine motor control at six to forty months of age compared to children of untreated depressed mothers (Casper et al., 2003). Additional research is needed to better understand potential long term consequences of in utero exposure to SSRIs.

Given the current body of research demonstrating the fetal effects of maternal SSRI treatment during pregnancy, providers are left with a dilemma. It is imperative that the potential risks and benefits of discontinuing (or continuing) SSRI treatment during pregnancy be assessed thoroughly and discussed with each patient. Guidelines for the

treatment of depressed pregnant women have been published and are available to providers (Yonkers et al., 2009).

Tricyclic antidepressants (TCAs). While an increasing number of women are being treated with SSRIs during pregnancy, tricyclic antidepressants remain a common alternative. They tend to be less frequently prescribed due to adverse side effects for the mother, particularly constipation and orthostatic hypotension, which may be exacerbated by pregnancy (Payne & Meltzer-Brody, 2009). Early case reports suggested a possible association between 1st trimester exposure to TCAs and limb malformation; however, more recent studies have failed to find a significant association between fetal exposure to TCAs and risk for any major structural malformations (Altshuler et al., 1996; Cohen & Altshuler, 1997; Simon et al., 2002). Research has found evidence for neonatal toxicity in infants exposed to TCAs at or near the time of delivery. These have included TCA withdrawal syndromes with characteristic symptoms of jitteriness, irritability and seizure (Kallen, 2004) as well as increased preterm delivery, increased risk for respiratory distress syndrome, endocrine and metabolic disturbances, and temperature regulation disorders (Davis et al., 2007). Finally, with regard to longer term neurobehavioral effects, further investigation is needed.

Psychotherapeutic Treatment

Due to maternal treatment preferences and concerns about fetal and infant health outcomes associated with antidepressant treatment, non-pharmacological treatment options are needed. A systemic review reported that patients with MDD in primary care prefer psychotherapy over antidepressant medication for treatment if psychotherapy is available (van Schaik et al., 2004). In addition, mothers remain hesitant to take

medication without definitive studies regarding long-term effects on child development (Sit & Wisner, 2005). Given these preferences, and the incomplete knowledge regarding the risks of prenatal exposure to psychotropic medications, the need for controlled psychotherapeutic treatment trials is great.

Despite the need for controlled clinical trials, research in this area is limited. The only form of psychotherapy that has been specifically tested during pregnancy and found to be effective for pregnant women is interpersonal therapy (IPT) (Grote et al., 2009; Spinelli & Endicott, 2003). IPT is a well established treatment for depression in the general population (Klerman, Weissman, Rounsaville, & Chevron, 1984). It became attractive as a treatment for perinatal depression because of its focus on disrupted interpersonal relationships, disputes, and making role transitions. Because of the unique and developmental problems associated with gestation, IPT trials with pregnant women also include an additional focus on pregnancy complications in which problems specific to pregnancy are addressed (undesired pregnancy, complications, multiple births, and congenital anomalies) (Grote et al., 2009; Spinelli & Endicott, 2003).

The initial results of studies using IPT to treat depressed pregnant women are promising. To date, there have been few randomized controlled clinical trials of interpersonal therapy with depressed pregnant women (Grote et al., 2009; Spinelli & Endicott, 2003). Spinelli and Endicott (2003) found a significant improvement in depression scores among women assigned to 16 weeks of interpersonal psychotherapy as compared to a parenting education control program on all measures of mood at termination (Edinburgh Postnatal Depression Scale, the Beck Depression Inventory, and the Hamilton Depression Rating Scale) and recovery criteria were met in 60% of the

women treated with interpersonal psychotherapy. Grote et al. (2009) found a significant reduction in depression diagnoses (using SCID criteria) among participants assigned to IPT compared to those in enhanced usual care following an eight week treatment during pregnancy which was maintained at six months postpartum. Additional uncontrolled trials of IPT during pregnancy have demonstrated significant improvement in depressive symptoms following treatment in pregnancy (Grote et al., 2004; Swartz, 2004). Further research is necessary to support the efficacy of other forms of psychotherapy for depression during pregnancy.

While psychotherapy is a safe treatment option during pregnancy, it is not readily available in the HMO dominant market and may not be acceptable to all pregnant women. Moreover, while initial evidence suggests that depressed pregnant women may benefit from interpersonal therapy during pregnancy, additional research is needed to further support this finding and identify other efficacious psychotherapies. Thus the current treatment options for depressed pregnant women are quite limited and studies of novel, safe and accessible treatments are needed for depression during pregnancy.

Complementary and Alternative Medicine (CAM)

In addition to depressed pregnant women, non-pregnant depressed individuals are also dissatisfied with current treatment options. Surveys of the United States population report that 22.4% of patients meeting criteria for major depressive disorder used alternative treatments in the past 12 months (Unutzer et al., 2000), either alone or in conjunction with traditional treatments, and women were more likely than men to use alternative treatments (Mackenzie, Taylor, Bloom, Hufford, & Johnson, 2003; Tindle, Davis, Phillips, & Eisenberg, 2005). The popularity of alternative treatments for

depression may, in part, suggest that conventional treatments fail to provide full or lasting remission. For example, the NIMH Collaborative Study of the Psychobiology of Depression, a prospective, naturalistic, longitudinal investigation on the course of illness in MDD, identified the cumulative probability of recurrence at nearly 30% after six months follow-up, almost 40% after 12 months of follow-up (Keller, Lavori, Lewis, & Klerman, 1983), and the risk of recurrence increased by 16% with each successive recurrence (Solomon et al., 2000). The high rate of recurrence among those suffering from MDD may be attributed to a number of factors, but certainly suggests a need for additional, efficacious and accessible treatments.

While there is a paucity of research in this area, complementary and alternative medicine (CAM) interventions have generated recent interest. Many of these interventions are desirable because they can easily be added to a conventional treatment plan with little risk and general health benefits for the patient. Research in this area has focused on supplements including St. John's Wort, S-Adenosyl-Methionine (SAMe), folate, 5-Hydroxytryptophan (5-HTP), and omega 3 fatty acids as well as bright light therapy, massage and exercise. Among CAM treatments, one of the more compelling areas of research has been on the use of acupuncture as a treatment for depression, including depression among pregnant women.

Acupuncture. Among CAM treatments, acupuncture has become a subject of great interest and one of the most researched complementary therapies. Acupuncture, as practiced in the United States, is used within the framework of traditional Chinese medicine, in which organ systems are thought to be connected via energetic channels and disease states are thought to arise from imbalance within those channels. Within the

context of Chinese medicine, the focus is not on the diagnosis and treatment of disease but rather on the detection of energetic imbalances. Because the patient is considered as a totality of body and mind, both physiological and psychiatric symptoms are equally important in the evaluation of patients and the resulting treatment prescribed.

Theory behind the use of acupuncture for depression. Chinese medicine views health as a balance between the Yin and Yang forces, which are dependent upon the proper circulation of vital energy, or Qi (chee), along the energetic pathways. Yin, which is nourishing, grants the qualities of rest, tranquility, and quiescence. When Yin is deficient, patients lack the qualities of receptivity and contemplation leading to agitation, nerves, and uneasiness (Kaptchuk, 1983). Yang, which by contrast is activating, causes transformation and change, providing the capacity to engage in life, react, and respond. When Yang is deficient, patients are often paralyzed in fear, confusion, indecision, and hopelessness (Kaptchuk, 1983). The balance of Yin and Yang depends on the capacity to adapt and change and is related to health and disease. When Yin and Yang are in balance, it allows for the natural flow of Qi which in turn optimizes health.

The proper circulation of Qi along energetic pathways, or meridians, sustains the homeostasis of an individual (the balance of Yin and Yang). The experience of a disorder, the nature of its symptoms, and the protocol for its treatment are determined by the specific tendencies in every person toward a deficiency or excess of Yin or Yang (Beinfeld & Korngold, 1991). These tendencies precipitate personal patterns of reaction. For example, in depressed individuals, patients may present with symptoms such as lethargy, weakness, lack of motivation, and loss of appetite. Chinese medicine would conceptualize these patients as manifesting a pattern of symptoms in which Yin is

predominant and Yang is deficient. However, in patients presenting with anxiety, irritability, agitation, increased appetite, and insomnia, Yang is predominant and Yin is deficient. In many cases of depression, imbalances between Yin and Yang are often linked to the liver network, the kidney network, and the heart network. The acupuncture points utilized will therefore consist of points activating the meridians linked to each of these organ systems depending on the symptom profile presented. Through activation and subsequent circulation of Qi along these meridians in one's body, homeostasis can be reestablished and health optimized (Beinfeld & Korngold, 1991).

In addition to releasing Qi stagnation in patients with depression, Chinese medicine also links Shen, or the organizing force of the self, to emotional imbalance. Shen organizes the emotional, mental and expressive life of the individual and is housed by the heart organ network (Beinfeld & Korngold, 1991). Therefore, an individual's response to the environment is determined by the health of the Shen. Inability to express joy and fulfillment, confusion, and agitation that are experienced during a depressive episode are conceptualized in Chinese medicine as manifestations of the heart's lost ability to enfold the Shen.

In summary, within the context of Chinese medicine, depression can be understood as a complex energetic reaction pattern that involves a predisposing tendency towards excess or deficiency of Yin or Yang, combined with varying degrees of Qi stagnation and Shen disturbance. Chinese medicine provides a framework for understanding symptom patterns from which an individually tailored acupuncture treatment plan can be developed. Thus, acupuncture treatments for depression focus on

the symptom profile presented, which may include both physiological and psychiatric symptoms.

Research on acupuncture treatment and depression. As a result of the need for safe, effective, and affordable alternative treatments for depression, researchers began to design and conduct randomized controlled trials to assess the efficacy of acupuncture as a treatment for depression. Although several randomized clinical trials have reported a significant reduction of depressive symptoms following treatment with acupuncture (Allen, Schnyer, & Hitt, 1998; Eich, Angelink, Lehmann, Lemmer, & Kleiser, 2000; Luo et al., 2003; Quah-Smith, Tang & Russell, 2005), generally the results appear contradictory. For example, the first randomized controlled trial, which recruited a small sample of woman with major depression, reported that after eight weeks of treatment there was a significant difference in symptom reduction between acupuncture and placebo groups on HRSD depression scores (Allen et al., 1998). However, in a larger scale clinical trial published in 2006, including both men and women, the same authors failed to support the efficacy of acupuncture as a monotherapy for major depression as greater decreases in depressive severity were exhibited by both groups (acupuncture and sham acupuncture) and the two groups did not differ from each other (Allen et al., 2006).

Because of the conflicting results achieved in different trials, several systemic reviews have been completed. A 2007 systematic review, which compared acupuncture with control conditions within nine randomized controlled trials, suggested some evidence for the utility of acupuncture in depression. Acupuncture modalities were as effective as antidepressants (Leo & Ligot, 2007). Additionally, a meta-analysis of eight randomized, controlled trials supported acupuncture as an effective treatment that could

significantly reduce the severity of depression (Wang et al., 2008). In a more recent systemic review, containing data from 30 studies, overall there was insufficient evidence of a consistent beneficial effect from acupuncture compared to controls or sham acupuncture (Smith, Hay, & Macpherson, 2010). However, among the studies included, two trials found acupuncture had an additive benefit when combined with medication compared to medication alone and a subgroup of participants experienced a reduction in depression with acupuncture alone compared to SSRIs. Generally, these conflicting results are complicated by the variability across studies with regard to differences in methodology, degree of experimental control, type of depression treated, and the outcome measures used.

While the majority of research has examined the efficacy of treating depressed men and women, a subset of research is developing that focuses specifically on the treatment of women. As mentioned above, the first randomized, double-blind, placebo-controlled study on the efficacy of acupuncture for depression consisted of a sample of 38 women ages 18-45. In the study, Allen, Schnyer, and Hitt (1998) found acupuncture provided significant depressive symptom relief; however, in a follow-up study (Allen et al., 2006) in which men were also included in the sample, the results were not significant. This may suggest that women of a childbearing age respond differently to the treatment and perhaps the use of acupuncture is more beneficial to them. Based on the initial findings of Allen and colleagues (1998), additional research has focused specifically on the treatment of depressed pregnant women with the use of acupuncture.

Research focused on the use of acupuncture to treat depressed pregnant women appears promising. In a prospective, controlled study of 51 mild to moderately depressed

pregnant women, researchers reported depressive symptoms decreased by 60% in women receiving acupuncture compared to 26% in the control group (Silva, 2007). In a randomized, double-blind, placebo-controlled pilot study on the efficacy of treating major depression during pregnancy with acupuncture, a significantly larger proportion of participants responded to an acupuncture treatment specific for depression (68.8% response rate) than to massage (31.6%); however, a statistically significant difference was not found between acupuncture treatment specific for depression and the control acupuncture group (47.4% response rate) (Manber, Schnyer, Allen, Rush & Blasey, 2004). These rates demonstrated comparable response rates to clinical trials of standard treatments for depression, typically 50-70% (Elkin et al., 1989). Furthermore, in Manber and colleagues (2004) pilot study, they found a significant main effect for response status to the acute phase of treatment on depression symptom severity at ten weeks postpartum, suggesting successful treatment with acupuncture or massage may result in an enduring effect into the postpartum phase.

Based on these preliminary results, Manber et al. (2010) completed a randomized controlled trial to examine the efficacy of acupuncture for treatment of depression during pregnancy. Women who received eight weeks of acupuncture specific for depression experienced a greater rate of decrease in depressive symptom severity compared to the combined control groups (control acupuncture and massage) and compared to control acupuncture alone. The women in the acupuncture specific for depression group also had a significantly greater response rate (63%) compared to the combined controls (44.3%). These results are meaningful when considering the length of treatment (eight weeks) is

shorter than the duration of most psychotherapy trials (12-16 weeks), yet the response rate remains comparable to those same trials.

Based on these findings, the use of acupuncture to treat depressed pregnant women appears promising. Because of the lack of psychotherapies examined for efficacy in pregnancy, along with concerns related to the use of antidepressants taken during pregnancy, alternative treatments are needed for depressed pregnant women. The current popularity and emergence of alternative treatments further suggests women are dissatisfied with conventional treatments for depression during pregnancy. Based on these concerns, it is imperative that researchers continue to pursue early detection of depressive symptoms and prompt initiation of treatment during pregnancy, including promising alternative treatments, in order to reduce the adverse consequences associated with PPD.

Treatment During Pregnancy to Prevent Postpartum Depression

To date, the majority of studies completed examining treatment interventions for postpartum depression have been implemented during the postpartum phase. Among these, medication, alone or with CBT (Appleby, Warner, Whitton & Faragher, 1997; Appleby et al., 1997; Suri, Burt, Altshuler, Zuckerbrow-Miller, & Fair, 2001), interpersonal therapy (O'Hara, Stuart, Gorman, & Wenzel, 2000), and CBT (Chabrol et al., 2002; Murray, Cooper, Wilson, & Romaniuk, 2003) produce the largest effect sizes (Bledsoe & Grote, 2006). Few studies have examined whether treatment during pregnancy can have an enduring effect into the postpartum. Although prevention approaches hold much promise to improve the outcomes of both mother and offspring, the field is in its developing stages. There have been few studies of prevention of

depression in pregnant women and there are even fewer randomized control trials focusing on depression during pregnancy through the postpartum period (Boyd, Pearson, & Blehar, 2002). Despite this, postpartum depression provides an ideal opportunity for prevention because its onset is preceded by a clear marker (giving birth), the period of risk for illness onset is well defined, and a high-risk sample of mothers can be identified. Additionally, pregnant women may be unusually open to making changes to improve their mental health before their baby is born (Cowan & Cowan, 2000).

Empirical research targeting high-risk women during pregnancy, in attempt to prevent postpartum depression, has been conducted largely in the last decade. The categorization of high-risk women has varied across studies, but commonly requires women meet one of the following: a personal history of depression, family history of psychiatric disturbance, depressive symptoms during pregnancy, marital conflict, or current negative life events. Interventions begin during pregnancy, often extend into the early postpartum phase, and are largely psychotherapeutic.

Among interventions, initial support for the use of interpersonal therapy as a preventative measure in asymptomatic high-risk women has been documented. Gorman (1997) randomly assigned 45 high-risk women to receive preventive IPT or a no treatment control. The intervention consisted of two individual sessions during pregnancy (from 32 weeks to delivery) and three weekly sessions between two to four weeks postpartum. At one month postpartum women in the intervention group were significantly less likely to have experienced major depression compared to women in the no treatment group (0% vs. 25%); however, at six months postpartum the difference between groups was no longer significant (Gorman, 1997). Similarly, Zlotnick, Johnson,

Miller, Pearlstein, & Howard (2001) randomized 37 low-income, high-risk women to four weekly sessions of group interpersonally-oriented psychoeducation or to a control. At three months postpartum women in the intervention group were significantly less likely to have developed PPD (0%), compared to the control (33%) (Zlotnick et al., 2001). These initial studies using a modified version of interpersonal therapy for prevention provide promising results.

In addition to interpersonally-oriented interventions, group psychoeducation has also been researched as a means to prevent PPD. While the format and content of the groups varies greatly by study, the general focus tends to be on problem solving strategies to avoid PPD as well as the importance of social support networks. Within this line of research, the results are inconclusive. Brugha et al. (2000) conducted a randomized prevention trial with 190 asymptomatic high-risk pregnant women consisting of six 2-hour session groups during pregnancy and an additional session at eight weeks postpartum. The intervention had no effect with respect to depressive symptomatology or diagnostic status at three months postpartum (Brugha et al., 2000). Additionally, Elliott Leverton, Sanjack, and Turner (2000) conducted an intervention with 99 asymptomatic high-risk pregnant women consisting of five group sessions during pregnancy (beginning at 24 weeks) and six group sessions postpartum. The results indicated significant differences with respect to level of depressive symptomatology at three months postpartum, only for first time mothers, and no differences were found at 12 months postpartum (Elliott et al., 2000).

Finally, there has been some research suggesting that the presence of antidepressants during a high-risk period may prevent the onset of another depressive

episode. Wisner and colleagues (Wisner et al., 2001; Wisner et al., 2004; Wisner et al., 2006), have studied the effects of starting treatment immediately after delivery compared to watchful waiting in samples of asymptomatic high-risk mothers with a history of PPD. In their initial study (Wisner et al., 2001), they compared nortriptyline to placebo and found no significant differences between groups with regard to depressive symptomatology. However, in their second study (Wisner et al., 2004), a 17 week trial of sertraline (compared to placebo) resulted in significantly fewer recurrences of major depression in those taking sertraline (7%) compared to placebo (50%). In addition, the time to recurrence was also significantly longer in the sertraline treated women than in the placebo treated women. Finally, in a more recent, definitive study, (Wisner et al., 2006), the researchers found no differences between nortriptyline and sertraline in an eight week comparative trial with a 16 week continuation phase. The proportion of women who responded and remitted did not differ, nor did the times to response and remission.

Both psychosocial and pharmacologic treatment trials designed to prevent postpartum depression in asymptomatic high-risk pregnant women have demonstrated mixed results. The interpersonally-based interventions have fared better than more general, educational based groups. The administration of an antidepressant for prevention of postpartum depression in high-risk, asymptomatic women remains inconclusive. The differences in outcome across studies may be due to variability on a number of dimensions. These include: the qualification of high-risk, the type of professional conducting the intervention, the type of intervention provided, as well as the number of sessions and timing of the intervention. Researchers have recommended

prevention interventions are conceptualized such that high-risk pregnant women continue to be targeted (Stuart, O'Hara, & Gorman, 2003). This conceptualization suggests that while interventions aimed at preventing acute PPD have not yet demonstrated strong support, it is clinically compelling to promote advances for women at risk during pregnancy.

While researchers have recommended that prevention interventions of postpartum depression target asymptomatic high-risk pregnant women (Stuart et al., 2003), others have taken this rationale further by recommending that treatments be directly targeted at depressed pregnant women (Austin, 2003). It is hypothesized that treating one of the strongest predictors of postpartum depression, namely depression during pregnancy, may reduce the likelihood of developing postpartum depressive symptomatology. In fact, recent studies have suggested that the prevalence of depressive symptoms during pregnancy is similar to that seen postpartum (Evans, Heron, Francomb, Oke, & Golding, 2001; Fergusson, Horwood, & Thorpe, 1996) and one study found that up to 40% of postpartum depression began prenatally (Green & Murray, 1994). According to Austin (2003), these figures suggest that evaluating 'indicated' interventions (i.e. in women with existing symptoms or early diagnosis of depression in pregnancy) will likely be a more fruitful approach than simply targeting high-risk women without current symptoms.

Furthermore, while the onset of depressive symptoms may occur after delivery, there are a significant proportion of women who experience symptoms during pregnancy that carry over into the postpartum period. Research indicates that the incidence and severity of depression increases from the first to the third trimesters of pregnancy (Gotlib, Whiffen, & Wallace, 1991) and several studies have found higher depressive scores

during the last two months of pregnancy compared to the first eight weeks postpartum (Evans et al., 2001; Hayes, Muller, & Bradley, 2001; Josefsson et al., 2001). Therefore, when women develop symptoms during the latter part of pregnancy, prevention becomes more difficult if treatment is implemented after delivery and an earlier intervention becomes increasingly important.

In order to alleviate depression during pregnancy and prevent depressive relapse postpartum, efficacious interventions are needed for high-risk, depressed pregnant women. Moreover, because many women wish to avoid the risks associated with taking antidepressants, the need for alternative interventions is great. While it is not clear whether the treatment of a major depressive episode during pregnancy and the early postpartum phase can protect women from the development of PPD, there is some evidence to support this theory.

To date, there have been few studies to test this theory directly. Most notably, Grote and colleagues (2004; 2009) have completed two studies examining the treatment of depressed pregnant women using brief interpersonal therapy to prevent postpartum depression. In 2004, they completed a pilot study with twelve women who received eight sessions of brief IPT during pregnancy, followed by monthly maintenance IPT sessions up to six months postpartum. The results indicated significant improvement on depression symptom severity measures (EPDS, BDI, and HRSD) both at post-treatment as well as at the six month postpartum assessment. Furthermore, in their 2009 study, 53 pregnant women received eight sessions of brief IPT during pregnancy, followed by monthly maintenance IPT sessions up to six months postpartum. Similar to their pilot study, the results indicated significant reductions in depression diagnoses and depressive

symptoms before childbirth (three months post-baseline) and at six months postpartum.

These studies provide initial support for the notion that treating depressed pregnant women may help to avert a postpartum depressive relapse.

While Grote and colleagues (2004; 2009) examined whether treatment during pregnancy can reduce the likelihood of postpartum depression, more commonly studies focusing on the treatment of depressed pregnant women have not instituted a postpartum follow-up. As a result, there is very limited data in this area. In Manber and colleagues (2004) pilot study examining the use of acupuncture treatment for depressed pregnant women, the authors found a significant main effect for response status to the acute phase of treatment on depression symptom severity at ten weeks postpartum. Few studies, however, have offered exploratory information regarding postpartum follow-up subsequent to treatment if assessed. For example, in Spinelli & Endicott's (2003) controlled trial of IPT compared to a parenting education program for depressed pregnant women, they reported that seven of eight women treated with IPT had no postpartum depression. In their control group, they found two of three available subjects had postpartum depression, and one did not. Few inferences can be made from these data; however, it emphasizes the importance of postpartum follow-up and the potential for prevention.

Present Study and Hypotheses

Given the deleterious costs of postpartum depression to individuals and society, prevention and treatment are large concerns. In the past decade, research has begun to examine viable treatment options during pregnancy in order to prevent depressive symptomatology in the postpartum phase. This research has largely included

interventions targeting high-risk, asymptomatic, pregnant women. More recently, recommendations have been made to direct these treatments at depressed pregnant women because of the large number of cases of PPD which are carried over from pregnancy, and also because depression during pregnancy is the strongest predictor of PPD. Therefore, it has been hypothesized that treating depressed pregnant women will be a more fruitful approach than simply targeting high-risk women without current symptoms.

In addition to intervention timing, researchers have begun to expand viable treatment options for pregnant and postpartum women. Studies of novel, safe, and accessible treatments are greatly needed. Many mothers remain hesitant to take medication both during pregnancy and in the postpartum phase without definitive studies regarding long-term effects on child development (Sit & Wisner, 2005). Given these preferences, and the incomplete knowledge regarding the risks of exposure to psychotropic medications, the need for controlled alternative treatment trials is great.

The present study was designed to extend this recent body of work by examining the role of treating depressed pregnant women with acupuncture and massage as a means to manage mood symptoms and reduce the likelihood of postpartum depression. It was conducted as part of a larger study which examined the efficacy of acupuncture for the treatment of depression in pregnant women and included three treatment group assignments: acupuncture needling points specific to depression, acupuncture needling non-specific points, and massage. Therefore, the current study did not examine treatment effects following the acute phase of treatment, but focused solely on the postpartum period. More specifically, the study examined whether or not successful treatment of

depression during pregnancy provided protection against the development of postpartum depression and explored factors that predicted postpartum depression in this high-risk group.

Rationale and Hypothesis One: Treatment Response and Postpartum Depression

Among interventions targeting asymptomatic high-risk women during pregnancy to prevent postpartum depression, studies have demonstrated initial support. Interventions using interpersonal therapy during pregnancy found participants were significantly less likely to develop postpartum depression, compared to those who did not receive treatment, at one month postpartum (Gorman, 1997) and three months postpartum (Zlotnick et al., 2001). Additionally, among IPT studies targeting women already experiencing depression during pregnancy, results have found participants maintain the treatment effect up to six months postpartum (Grote et al., 2004; Grote et al., 2009). The results of these studies, targeting both asymptomatic high-risk women and women already experiencing depression during pregnancy, suggest that interventions focused on high-risk women may result in an enduring effect in the postpartum phase.

Furthermore, in Manber and colleagues (2004) pilot study of treating 61 depressed pregnant women with acupuncture, they found a significant main effect for response status to the acute phase of treatment on depression symptom severity at ten weeks postpartum. These results revealed that, regardless of treatment modality, early successful treatment of depression during pregnancy incurred protection against postpartum depression in a high-risk group. A similar effect is expected in the current study. Therefore, it is hypothesized that responders to the acute phase of treatment during pregnancy, across all three treatment groups, will have better clinical outcomes at

ten weeks, six months, and nine months postpartum than those who have not responded to the acute phase of treatment. More specifically, the following hypotheses are proposed with regard to clinical status in the postpartum phase:

Hypothesis 1(a): It is hypothesized that responders to the acute phase of treatment during pregnancy, across all three treatment groups, will have better clinical outcomes as measured by the Hamilton Rating Scale for Depression (the study's main outcome measure) compared to non-responders at ten weeks, six months, and nine months postpartum.

Hypothesis 1(b): It is hypothesized that responders to the acute phase of treatment during pregnancy, across all three treatment groups, will have lower depression symptom severity scores as measured by the BDI-II compared to non-responders at ten weeks, six months, and nine months postpartum.

Rationale and Hypothesis Two: Treatment and Relapse Rate

While it is hypothesized that responders will endure longer-term benefits from their treatment, some women might experience re-occurrence of MDD during the postpartum phase. The re-occurrence might represent a relapse into the index episode or a recurrence into a new episode, depending on the time in remission. Women with a history of depression, and particularly postpartum depression, are at increased risk for relapse. Cohen et al. (2006) recently reported a five times higher rate estimate of relapse among pregnant women who discontinued antidepressants compared to women who did not discontinue antidepressants. Additionally, 26% of women who continued antidepressants throughout pregnancy relapsed. The women in their study were considered high-risk based on multiple episodes of MDD and early onset of illness;

therefore, the results of the study cannot be extrapolated to the general population.

However, the cumulative probability of relapse among patients with MDD in the general population after six months follow-up is nearly 30% and almost 40% after twelve months of follow-up (Keller, Lavori, Lewis, & Klerman, 1983). Based on these estimates, it is expected that a subset of women may experience re-occurrence of MDD during the postpartum phase. Despite this, it is expected that the acupuncture group specifically tailored to address depression related patterns of disharmony (SPEC) will fare better in terms of time to relapse. More specifically, the following hypothesis is proposed:

Hypothesis 2: It is hypothesized that time to relapse will be sooner for participants in the two control groups (acupuncture addressing nonspecific depression pressure points (NSPEC) and prenatal massage (MSSG)) than for participants in the SPEC group when examined at ten weeks, six months, and nine months postpartum.

Rationale and Hypothesis Three: Predictors of Postpartum Depression.

Historically, research on PPD has focused largely on its predictors. Among those studies, a history of depression consistently emerges as one of the strongest predictors of PPD. This includes a history of recurrent depression and a history of perinatal and postpartum depression. Verkerk and colleagues (2003) reported a personal history of depression and high depressive symptomatology during mid-pregnancy were the two largest risk factors which predicted PPD. Depression during pregnancy, which is often cited as the strongest predictor of PPD, is the most important inclusion criteria for women to participate in the current study. It is expected that additional depressive history, will lead to increased levels of depressive symptomatology in the postpartum period.

Additionally, women who have suffered from one episode of PPD experience increased

risk for recurrence in the year following another birth (Wisner et al., 2004). Therefore, it is hypothesized that clinical status during the postpartum will be worse for women with a history of previous depressive episodes. More specifically, the following hypotheses are proposed:

Hypothesis 3(a): It is hypothesized that women with a history of previous episodes of depression will be more likely to meet criteria for MDD at any time point during the postpartum (ten weeks, six months, or nine months postpartum).

Hypothesis 3(b): It is hypothesized that women with greater numbers of past episodes of depression during pregnancy and postpartum will have higher depression symptom severity scores as measured by the Hamilton Rating Depression Scale at six months and nine months postpartum.

Exploratory Analyses: ROC Curve Approach

While a history of previous depressive episodes has consistently emerged as a strong predictor of PPD, a second factor that appears to be associated consistently with PPD is support. Several studies have found that women who were depressed postpartum reported greater marital tension and more persistent feelings of being unloved by their husbands (Eberhard-Gran et al., 2002; Gotlib et al., 1991; O'Hara, 1986; Whiffen, 1988). These studies suggest that a lack of spousal support may play an important role in the development of PPD.

Other factors related to the onset of PPD have not demonstrated consistent, strong associations as compared to the evidence for history of depression and lack of support. For example, some investigators have found a strong association between PPD and not breastfeeding (Eberhard-Grad et al., 2002; Misri et al., 1997); however, others have not

found the same relationship (McCoy et al., 2008). Socioeconomic status has also been inconsistently linked to PPD with some researchers reporting strong associations (Bernazzani et al., 1997; Righetti-Veltima et al., 1998) and others finding small effect sizes (Beck, 2001). Additionally, inconclusive results have been reported with regard to unwanted pregnancy (Beck, 2001; O'Hara & Swain, 1996), childhood sexual abuse (Buist & Barnett, 1995), childcare stress (Honey et al., 2003), and obstetrical complications (Campbell et al., 1992).

Because of the importance of understanding how factors beyond treatment influence the development of PPD, exploratory analyses examine potential predictors of PPD. While it is clear in the literature that both history of depression and spousal support are strong predictors of PPD, other variables have not demonstrated conclusive results. Thus, there was not a compelling rationale upon which to build a hypothesis for these predictors of PPD. As a result, exploratory analyses were completed to ascertain the extent to which these variables impact clinical status in the postpartum phase.

CHAPTER THREE

METHOD

Participants

Recruitment and Selection

Data for the present study was obtained from a research project funded by the Agency for Healthcare Research and Quality, at the U.S. Department of Health and Human Services, designed to examine the efficacy of acupuncture in the treatment of depression during pregnancy. Data were collected at Stanford University, where the Institutional Review Board approved the protocol. Written informed consent was obtained from all participants at the time of enrollment.

Participants consisted of pregnant women with a current Major Depressive Episode and were recruited between 2003-2007 in the San Francisco Bay Area community. They were recruited from obstetric clinics and from advertisements in local parent and baby magazines. Efforts were made to ensure adequate minority representation in the study. These included initiating contacts in hospitals and clinics with high attendance by minorities, placing brochures in libraries with higher minority populations, and advertising in publications geared toward minorities.

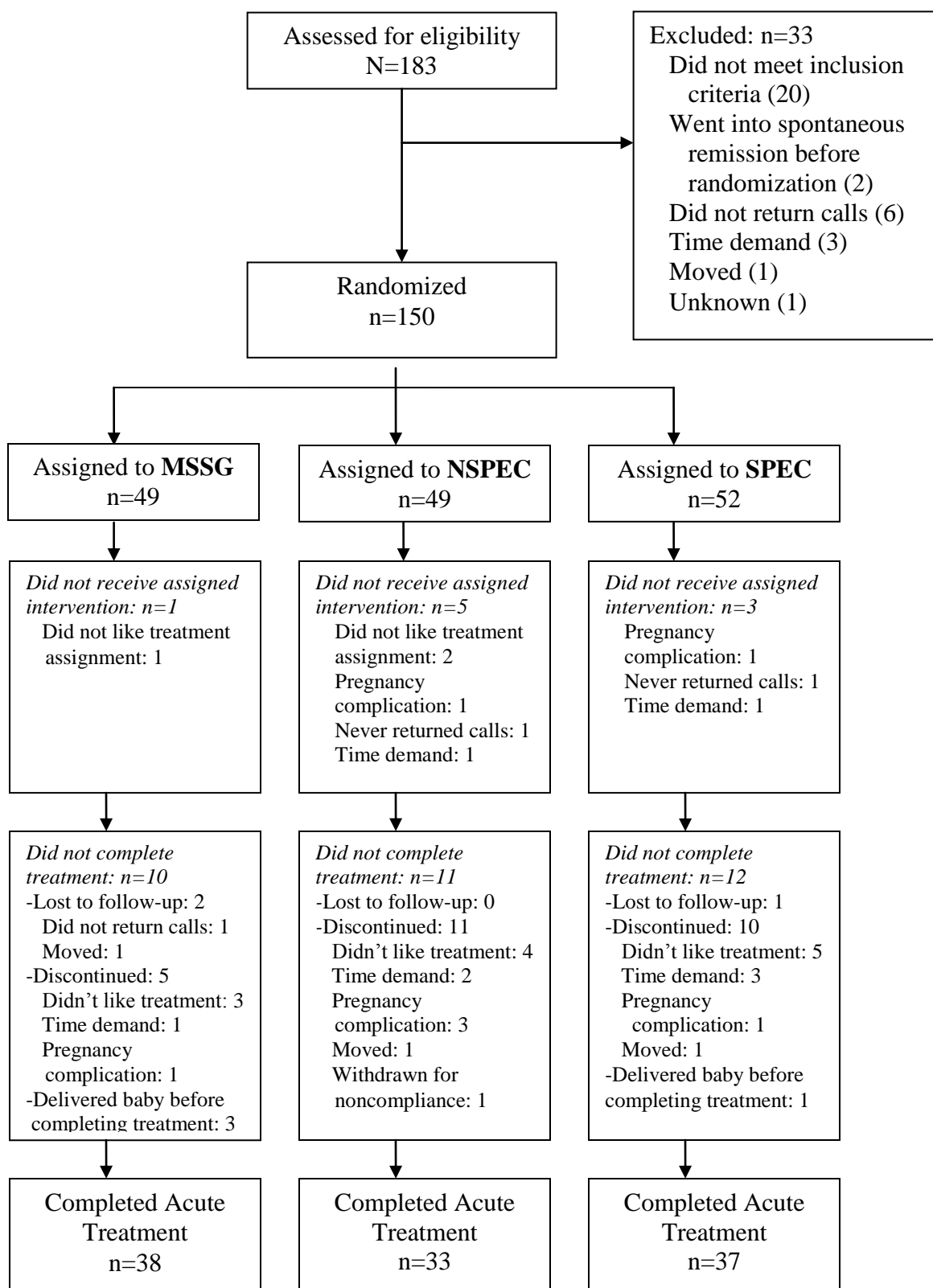
Participants met the following criteria for inclusion: (a) ambulatory women (age \geq 18) with viable pregnancy, and between 12-30 weeks of gestation; (b) satisfy DSM-IV criteria for a current Major Depressive Episode; and (c) obtain a minimum score of 14 on

the 17-item Hamilton Rating Scale for Depression. Participants were excluded from participation for the following reasons: (a) meeting criteria for a primary Axis I disorder in the past two months, other than unipolar depression or social phobia; (b) Seasonal Affective Disorder or current episode duration of two years or more (chronic depression); (c) abnormal results on a laboratory screen including a thyroid panel and drug screen; (d) serious uncontrolled medical conditions or conditions that may have a medical basis for depression; (e) cluster B personality disorders; (f) confounding treatments for depression, including any psychotherapy, herbs, or pharmacotherapy; (g) current use of any prescribed psychotropic medication or any medication that impacts mood; (h) treatment with ECT or vagal nerve stimulation in the past year; (i) current active suicidal potential necessitating immediate treatment; (j) absence of prenatal care from an OB-GYN practitioner in the community; and (k) any condition necessitating bed rest.

A total of 183 participants enrolled in the research project between February 2003 and June 2007 and were assessed for eligibility. Thirty-three were excluded for a variety of reasons (see Figure 1) and 150 were randomly assigned to a treatment condition. Among these 150, the mean age was 32.86 ($SD=4.88$). The majority were living with their husband (76%), employed (61%), and had obtained a college degree (42%) or graduate degree (33%). Participants identified primarily as Caucasian (65%), Asian (10%), and African American (5%). Hispanic and/or Latino ethnicity represented 18% of the sample.

Participation in the larger project involved three phases: the acute phase of treatment, a continuation phase of treatment, and a follow-up phase. Only those women

Figure 1. Flow of participants: Acute phase of treatment



who participated in the follow-up phase were included in the present study. This sample of women is thus described in more detail below.

Participant Characteristics: Follow-up Sample

Participants who completed at least one assessment during follow-up were included in the analyses for the present study ($n=120$). The 120 participants averaged 32.9 years old ($SD=5.01$), were predominately married (82%), employed (63%), obtained a college education (42%) or graduate degree (35%), and reported an annual income level of greater than \$60,000 (67%). They identified primarily as Caucasian (67%), Asian (9%), and African American (3%). Hispanic and/or Latino ethnicity represented 18% of the sample. See Table 1 for further details related to participant information.

Clinical information related to depression history is listed in Table 2. The 120 participants experienced an average of 3.06 ($SD=2.49$) previous episodes of depression and age of first onset of depression was 20.01 years ($SD=9.12$). Many participants had experienced an episode of depression associated with a previous pregnancy (35%) and the majority of participants developed the current episode of depression during pregnancy (61%) as opposed to before pregnancy (39%). Depression rating scores at intake included the HRSD₁₇ ($M=20.55$, $SD=3.30$), which fell in the severe range, and the BDI-II ($M=30.16$, $SD=7.92$), which also fell in the severe range.

Study Design and Procedure

As already indicated, although the present study focused on follow-up participants, the larger project involved three phases. Prior to the follow-up phase, an acute treatment phase and a continuation phase took place. Each of the three phases is described briefly below.

Table 1. Demographics of sample

	Randomization n = 150	Follow-up n = 120
Age (in years)	32.86 (4.88)	32.90 (5.01)
Marital Status		
Married	75.8%	81.7%
Unmarried, live with partner	12.1%	8.3%
Hispanic ethnicity	18.1%	17.5%
Racial distribution		
Native American/Alaska Native	0.7%	1.7%
Black or African American	5.4%	3.4%
Asian	10.2%	9.3%
White	65.3%	66.9%
Other	18.4%	18.6%
Highest education level		
High school	4.1%	2.5%
Some college	20.8%	20.8%
College	42.3%	41.7%
Graduate school	32.9%	35.0%
Work status		
Work	61.3%	62.5%
Student	2.7%	2.5%
Unemployed/homemaker	36.0%	35.0%
Household income bracket		
Less than \$20,000	8.1%	9.2%
\$20,001–\$59,999	25.2%	24.2%
More than \$60,000	66.7%	66.6%

Data are presented as mean, (SD), and percentages.

Table 2. Clinical information of sample

	Randomization n = 150	Follow-up n = 120
<i>Depression History</i>		
No. of past depressive episodes	3.03 (2.93)	3.06 (2.49)
Age of onset of first depressive episode	20.34 (8.78)	20.01 (9.12)
Patients with history of chronic depression	24.8%	25.8%
Patients with history of maternal depression	46.0%	44.1%
Depression severity at intake		
Hamilton Rating Scale for Depression	20.17 (4.45)	20.55 (3.30)
Beck Depression Inventory	29.62 (7.91)	30.16 (7.92)
Length of index episode (in months)	5.41 (5.85)	5.40 (6.20)
Onset of current episode of depression		
Before pregnancy	39.6%	39.2%
During pregnancy	60.4%	60.8%
Patients with history of postpartum depression		
Onset during postpartum	17.7%	20.0%
Onset either during pregnancy or postpartum	31.9%	34.8%
Patients taking antidepressant medication prior to or during pregnancy	24.6%	24.4%
Stopped when began trying to conceive	9.3%	8.4%
Stopped once pregnancy confirmed	15.3%	16.0%
<i>Pregnancy/Postpartum Information</i>		
Gestation week at intake	20.53 (5.75)	20.58 (5.76)
No. of previous pregnancies/births	2.26 (1.50)	2.18 (1.45)
Planned pregnancy	60.1%	63.9%
Breastfed baby		69.0%
Treatment postpartum (postpartum use after study)		
Antidepressant use		18.3%
Psychotherapy		11.7%
CAM: Massage or Acupuncture		6.7%

Data are presented as mean, (SD), and percentages.

Acute Phase of Treatment

Of the 183 participants who were enrolled in the study, 161 were eligible for randomization, and 150 were randomized. Participants were randomly assigned to treatment designed to specifically target acupuncture pressure points linked to the symptoms of depression (SPEC) or to one of two control treatments: treatment using acupuncture points which did not directly target depression-relevant patterns of disharmony (NSPEC) or prenatal massage (MSSG). Massage was conceptualized as a control treatment because, although it improves mood immediately after a session, there is insufficient evidence to support its efficacy as a treatment for depression (Coelho, Boddy, & Ernst, 2008). Participants who received acupuncture were not told which of the two types of acupuncture they were receiving. The consent stated that, “participants in one of these two groups will receive acupuncture that focuses on depression symptoms and the other treatment will not.” Participants assigned to massage were not blinded to treatment assignment. There were no significant differences between treatment groups on expectations of benefit associated with treatment assignment. Expectancy of treatment success was assessed after the first treatment, with one item, on a 0-5 point Likert scale.

Among the 150 participants who were randomized to treatment, 141 began treatment, and 108 completed treatment (77%). Participants received 12 treatments over a period of eight weeks (two times per week for the first four weeks and weekly for an additional four weeks) during the acute phase of treatment. See Figure 1 for a more detailed flow of participants through the acute phase of treatment.

Participants in the **SPEC** group received acupuncture treatment specifically tailored to individually address their depression related patterns of disharmony according to the principles of Chinese medicine and following a published standardized treatment manual (Schnyer, Allen, Hitt, & Manber, 2001). During each session, seven to twelve points were needled and were distributed across the same general areas of the body. The points were selected from a set of five main points that together addressed Qi stagnation and Shen disturbance. Other points were selected to further address differential diagnosis and emphasize the treatment of the underlying pattern of disharmony. Within the SPEC treatment assignment, 37/49 (76%), completed the acute phase of treatment. Attrition in the acute phase among SPEC participants was 24%, with 12/49 participants discontinuing.

Participants in the **NSPEC** group received acupuncture treatment which was also standardized and needles were inserted in real acupuncture points that did not address depression relevant patterns of disharmony according to traditional Chinese medicine. The nonspecific treatment was designed to address any pertinent complaint, unrelated to depression, presented by the participant: i.e. back ache, allergies, sciatica, etc. Treatments also consisted of seven to twelve total points, which were distributed across the same general areas of the body. Within the NSPEC treatment assignment, 33/44 (75%), completed the acute phase of treatment. Attrition in the acute phase among NSPEC participants was 25%, with 11/44 participants discontinuing.

To blind treating acupuncturists, needling of the patient was separated from the determination of which acupuncture points should be needled. Each participant was evaluated monthly by a senior acupuncturist (assessing acupuncturist), with at least five

years experience, who followed a standardized algorithm for assessing participants' signs and symptoms according to traditional Chinese medicine and designed a treatment plan specifying the points to be needled at each session in the month that followed. The senior acupuncturists were not told to which treatment the participant was assigned and developed both acupuncture not specific for depression and acupuncture specific for depression treatments for each participant. Junior acupuncturists, with less than two years experience, provided the treatments prescribed by the senior acupuncturist. All acupuncturists were nationally board certified (National Certification Commission for Acupuncture and Oriental Medicine) and licensed in the state of California.

Participants in the **MSSG** group received prenatal massage designed to alleviate common physical discomforts associated with pregnancy. The treatment consisted of Swedish massage provided in a standardized fashion and included effleurage and petrissage strokes. Approximately five minutes was spent on each of the following areas: back, face, head, neck and shoulders, and feet while the participant was lying on her side. The strokes were general, nonspecific, and always applied to the participants' comfort level. All massage therapists were California state board certified and trained in the treatment protocol by a senior massage therapist. They were also members of the American Massage Therapy Association. Within the MSSG treatment assignment, 38/48 (79%), completed the acute phase of treatment. Attrition in the acute phase among MSSG participants was 21%, with 10/48 participants discontinuing.

Participants in each of the three treatment conditions received the same number of sessions. They all followed the same schedule and the lengths of sessions were equal (approximately 25 minutes). Treatment providers were instructed to minimize verbal

communication and refrain from providing any counseling, dietary or other advice, or playing background music during the treatment session.

Continuation Phase of Treatment

Upon completion of the acute phase of treatment, participants were categorized as a responder or non-responder. Response was defined using the protocol outlined by Frank and colleagues (1990) and Rush and colleagues (2006) (Frank et al., 1990; Rush et al., 2006). Response was defined jointly by (a) failure to meet full criteria for MDD; (b) at least 50% reduction from baseline HRSD₁₇ score; and (c) HRSD₁₇ more than 7 and less than 14.

Responders to the acute phase of treatment entered a continuation phase during which they received weekly treatments that continued until ten weeks postpartum. Participants in the continuation phase received the same treatment to which they were randomly assigned in the acute phase of treatment. Participants who did not qualify for continuation treatment no longer received a study treatment.

Based on the number of participants who qualified for the continuation phase of the study (70 of 108 participants), 65% of participants responded to the acute phase of treatment. Among the 70 participants who qualified for the continuation phase of the study, the break down across treatments was 31/49 (63%) in specific acupuncture, 16/44 (36%) in nonspecific acupuncture, and 23/48 participants in massage (48%). The number of continuation treatments completed during pregnancy within each group was as follows: specific acupuncture ($M=3.45$, $SD=5.20$), nonspecific acupuncture ($M=4.56$, $SD=5.35$), and massage ($M=4.57$, $SD=6.37$). The number of continuation treatments

completed postpartum, prior to the follow-up phase which began at ten weeks postpartum, was as follows: specific acupuncture ($M=5.94$, $SD=3.38$), nonspecific acupuncture ($M=3.69$, $SD=3.60$), and massage ($M=5.52$, $SD=3.42$). Among the 70 participants eligible for continuation sessions, 45 completed at least 75% of the sessions (64%), eight completed fewer than 50% (11%), and 17 did not complete any sessions (24%). See Table 3 for information related to number of sessions participants completed during the continuation phase.

Follow-up Phase of Study

At the end of the continuation phase, the follow-up phase of the study began and participants were clinically assessed at ten weeks, six months, and nine months postpartum. Attempts were made to collect follow-up data on all participants who had been randomized to treatment ($n=150$), regardless of whether they completed treatment or participated in the continuation phase. The follow-up phase of the study was completed in August of 2008.

A total of 120 participants completed at least one assessment during the follow-up phase: 116 at ten weeks, 101 at six months, and 106 at nine months postpartum. The majority of participants completed all three HRSD₁₇ assessments during follow-up; 95/120 (79%) and 108/120 (90%) completed two or more of the HRSD₁₇ follow-up assessments. Only 12/120 participants (10%) completed merely one follow-up HRSD₁₇ assessment. In terms of the number of participants completing all three BDI-II assessments during follow-up, 69/116 (59%) completed all three, 99/116 (85%)

Table 3. Number of participants completing each phase of the study

	SPEC	NSPEC	MSSG	Total
<i>Acute Phase of Treatment</i>				
Completed	37/49 76%	33/44 75%	38/48 79%	108/141 77%
Discontinued	12/49 24%	11/44 25%	10/48 21%	33/141 23%
<i>Continuation Phase of Treatment</i>				
Completed (all sessions)	21/31 68%	5/16 31%	12/23 52%	38/70 54%
Completed ($\geq 75\%$ of sessions)	23/31 74%	6/16 38%	16/23 70%	45/70 64%
Completed ($\leq 50\%$ of sessions)	2/31 6%	4/16 25%	2/23 9%	8/70 11%
Discontinued (0 sessions)	6/31 19%	6/16 38%	5/23 22%	17/70 24%

completed two or more, and 17/116 (15%) completed only one BDI-II during follow-up. There were four participants who did not complete any BDI-II assessments, but had completed at least one HRSD₁₇ assessment during follow-up.

Among the 150 participants who were randomized to the acute phase of treatment, thirty did not complete any assessments during the follow-up phase (20%). Of the participants, 18/30 (60%) dropped out of the acute phase of treatment and completed no further follow-up. Other reasons participants did not complete any follow-up assessments were: refusal (n=4), not returning phone calls (n=4), pregnancy complication resulting in no further follow-up (n=3), and investigator initiated discontinuation from the study due to non-compliance (n=1). See Figure 2 and Figure 3 for the flow of participants during the follow-up phase, by treatment assignment and response status, respectively.

Assessment and Measures

Schedule of Mood Assessments

During the acute phase of treatment, depression symptomatology was assessed at baseline (prior to beginning treatment), at four weeks (halfway through treatment), and at eight weeks (at the completion of treatment). At each of these assessments, three depression outcome measures were given: HRSD₁₇, BDI-II, and the major depression episode section of the SCID-IV.

Participants qualifying for the continuation phase of treatment (responders to the acute phase of treatment) were clinically assessed at intervals of eight weeks during their continuation treatment and at 36 weeks gestation. Non-responders, or participants who

Figure 2. Flow of participants: Follow-up phase of study (treatment assignment)

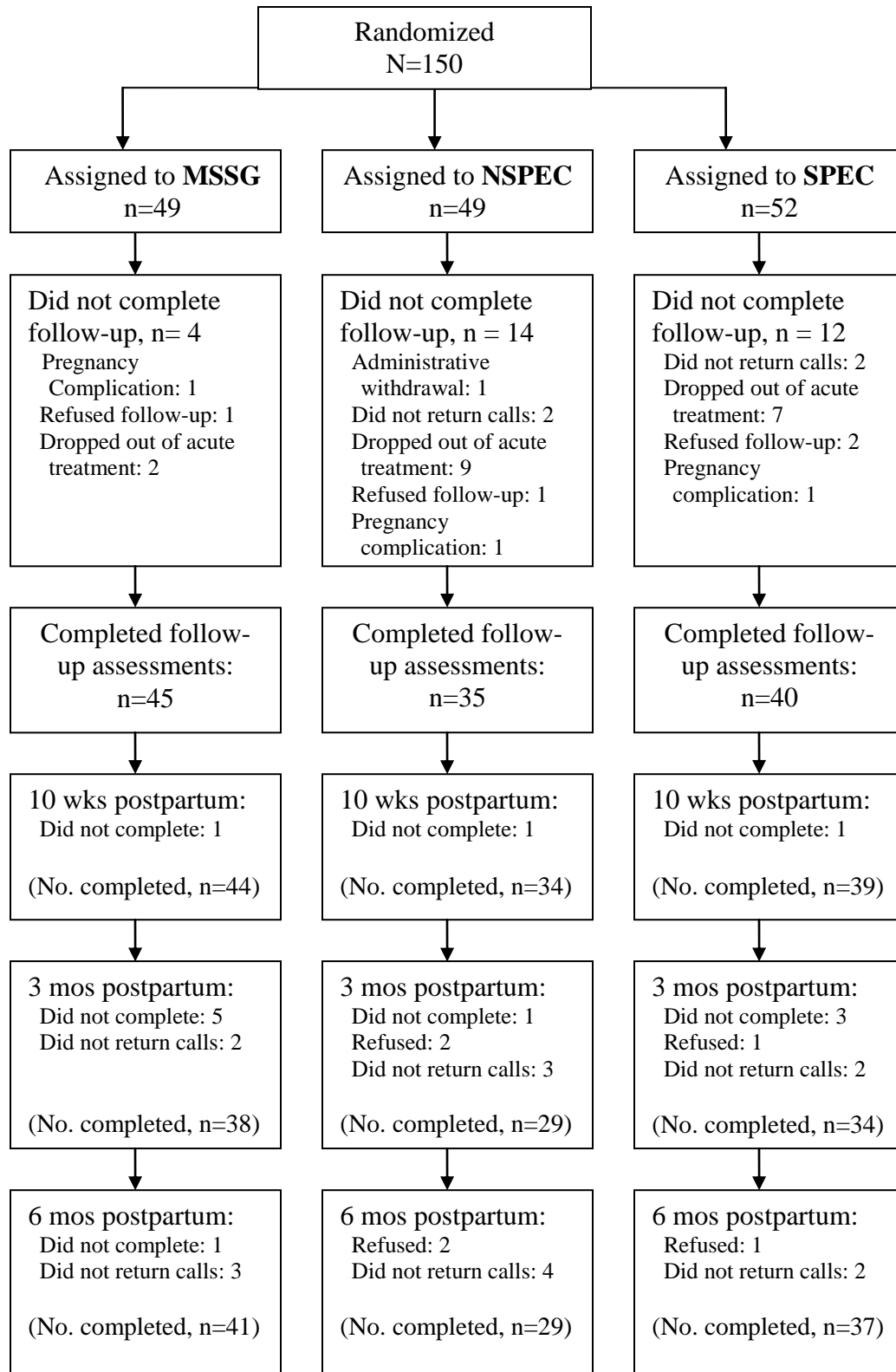
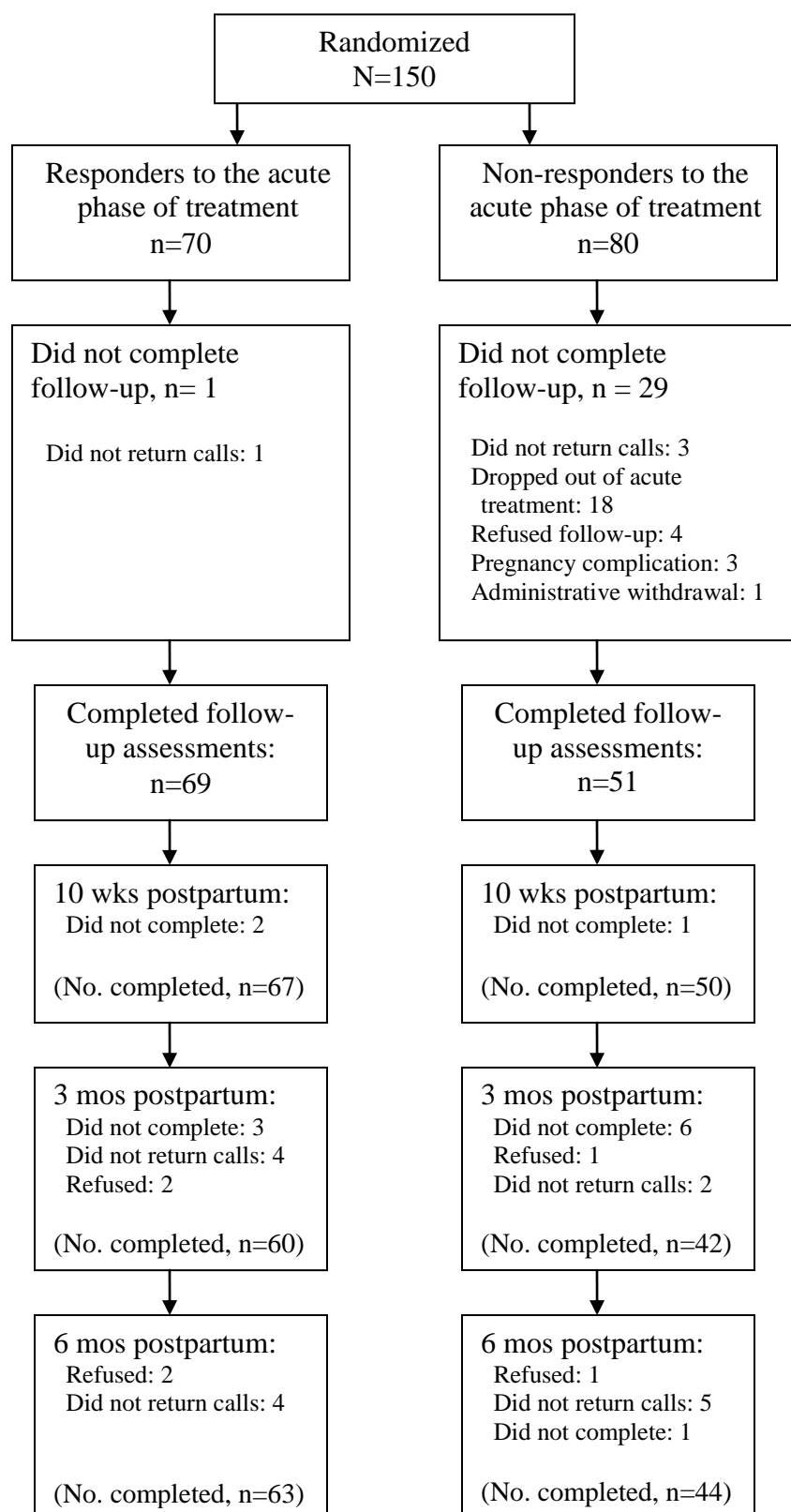


Figure 3. Flow of participants: Follow-up phase of study (response status)



did not qualify for continuation treatment, were assessed only at 36 weeks gestation. At each of these time points, the three depression outcome measures were given: HRSD₁₇, BDI-II, and the major depression episode section of the SCID-IV.

During follow-up, participants were clinically assessed at intervals of twelve weeks (three time points: ten weeks, six months, and nine months postpartum). Again, at each of these time points, the three depression outcome measures were given: HRSD₁₇, BDI-II, and the major depression episode section of the SCID-IV.

Outcome Measures

Outcome measures for the current study consist of three measures of depression which were administered at ten weeks, six months, and nine months postpartum. These include two clinician administered assessments: the Hamilton Rating Scale for Depression and the major depression episode section of the Structured Clinical Interview for DSM-IV. In addition, one self-administered measure was administered: the Beck Depression Inventory. Each of these instruments has been widely used in research for diagnosing depression.

Hamilton Rating Scale for Depression (HRSD₁₇). The 17-item version of the Hamilton Rating Scale for Depression is a widely used observer-rated scale. It is concerned primarily with the behavioral and somatic features of depression (Hamilton, 1960) and has been validated for use in postpartum populations (Thompson, Harris, Lazarus, & Richards, 1998). The scale is clinician administered and rated. The patient is asked to respond to questions concerning symptoms of depression that have been experienced during the past week. Nine items include 5-point scales ranging from 0-4,

representing ascending levels of symptom severity. The remaining eight items include 3-point scales ranging from 0-2, also representing ascending levels of severity. Higher scores indicate greater levels of depression. Interpretation of scores is as follows: less than 7, non-depressed; 8-13, mild depression; 14-18, moderate depression; 19-22, severe depression; more than 23, very severe depression.

The HRSD₁₇ has demonstrated high reliability in patients with depression. Its internal consistency has been found to range from 0.45-0.78 (Schwab, Bialon, & Holzer, 1967) and studies with depressed patients have reported high correlations (.84 and .90) between HRSD₁₇ scores and global clinical ratings of severity at the time of hospital admission (Hamilton, 1960; Paykel, Prusoff, & Tanner, 1976). Interrater reliability coefficients for the HRSD₁₇ have been found to be 0.84 or higher (Hedlund & Vieweg, 1979). A wide range of instruments has been used to examine the convergent validity of the HRSD₁₇. One meta-analysis found that convergent validity of the HRSD₁₇ with the BDI-II ranged from 0.27 – 0.89, and 0.37 with the major depression section of the SCID (Bagby, Ryder, Schuller, & Marshall, 2004). Lastly, two factors have emerged in analyses: endogenous depression and agitated depression (Hedlund & Vieweg, 1979).

In the current study, the HRSD₁₇ is the primary outcome measure of depression. The HRSD₁₇ was administered at baseline, following the acute phase of treatment (eight weeks), 36 weeks gestation, ten weeks postpartum, six months postpartum, and nine months postpartum by trained clinical interviewers who were blinded to treatment assignment. Assessments used for the current study included those administered during follow-up at ten weeks, six months, and nine months postpartum. The internal consistency of the HRSD₁₇ at intake was 0.79 and the intraclass correlation among

clinical raters administering the HRSD₁₇ in the current study was 0.96. During follow-up, participants were most frequently assessed over the phone; however, some chose to complete the interview in person.

Structured Clinical Interview for DSM-IV (SCID). The SCID is a semi-structured interview for making Axis I DSM-IV diagnoses (First, Spitzer, Gibbon & Williams, 1994). It is administered by a clinician and includes an introductory overview followed by modules which represent the major Axis I diagnostic classes. The output of the SCID is a record of the presence or absence of each of the disorders being considered, for current episode (past month), and for lifetime occurrence. The reliability of the SCID in adult populations with diverse disorders has indicated generally high reliability (Segal, Hersen & Van Hasselt, 1994). In an extensive multi-site project which involved test-retest reliability of 592 interviews, the kappa for major depression was 0.64 (Williams et al., 1992). In two more recent studies which examined the test-retest reliability of SCID diagnoses, the kappas for major depression were 0.61 and 0.73 respectively (Zanarini et al., 2000; Zanarini & Frankenburg, 2001). The SCID is also considered to have superior validity across a number of studies examining intake diagnoses (Basco et al., 2000).

In the current study, the SCID-I clinical version was used. The clinical SCID version is trimmed down to encompass only those DSM-IV disorders that are most typically seen in clinical practice. The SCID-I clinical version was given at baseline to diagnose current Major Depressive Episode (MDE) and other Axis I psychopathology. Subsequent to baseline, the MDE module was administered following the HRSD₁₇ assessment: following the acute phase of treatment (eight weeks), 36 weeks gestation, and at ten weeks, six months, and nine months postpartum. Similar to the HRSD₁₇, the

administration of the SCID was completed by a trained interviewer who was blinded to treatment condition. All interviewers underwent standardized training until item-level intraclass agreement exceeded 0.85. During follow-up, participants were most frequently assessed over the phone; however, some chose to complete the interview in person.

Beck Depression Inventory-II (BDI-II). The BDI-II is a 21 question multiple-choice self-report inventory that is one of the most widely used instruments for measuring the severity of depression (Beck, Steer, & Brown, 1996). Each of the 21 items corresponds to a symptom of depression and is summed to give a single score. There is a four-point scale for each item ranging from 0 to 3. On two items (16 and 18) there are seven options to indicate either an increase or decrease of appetite and sleep. Cut score guidelines for the BDI-II are given with the recommendation that thresholds be adjusted based on the characteristics of the sample, and the purpose for use of the BDI-II. A total score of 0-13 is considered minimal range, 14-19 is mild, 20-28 is moderate, and 29-63 is severe.

The BDI-II has demonstrated high reliability. Beck and colleagues (1996) reported a coefficient alpha of 0.91 and test-retest reliability of 0.96. The same authors also reported a positive correlation of 0.71 with the HRSD, thereby demonstrating convergent validity. In addition, researchers have confirmed a two factor structure of the BDI-II. The first factor, non-cognitive, consists of somatic and affective symptoms, while the second factor, cognitive, is comprised mostly of psychological symptoms (Steer, Rissmiller, & Beck, 2000). The BDI-II is considered to have strong content and convergent validity. It has been found to correlate 0.71 with the HRSD (Beck, Steer, & Brown, 1996).

In the current study, the BDI-II was administered weekly during the acute treatment (eight weeks) and bimonthly during the continuation treatment. It was completed at ten weeks, six months, and nine months postpartum. Internal consistency of the BDI-II at intake was 0.83. The patient's response to the suicide item of the BDI-II was always inspected on the day of its completion. Because of participants' preference to complete the follow-ups via phone, the BDI-II was mailed to participants and upon completion was returned to the study coordinator.

Additional Measures and Demographic Information

Childhood Trauma Questionnaire – Short Form (CTQ-SF). The CTQ-SF is a 25-item self-report questionnaire that assesses both abuse and neglect and has three abuse scales (physical, emotional, and sexual) (Bernstein et al., 2003). Each abuse scale includes five items, one of which inquires broadly as to whether the respondent was physically, emotionally, or sexually abused. At least three items on each abuse scale inquire about specific, behaviorally defined examples of childhood abuse (e.g., for emotional abuse, "I believe that I was emotionally abused."). The broad items explicitly use the term abuse and the items that inquire about specific examples of abusive experiences are dispersed throughout the 25-item questionnaire. The item response options of the CTQ-SF define the frequency of maltreatment experiences using a Likert scale ranging from 0 to 5: never, rarely, sometimes, often, or very often. Bernstein et al. (2003) have reported good internal consistency of the CTQ-SF for each of the abuse scales across four heterogeneous samples (physical abuse, 0.83-0.86; emotional abuse, 0.84-0.89; and sexual abuse, 0.92-0.95). The CTQ-SF was mailed to participants during follow-up to determine history of childhood abuse.

Descriptive information obtained at baseline. The following information was obtained at baseline and was used to describe the sample: gravidity/parity, past obstetrical history, maternal age, planned pregnancy, presence of the father of the baby, involvement of the father of the child emotionally and financially, health habits, client and providers expectations about treatment (higher scores indicating greater expectations), history of depression (e.g., number of prior depressive episodes, age at onset of first depressive episode, history of PPD, history of depression during pregnancy, history of maternal depression), treatment information about the current episode of depression (e.g., antidepressant use prior to and subsequent to conception), and socioeconomic status (see Appendix A).

Descriptive information obtained at follow-up. During the follow-up phase, additional information was collected on the following topics: breast feeding/bottle feeding, weight loss, alcohol consumption, smoking, prescription and nonprescription medication use, medications taken for the treatment of anxiety or depression (including herbal supplements), as well as other treatments for stress reduction, anxiety, or depression (e.g. massage, rake, therapy, counseling).

CHAPTER FOUR

RESULTS

Preliminary Analyses

Participants: Follow-up Sample

The sample for the primary analyses consisted of participants for whom at least one postpartum assessment was completed during the follow-up phase (n=120). Chi square and analyses of variance were performed to compare demographic and clinical characteristics of participants who completed at least one assessment during the follow-up phase (n=120) to participants who were randomized, but did not complete any assessments during the follow-up phase (n=30).

In terms of demographic characteristics, there was a higher percentage of participants married and living with their husband among those who completed a follow-up assessment (82%) compared to those who did not complete a follow-up assessment (52%), $\chi^2 (3, N=149) = 13.05, p = .005$. However, there were no differences between groups on feeling emotionally supported by the father of their child, $\chi^2 (1, N=147) = 0.13, p = .67$, or feeling happy in their relationship, $F(1, 147) = 0.13, p = .72$. Also, although not significant, there was a trend for participants who completed a follow-up assessment to have a higher percentage of planned pregnancies (64%) compared to those who did not complete a follow-up assessment (33%), $\chi^2 (1, N=148) = 3.53, p = .06$. No other significant differences were found between these groups on demographic variables.

In terms of clinical variables, several significant differences emerged between the two groups. Participants who completed a follow-up assessment endorsed experiencing a greater number of previous episodes of depression that began during pregnancy compared to those who did not complete a follow-up assessment, $F(1, 138) = 6.195, p = .014$. There was not a significant difference between groups on the number of previous episodes of depression beginning in the postpartum, $F(1, 140) = 1.001, p = .32$. Intake scores on the BDI-II were significantly higher for those who completed a follow-up assessment ($M=30.16, SD=7.92$) compared to those who did not complete a follow-up assessment ($M=26.79, SD=3.14$), $F(1, 148) = 4.17, p = .043$. There was not a significant difference between groups on HSRD scores at intake, $F(1, 149) = 1.64, p = .203$. No other significant differences were found between these groups on clinical variables.

Participants: Responders and Non-responders

Two groups were formed based on response status following the acute phase of treatment: responders and non-responders. Response to treatment was defined jointly by (a) failure to meet full criteria for MDD; (b) at least 50% reduction from baseline HRSD₁₇ score; and (c) HRSD₁₇ more than seven and less than 14, at the end of the acute treatment. Participants that did not meet the criteria for response were categorized as non-responders. Participants who did not complete treatment were categorized based on their last available data point. Based on this definition, the randomized sample consisted of 70 participants who were classified as responders and 80 as non-responders. At the end of the acute phase of treatment, responders and non-responders differed significantly on their HRSD₁₇ scores, $F(1, 107) = 96.90, p = .000$, and on their BDI-II scores, $F(1, 107) = 41.79, p = .000$. Thus the process for determining responders and non-responders

following acute treatment did in fact yield two different groups. Among the 120 participants included in the follow-up sample, 69 participants were categorized as responders and 51 as non-responders.

Chi square and analyses of variance were performed to compare responders ($n=69$) and non-responders ($n=51$) to the acute phase of treatment who were included in the follow-up sample ($n=120$). There were several statistically significant differences between the two groups. A larger percentage of responders were: (a) employed (72% vs. 49%), $\chi^2 (2, N=120) = 6.98, p = .03$; (b) exclusively breastfeeding their babies postpartum (75% vs. 55%), $\chi^2 (2, N=116) = 9.16, p = .01$; and (c) randomized to SPEC treatment (43% vs. 20%), $\chi^2 (1, N=120) = 7.52, p = .006$.

Although not significant, there were two variables trending towards significance: stopping antidepressant use after conception and endorsing a history of maternal depression. A larger number of non-responders endorsed discontinuing antidepressant medication following conception (24%) compared to responders (10%), $\chi^2 (1, N=119) = 3.805, p = .051$, and also were more like to endorse a history of maternal depression (53%) compared to responders (36%), $\chi^2 (1, N=118) = 3.47, p = .062$. There were no other differences found between the responders and non-responders included in the follow-up sample, on other demographic or clinical variables (see Table 4).

Measures

Preliminary analyses were completed to examine the correlations between BDI-II and HRSD₁₇ scores at each of the three postpartum time points. The correlation between the BDI-II and HRSD₁₇ at ten weeks postpartum was, $r(106)=0.677, p=.000$; at three

Table 4. Clinical information of responders and non-responders

	Responders n = 69	Non-responders n = 51
<i>Depression History</i>		
No. of past depressive episodes	3.26 (2.78)	2.78 (2.02)
Age of onset of first depressive episode	20.76 (9.21)	18.98 (8.98)
Patients with history of chronic depression	27.5%	23.5%
Patients with history of maternal depression	36.2%	52.9%
Depression severity at intake		
Hamilton Rating Scale for Depression	20.09 (3.21)	21.18 (3.34)
Beck Depression Inventory	29.26 (7.47)	32.02 (7.27)
Length of index episode (in months)	5.49 (7.68)	5.27 (3.16)
Onset of current episode of depression		
Before pregnancy	42.0%	35.3%
During pregnancy	58.0%	64.7%
Patients with history of postpartum depression		
Onset during postpartum	21.7%	15.7%
Onset either during pregnancy or postpartum	37.9%	30.6%
Patients taking antidepressant medication prior to or during pregnancy	18.8%	31.4%
Stopped when began trying to conceive	8.7%	7.8%
Stopped once pregnancy confirmed	10.1%	23.5%
<i>Pregnancy/Postpartum Information</i>		
Gestation week at intake	21.15 (5.97)	19.80 (5.44)
No. of previous pregnancies/births	2.24 (1.51)	2.10 (1.39)
Planned pregnancy	68.1%	56.9%
Breastfed baby	75.4%	54.9%
Treatment postpartum (postpartum use after study)		
Antidepressant use	15.9%	21.6%
Psychotherapy	10.1%	13.7%
CAM: Massage or Acupuncture	7.2%	9.8%
<i>Treatment Assignment</i>		
SPEC	43.4%	19.6%
NSPEC	23.2%	37.3%
MSSG	33.3%	43.1%

Data are presented as mean, (SD), and percentages.

months postpartum, $r(88)=0.732$, $p=.000$; and at six months postpartum, $r(84)=0.711$, $p=.000$. The two scales were significantly correlated at each time point suggesting strong convergent validity. Additionally, each measure was highly correlated with itself across the three time points (see Table 5).

Acute Treatment Response and Postpartum Depression

It was hypothesized that, regardless of assigned treatment modality, early successful treatment of depression during pregnancy would incur protection against postpartum depression. More specifically, hypothesis 1(a) predicted that responders to the acute phase of treatment during pregnancy would have better clinical outcomes on the HRSD₁₇ at ten weeks, six months, and nine months postpartum compared to those who did not respond to the acute phase of treatment. Hypothesis 1(b) predicted that responders to the acute phase of treatment would have lower depressive symptom severity scores on the BDI-II at ten weeks, six months, and nine months postpartum compared to non-responders to the acute phase of treatment. Tables 6 and 7 present descriptive data for the HRSD₁₇ and BDI-II, respectively, across the three postpartum time points.

Mixed model regression analyses were used to test for differential effects of response to treatment on the main outcome measure (Hamilton Rating Scale for Depression) and separately using the self-report symptom severity measure (Beck Depression Inventory) using follow-up data provided at ten weeks, six months and nine months postpartum. In the mixed effects analyses, the following variables were included as fixed effects: response status (responders, non-responders), time (ten weeks, six months, and nine months postpartum), and the response status by time interaction.

Table 5. Correlation coefficients: HRSD₁₇ and BDI-II across the postpartum phase

Variable	1.	2.	3.	4.	5.
1. BDI-II 10 weeks postpartum	-----				
2. BDI-II 6 months postpartum	0.695**	-----			
3. BDI-II 9 months postpartum	0.637**	0.782**	-----		
4. HRSD ₁₇ 10 weeks postpartum	0.677**	0.505**	0.513**	-----	
5. HRSD ₁₇ 6 months postpartum	0.620**	0.732**	0.607**	0.614**	-----
6. HRSD ₁₇ 9 months postpartum	0.323**	0.506**	0.711**	0.289*	0.537**

** $p \leq .001$, * $p \leq .005$

Table 6. Mean HRSD₁₇ scores during the postpartum

	10 weeks postpartum N=116	6 months postpartum N=101	9 months postpartum N=106
<i>Follow-up Sample</i>			
	6.51 (5.78) n= 116	6.87 (5.96) n= 101	7.32 (5.24) n= 106
<i>Response Status:</i>			
Responder	5.88 (5.12) n= 66	5.93 (5.39) n= 60	7.19 (5.14) n= 62
Non-Responder	7.34 (6.52) n= 50	8.24 (6.54) n= 41	7.50 (5.44) n= 44
<i>Treatment v. Collapsed Control:</i>			
SPEC	7.25 (5.23) n= 39	6.18 (4.90) n= 34	6.57 (4.87) n= 37
Control	6.13 (6.04) n= 77	7.22 (6.44) n= 67	7.72 (5.42) n= 69

Data are presented as mean and (SD).

Table 7. Mean BDI-II scores during the postpartum

	10 weeks postpartum N=109	6 months postpartum N=89	9 months postpartum N=86
<i>Follow-up Sample</i>			
	9.66 (7.32) n= 109	9.93 (8.69) n= 89	10.09 (7.93) n= 86
<i>Response Status:</i>			
Responder	7.55 (5.95) n= 63	8.31 (8.18) n= 56	9.39 (7.58) n= 49
Non-Responder	12.56 (8.07) n= 46	12.70 (8.95) n= 33	11.02 (8.40) n= 37
<i>Treatment v. Collapsed Control:</i>			
SPEC	9.02 (6.43) n= 35	9.41 (8.13) n= 34	9.81 (6.89) n= 31
Control	9.96 (7.73) n= 74	10.26 (9.07) n= 55	10.25 (8.52) n= 55

Data are presented as mean and (SD).

Mixed effects models allow flexible modeling of the covariance structure of data and thus can adequately model data in which observations are not independent. They are also less restrictive in that they can be applied to repeated measurements taken at unequal (between and within subjects) time intervals (Charkroborty & Gu, 2009). Mixed models provide a contemporary approach to missing data by using estimated individual time trend lines based on all available data for each individual, augmented by information from data for all other individuals in the sample (Gibbons et al., 1993). These analyses were completed using Predictive Analytics SoftWare (PASW) SPSS Version 18.

First, mixed effects analyses tested hypothesis 1(a) by examining Hamilton Rating Scale for Depression scores at ten weeks, six months, and nine months postpartum based on response status (responder or non-responder) to the acute phase of treatment. There was not a significant response status by time interaction, $F(1, 116) = .97, p = .327$ nor was there a significant main effect for time, $F(1, 116) = .99, p = .320$. Although trending towards responders faring better in the postpartum phase, there was not a significant main effect for response status in this analysis, $F(1, 118) = 2.80, p = .097$. Thus, hypothesis 1(a) with regard to clinical outcome, as measured by the HRSD₁₇, was not supported.

Second, hypothesis 1(b) was tested using a similar analysis to examine Beck Depression Inventory scores at ten weeks, six months, and nine months postpartum based on response status (responder or non-responder) to the acute phase of treatment. There was not a significant response status by time interaction, $F(1, 90) = 1.99, p = .162$, suggesting little change in slope over time between responders and non-responders on self-reported depression symptom scores. There was also not a significant main effect for

time, $F(1, 90) = .024, p = .877$. However, there was a significant main effect for response status such that responders endorsed fewer depressive symptoms on the BDI-II compared to non-responders, $F(1, 113) = 13.37, p = .000$.

In order to test if treatment during continuation was driving the significant main effect for response status, two covariates were added to the mixed effects model: number of continuation treatment sessions during pregnancy and number of continuation treatment sessions during the postpartum phase. The main effect for response status remained significant; responders reported lower depressive symptoms severity scores on the BDI-II in the postpartum phase compared to non-responders, even when controlling for number of continuation treatment sessions during pregnancy and number of continuation treatment sessions during the postpartum phase, $F(1, 120) = 7.20, p = .008$. This finding supported the hypothesis that participants who responded to acute treatment would have lower depression symptom severity scores during the postpartum compared to non-responders, as measured by the BDI-II.

Finally, although there was not a significant main effect for HRSD₁₇ scores across the postpartum, there was some evidence that treatment assignment may have been influencing this result. In examining the observed means for HRSD₁₇ scores (see Table 6), responders' scores appeared to be increasing over time whereas non-responders' scores were not. However, the observed HRSD₁₇ means for treatment assignment indicated that the SPEC group was remaining consistent across time, whereas the combined control group was worsening (increasing HRSD₁₇ scores). Based on this observation, it appeared that among responders, different patterns of scores may have emerged based on treatment assignment to SPEC or control. A post-hoc analysis was

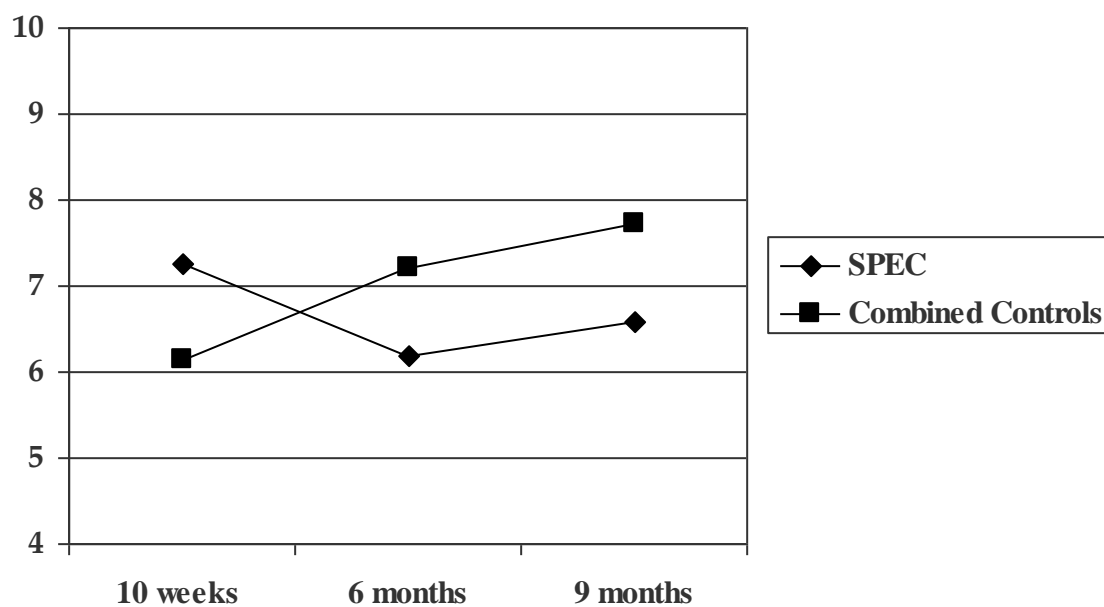
completed to test this observation. A mixed effects analysis was limited to responders, and examined Hamilton Rating Scale for Depression scores at ten weeks, six months, and nine months postpartum based on treatment assignment (SPEC or control). There was a significant treatment assignment by time interaction, $F(1, 64) = 4.29, p = .04$, which suggested that although the control group had lower scores at the first postpartum time point, ten weeks, their scores worsened across the postpartum phase while the SPEC group maintained improvement (see Figure 4).

Treatment and Relapse Rate

It was expected that a subset of women would experience re-occurrence of MDD during the postpartum phase. More specifically, hypothesis two predicted that time to relapse, following remission, would be sooner for participants in the control groups (NSPEC and MSSG) than for participants in the SPEC group when examined at ten weeks, six months, and nine months postpartum. Responders were classified as being in remission if the following two criteria were met jointly: a) the absence of the core symptoms of depression being reported at threshold or subthreshold level using the SCID (depressed mood and anhedonia); and, b) HRSD₁₇ score of seven or less. Rate of relapse was determined based on SCID criteria and included any participant who met criteria for MDD at any one of the three follow-up time points (ten weeks, six months or nine months postpartum).

It was proposed that the differential likelihood of relapse would be analyzed via survival analysis using the Cox proportional hazards method, which models event rates as a log-linear function of predictors, or covariates. However, this analysis was not performed because among the 65 participants who remitted from their index episode of

Figure 4. Mean HRSD₁₇ scores among responders: Treatment assignment by time interaction ($p=.04$)



MDD by 36 weeks gestation only four (6%) relapsed during the postpartum phase. In order to arrive at reliable estimates of survival and standard errors at each time interval, the minimum recommended sample size is 30, thus precluding a test of the second hypothesis (time to relapse following remission).

It should be noted that, although few met strict criteria for MDD following remission, descriptive statistics for other variables suggest some re-emergence of depressive symptoms. One such variable is having a score ≥ 14 on the HRSD₁₇ during the postpartum phase, which was the minimum HRSD₁₇ score required for inclusion in the study; 13 of the 65 (20%) scored ≥ 14 on the HRSD₁₇ during the postpartum phase, suggesting sub-criteria depressive symptomatology. Additionally, 7 of the 65 participants (11%) who remitted began taking antidepressant medication during the postpartum phase which may also suggest return of depressive symptoms.

Predictors of Postpartum Depression

The current study expected to replicate previous research indicating increased risk for postpartum depression. Three variables measuring history of depression were tested: having ever experienced a previous episode of depression, having a history of depression during pregnancy, and previous episodes of postpartum depression. Hypothesis 3(a) predicted that women with a previous history of depression would be more likely to meet criteria for MDD during the postpartum phase (at ten weeks, six months, or nine months postpartum). The relationship between prior history of depression and the absence or presence of MDD during postpartum was analyzed using Chi square. The analysis was completed by generating two categorical variables: the presence of MDD (yes/no) at any time point during the postpartum and prior history of depressive episodes (none/some).

Only nine participants did not have a past history of depression and few participants experienced MDD during the postpartum ($n=20$); therefore, Fisher's exact test was utilized for this analysis as it does not assume cell values have an expected frequency of five or greater and is typically used for a 2x2 table. In this analysis, there was not a significant difference between previous history of depression and presence or absence of MDD during the postpartum phase, $\chi^2 (1, N=109), p = .48$. Thus, hypothesis 3(a) was not supported.

Hypothesis 3(b) predicted that a greater number of past episodes of depression during pregnancy and postpartum would be associated with higher depression symptom severity scores as measured by the HRSD₁₇ at six months and nine months postpartum. The relationship between number of past depressive episodes during pregnancy and the postpartum phase and symptom severity at six and nine months postpartum was analyzed using Spearman's correlation. Two separate correlations were computed. Spearman's correlation was computed for HRSD₁₇ scores at six months and nine months postpartum with both number of past episodes of depression during pregnancy and past number of episodes of postpartum depression (see Table 8).

HRSD₁₇ scores at six months postpartum were significantly correlated with number of previous episodes of depression during pregnancy, $r(97)=.264, p=.008$. Similarly, HRSD₁₇ scores at six months postpartum were significantly correlated with number of previous episodes of postpartum depression, $r(96)=.286, p=.004$. However, at nine months postpartum, HRSD₁₇ scores were not significantly correlated with number of previous episodes during pregnancy, $r(102)=.089, p=.370$, or with number of previous

Table 8. Correlation coefficients: HRSD₁₇ postpartum and depression history

Variable	1.	2.	3.	4.
1. HRSD ₁₇ 6 months postpartum	-----			
2. HRSD ₁₇ 9 months postpartum	.422**	-----		
3. Number of previous episodes of depression during pregnancy	.264*	.089	-----	
4. Number of previous episodes of postpartum depression	.286*	.090	.590**	-----
5. Number of previous episodes of depression	.179	-.110	.184	.272*

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

episodes of postpartum depression, $r(101)=.090, p=.367$. The two predictor variables, history of depression during pregnancy and history of postpartum depression, were highly correlated, $r(139)=.590, p=.000$, suggesting significant overlap among the two variables. These findings support the hypothesis that participants with histories of depression during pregnancy and/or postpartum would experience greater depression symptom severity scores at six months postpartum; however, by nine months postpartum the predictive relationship did not hold.

Exploratory Analyses: ROC Curve Approach

Exploratory analyses to examine potential predictors of postpartum depression utilized the receiver operating characteristic (ROC) curve approach with the ROC4 program (Kraemer, 2004). ROC is a nonparametric technique that evaluates multiple potential predictors without making restrictive assumptions (e.g., linearity, additivity, homoscedasticity) that are required of parametric linear models. A unique feature of the ROC program is that it allows the user to designate the criterion for identifying the best variable by adjusting the weight in kappa in order to optimize sensitivity (i.e., emphasis placed on avoiding false negatives), specificity (i.e., emphasis placed on avoiding false positives), or efficiency (i.e., equal emphasis placed on both types of errors). The decision to adjust the weighted kappa is based on clinical importance of false negatives vs. false positives. For each independent variable (IV), the program searches for a cut-point that optimizes the balance between sensitivity and specificity for predicting the outcome of interest (e.g., postpartum depression). The groups identified in each round of the analysis are then re-tested to see which variable best predicts outcome within that group, until groups are too small for significant predictors to be found ($p > 0.01$). This

hypothesis-generating technique has been used in other naturalistic studies as it is well suited for identifying predictors or characteristics of those at risk for a particular dichotomous outcome, such as postpartum depression (Ackerman, Greenland, & Bystriksy, 1996; Manber et al., 2008; O'Hara et al., 2002).

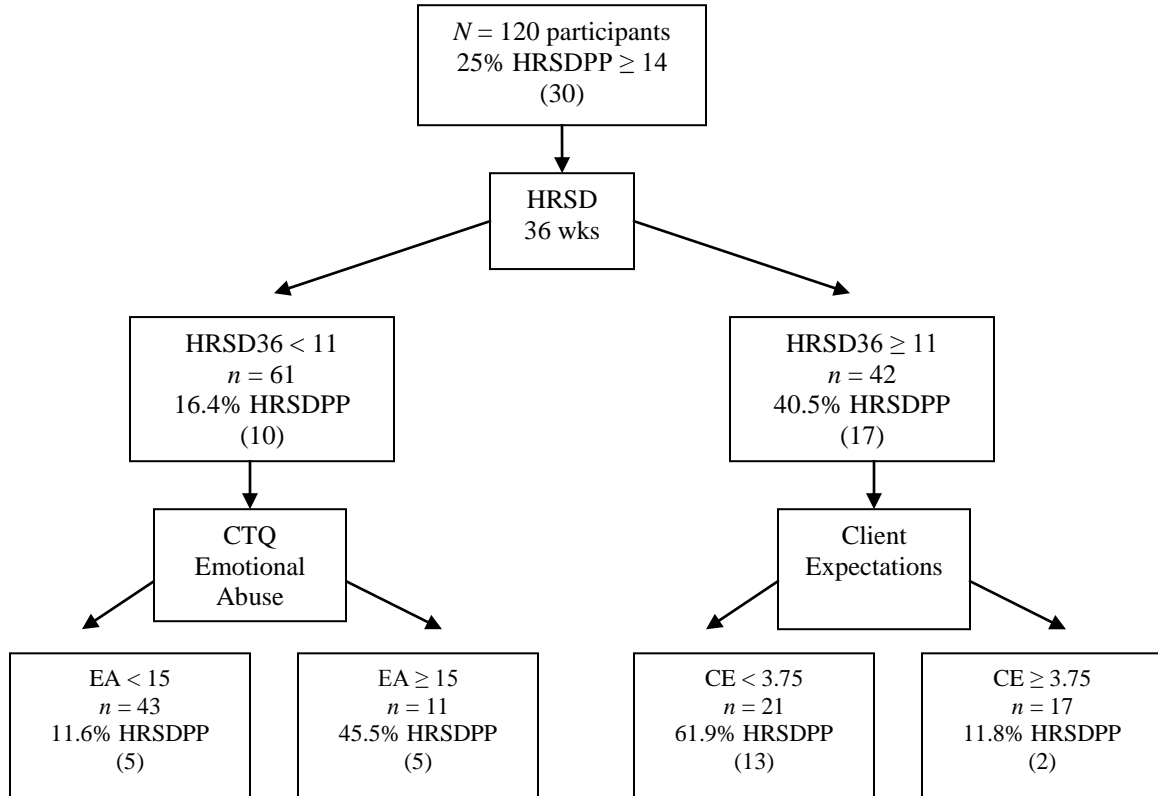
In the present study, two separate ROC analyses were conducted. In the first, the criterion variable was $\text{HRSD}_{17} \geq 14$ (yes / no), at any of the following three postpartum time points: ten weeks, six months, and nine months postpartum. The cutoff of $\text{HRSD}_{17} \geq 14$ is common in the depression literature and was used as one of the inclusion criteria in this study. In the second ROC analysis the criterion variable was meeting MDD criteria on the SCID at any of the same three time points: ten weeks, six months, and nine months postpartum. Thus, the two analyses served to identify predictors of depressive symptom severity, and separately, clinical diagnoses of MDD during the postpartum. In the ROC analysis, the weight for kappa was set at 0.50 so that false negatives and false positives were given equal consideration.

The predictors (IVs) entered into the model included treatment group, response to acute treatment, income level, planned pregnancy (yes/no), number of previous depressive episodes, number of previous pregnancies, HRSD_{17} score at baseline, HRSD_{17} score at the end of pregnancy (36 weeks gestation), breast / bottle feeding, emotional support from father of the child, relationship satisfaction with father of the child, client expectations of treatment benefits, provider expectations of treatment benefits, receiving depression treatment postpartum, four subscales of the CTQ (childhood physical neglect, childhood emotional neglect, childhood emotional abuse, childhood physical abuse), history of maternal depression, age, age of onset of first depressive episode, whether

antidepressants were stopped when planning to conceive, and whether antidepressants were stopped following conception (consistent with the exclusion criteria, no study participants used antidepressants subsequent to enrollment, but may have taken an antidepressant earlier in pregnancy).

The results of the ROC analysis for HRSD₁₇ score ≥ 14 (yes/no) during the postpartum period (n=120) are depicted in Figure 5. Three significant predictors were identified. At the first level, the best predictor variable was HRSD₁₇ score at 36 weeks gestation with an optimal cut-point of 11, $\chi^2 = 7.46$, $p < .01$. Of the 42 participants who scored ≥ 11 , 40.5% experienced clinically significant depressive symptoms at some point during the postpartum. This group was further differentiated by client expectation of treatment benefits (CE), with a cut-point of client expectation < 3.75 , $\chi^2 = 9.89$, $p < .01$. Of the 21 participants who scored 11 or more on the HRSD₁₇ at the end of pregnancy and also had low expectations of benefits early in treatment (< 3.75), 61.9% experienced clinically significant depressive symptoms during the postpartum. In contrast, of the 17 participants who scored 11 or more on the HRSD₁₇ at the end of pregnancy but had high expectations of benefits early in treatment (≥ 3.75), only 11.8% experienced clinically significant depressive symptoms during the postpartum. Of the 61 participants whose HRSD₁₇ score at 36 weeks gestation was < 11 and also scored ≥ 15 on the emotional abuse subscale of the CTQ, 45.5% experienced clinically significant depressive symptoms during the postpartum. In contrast, of the participants who scored < 11 on the HRSD₁₇ at 36 weeks gestation and < 15 on the emotional abuse subscale of the CTQ, only 11.6% experienced clinically significant depressive symptoms during the postpartum. No other significant variables were found.

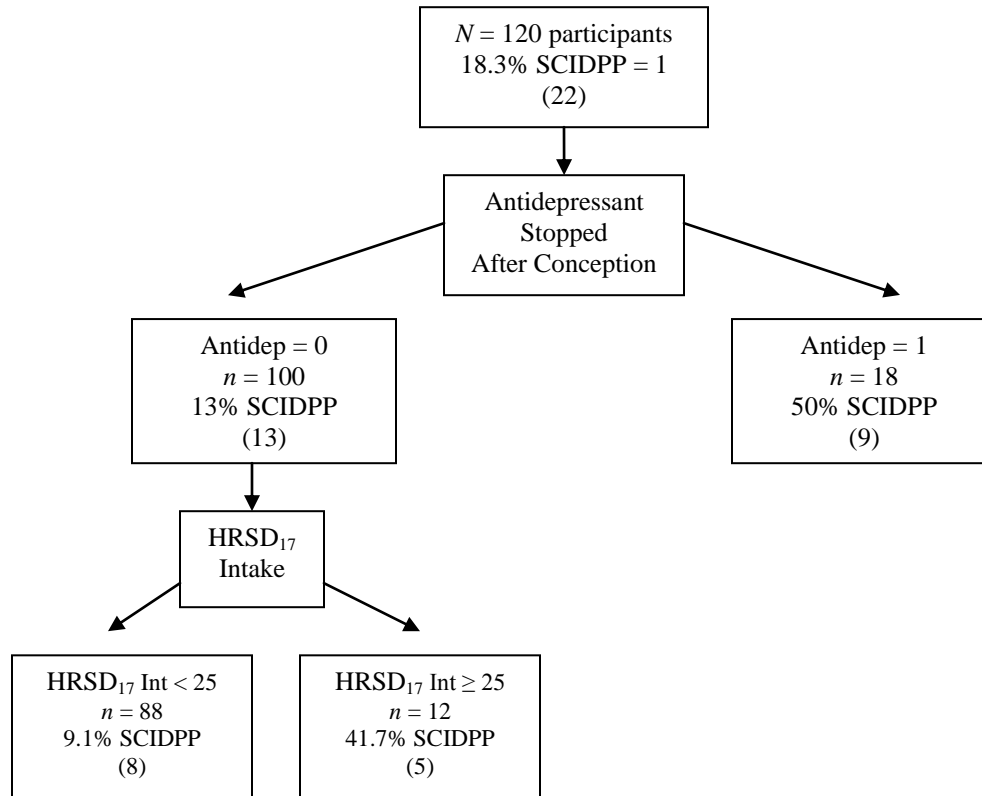
Figure 5. ROC tree of subgroups with different risk for experiencing HRSD_{17} scores ≥ 14 in the postpartum phase. The number of participants who meet criteria are denoted in parentheses.



Thus, the ROC analysis identified HRSD₁₇ score at 36 weeks gestation as the best predictor of experiencing an HRSD₁₇ score ≥ 14 during the postpartum phase. For those who met this initial cut-point (HRSD₁₇ at 36 weeks ≥ 11), the next best predictor was client expectations of their assigned treatment, such that more favorable expectations (≥ 3.75) resulted in better outcomes. For those who did not meet the initial cut-point (HRSD₁₇ at 36 weeks < 11), the next best predictor was childhood emotional abuse, such that higher levels of emotional abuse (EA ≥ 15) resulted in greater likelihood of experiencing an HRSD₁₇ score ≥ 14 during the postpartum phase.

A second ROC analysis was completed using SCID criteria for MDD (yes/no) during the postpartum as the criterion variable (n=120). This exploratory analysis revealed two predictors (see Figure 6 for ROC tree). At the first level, the best predictor was stopping antidepressant use after conception (and prior to beginning the study), $\chi^2 = 13.77$, $p < .001$. Of the 16 participants who stopped an antidepressant after conception, 50% met criteria for MDD during the postpartum. For this small group, the stopping rule went into effect and the ROC analysis did not further differentiate any subgroups. In contrast, out of the 100 participants who never used an antidepressant during pregnancy, only 13% met criteria for MDD during the postpartum. Subsequently, this group was further differentiated by the severity of depressive symptoms at baseline, with a cut-point of intake HRSD₁₇ ≥ 25 , $\chi^2 = 9.91$, $p < .01$. Of the 12 participants who did not use antidepressants during pregnancy and had intake HRSD₁₇ scores ≥ 25 , 41.7% met criteria for MDD during the postpartum. In contrast, of the 88 participants who did not use antidepressants during pregnancy and had intake HRSD₁₇ scores < 25 , only 9.1% met criteria for MDD during the postpartum. No other significant variables were found.

Figure 6. ROC tree of subgroups with different risk for meeting SCID criteria for Major Depression in the postpartum phase. The number of participants who meet criteria are denoted in parentheses.



In summary, this second exploratory analysis using ROC indicated that the best predictor for meeting MDD criteria postpartum was stopping antidepressant use after conception. For participants who had not taken an antidepressant medication during pregnancy, the next best predictor was intake HRSD₁₇ score, with higher scores (≥ 25) resulting in a greater likelihood of meeting criteria for MDD postpartum.

CHAPTER FIVE

DISCUSSION

Introduction

The current project examined the impact of treating major depressive disorder during pregnancy to reduce the likelihood of postpartum depression and explored factors that predict postpartum depression in a high-risk sample. The results partially support the hypothesis that successful treatment of depression during pregnancy can reduce depressive symptom severity in the postpartum phase. The results also provide replication of previously identified predictors of postpartum depression, including a strong emphasis on depressive symptoms during pregnancy increasing risk in the postpartum. In combination, the findings suggest women at risk for postpartum depression may be identified during pregnancy and successful treatment prior to birth may reduce the occurrence of postpartum depressive symptomatology.

Acute Treatment Response and Postpartum Depression

It was hypothesized that responders to the acute phase of study treatment, during pregnancy, would benefit from enduring effects of their treatment into the postpartum phase. In other words, successful treatment of depression during pregnancy would incur protection against postpartum depression. This hypothesis was tested using mixed effects modeling, analyzing both BDI-II and HRSD₁₇ data. The results partially supported the proposed hypothesis.

Depression symptom severity scores in the postpartum, as measured by the self-reported BDI-II, were significantly lower for responders to the acute phase of treatment compared to non-responders. This suggests that participants who responded to treatment endorsed fewer depressive symptoms in the postpartum phase. When examining clinical status scores in the postpartum phase, as measured by the HRSD₁₇, there was not a main effect for response status. However, the analysis was trending towards significance, such that responders had better clinical outcomes postpartum. This trend, although not statistically significant, mirrors the finding associated with the BDI-II data. Taken together, these results suggest that successful response to treatment during pregnancy may provide enduring effects into the postpartum phase, up to nine months in the current study, which is supportive of the main hypothesis of this project. Before discussing possible reasons for the inconsistent findings with respect to the BDI-II and HRSD₁₇, the available literature on the effects of response to treatment during pregnancy and depressive symptom severity during the postpartum will be addressed.

To date, the majority of studies examining treatment interventions for postpartum depression are implemented during the postpartum phase. Among these, medication, alone or with CBT; interpersonal therapy; and CBT produce the largest effect sizes (Bledsoe & Grote, 2006). Few studies have examined whether treatment during pregnancy can have a lasting effect into the postpartum. Among the existing published studies, many have provided group psychoeducation classes, as opposed to psychotherapeutic intervention, and others inherently exclude medication, which may result in lower effect sizes. Two studies provided IPT during pregnancy to depressed women and included maintenance sessions postpartum. These studies demonstrated

lasting effects up to six months postpartum (Grote et al., 2004; Grote et al., 2009). The current study results are consistent with the previous findings associated with IPT, even while controlling for continuation sessions both during pregnancy and postpartum, which is noteworthy given the women who responded to treatment predominately stayed well through nine months postpartum. The current results provide further evidence that timing of implementation of interventions during pregnancy, as well as response to treatment regardless of treatment assignment, may produce enduring effects in the postpartum phase.

Lastly, although responders in the current study were comprised of participants among all three treatment groups, post-hoc analyses limited only to responders, provided evidence that the active treatment may have contributed to the maintenance of change postpartum. Among HRSD₁₇ responders, there was a significant interaction based on treatment group assignment across time in the postpartum. While responders assigned to the control treatments began with lower HRSD₁₇ scores at ten weeks postpartum compared to responders to SPEC, across the postpartum phase SPEC responders' HRSD₁₇ scores remained consistent, whereas the responders assigned to one of the control treatments experienced a worsening of symptoms over time.

This interaction effect is consistent with the findings of both Manber et al.'s (2004) pilot study and Manber et al.'s (2010) larger randomized clinical trial examining the efficacy of treating depressed pregnant women with acupuncture. Both studies found a greater rate of decrease in depression symptom severity and a greater response rate for the acupuncture specific for depression (SPEC) compared to control groups following the acute phase of treatment (Manber et al., 2004; Manber et al., 2010). The current finding

builds upon these earlier results in that responders assigned to SPEC maintained their improvement on HRSD₁₇ scores across the postpartum time frame compared to responders assigned to the control groups. In sum, this suggests that not only was response to treatment important for reducing postpartum depressive symptoms, but also successful treatment with the active treatment, SPEC, was influential in maintenance of change and improvement up to nine months postpartum. Thus, the current research provides preliminary evidence that successful treatment with acupuncture may provide protection against reemergence of mood symptoms postpartum.

BDI-II and HRSD₁₇ Differences

The differences in statistical significance between the BDI-II and HRSD₁₇ mixed effects analyses might be explained in a number of ways. First, the BDI-II and HRSD₁₇ vary in administration. The BDI-II is a self-report measure and the HRSD₁₇ a clinician administered rating. Research has indicated that the prevalence of depression may vary systematically as a function of the method used to identify depression cases.

Self-report measures introduce the possibility of participant bias. This may present as an exaggeration of symptoms by participants, compared to objective measurements in which trained clinicians provide ratings; however, it is also possible participants might underreport symptoms. It has been hypothesized that self-report measures may identify a broader spectrum of depressive disorders or symptoms that reflect comorbid psychiatric illness (e.g., anxiety) or general distress whereas clinician administered ratings adhere to strict guidelines when determining whether or not a symptom is rated. In the current study, responders tended to rate fewer postpartum depressive symptoms on the BDI-II, a self-report measure, whereas on the HRSD₁₇,

responders and non-responders did not significantly differ. This suggests that participants perceived themselves as less depressed than the clinician.

In addition to participant bias, it is possible that response bias may have influenced the results. The BDI-IIs were predominately sent by mail during the follow-up phase whereas HRSD₁₇ assessments were completed over the phone. As a result, there were fewer BDI-IIs completed at each time point in the postpartum compared to HRSD₁₇ assessments. There were no significant differences in HRSD₁₇ scores during follow-up between those who returned the BDI-II and those who did not at 10 weeks ($p>.509$), six months ($p>.946$), and nine months postpartum ($p>.939$), suggesting a lack of difference in patterns of response between the two groups, and giving greater confidence to the overall main effect found when examining BDI-II scores using mixed effects analyses.

Second, while both are measures of depression, the BDI-II and HRSD₁₇ assess slightly different dimensions of a multi-dimensional construct. The HRSD₁₇ focuses primarily on behavioral and somatic symptoms of depression, while the BDI-II includes a large focus on cognitive aspects of depression. The HRSD₁₇ assesses symptoms such as early, middle, and late insomnia; psychomotor retardation; agitation; loss of appetite and weight loss; and muscular aches and pains. There is less emphasis on cognitive factors influencing depressive symptomatology on the HRSD₁₇ compared to the BDI-II. Although the two measures may tap slightly different dimensions of depression, they tend to be highly correlated (Beck et al., 1996). Consistent with the literature, in the current study the BDI-II and HRSD₁₇ were highly correlated with each other at each of the three

postpartum time points: ten weeks postpartum (0.68), six months postpartum (0.73), and nine months postpartum (0.71).

Furthermore, the BDI-II has been consistently found to consist of two factors: somatic and cognitive. Research examining the factor structure of the BDI-II in a non-clinical pregnancy sample found a two factor solution explained 43% of the variance with somatic and cognitive factors contributing equally (Carvalho Bos et al., 2009). However, a three factor solution was preferred during the postpartum, explaining 50% of the variance, and included a factor for guilt in addition to somatic and cognitive factors. Moreover, research has indicated that women with postpartum depression appear to be significantly more likely than nonpostpartum women to present with anxious features (Hendrick, Altshuler, Strouse, & Grosser, 2000), and during pregnancy, somatic symptoms such as appetite increase, sleep disruption, and agitation have been found to be uninformative with regard to a MDD diagnosis (Yonkers et al., 2009). In fact, Spinelli and Endicott's (2003) controlled clinical trial testing the efficacy of IPT during pregnancy excluded the somatic cluster from the BDI-II, as it did not reduce the psychometric stability of the BDI-II in this patient population, and the cognitive cluster of symptoms on the BDI-II was most sensitive to depression during pregnancy.

Extrapolating from these findings, it may be that in the current study responders fared better than non-responders as measured by the BDI-II based on enduring effects related to cognitive aspects of depression as opposed to somatic. Post-hoc analyses using mixed effects analyses to examine changes in the cognitive cluster of items on the BDI-II, across the three postpartum time points, revealed responders to treatment endorsed fewer cognitive based depressive symptoms compared to non-responders, even when

covarying for number of continuation sessions during pregnancy and postpartum, $F(1, 120) = 8.11, p = .005$. A main effect for the somatic cluster of BDI-II items across the three postpartum time points was also significant, such that responders endorsed fewer somatic depressive symptoms postpartum, with continuation sessions during pregnancy and postpartum included as covariates, $F(1, 120) = 4.56, p = .04$. However, in comparison to the cognitive factor the significance was not as strong. This trend suggests that while responders experienced fewer cognitive and somatic depressive symptoms postpartum compared to non-responders, the difference in somatic symptoms between the groups was not as large. Thus, somatic symptoms may remain higher in both responders and non-responders in the postpartum phase based on recovery from birth, delivery, and lasting somatic concerns related to changes in sleep, appetite, and fatigue.

Influence of Remission on Enduring Response Effect

While responders endorsed fewer depressive symptoms during follow-up compared to non-responders, the long-term outcomes for treatment during pregnancy and postpartum are not well characterized. Depression runs a chronic course in which relapse and recurrence rates following treatment are high (Solomon et al., 2000). A meta-analysis completed by Vittengl et al. (2007) found that among responders to cognitive behavior therapy, 29% experienced a relapse or recurrence by the end of the first year following treatment. In a high-risk pregnancy sample, Cooper et al. (2003) found that the effects of therapeutic treatment (compared with usual care) faded within about five months. The postpartum period presents significant challenges to women as they balance infant care with responsibilities in the home and at work. Despite these challenges, women in the current study tended to remain well in the postpartum phase.

In fact, the rates of depression as measured by both the BDI-II and HRSD₁₇, regardless of response status, fell in the minimal range during the postpartum phase suggesting overall improvement on depression scores compared to during pregnancy. The overall decline in depression symptom severity scores from pregnancy and maintained throughout the postpartum may be attributable to continued rates of recovery among those who did not respond to the acute treatment. Alternatively, these findings may simply reflect the episodic course of depression whether or not it is treated (Solomon et al., 2000) or may also reflect accelerated improvement over time independent of recovery following acute treatment. Whether participants improved following pregnancy due to remission or due to the effects of their treatment, overall women in this project remained well during the postpartum phase, despite facing challenges which might undermine the sustainability of recovery.

Treatment and Relapse Rate

It was hypothesized that a subset of women would experience re-occurrence of MDD in the postpartum and specifically, that time to relapse would be sooner for participants in the control groups (NSPEC and MSSG) than for participants in the SPEC group. In the current study, few women experienced re-occurrence of MDD during the postpartum phase; therefore, a survival analysis testing differential likelihood of relapse was not tested.

Among the 65 who remitted from their index episode of MDD by 36 weeks gestation, 6% (4/65) met criteria for MDD postpartum, 20% (13/65) scored ≥ 14 on the HRSD₁₇ postpartum, and 11% (7/65) began taking antidepressant medication postpartum. The incidence of postpartum depression in the current study is similar to rates reported in

the literature, which range from 3.5% to 17%. However, given this was a high-risk sample and that the probability of relapse among patients with MDD in the general population after six months is nearly 30% (Keller et al., 1983), it was anticipated that a larger group of women would experience re-occurrence of MDD postpartum.

The lack of re-occurrence in the postpartum may be due to a number of reasons. The current sample consisted of women with high levels of education (35% obtained a graduate degree), were largely employed (63%), and reported a household income greater than \$60,000 (67%) and therefore have had both the resourcefulness and finances to access support in the postpartum phase to balance the responsibility of infant care with work and family. Additionally, some women sought treatment for depression during the postpartum phase including antidepressant medication (18%), psychotherapy (12%), and massage and/or acupuncture (7%), which have likely reduced depressive symptoms. Furthermore, the sample consisted of women who identified as living with their husband (82%) or with the father of their child (8%). Given the increasing rates of births to single mothers, and their associated stressors, the fact that 90% of the women in the current study were in committed relationships with the father of their child may have resulted in increased benefit from emotional support and a sense of stability. Another source of support for the participants may have been the continuous attention they received through their involvement in the study. These factors may have improved outcomes. Finally, while research points to a continuity of depressive symptoms from pregnancy into the postpartum phase, it also documents a decrease in depressive symptoms from pregnancy to the postpartum period (Evans et al, 2001). This pattern is similar to the reduced rates of depression following birth in the current project.

Predictors of Postpartum Depression

It was hypothesized that previously identified predictors of postpartum depression including a history of depression during pregnancy and a previous episode of postpartum depression would emerge as significant predictors in the current study. The results were largely consistent with this hypothesis and replicated findings in the literature related to predictors of postpartum depression. Spearman's correlations indicated that both number of previous episodes of depression during pregnancy and number of previous episodes of postpartum depression were significantly correlated with HRSD₁₇ scores at six months postpartum. This finding is consistent with previous research that has demonstrated depression during pregnancy is one of the strongest predictors of postpartum depression (Milgrom et al., 2008; Verkerk et al., 2003) and that previous episodes of postpartum depression increase the risk for future postpartum episodes (Wisner et al., 2004).

While the correlations among number of previous episodes of depression during pregnancy and number of previous episodes of postpartum depression were significantly correlated with HRSD₁₇ scores at six months postpartum, they did not demonstrate statistical significance at nine months postpartum. Research has indicated that the timing of onset of postpartum depression tends to cluster closer to birth, and 90% of women experience onset within 28 weeks postpartum (Wisner et al., 2004); therefore, it is possible that by nine months postpartum there were fewer depression symptoms emerging, thereby decreasing the likelihood of finding statistically significant positive correlations. Additionally, over half of infants are sleeping concurrently with their parents at five months old (Henderson, France, Owens, & Blampied, 2010) and because sleep and mood are highly correlated, it is possible that improved sleep may have also

resulted in reduced depressive symptomology at nine months postpartum. Overall, the findings are consistent with the literature and suggest that previous history of depression, occurring during pregnancy and the postpartum, places a woman at greater risk for experiencing postpartum depression following a future pregnancy.

Exploratory Analyses: ROC Curve Approach

Exploratory analyses were completed to identify characteristics of women who were at risk for experiencing clinically significant depression symptoms postpartum ($\text{HRSD}_{17} \geq 14$ during the postpartum phase). Overall, the findings revealed that greater depression symptoms in late pregnancy (36 weeks gestation) increased the likelihood of experiencing depression in the postpartum phase. Furthermore, low client expectations of their treatment assignment during the acute phase and childhood emotional abuse also emerged as significant predictors. More specifically, the ROC analysis revealed that participants who scored ≥ 11 on the HRSD_{17} assessment (mild depression) at 36 weeks gestation were more likely to experience clinically significant depressive symptoms postpartum. Among participants who scored at least 11 on the HRSD_{17} at 36 weeks gestation, those who also provided a less favorable expectation of assigned treatment (< 3.75) were most likely to experience postpartum depression. Lastly, those who scored below 11 on the HRSD_{17} at 36 weeks gestation, yet endorsed greater levels of childhood emotional abuse (≥ 15), were more likely to experience postpartum depression. These findings demonstrate the importance of addressing depressive symptoms during pregnancy as well as provide evidence of increased risk for postpartum symptoms associated with negative client expectations of treatment and childhood emotional abuse.

The best predictor of clinically significant postpartum symptomatology was experiencing $\text{HRSD}_{17} \geq 11$ (mild depression) at the end of pregnancy. This finding is consistent with the literature in which previous studies have found that many cases of postpartum depression, up to 40%, are preceded by depression during pregnancy (Milgrom et al., 2007; Verkerk et al., 2003). The implication is that it is important to target women who are at risk for postnatal depression and begin screening during mid-to-late pregnancy.

It is well documented that patients' expectations of treatment effectiveness can have a powerful influence on outcome in terms of predicting full recovery and reduction of depression symptoms post-treatment (Sotsky et al., 1991). Also, because of their potency, expectations have been conceptualized as a "common factor" of intervention (Frank & Frank, 1991). In exploring predictors of postpartum depressive symptoms, expectancy played an influential role, as participants with $\text{HRSD}_{17} \geq 11$ (mild depression) during late pregnancy and with low expectations of their assigned treatment were at greatest risk for experiencing postpartum depression. This suggests that expectations may not only influence treatment outcome, but may result in enduring depressive symptoms through pregnancy and into the postpartum. Thus when considering viable treatment options for high-risk women during pregnancy, it will be important to promote favorable expectations of the treatment to increase the likelihood of success in the postpartum.

Interestingly, among women who did not exhibit increased levels of depression symptomatology during late pregnancy, the findings provided evidence of increased risk for postpartum depression associated with a history of childhood emotional abuse.

Literature on predictors of postpartum depression have yielded mixed results when examining the effect of exposure to abuse in childhood and its impact on postpartum depression. Furthermore, it is not clear that these studies have separated forms of abuse, whether sexual, physical or emotional. There have been a number of studies indicating that emotional abuse is associated with increased levels of depression (Mullen, Martin, Anderson, Romans, & Herbison, 1996; Rich, Gingerich, & Rosen, 1997; Spertus, Yehuda, Wong, Halligan, & Seremetis, 2003). Additionally, there is emerging evidence that history of childhood emotional abuse predicts depressive symptomatology even when controlling for other types of abuse, suggesting it is more severe than overt forms of abuse and has implications for women's mental health (Spertus et al., 2003). The mechanism by which this is operating in the current study is unclear; however, it may be that the psychological and social adjustments associated with childbirth and parenthood activate preexisting negative beliefs and fears. Given the current finding, future studies might further evaluate the impact of childhood trauma and the role of emotional abuse, particularly in women who otherwise do not appear to be at risk for postpartum depression.

Exploratory analyses were also completed to identify characteristics of women who were at risk for experiencing MDD during postpartum (meeting SCID criteria). The findings revealed that women who stopped taking an antidepressant medication after conception were most likely to experience MDD during postpartum. Furthermore, women who had not taken an antidepressant early in pregnancy, yet scored at or above 25 on their intake HRSD₁₇ (very severe range) were also more likely to experience MDD during postpartum.

These findings demonstrate that women with recent and severe depressive symptomatology were at greatest risk of meeting criteria for MDD during the postpartum phase. In this analysis, the best predictor was stopping antidepressant medication subsequent to conception. This finding is consistent with research by Cohen et al. (2006) in which he reported a five times higher rate estimate of relapse among pregnant women who discontinued antidepressant medication. Furthermore, women enduring higher rates of depressive symptomatology at intake, in the very severe range, were more likely to experience MDD postpartum. Again, this finding is consistent with the literature which indicates that high depressive symptomatology during mid-pregnancy (mean gestation age for the current study was 20.58 weeks) is one of the largest risk factors predicting postpartum depression (Verkerk et al., 2003) and that depression-related variables at the time of acute treatment appear to be the most reliable predictors of long-term outcome (Szadoczky, Rozsa, Zambori, & Furedi, 2004).

Taken together, the findings from the second ROC analysis support previous research and suggest that women who appear at high-risk during pregnancy, due to recent antidepressant use and significant depression symptomatology, are at increased risk for MDD postpartum. In turn, these findings highlight the dilemma women face who are taking antidepressants or who are severely depressed, particularly given the risks associated with antidepressant use during pregnancy. Thus, it will remain important for further research to continue to investigate both the short and long term effects of antidepressant treatments as well as continue to focus on the development of efficacious alternative treatments in this population.

Finally, it is important to emphasize that the ROC analysis is a method for generating hypotheses rather than testing hypotheses and that the number of participants in these analyses were small. Therefore, replication of the above listed findings should be tested in other samples before the predictor variables and cut-points identified in this study can be adopted as recommendations for clinical decision making. Future analyses might also attempt to explore predictors of postpartum depression with a larger sample. Though the current findings are consistent with the literature, they should be viewed as providing preliminary guidelines for identifying patients at risk for experiencing postpartum depression.

Limitations and Directions for Future Research

Generalizability

Several sample characteristics may limit the generalizability of the results. These include the high education and socioeconomic status as well as the predominance of Caucasians (66.9%). This may have impacted the results twofold in that (1) the data may not generalize to minority populations, and (2) participants in this study may have had access to resources which served to reduce stress following childbirth. Additionally, the exclusion criteria for this study included comorbid psychiatric conditions and medical disorders. The results may not generalize to pregnant women with other comorbid mental health conditions or to those with severe depression which might include psychotic features or suicide plans. Excluding participants with comorbid Axis I and Axis II disorders may have reduced complications in adherence to and potential response to treatment, which might also be considered when reflecting on the current study results. Finally, follow-up data was not obtained for all randomized participants in the study and

although there were few differences between those who completed follow-up and those who did not, further replication is needed to ensure generalizability of the results. Future research might focus efforts on including a more diverse participant population both in terms of ethnic background and socioeconomic status, as well as including a more diverse psychiatric population particularly given the high rates of comorbid psychiatric conditions among individuals diagnosed with major depression.

Measurement

Several measurement concerns must be considered when weighing the current results. First, the majority of follow-up HRSD₁₇ interviews were completed over the phone as opposed to in person. Great attempts were made to reach participants and as a result, few were lost to follow-up. However, it may be more difficult to determine certain behavioral aspects of depression when the assessment is not completed in person resulting in a less sensitive measurement. Conversely, there is an emerging body of evidence supporting telemedicine interventions which suggests that face-to-face contact may not be necessary to complete a competent assessment of mood functioning. Additionally, the majority of BDI-II follow-ups were mailed to participants. As a result, the number of completed BDI-IIs at each postpartum time point is lower than the number of HRSD₁₇ assessments completed. Despite this, there was no evidence of differences in patterns of response on the HRSD₁₇ when examining those who returned BDI-IIs compared to those who did not. Again, while efforts were made to encourage participants to complete the BDI-II, the number returned was likely impacted by the medium used for measurement.

A second limitation of the current study was the lack of a continuous timeline of depression symptoms over the course of the postpartum phase. Participants were assessed at three time points (ten weeks, six months, and nine months postpartum). Data was not available for the periods of time falling between these assessments. It is possible periods of depressive symptomatology may have been missed, and perhaps rates of postpartum depression are lower, because of the frequency of mood assessments or the incidence of depression peaked at a time not measured in this study.

Third, a potential confound to the current results was the influence of continuation sessions on the maintenance of response to treatment. Attempts were made to control for continuation sessions, both during pregnancy and postpartum, by including these sessions as a covariate in the major statistical analyses and when doing so, the results held. Controlling for continuation sessions was not meant to suggest that continuation sessions are not effective or do not play a role in maintaining either a treatment or response effect. This was not specifically tested; rather, in the current study it was demonstrated that response to treatment can have an impact in the postpartum above and beyond the impact of continuation. Further research might look specifically at the role continuation sessions play in preventing further reemergence of mood symptoms postpartum following completion of the acute phase of treatment.

Emphasis on Response Status

The main analyses for the current study focused on response status to the acute phase of treatment. Several limitations are associated with this categorization. Although conclusions can be made about the role of response status on depressive symptoms throughout the postpartum, these conclusions are complicated by the differing treatments

participants received within the current study. Because the study did not focus specifically on treatment effects, less conclusive findings can be made with regard to the impact of treatment itself on the postpartum presentation. Post-hoc analyses in the current study, limited solely to responders, provided initial evidence supporting the impact of acupuncture specific for depression on maintaining change and improvement postpartum. However, limiting analyses solely to responders, reduced the sample size to a smaller number which may have decreased power. While the current research builds upon the findings of Manber et al. (2004; 2010), conclusive statements regarding treatment effects and in relation to enduring effects of acupuncture must be cautiously interpreted. Future research might explore more specifically the role of acupuncture in both treating and preventing postpartum depressive symptoms and focus on including larger sample sizes.

Finally, in the current study, analyses comparing treatments collapsed the control groups to include both massage and acupuncture not specific to depression (NPEC). It cannot be assumed that the placebo response for massage and control acupuncture are the same. Furthermore, while attempts were made to double blind acupuncturists, the massage condition was unblinded. The massage group may have generated a downward or upward effect on the control mean. In fact, the massage and NSPEC groups differed in their depression score means such that the massage group tended to fare better, potentially demonstrating a therapeutic effect, which made it more difficult to find a treatment effect when comparing SPEC to the combined control groups.

Implications for Clinical Practice and Conclusion

The long term consequences of postpartum depression suggest that preventative approaches are warranted. The implications of the current study results in relation to potential prevention of postpartum depression are twofold: (1) identify and target high-risk women during pregnancy, and (2) successful treatment during pregnancy may have enduring effects into the postpartum phase. Therefore, the current results provide information related to both characteristics placing women at greatest risk as well as the timing of greatest risk.

In the current study, women were most at risk for experiencing postpartum depressive symptomatology based on the level of their depressive symptoms during pregnancy. This finding is consistent with the literature and suggests that evaluating indicated interventions (e.g., targeting women with existing symptoms or early diagnosis of depression during pregnancy) may be a more fruitful approach to preventing postpartum depression rather than waiting until symptoms emerge postpartum. In fact, pregnancy provides an ideal opportunity for prevention because its onset is preceded by a clear marker (giving birth), the period of risk for illness onset is well-defined, and a high-risk sample of mothers can be identified. Additionally, pregnant women may be unusually open to making changes to improve their mental health before their baby is born (Cowan & Cowan, 2000). Therefore, emphasis on screening and identifying women at high-risk during pregnancy are critical components for prevention measures.

Additionally, once a high-risk group is identified, interventions might be targeted at them and implemented during pregnancy. The current findings are aligned with emerging research which suggests that successful treatment during pregnancy may have a

lasting effect into the postpartum phase. In the context of the current study, alternative treatments were utilized; and, while response to any of the study treatments resulted in an enduring effect postpartum, preliminary evidence suggested women treated with acupuncture specifically tailored for depression experienced the most enduring effect. Eliciting from women their preferred choice of treatment, if determined to be high-risk, may also be beneficial given response may be equally important as treatment assignment. Future studies might expand upon the emerging body of research which demonstrates enduring response to treatment postpartum, following implementation during pregnancy.

Given the deleterious costs of postpartum depression to individuals and society, prevention and treatment remain concerns. Efforts have begun to focus on better understanding the illness; however, there remains a significant lack of substantial high-quality research in this area (Wisner et al., 2006). Continued focus on both increased surveillance of postpartum depression and better implementation of preventative and treatment options for childbearing women is needed. Future research might continue to focus on addressing critical questions related to the course and treatment of maternal depression, which in turn might inform approaches of preventive interventions with pregnant and postpartum women.

APPENDIX A
PERINATAL SURVEY

Perinatal Survey

IN: _____ ID#: _____ Date: _____

Pregnancy Information

Date of first day of last menstrual period (LMP): _____

How certain are you of this LMP? _____

Estimated number of weeks of pregnancy _____

Due Date: _____

Due Date is based on: ☐ LMP

☐ Ultrasound: date of ultrasound _____

☐ Conception date

☐ Other _____

Weight before pregnancy _____

SECTION I - Background Information

Date of your birth: _____ (mm/dd/yyyy)
(please make sure the year is the year of birth)

1) Living arrangement

- ☐ Live with husband
☐ Not married but live with a partner
☐ Live without a partner but with other adults
specify relationship of others to you

☐ I am the only adult in my household

We must report to the government, which funds this study, a summary of both ethnicity and race of participants in our study. Therefore, please answer **BOTH** questions #2 (Ethnicity) and #3 (Racial Background) though it may seem redundant.

2) Select Ethnicity: ☐ Hispanic or Latino or ☐ Not Hispanic or Latino

3) Select Racial Background:

<input type="checkbox"/> American Indian/ Alaska Native	<input type="checkbox"/> Asian
<input type="checkbox"/> Native Hawaiian or Other Pacific Islander	<input type="checkbox"/> White
<input type="checkbox"/> Black or African American	<input type="checkbox"/> Other _____

4) Health Insurance

a) Do you have a general health insurance plan?

☐ Yes ☐ No

b) Do you have any insurance coverage of mental health?

☐ Yes ☐ No

c) Do you have coverage of prenatal care?

☐ Yes ☐ No

d) Do you have insurance coverage of post-partum care? (For at least 2 months after delivery)

☐ Yes ☐ No

5) Transportation

When it comes to getting transportation to clinic/study appointments would you say that:

- ☐ Transportation is not a problem.
- ☐ You will usually be able to get transportation.
- ☐ You might have occasional trouble getting transportation.
- ☐ Transportation will always be a problem.

Education & employment

6) How many years of formal education have you completed?

<input type="checkbox"/> No High School	<input type="checkbox"/> Some College
<input type="checkbox"/> Some High School	<input type="checkbox"/> College Degree
<input type="checkbox"/> High School Diploma	<input type="checkbox"/> Additional Education – Please specify _____

7) What is your usual profession? _____

8) Present employment status:

- | | |
|--|--|
| <input type="checkbox"/> Working full-time | <input type="checkbox"/> Student |
| <input type="checkbox"/> Working part-time | <input type="checkbox"/> Disabled |
| <input type="checkbox"/> Homemaker | <input type="checkbox"/> On Leave because of pregnancy |
| <input type="checkbox"/> Unemployed | <input type="checkbox"/> On Leave for reasons other than pregnancy |
- ☐ Other _____

9) From all sources of income, including your own, what is the total combined income of all people you live with?

- | | | |
|--|---|--|
| a) <input type="checkbox"/> Less than \$10,000 | <input type="checkbox"/> \$30,000 to \$40,000 | <input type="checkbox"/> \$70,000 to \$100,000 |
| <input type="checkbox"/> \$10,000 to \$20,000 | <input type="checkbox"/> \$40,000 to \$50,000 | <input type="checkbox"/> Over \$100,000 |
| <input type="checkbox"/> \$20,000 to \$30,000 | <input type="checkbox"/> \$50,000 to \$60,000 | |

b) How many people are supported by this income? _____

c) Do you receive SSI or child support?

- ☐ Yes ☐ No

SECTION II - Pregnancy

1) a. Was this pregnancy planned?

- ☐ Yes ☐ No

b. If not, were you planning to have children ever?

- ☐ Yes ☐ No

2) a. Does the father of the baby live with you?

- ☐ Yes ☐ No

b. If not, do you live with a partner committed to parenting the child with you?

- ☐ Yes ☐ No

3) Is the father of the baby involved?

- ☐ Yes ☐ No

- 4) Please check all that apply regarding the involvement of the father of the baby.

The father of the baby:

- a. Provides some financial support

☐ Yes ☐ No

- b. Has excitement and interest in having this child

☐ Yes ☐ No

- c. Understands my emotional needs related to this pregnancy

☐ Yes ☐ No

- 4) The numbers on the following line represent different degrees of happiness in your relationship. The middle point, "happy", represents the degree of happiness of most relationships. **Please circle the number** which best describes the degree of happiness, all things considered, of your relationship.

0	1	2	3	4	5	6
Extremely	Fairly	A Little	Happy	Very	Extremely	Perfectly
Unhappy	Unhappy	Unhappy		Happy	Happy	Happy

- 5) Do you have friends or family members who will be able to help with the physical care of your baby after delivery?

☐ Yes ☐ No

If yes, specify how many people will be able to assist you _____

- 6) Do you have friends or family members who will be available to you for emotional support after you deliver your baby?

☐ Yes ☐ No

If yes, specify how many people will provide emotional support _____

- 7) Are you attending or planning to attend childbirth classes?

☐ Yes ☐ No

If yes, please specify type of class _____

8) Are you attending or planning to attend parenting classes?

☐ Yes ☐ No

9) Are you taking vitamins daily or almost daily?

☐ Yes ☐ No

10) How do you plan to feed the baby?

☐ Bottle (formula) ☐ Breast feed ☐ Both bottle and breast feed

11) Have you experienced any of the following in association with this pregnancy?

☐ Nausea ☐ Vomiting
☐ Heartburn ☐ Constipation
☐ Food cravings (What do you crave? _____)

12) Please indicate below the type of delivery and pain control that is anticipated?

a) Type of delivery ☐ Vaginal ☐ C-section
b) Type of pain control ☐ None ☐ Epidural ☐ Other _____

SECTION III – Depression

Depression and Nausea Timeline

Month/year depression began: _____

1. When did this depressive episode begin in relation to the present pregnancy?

☐ Before current pregnancy ☐ During current pregnancy

2. Do you believe that being pregnant has affected the present depressive episode?

☐ No, pregnancy had no impact on my depression - the two are independent
☐ Yes, pregnancy made it somewhat better
☐ Yes, pregnancy made it somewhat worse
☐ Yes, pregnancy is the cause of my depression

3. Have you experience nausea during this pregnancy? ☐ Yes ☐ No

If you answered yes to question 3 then please continue. If No, then skip to SECTION IV.

3b. Month/year your nausea began? _____

3c. Please check the box that applies best:

- ☐ Nausea preceded depression ☐ Nausea began after the onset of depression
☐ Nausea and depression emerged simultaneously

4a. What role do you think nausea has played in **causing** your depression?

- ☐ Nausea is the primary cause of my depression
☐ Nausea is partially responsible for my depression
☐ My depression is not related to nausea

4b. If you think nausea did play a role in causing the onset of your depression, please check the statement that best describes your experience at that time:

- ☐ Unable to keep meals, or vomited more than once a day
☐ Vomited once a day
☐ Nausea was present most of the day, but no vomiting
☐ Nausea was present, but not all day

5. What relationship, if any, have you noticed between the experience of nausea and depressed mood?

- ☐ Strong relationship ☐ Minimal relationship ☐ No relationship

6. Do you experience nausea at this point in your pregnancy?

☐ No If no, when did your nausea end _____ (month/year)

☐ Yes If yes, please check the box that applies best:

- ☐ Unable to keep meals, or vomit more than once a day
☐ Vomit once a day
☐ Nausea present most of the day, but no vomiting
☐ Nausea present, but not all day

SECTION IV – Hospitalizations and Medications

1. List all your hospitalizations stating specific type of illness/surgery/hospitalization.
Please be specific.

_____ Year _____
_____ Year _____
_____ Year _____
_____ Year _____

2. What prescription medications are you presently taking? List name, dosage, and how often you take each medication.

Name	Dosage	Frequency

Comments _____

3. What non-prescription medications do you presently take? For example – herbs, laxatives, diet pills, vitamins, antacids or cold remedies. List name, dosage, and how often you take each.

Name	Dosage	Frequency

Comments _____

SECTION V - Smoking Habits

1. Do you smoke now? ☐ Yes If so, how much? _____
☐ No

2. Have you quit recently? ☐ Yes ☐ No

3. Did you stop because of pregnancy? ☐ Yes ☐ No ☐ not applicable

If so, when? ☐ before pregnancy (date _____)
☐ during pregnancy

SECTION VI – Health Maintenance

For each of the statements below please indicate how often you do each of the following.

In the left column, indicate frequency prior to this pregnancy. In the right column, indicate frequency during this pregnancy.

	Before pregnancy	During pregnancy
1) use seat belt?	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable
2) comply with a routine dental check up schedule (every 6-12 months)?	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable
3) wear a helmet when riding a bike or motorcycle?	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable
4) have sexual partners use condoms?	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable
5) eat nutritional meals	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable
6) get enough rest	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable
7) drink and drive	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable
8) exercise for at least 20 minutes at least 3 times a week?	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable
9) take vitamins / minerals	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable	<input type="checkbox"/> always <input type="checkbox"/> usually <input type="checkbox"/> rarely <input type="checkbox"/> never <input type="checkbox"/> not applicable

Section VII Acupuncture/Massage Treatments:

- 1) Have you ever received acupuncture treatment in the past?
☐ Yes ☐ No

If Yes,

When was your **last** acupuncture treatment (month/year)?

What was the purpose of the treatment?

Was it helpful? ☐ very much ☐ somewhat ☐ not at all

- 2) Have you received massage from a professional massage therapist before?
☐ Yes ☐ No

If Yes,

When was your **last** massage session?

If it was part of a series of massage sessions-

How many sessions did you have? _____

How often did you have them (eg. once/week, twice/week)? _____

Did massage help your mood? ☐ very much ☐ somewhat ☐ not at all

- 3) The answers to the following questions will not determine which group you will be assigned to in the study. This is determined by a completely randomized system. We would just like to know what your preference would be.

- a. Do you have a preference for receiving acupuncture or massage as your treatment in the study?

☐ Acupuncture

☐ Massage

- b. Which do you think would be more helpful in treating your depression?

☐ Acupuncture

☐ Massage

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VITA

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