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MATERNAL POSTPARTUM DEPRESSION AND FATHER INVOLVEMENT ACROSS THE TRANSITION TO PARENTHOOD

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**MATERNAL POSTPARTUM DEPRESSION AND FATHER INVOLVEMENT
ACROSS THE TRANSITION TO PARENTHOOD**

A Dissertation Presented

by

KATHERINE NEWKIRK

Submitted to the Graduate School of the
University of Massachusetts Amherst in partial fulfillment
of the requirements for the degree of

DOCTOR OF PHILOSOPHY

September 2018

Clinical Psychology

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A Dissertation Presented

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DEDICATION

To my husband, Tejas Bhatt, who supported my work with patience and his own involvement in childcare. To my daughter, Selene Bhatt, who patiently waited until my proposal was finished to make her own appearance. To my father, Jim Newkirk, who provided both instrumental and emotional support for my many years of education. And to my mother, Barbara Newkirk, who inspired me and supported me and would have enjoyed hearing about this project.

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ABSTRACT

MATERNAL POSTPARTUM DEPRESSION AND FATHER INVOLVEMENT ACROSS THE TRANSITION TO PARENTHOOD

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Maternal postpartum depression is a common complication of childbirth that affects the whole family. Fathers' greater involvement in childcare can buffer children from the negative effects of mothers' depression, and aid in mothers' recovery, so it is important to understand under what conditions fathers become more or less involved when mothers are depressed. Prior research has supported both a compensation hypothesis, whereby fathers compensate for the effects of mothers' depression on mothers' parenting by being more involved in parenting, and a spillover hypothesis, whereby mothers' negative emotionality causes fathers to pull back from family life and be less involved in childcare. The present study addressed three factors that could moderate the relationship between mothers' postpartum depression and fathers' involvement in childcare: the timing and duration of mothers' postpartum depression, the presence of mothers' comorbid anxiety symptoms, and whether couples work the same shift or opposite-shifts from each other. These questions were addressed using a sample

of 182 dual-earner, working-class, married or cohabiting couples having their first child. Findings supported a compensation effect when mothers' were depressed at one time point, but not two, with a stronger interaction between early and later maternal depression for couples working opposite-shifts than those working same-shifts. Comorbid anxiety moderated the effect of maternal depression on father involvement at 1-month postpartum, with fathers more involved when mothers were depressed with low anxiety, and less involved when mothers were depressed with higher anxiety. Implications for research and practice are discussed.

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CHAPTER 1

INTRODUCTION

Maternal depression affects 1 in 11 infants during the first year of life, with rates much higher among families facing stressors such as financial hardship (Early Head Start Research and Evaluation Project, 2006; Miller, 2002). The high prevalence of postpartum depression (PPD), defined as a depressive episode occurring within one year of the birth of a new child, is of great concern not only for the negative effects on mothers, but also because of its long-term detrimental effects on children's cognitive functioning, emotion regulation, internalizing problems, externalizing problems, and behavior problems (Barker, Jaffee, Uher, & Maughan, 2011; Feldman, Granat, & Pariente, 2009; Goodman, Rouse, Connell, Borth, Hall, & Heyward, 2011; Hay, Pawlby, Waters, & Sharp, 2008; Korhonen, Luoma, Salmelin, & Tamminen, 2012; O'Hara & McCabe, 2013). More recently, scholars have called for research to examine possible mechanisms linking maternal PPD to children's developmental outcomes (O'Hara & McCabe, 2013).

One mechanism whereby maternal postpartum depression has been shown to predict children's developmental outcomes is through its effects on parenting. Studies have found that parenting is impaired among mothers with PPD, leading to reduced maternal engagement and withdrawn maternal behaviors. More recent research has examined how maternal PPD extends beyond mothers' own parenting to influence fathers' parenting, particularly in terms of involvement with their infants (Cabrera, Hofferth, & Chae, 2011; Goodman, Lusby, Thompson, Newport, & Stowe, 2014; Jia, Kotila, Schoppe-Sullivan, & Dush, 2016; Paulson, Dauber, & Leiferman, 2006; Planalp

& Braungart-Rieker, 2016). Studies on this topic suggest that father involvement can buffer some of the deleterious effects of maternal PPD on child development (Chang, Halpern, & Kaufman, 2007; Lewin, Mitchell, Waters, Hodgekinson, Southammakosane, & Gilmore, 2014; Mezulis, Hyde, & Clark, 2004); thus, it is important to understand whether, and under what conditions, fathers' involvement with the care of their infants is associated with maternal postpartum depression.

Mothers with postpartum depression frequently experience additional mental health problems. One study estimates that 38% of women with postpartum depression experience a comorbid anxiety disorder (Austin et al., 2010). A large U.S. study found that 6% of women experience significant depressive symptoms with significant comorbid anxiety symptoms in the first three months postpartum (Farr, Dietz, O'Hara, Burley, & Ko, 2014). While research has examined the deleterious effects of postpartum anxiety alone (For reviews see Glasheen, Richardson, & Fabio, 2010; Ross & McLean, 2006), there is a dearth of research on how the presence of comorbid anxiety may alter the consequences of maternal PPD. Given that some research has shown that mothers with PPD and comorbid PPA may recover more slowly than those with PPD symptoms alone (Yawn, Bertram, Kurland, & Wollan, 2015) and have more severe symptoms (Farr et al., 2014), it is important to understand how this comorbidity could alter the effects of PPD on the family.

The current study builds on the existing literature on maternal PPD and father involvement in several key ways. First, this research contributes to the literature by examining longitudinal data across the first year of parenthood. Existing literature suggests that associations between mothers' PPD and fathers' involvement may differ

depending on when, during the postpartum year, they are measured. Longitudinal research can help to clarify these associations, and determine whether associations change across the postpartum period. In addition, the current study also examines how change in mothers' depressive symptoms across the postpartum year may be associated with change in father involvement – a question that has received little attention in the literature.

A second contribution is the focus on how comorbid maternal postpartum anxiety (PPA) symptoms may moderate the associations between maternal PPD and fathers' involvement. Several authors have noted the lack of research on comorbid PPD and PPA (Leach, Poyser, & Fairweather-Schmidt, 2015; Seymour, Giallo, Cooklin, & Dunning, 2015; Yawn et al., 2015), despite the fact that comorbidity rates are even higher in the postpartum population than they are in the general population (Ross, Evans, Sellers, & Romach, 2003).

A third way the current study contributes to the literature is by examining the relationship between mothers' mental health and father involvement in an understudied sample of dual-earner, working-class parents having their first child. Much of the literature on maternal PPD and father involvement has ignored how parents' work may influence the ways in which maternal PPD is associated with father involvement. For example, few working-class jobs offer paid leave, so new mothers generally take shorter maternity leave, which has been related to higher levels of depressive symptoms (Chatterji, Markowitz, & Brooks-Gunn, 2013). Moreover, parents' work hours necessarily dictate the amount of time that both mothers and fathers are at home and

available for childcare, and it is common for working-class parents to work different shifts from each other to cover their childcare needs.

The present study aims to illuminate how maternal postpartum depression is related to both levels of and changes in father involvement across the first year of parenthood, and whether these associations differ if mothers have significant comorbid anxiety symptoms in a sample of dual-earner, working-class new parents. The following literature review first addresses the definition and consequences of maternal postpartum depression and comorbid anxiety. Second, it reviews research examining the connections between maternal postpartum depression and father involvement with infants. Third, it provides a rationale for why associations between father involvement and maternal postpartum depression might differ depending on the presence of comorbid anxiety. Finally, it addresses how the ecological context of dual-earner working-class new parents may shape these associations.

Literature Review

Defining Postpartum Depression

Postpartum depression is broadly defined as a depressive episode that occurs following childbirth. PPD differs from postpartum blues, which is a mild and temporary mood disturbance occurring three to five days after childbirth and typically resolving by the tenth day. Postpartum blues occurs after the majority of births (40 – 80% incidence) and is thought to be a consequence of hormonal changes that occur in the days following childbirth (Buttner, O’Hara, & Watson, 2012). Postpartum depression, on the other hand, is defined as a major depressive episode that occurs during the postpartum period. A

major depressive episode, by definition in the DSM-5, lasts for a minimum of two weeks and is characterized by either depressed mood or loss of interest or pleasure in almost all activities as well as at least four other symptoms such as feelings of worthlessness or guilt, difficulty making decisions, suicidal ideation, and changes in appetite or sleep. To be diagnosed as a major depressive episode, these symptoms must cause significant impairment or distress in at least one area of functioning, or, in the case of a minor depressive episode, may require a great deal of effort to function adequately (American Psychiatric Association [APA], 2013).

There is some debate as to the timing of onset of PPD. The DSM-5 uses a definition of a depressive episode with an onset occurring during pregnancy or within four weeks of the birth of a new child, as many depressive episodes begin during pregnancy (APA, 2013). In contrast, the World Health Organization and Centers for Disease Control and Prevention use a period of 12 months postpartum (Centers for Disease Control and Prevention, 2008; World Health Organization, 2010). In practice, as noted by O'Hara and McCabe (2013), most clinicians and many researchers define PPD as having an onset within the first 12 months postpartum. The current study uses a definition of 12-months postpartum due to it being the most commonly used definition in the literature (O'Hara & McCabe, 2013).

Prevalence estimates for postpartum depression have varied widely due to differences in measurement, timing of studies, and prevalence periods used. In a systematic review including only studies from "developed countries" and that used a diagnostic interview, a period prevalence rate of 19.2% was found for major and minor depression across the first 3 months combined, with point prevalence rates in the full

postpartum year ranging from 12.9% at 3-months to 6.5% at 12-months postpartum (Gavin, Gaynes, Lohr, Meltzer-Brody, Gartlehner, & Swinson, 2005). More recently, a study using a nationally representative sample of U.S. women found a prevalence rate of 10.2% for major depressive episodes across the first postpartum year using both a diagnostic interview and women's retrospective reports (Hoertel et al., 2015). Although there is some debate as to whether the incidence of major depressive episodes is higher among women during the postpartum year than it is for non-postpartum women of childbearing age, the deleterious effects of PPD on women and their families make PPD a recognized major public health concern that is increasingly recognized by professional organizations in medicine (Joseph, 2009) and legislative bodies in the U.S. (Rhodes & Segre, 2013).

Peak rates of postpartum depression seem to occur during the first 6 months postpartum (Paulson & Bazemore, 2010), although this finding could be due, in part, to a greater number of studies examining PPD in the first 6 months than 6-12 months postpartum. A 2010 meta-analysis looking at mothers' PPD found the peak rate of maternal depression occurred during the 3- to 6-month postpartum period (Paulson & Bazemore, 2010). Other studies have found onset typically occurs within 5 weeks (Cox, Murray, & Chapman, 1993) or 6 weeks postpartum (Kettunen, Koistinen, & Hintikka, 2014). Although there is no conclusive agreement on the timing of peak prevalence of PPD, it is generally assumed to occur during the first 6 months postpartum. Some mothers may recover within the first 6 months postpartum, whereas others' depressive symptoms may persist throughout the postpartum year (O'Hara & McCabe, 2013), making it important to consider the whole postpartum year when studying PPD.

The literature is further complicated by the ways in which depression is measured, with some studies using diagnostic interviews, some using continuous scores on self-report measures, and some using the cutoff criteria on self-report measures. These different approaches further explain the wide range of prevalence reported in existing literature. Diagnostic interviews are considered the most conservative method of defining PPD, as the diagnosis includes only cases that meet the clinical criteria for a depressive episode. These criteria require the individual not only to experience a minimum number of symptoms, but also that symptoms persist most of the day, nearly every day, for at least 2 weeks, and that they impair the ability to function at work, home, or in other important domains (APA, 2013).

Validated, self-report measures of depression, on the other hand, allows researchers to capture associations between depressive symptoms that may not reach diagnostic criteria, due to number, severity, or duration of symptoms, or due to someone being able to compensate and continue functioning adequately. The benefits of the self-report approach are that symptoms that do not meet the diagnostic criteria, but are still distressing to the person, have been found to be associated with less optimal outcomes for mothers and their families.

Self-report measures are frequently used in clinical settings to screen patients for PPD using a clinical cutoff, or score above which a patient is flagged as potentially having PPD. This approach provides an alternative between using a strict diagnostic criteria and using the scale as a continuous variable. Group differences above and below the cutoffs are better able to represent differences between people who are not depressed and those who likely are.

PPD and Comorbid Anxiety

Research has examined PPD and comorbid anxiety both in terms of whether or not individuals with PPD exhibit some anxiety symptoms, and in terms of individuals with a PPD diagnosis that also have a diagnosable anxiety disorder. In terms of comorbid symptoms, research has pointed to higher rates of anxious features among women with PPD than among women with depression outside of the postpartum period (Hendrick, Altshuler, Strouse, & Grosser, 2000). Other studies looking at anxiety-related symptoms among women with PPD have found higher rates of panic attacks (Miller, Pallant, & Negri, 2006), restlessness and agitation (Bernstein et al., 2008), phobias (Pitt, 1968), and obsessive-compulsive symptoms including intrusive thoughts primarily concerning harm coming to the baby (Abramowitz et al., 2010; Jennings, Ross, Popper, & Elmore, 1999), than occur in depression outside of the postpartum period. Turning to a diagnosis of an anxiety disorder, rather than merely the presence or absence of anxiety symptoms, research has documented higher comorbidity rates between PPD and postpartum anxiety disorders than in depression occurring outside of the perinatal period (Ross et al., 2003). An Australian study of postpartum women using a diagnostic interview found that 37.7% of women with major depressive disorder within the first 6-8 months postpartum had a comorbid anxiety disorder (Austin et al., 2010). In a smaller community sample in the U.S. of women at 8-weeks postpartum using a structured diagnostic interview to assess for anxiety disorders, 75% of women with postpartum Generalized Anxiety Disorder, the most prevalent postpartum anxiety disorder in the study, reported clinically significant levels of depressive symptoms (Wenzel, Haugen, Jackson, & Brendle, 2005). Although

the comorbidity rates seem to vary somewhat across studies and types of anxiety symptoms, it is clear that there is significant comorbidity.

An emerging body of research suggests that the presence of anxiety symptoms may reflect a more severe form of PPD. Though this is a relatively new area of study, the evidence suggests that there may be different types of PPD. A 2015 study using data from 17,912 mothers examining antepartum and postpartum depression highlighted the heterogeneity of perinatal depression in terms of timing of onset, severity, prior history of mood disorders, and symptom constellation. This study demonstrated that women with more severe PPD symptoms were more likely to have a history of comorbid anxiety and mood disorder. In addition, more severe PPD was characterized by higher reports of feeling scared or panicky and higher reports of suicidal ideation. A second group, with less severe PPD, was not characterized by higher reports of anxiety symptoms, and typically had an onset within the first 4 weeks of birth (Postpartum Depression: Action Towards Causes and Treatment (PACT) Consortium, 2015). Similarly, a 2014 study found greater depressive symptom severity, as well as higher levels of anxiety symptoms, among mothers with postpartum depression who had a prior history of depressive episodes or whose depressive symptoms began during pregnancy (Kettunen et al., 2014). Moreover, comorbid PPD with PPA has been found to take longer to remit when compared to PPD alone (Yawn et al., 2015) and symptoms are more severe among women with comorbid PPD and PPA than those who have only PPD or only PPA (Farr et al., 2014). In sum, it appears that mothers with PPD characterized by anxiety symptoms may be a different group than those with PPD absent anxiety symptoms. It has been noted that little research has examined differences between PPD and comorbid PPD and PPA

beyond examining comorbidity rates and symptom severity (Leach et al., 2015). The literature has done little to address how this comorbidity between PPD and PPA may have differing impacts on the mother and family, as compared to PPD alone.

Maternal Postpartum Depression and Father Involvement

Belsky's process model of parenting (1984) provides a useful framework to conceptualize the associations between mothers' PPD, comorbid PPA and fathers' parenting. Belsky posits that parenting is best explained by examining a combination of parents' personal psychological resources, child characteristics and the context in which they interact. Thus, this model of parenting includes parent's own mental health, family relationships, and contextual sources of stress and support.

The figure from Belsky's model (Figure 1) uses parents' personality, a trait-like variable, to embody parent characteristics, but Belsky also notes the importance of parent's psychological well-being in shaping parenting (Belsky, 1984). The current study focuses specifically on the parent characteristic of mothers' mental health, a state-like variable, as a stressor on the family system that spills over to influence fathers' parenting. Thus, mothers' mental health affects not only her own parenting in this model, but, fathers' involvement as well. Belsky also contends that child characteristics play a role in the system, arguing that child temperament and gender shape parent mental health and parenting. The current study examines parents' work hours and shift work as additional contextual sources of stress that shape parenting.

Mothers' parenting is compromised by PPD and consequently fathers' involvement can be influenced in two distinct ways by PPD. One hypothesis, the

spillover hypothesis, is based on the premise that the stress fathers experience in relation to their wives' postpartum depression increases their negative emotionality, which leads fathers' to disengage from family life and, thus, be less involved with their infants (Goodman et al., 2014). Coyne's (1976) theory on the interactional nature of depression would also point to a spillover effect, as it suggests that depressed persons induce a negative mood in others through excessive reassurance seeking behaviors, which could cause partners to withdraw from the depressed person. Among new parents, this withdrawal could reduce fathers' time spent engaging with family life and involvement with childcare. Coyne's research on the interpersonal nature of depression links depression with marital discord (Anderson, Beach, & Kaslow, 1999) which is linked to lower father involvement (Planalp & Braungart-Rieker, 2016). In contrast, the *compensation hypothesis* holds that fathers will increase their involvement with their infants when their wives with PPD withdraw from parenting, in order to make sure that the family system functions (Goodman et al., 2014; Minuchin, 1985). To understand these hypotheses, it is first important understand how PPD and comorbid PPA relate to mothers' own parenting, that in turn influences fathering.

A wealth of research has demonstrated negative effects of maternal postpartum depression on mothers' parenting (for reviews see Field, 2010; Lovejoy, Graczyk, O'Hare, & Neuman, 2000). Qualitative research has described mothers with PPD as disengaged from childcare, noting, "maternal unavailability was overwhelmingly identified as the most toxic effect of the depression, and often appeared resistant to enticements to re-engage in family life" (Beestin, Hugh-Jones, & Gough, 2014, p. 722). Mothers with PPD see themselves as less competent (Dix & Meunier, 2009), are more

disengaged from the child (Field, 2010; Lovejoy et al., 2000; Paulson et al., 2006) and are less involved with the care of their infants (Jennings et al., 1999; Lyons-Ruth, Wolfe, Lyubchik, & Steingard, 2002). This withdrawal from involvement shapes the context in which fathers parent their children.

One factor that could alter how PPD is related to mothers' parenting is the presence of comorbid anxiety. No research has examined how comorbid PPD and PPA are related to mothers' involvement with routine parenting tasks, such as changing diapers, bathing, and soothing the infant. In the absence of research on comorbid PPD and PPA, studies on PPA alone can provide clues as to how comorbid PPA could influence the associations between PPD and parenting. Research suggests that symptoms of the most common type of PPA disorder, Generalized Anxiety Disorder (Wenzel et al., 2005), may be associated with maternal over-involvement, rather than withdrawal, from infant care (Möller, Majdandžić, & Bögels, 2014). Postpartum mothers with anxiety experience excessive worry about their infants' well-being and intrusive, worried thoughts about harm coming to their babies (Abramowitz, Schwartz, & Moore, 2003). Thus these mothers may take on the majority of infant care to alleviate their anxiety when not in control of their infant. In support of this hypothesis, Jia et al. (2016) found that mothers' higher postpartum anxiety symptoms were related to increases in their time spent in childcare activities from 3-9 months postpartum.

In sum, the existing literature suggests that mothers may withdraw from involvement in parenting when they experience PPD, whereas some evidence points to anxious mothers becoming more involved in infant care. Mothers' parenting necessarily influences fathers' parenting. Fathers may be pulled to fill in for mothers' withdrawal

from parenting, may withdraw themselves due to their negative feelings brought about by mothers' impaired mental health, or may be deterred from being involved when mothers' feel the need to be in control. Due to the aforementioned effects of PPD and PPA on mothers' own parenting, a growing body of research has addressed how maternal PPD and, to a lesser extent, PPA, is related to fathers' involvement in parenting.

Defining Father Involvement

Father involvement is composed of three primary components: *engagement*, *availability*, and *responsibility* (Lamb, Pleck, Charnov, & Levine, 1985; Lamb, 1987). Engagement constitutes time directly spent interacting with and caring for the child. Availability is defined as time when the parent is present and could potentially interact with the child. Responsibility represents the parent's role in arranging for the care of the child and ensuring the child has needed resources, such as scheduling doctors' appointments and making sure the child has appropriate clothing (Lamb et al., 1985; Lamb, 1987). Father engagement is the component of involvement that is most consistently associated with children's later developmental outcomes (Sarkadi, Kristiansson, Oberklaid, & Bremberg, 2008). The current study focuses specifically on the engagement component of father involvement, and this is what is meant by the term "involvement" in the context of the present study.

Father involvement is typically measured either using diary data reporting hours spent engaged with the infant (Goodman et al., 2014; Jia et al., 2016), self-report measures using Likert scales to indicate how frequently fathers engage in tasks and activities with their infants (Cabrera et al., 2011; Lewin et al., 2014), or proportional measures indicating how often fathers perform child care tasks compared to how often

mothers perform these tasks resulting in an indicator of fathers' "share" of child care involvement (Adamsons, O'Brien, & Pasley, 2007; Barry, Smith, Deutsch, & Perry-Jenkins, 2011). Measures focusing on the amount of time spent involved with the infant or frequency of childcare tasks have some advantages in terms of reducing social acceptability bias (Jia et al., 2016). When looking at change in involvement over time, these methods also reflect changes in demand for involvement inherent in child development; thus a decrease in involvement using these measures may simply reflect a reduced need for certain childcare activities as the infant develops. A share of involvement measure is less susceptible to changing demands, making a share of involvement measure potentially more useful in models examining change in involvement as a dependent variable. Who reports father involvement is another important factor in measuring involvement. Mothers' reports of father involvement have been found to be biased by maternal depressive symptoms, making father reports more reliable when examining maternal depression and father involvement (Raskin, Fosee, & Easterbrooks, 2015).

Involvement is important to examine, particularly among fathers of infants, as greater father involvement early on has been linked to better psychological, behavioral, social, and cognitive outcomes for children (as reviewed in Sarkadi et al, 2008). Early involvement is also a predictor of fathers' parenting skill development (Barry et al., 2011), which, in turn, predicts fathers' greater involvement and skill over time (McHale & Huston, 1984). Father involvement is especially important to examine in the context of maternal postpartum mental health, as some studies have found that it may help buffer

negative effects of maternal postpartum depression on children's outcomes (Chang et al., 2007; Mezulis et al., 2004).

Competing Hypotheses for Father Involvement

As noted earlier, the literature has focused on two competing hypotheses for how father involvement is associated with maternal postpartum depression: the spillover hypothesis and the compensation hypothesis. Given that these two hypotheses predict opposite associations, it is surprising that both have received empirical support in the literature.

A number of studies have found evidence for a spillover effect, whereby fathers' parenting is impaired when mothers' have PPD (Paulson et al., 2006; Séjourné, Vaslot, Beaumé, Goutaudier, & Chabrol, 2012). One U.S. birth cohort study of 5,089 fathers found that fathers were less involved with their infants when their partners had higher levels of depressive symptoms at nine-months postpartum. In this study, fathers reported on their own levels of involvement (Paulson et al., 2006). Of note, this study found an effect of mothers' PPD symptoms when using a dichotomous clinical cutoff, but not when depression was measured as a continuous variable. Similarly, Paulson, Dauber, and Leiferman (2011) found concurrent associations at 9-months postpartum between maternal depression and lower levels of father involvement among non-residential fathers using combined mothers' and fathers' reports of father involvement. The only study to find spillover when examining maternal PPD prior to 6-months postpartum was a small French study using both parents' reports of father involvement and concurrent maternal depressive symptoms at 2-months. This study found support for a spillover hypothesis

whereby mothers' PPD was negatively related to mothers' reports of father involvement, but found no association when examining fathers' reports (Séjourné et al., 2012).

Other studies have found support for the compensation hypothesis, whereby fathers are more involved when mothers are depressed. One such study found that when mothers had depressive symptoms exceeding the clinical cutoff at 2-months postpartum fathers engaged in more positive involvement at 15-18 months postpartum (Edhborg, Lundh, Seimyr, & Widström, 2003), suggesting a long-term effect of depression in the early postpartum period. Similarly, a Brazilian study of low-income families found that when mothers' PPD symptoms exceeded the clinical cutoff at 3-months postpartum, fathers were more involved when children were 3 years old (de Mendonça, Bussab, Rodrigues, Siqueira, & Cossette, 2012). Goodman et al. (2014) assessed both involvement and availability, finding associations with involvement early on, and availability later in the postpartum year. The authors found a compensation effect when looking at maternal depressive symptoms in the first 6 months postpartum and fathers' time spent engaging with their infants, but found a spillover effect on fathers' availability for childcare, but not involvement, when looking at mothers' depressive symptoms from 7- to 12-months postpartum in a sample at high risk for PPD due to prior mental health history. A large birth cohort study found that mothers' higher self-reported depressive symptoms were concurrently related to greater levels of father involvement in childcare at 10-months postpartum (Cabrera et al., 2011). Planalp and Braungart-Reiker (2016) large nationally representative study found mothers' higher self-reported depressive symptoms were related to higher levels of father involvement with childcare at 9-months postpartum.

In a qualitative study, fathers most frequently reported compensating for their partners' depression by becoming more involved themselves and taking over responsibility for the functioning of the family (Beestin et al., 2014), a finding that is consistent with other qualitative studies (Barclay & Lupton, 1999; Boath, Pryce, & Cox, 1998; Meighan, Davis, Thomas, & Droppleman, 1999). It should be noted that all of the quantitative studies finding compensation relied on fathers' reports of their own time involved with their infants or third party observation of positive involvement rather than mothers' reports.

One reason for the mixed results when examining spillover and compensation hypotheses may be related to how and when depression and father involvement are measured. Goodman et al. (2014) found a spillover effect for fathers' availability – the amount of time when fathers are present to potentially provide childcare – when PPD occurred later in the postpartum period. Specifically, PPD from 7- to 12-months was related to less availability; however, a compensatory effect for involvement was found when PPD occurred in the early postpartum period. Though Goodman et al.'s spillover effect at 12-months postpartum was for availability, with no significant association for direct involvement with childcare, this is still a notable finding, given that fathers were spending less time at home, and thus seemed to be withdrawing from family life more when mothers were depressed between 7- and 12-months postpartum. The studies that have looked at concurrent or longitudinal associations of father involvement with mothers' depression prior to 6-months postpartum have, for the most part, found support for the compensation hypothesis (de Mendonça et al., 2012; Edhborg et al., 2003; Goodman et al., 2014). Most studies that have looked at mothers' postpartum depression

after 6 months have supported a spillover effect on father involvement (Paulson et al., 2006, 2011) or fathers' availability to be involved (Goodman et al., 2014). Thus, for the early postpartum period, existing research lends more support to the compensation hypothesis. For maternal depression occurring in the later postpartum period, the literature supports a spillover hypothesis.

Maternal Comorbid Anxiety with PPD and Father Involvement

None of the studies reviewed above examined or controlled for maternal postpartum anxiety symptoms. I found only one study that looked at the association between maternal PPA and father involvement. In a low-risk sample of highly educated dual-earner couples having their first child, an increase in mothers' anxiety symptoms from 3-months to 9-months postpartum was associated with a reduction in fathers' time involved in childcare activities independently of maternal depression (Jia et al., 2016). As previously noted, there is some evidence that mothers with anxiety may become overinvolved with their children, perhaps due to fear of harm coming to the child when the mother is not in control, which would be expected to hinder fathers from becoming more involved. Jia et al.'s (2016) findings lend support to this hypothesis. This study, the only one that has looked at both maternal postpartum anxiety and depressive symptoms as they relate to father involvement, did not look at the comorbidity or interaction between these two types of symptoms.

The question of what happens when mothers with PPD have significant comorbid PPA symptoms has not been addressed in the quantitative literature; however, one qualitative study of fathers whose wives experienced PPD noted a theme whereby fathers perceived that depressed mothers' anxious feelings often led them to undermine men's

involvement with infants, or engage in inhibitory gatekeeping behaviors (Beestin et al., 2014). Of importance in this study, fathers only described their wives preventing them from being involved when their wives were experiencing comorbid anxiety. These qualitative data lend support to the hypothesis that mothers with PPD may thwart fathers' efforts to be involved with childcare when mothers also have anxiety symptoms.

To my knowledge, no quantitative studies have examined mothers' comorbid postpartum anxiety and depressive symptoms as they relate to father involvement. Given the mixed findings concerning PPD and father involvement, and the limited data on PPA supporting a spillover hypothesis, the question of whether the combination of PPA and PPD is associated with father involvement differently from PPD alone merits study. Given the above theme found in the qualitative data, prior findings that the most common PPA symptoms are related to maternal over-involvement, and Jia et al.'s (2016) findings of a spillover effect of maternal PPA on father involvement, I expected comorbid PPD with PPA to be related to reduced father involvement with infant care.

Ecological Context: Social Class and Life Course

All family processes occur within a social context that extends beyond the interpersonal context of the family system. A broader view of context, taken from Bronfenbrenner's ecological perspective (Bronfenbrenner & Morris, 1998), suggests that both macro-level contextual factors, like social class, as well as more proximal contexts, such as family structure (e.g., dual-earner households), can shape the processes that occur within families. The particular context of the present study, dual-earner working-class couples transitioning to parenthood, has potential implications for how maternal

postpartum depression relates to father involvement. Many studies on the transition to parenthood overlook parents' work situations completely or simply statistically control for parents' work hours in their models (Cabrera et al., 2011; Jia et al., 2016; Paulson et al., 2006). Few address how parents' employment may be directly related to parental well-being and parenting or moderate the relationship between the two. In terms of social class, there has been literature looking at predominantly white, professional, married couples (Goodman, 2008; Goodman et al., 2014; Jia et al., 2016; Mezulis, Hyde, & Clark, 2004). Some have looked at lower-income samples with non-resident fathers (Paulson, Dauber, & Lierferman, 2011), and others have had more diverse married and cohabiting samples that were not exclusively working-class or lower-income (Cabrera et al., 2011) even going so far as to control for race or ethnicity and SES (Planalp et al., 2016). To my knowledge, no prior study has looked at the associations between maternal PPD and father involvement among a sample of low-income and working-class dual-earner married or cohabiting couples.

The experience of the transition to parenthood is likely different in some key ways for working-class families than it is for middle-class families; ways that could influence associations between maternal postpartum depression and father involvement. First, given the lack of an extended and paid leave policy for new parents in the U.S., low-wage families can rarely afford long parental leaves and most return to work within 3-months postpartum. For working-class mothers, maternity leave is often shorter than mothers would like due to a lack of paid parental leave benefits (Hegewisch & Gornick, 2011). Shorter maternity leave is related to increases in mothers' depressive symptoms following the return to work (Chatterji et al., 2013). In addition, mothers' return to paid

employment often pulls for more father involvement with the infant due simply to time availability. For working-class, dual-earner families, it is important to examine these associations over time during the postpartum years as families adjust to mothers' return to work.

Another characteristic of low-wage work is variable schedules and shift work. Most professional jobs have daytime work schedules while working-class jobs often require evening, overnight, and variable shifts, making it more likely that couples work opposite shifts. In fact, working-class couples may choose to work opposite shifts to reduce the need for external childcare (Presser, 1989). Thus, in shift work families, fathers are more involved in infant care due to structural constraints as opposed to internal family processes.

The timing of mothers' transition back to work may also shape father involvement, because upon their return to work mothers become less available for childcare. I expected associations between maternal PPD and father involvement to be stronger during the early postpartum period before mothers return to work. Whether couples work the same shift or different shifts from each other, and whether mothers return to work part-time or full-time are expected to be important influences on the association between maternal depression and father involvement in childcare after mothers return to work.

I also expected that the association between maternal PPD, comorbid PPA, and father involvement could vary depending on whether the couple is transitioning to parenthood for the first time or whether the couple are already parenting older children. Families are especially vulnerable during the transition to new parenthood. While it may

be a time of great joy for many families, the birth of a first child initiates a time of psychological strain as parents adjust to a new role with new responsibilities and a shift in focus from the couple relationship to the baby's needs (Huston & Homes, 2004). Research suggests that levels of father involvement established in infancy tend to be at least moderately stable over time, making patterns established at this stage likely to continue (Brown, 2009; Hwang & Lamb, 1997). It is of great import that the bulk of literature on associations between maternal PPD, PPA, and father involvement does not look specifically at the postpartum experiences of families having their first child (for an exception see Jia et al., 2016), so little is known about these associations at this particular life transition.

The Current Study

The current study aims to shed light on the concurrent and longitudinal associations between maternal PPD and father involvement with infant care. A second aim of the study is to test whether mothers' comorbid anxiety symptoms moderate the associations between PPD and father involvement. A third aim of the current study is to examine how work characteristics, specifically shift work, of working-class dual-earner parents having their first child, may shape the above associations.

This research is grounded in trends in the literature supporting compensation for early depression (Cabrera et al., 2011; de Mendonça et al., 2012) and spillover for later depression (Paulson et al., 2006, 2011). Building off of Goodman et al.'s (2014) study, which found support for a compensation hypothesis until 6-months postpartum, and spillover for father availability and maternal depression after 6 months, the current study

capitalizes on having multiple postpartum time points to test Goodman et al.'s explanation for this result. The authors proposed that fathers compensate for mothers' PPD early on, expecting that their wives will recover, but "give up" and become less involved when wives do not recover and fathers begin to see their wives' depression as a long-term state of affairs. They based this explanation on the fact that depressive symptoms were fairly stable over time in their study, and thus assumed that most mothers who were depressed in the 7-12 month period had been depressed in the early postpartum period as well. The current study tests this explanation by examining not only clinical levels of maternal depression, meaning depression scores that meet or exceed the clinical cutoff of 16 on the Center for Epidemiologic Studies Depression scale (CES-D), at 1-month postpartum as a predictor of concurrent father involvement and change in father involvement over the postpartum year, but also by examining how initial clinical levels of PPD interacts with change in PPD symptoms whether mothers' depressive symptoms increase or decrease, to predict father involvement in two different ways. This longitudinal question is addressed first by examining whether initial clinical levels of maternal depression at 1-month postpartum interact with change in maternal depressive symptoms, meaning change in the sum score of depressive symptoms on the CES-D, from 4- to 12-months postpartum to predict change in father involvement from 1- to 12-months postpartum. The second set of analyses directly test whether late onset of clinical levels of PPD has different implications for involvement compared to mothers who are continually depressed. Thus, analyses examine whether initial clinical levels of depression interact with clinical levels of depression at 4-months and at 12-months postpartum to predict levels of father involvement at 12-months postpartum.

Research Question 1: Are mothers' clinical levels of depressive symptoms at 1-month postpartum (PP) associated with concurrent levels of father involvement with infant care? (Figure 2)

Hypothesis 1: Consistent with prior research, a compensation effect is hypothesized for maternal depression at 1-month postpartum, such that mothers' high levels of depressive symptoms will be related to fathers' greater concurrent involvement.

Research Question 1a: Do mothers' clinical levels of depressive symptoms at 1-month postpartum interact with change in their depressive symptoms from 4- to 12-months postpartum to predict change in father involvement from 1- to 12-months postpartum? (Figure 3).

Hypothesis 1a: Consistent with Goodman's explanation, change in mothers' depressive symptoms is expected to moderate the association between mothers' 1-month postpartum depressive symptoms and change in father involvement from 1- to 12-months postpartum, such that fathers will stay involved when mothers have initial depression in the clinical range that decreases over time and become less involved when mothers have initial depression in the clinical range that stays the same or increases over time. In other words, compensation is expected if mothers are initially depressed but recover, whereas spillover is expected when mothers are initially depressed and stay depressed. When mothers were not depressed initially but become increasingly depressed, compensation is expected as fathers may still view mothers' depression as a temporary state if it has a later onset.

Research Questions 1b: Do mothers' clinical levels of PPD symptoms at 1-month postpartum interact with mothers' clinical levels of PPD symptoms at 4-months

postpartum to predict changes in father involvement and levels of said involvement at 12-months postpartum? While Question 1a examined change in depressive symptoms as a moderator, Question 1b addresses the issue of later onset PPD more directly by using clinical cutoffs rather than change in depressive symptoms measured continuously (Figure 4).

Hypotheses 1b: Clinical levels of PPD at 4-months postpartum will moderate the association between clinical PPD at 1-month and fathers' involvement at 12-months postpartum, such that presence of later PPD will be related to higher involvement and an increase in involvement when mothers did not have initial PPD, and lower involvement and a decrease in involvement when mothers did have initial PPD.

Research Question 1c: Do mothers' clinical levels of PPD symptoms at 1-month postpartum interact with mothers' clinical levels of PPD symptoms at 12-months postpartum to predict father involvement at 12-months postpartum? As in Question 1b, this question addresses the issue of later onset PPD by using clinical cutoffs rather than change in depressive symptoms measured continuously (Figure 5).

Hypotheses 1c: Clinical levels of PPD at 12-months postpartum will moderate the association between clinical PPD at 1-month and fathers' involvement at 12-months postpartum, such that presence of later PPD will be related to higher involvement and increasing involvement when mothers did not have initial PPD, and lower involvement and decreasing involvement when mothers did have initial PPD.

Research Question 2: How do clinical levels of maternal depressive symptoms at 1-month postpartum interact with clinical levels of comorbid anxiety symptoms at 1-month postpartum to predict concurrent father involvement? (Figure 6)

Hypothesis 2: Given that mothers with the most common form of postpartum anxiety symptoms, typically characterized by worries about the child's well-being, have been shown to be overinvolved, in contrast with the more withdrawn parenting of mothers with postpartum depression, it is hypothesized that mothers' anxiety symptoms will moderate the associations between maternal PPD symptoms at 1-month postpartum and father involvement, such that when mothers have comorbid anxiety symptoms, fathers will be less involved in childcare tasks, and fathers will be more involved when mothers have depressive symptoms only.

Research Question 2a: Do initial clinical levels of PPD and PPA interact with later clinical levels of PPD and PPA to predict father involvement at 12-months postpartum? This exploratory question proposes a 4-way interaction between initial and later clinically significant PPD and PPA symptoms. This question will be tested for later PPD and PPA at 4-months and 12-months postpartum (Figure 7).

Hypothesis 2a: This is an exploratory question to determine whether different combinations of early and later PPD and PPA are related to later father involvement. While no literature has looked at these possible interactions, I do have some tentative hypotheses based on prior literature. These hypotheses are summarized in Table 1.

- When mothers have initial comorbid PPD and PPA and still have significant PPA later, father involvement at 12-months will be low, as mothers will continue to feel the need to be in control of caring for the baby.
- When mothers have initial PPD only and continue to have only PPD later, father involvement will be lower, as these fathers will begin to see their partner's PPD as a more permanent state.

- When mothers have initial PPD only, but no longer have it later, father involvement will be higher. These fathers will have developed self-efficacy early on by being more involved during their partners' early depression, and it is known that more skilled fathers stay more involved (Barry et al., 2011; McHale & Huston, 1984)
- When mothers have initial PPA alone and later PPA alone, fathers will continue to be less involved, as mothers will still feel the need to be in charge.
- When mothers have neither PPD nor PPA initially, but develop PPA or comorbid PPD and PPA, fathers will be less involved, as these mothers will have developed the need to be in control.
- When mothers have neither PPD nor PPA initially but develop PPD alone later, fathers will be more involved. This is expected because fathers will view their partners' PPD as a temporary state if it had a recent onset and will, therefore, compensate.

Research Questions 3a, 3b, and 3c: Are associations found in the previous questions (1a, 1b, and 1c) moderated by couples' work shifts? This question capitalizes on the ecological context of this unique sample by examining a characteristic commonly found in working-class jobs: shift work. Couples working opposite shifts may do so in order to avoid having to use non-parental childcare. These parents' involvement with caring for their baby would, out of necessity, be shaped by work schedules more than any psychological or relational factors. Shift work will not be tested as a moderator for concurrent associations between maternal PPD at 1-month postpartum and father involvement, as this time point occurred before mothers' had returned to work, and thus fathers would not have had times when they were the sole available caretaker for the

baby during this time point. This moderation question will be tested for the models for Questions 1a and 1b.

Hypotheses 3a, 3b, and 3c: The hypothesized associations from questions 1a, 1b, and 1c will be weaker for families in which parents work opposite shifts from each other and stronger when parents work overlapping shifts, since opposite shifts require fathers to be involved regardless of mothers' mental health.

CHAPTER 2

RESEARCH DESIGN AND METHOD

Participants

Participants for this study are part of two waves of a larger longitudinal investigation examining the transition to parenthood among working-class parents. Data collection for Wave 1 began in 1996 and was completed in 2006 and Wave 2 took place from 2003 to 2009. Parents were recruited from prenatal classes at hospitals in the New England area during their third trimester of pregnancy. For the first wave, couples were included who met the following criteria: (a) both members of the couple were employed full-time (35 hours per week or more); (b) both members of the couple planned to resume working full-time within 6 months of the baby's birth; (c) both members of the couple were "working class" as defined by educational attainment of a two-year associates degree or less; (d) both members of the couple were expecting their first child; and (e) the couple was married or cohabiting for at least one year prior to participation in the study. Participants in Wave 2 comprised single, cohabiting, and married mothers and babies' biological fathers. Parents in this wave were also recruited from prenatal classes at hospitals in New England during their third trimester of pregnancy. In order to qualify for participation in Wave 2, mothers had to meet the following criteria: (a) worked full time prior to the birth of the baby, (b) intended to return to full-time employment within six months of giving birth, (c) deemed "working class", as defined by the restriction on educational level at no higher than an Associate's Degree, and employment in unskilled or semiskilled jobs.

For the current study, which looks at families with residential fathers having their first child, 147 participants from Wave 1 who remained in the study after the birth of their baby were included, but only 35 participants from Wave 2 who remained in the study and also met the Wave 1 criteria were included, making for a sample of 182 dual-earner, working-class cohabiting and married parents, who did not live with other adults, having their first child.

Design

The current study uses a longitudinal design with three time-points. Participants were interviewed in their homes by trained graduate students at 1-month, approximately 4-months (1-month after mothers returned to work), and 12-months postpartum, and completed a mail survey at 6-months postpartum.

Measures

Maternal Depressive Symptoms

The current study considers both continuous levels of depressive symptoms and clinical depression. Most research on maternal PPD and father involvement uses only continuous measures of depressive symptoms (Cabrera et al., 2011; Goodman et al., 2014; Jia et al., 2016), because research has demonstrated that even subclinical levels of these symptoms may affect the family. Less research has been done on clinical levels of maternal depression as it relates to father involvement, but Cabrera et al. (2011) used both a continuous and a dichotomous clinical cutoff measure of maternal depression, and only found associations with father involvement in enrichment activities for the clinical

cutoff. Given that there is a certain amount of stress and fatigue that is normative in the early postpartum period, it is possible that findings will differ when only examining clinical levels of depressive symptoms, as some of the normative experience of having a newborn may be captured as sub-clinical levels of depressive symptoms.

Depressive symptoms for participants were assessed at 1-, 4-, and 12-months postpartum using the Center for Epidemiological Studies-Depression Scale (CES-D) developed by the National Institute of Mental Health (Radloff, 1977). The 20-item scale indicated the frequency at which participants experienced certain thoughts and emotions in the past week. Respondents rated these items on a four-point scale from 0 (*rarely or none of the time/less than once a day*) to 3 (*most or all of the time/5-7 days*). The items included statements like “I thought my life has been a failure”, “I felt lonely”, and “I felt everything was an effort.” Higher scores indicated greater symptomatology. Participants completed this scale at all three time points. Hypotheses concerning levels of mothers’ depressive symptoms were tested using the clinical cutoff score of 16 or higher for this measure, which is considered to indicate clinically significant depressive symptoms. Hypotheses concerning change in depressive symptoms use the scale as a linear variable.

Maternal Anxiety Symptoms

Anxiety symptoms were measured at 1-, 4-, and 12-months postpartum using the state anxiety subscale of Spielberger’s (1972) State-Trait Anxiety Inventory (STAI). On a 4-point intensity scale ranging from 1 (*not at all*) to 4 (*very much so*), respondents rated the extent to which 20 statements (e.g., “I worry too much over something that really doesn’t matter; I am content; I feel nervous and restless”) represented their current feelings at the time of interview. A high score on this measure indicates greater

symptomatology. Hypotheses concerning comorbid anxiety symptoms use the STAI as a continuous variable, since I was interested in comorbid anxiety symptoms, and not necessarily an anxiety diagnosis.

Father Involvement

The research on father involvement and maternal PPD has used a wide range of measures and conceptualizations of involvement. Several studies have examined these day-to-day tasks as a measure of involvement (Cabrera et al., 2011; Jia et al., 2016), whereas others have exclusively focused on enrichment activities (Paulson et al., 2006), such as playing with, singing to, and reading to the baby, or did not differentiate between these types of activities (Goodman et al., 2014). The focus of this research is on instrumental childcare tasks for two reasons. First, the measure involves fathers' reports of how often they perform each task in relation to how often the mother performs each task, resulting in a proportion. Such reports should be more accurate for instrumental tasks than for enrichment activities, because spouses are likely to be less aware of how often their partner engages in enrichment activities, such as singing or reading to the baby, when the family is not all together. Parents can more accurately imagine how many times their spouses change diapers, feed the baby, and put the baby to bed when they are not there to witness these activities, because they know how often these activities must take place. A second reason for focusing on instrumental childcare tasks is that they are expected to be more influenced by maternal depression, due to the emotional stress of trying to soothe and physically manage a distressed, fussy baby in order to complete frequently required routine tasks. Instrumental childcare tasks may also be more influenced by maternal comorbid anxiety due to the fact that they are related to the health

and safety of the child, which are of particular concern to mothers with anxiety symptoms. While this measure of involvement addresses the proportion of childcare, rather than actual hours spent involved in childcare, this approach is advantageous in this longitudinal study that examines change in involvement, as the proportion of tasks is not affected by changes in childcare demands that occur from the very labor intensive newborn stage through 12-months postpartum.

This research uses fathers' reports of their own involvement with instrumental childcare tasks, such as changing diapers and bathing the baby, due to findings in prior research demonstrating that mothers' reports of father involvement are biased by mothers' depressive symptoms (Raskin et al., 2015).

Fathers' reports of their involvement in routine childcare tasks were assessed at 1-month, 4-months, and 12-months using a scale developed by Barnett and Baruch (1987). Contribution to the performance of 11 instrumental childcare tasks was assessed using a 5-point scale from 1 [*usually or always my spouse (0%-20% personal contribution)*] to 5 [*usually or always myself (80%-100% personal contribution)*]. Tasks on the scale were bathing, feeding, diaper-changing, soothing, dressing the baby, picking up after the baby, getting up with the baby at night, putting the baby to sleep, planning the baby's activities, taking the baby to doctor's appointments, and caring for the baby when he or she is sick. The mean of these 11 items was calculated with higher values indicating greater levels of father involvement.

Couple Work Hours and Shift

Given that parental involvement in childcare must be affected by the hours they work outside the home, when those hours occur, and whether they occur at the same time

for both parents, parents' work hours and work shifts are important to consider in this research. Mothers and fathers reported their work schedule for a typical week at 4- and 12-months postpartum. A dichotomous *shift* variable was created to indicate whether the couple worked alternating shifts from each other (1) or the same shift (0). Mothers' and fathers' weekly *work hours* were calculated from the same questions at 1-month postpartum for fathers, and at 4-months and 12-months postpartum for both parents.

Covariates

Several additional variables are included in this research as potential covariates due to their known influences on father involvement. Child *gender* was reported by mothers at 1-month postpartum and was coded as girl (0) and boy (1). Child gender is included, as some research has demonstrated that fathers are more involved with sons than with daughters (NICHD Early Child Care Research Network, 2000). *Negative infant temperament* was assessed using the 94-item Infant Behavior Questionnaire (IBQ) at 1-month postpartum (Rothbart, 1981). Both parents rated the frequency of particular infant behaviors in the last week, using a 7-point scale ranging from 1 = *never* to 7 = *always*. The scores for the subscales measuring infant distress (distress to limitation, distress and latency to approach intense or novel stimuli) were averaged, and then parents' reports were averaged together resulting in a negative temperament score that accounts for both mothers' and fathers' perceptions. Infant temperament is included as a covariate due to research demonstrating that fathers may be less involved when infants have negative temperaments (Mehall, Spinrad, Eisenberg, & Gaertner, 2009). *Fathers' depressive symptoms* were measured at 1-, 4-, and 12-months postpartum using the same scale described above for mothers. Depressive symptom scores at 1-month postpartum are used

in analyses looking at father involvement at 1-month postpartum, and fathers' depressive symptom scores at 12-months postpartum are used in analyses looking at change in father involvement across the postpartum year and levels of involvement at 12-months postpartum. Given that fathers' postpartum depressive symptoms have been associated with both mothers' depressive symptoms (Goodman, 2008; Paulson & Bazemore, 2010) and father involvement (Jia et al., 2016; Lyons-Ruth et al., 2002), it is important to control for fathers' own depression when examining associations between mothers' PPD and father involvement. *Fathers' anxiety symptoms* were measured at 1-, 4-, and 12-months postpartum using the same scale described above for mothers. Anxiety symptom scores at 1-month postpartum are used in analyses looking at father involvement at 1-month postpartum, and fathers' anxiety symptom scores at 12-months postpartum are used in analyses looking at change in father involvement across the postpartum year and levels of involvement at 12-months postpartum. As with depression, fathers' greater anxiety has been linked with less involvement with childcare (Jia et al., 2016), making it important to control for when examining associations between mothers' PPD and father involvement. Mothers' and fathers' *age* are also included as covariates as parents' age has been found to be negatively related to father involvement with infants (Feldman, Nash, & Aschenbrenner, 1983), and studies have linked maternal age with postpartum depression in multiple ways (McMahon et al., 2011; Mirowsky & Ross, 2002; Muraca & Joseph, 2014).

Analytic Plan

As excluding cases with missing data can reduce statistical power and bias parameter estimates, multiple imputation was used for missing values (Allison, 2002). Ten imputed data files were generated in SPSS and were used for analyses in SPSS and Mplus to maintain consistency in sample size across analyses. Hypothesis 1, 1c, 2, 2a, and 3c, which look at levels but not change in father involvement, were tested using multiple regression analysis with the multiply imputed data in SPSS. Simple slopes were tested for significant interactions to determine the conditional effects of the predictors on father involvement (Aiken & West, 1991; Dawson, 2014), by calculating T-values from the slopes and their standard errors at different levels of the moderator, per Aiken and West (1991).

Multilevel modeling was used to test Hypotheses 1a and 1b in Mplus (Muthén & Muthén, 1998-2012). MLM offers a robust way to model individual change over time (Raudenbush & Bryk, 2002). This approach allows for simultaneous estimates of levels of father involvement at 12-months postpartum and change in father involvement across 1-, 4-, and 12-months postpartum. Before testing my hypotheses, I fit a baseline model for level and change in father involvement to determine whether there was significant variability in fathers' level of involvement 12-months postpartum (the intercept) and in change in their involvement from 1- to 12-months postpartum (the linear slope).

Establishing between-person variability in both level and change is necessary in order to have a rationale for adding individual-level predictors to explain why fathers may differ on these constructs of interest (Raudenbush & Bryk, 2002). The second step was to test which covariates are related to levels and change in father involvement. The potential

covariates (parents' age, child gender, infant temperament, fathers' depressive and anxiety symptoms, and parents' work hours) were entered as a block in the equations for level and change in father involvement. The control model was trimmed to include only the significant covariates to conserve power and the following models were built on that trimmed covariate model. Of note, neither infant temperament nor child gender were significant predictors of father involvement in any model, so they were omitted in the control model and subsequent models. Predictors were then added, and nonsignificant predictors trimmed from the model, starting with the equation for the slope, as multilevel models do not allow predictors for the slope that are not included in the equation for the intercept. Moderator analyses included simple slopes coefficients calculated in Mplus based on Hayes' PROCESS methodology for testing conditional effects (Hayes, 2013; Stride, Gardner, Catley, & Thomas, 2015). Multigroup multilevel modeling (Asparouhov & Muthén, 2012) was used to test Hypotheses 3a and 3b, comparing couples who work the same shift with those who work opposite shifts on the interactions between early and later depressive symptoms put forth in Questions 1a and 1b.

CHAPTER 3

RESULTS

Descriptive Statistics

Descriptive statistics for participants' demographic characteristics and covariates can be found in Table 2. Participants were primarily married (70.3%) and 29.7 percent cohabiting. Just over half of babies were girls. Mothers (Mean age = 26.54) were slightly younger than fathers (Mean age = 28.41 years) on average. The sample was predominantly White (85.7% of mothers and 81.3% of fathers), with smaller proportions of Latino (6% of mothers and 9.3% of fathers) and African-American (5.5% of mothers and 5.5% of fathers) parents. Most mothers (53.8 %) and a smaller majority of fathers (50%) had some college or vocational training, and 26.4% of mothers and 12.1% of fathers held Associates degrees. Family income at 12-months postpartum was a median of \$52,500 before taxes, with fathers working a median of 45 hours and mothers working a median of 40 hours at 12-months postpartum.

Descriptive statistics were computed for all study variables and can be found in Table 3. Fathers typically reported performing less than half of instrumental childcare tasks at all three phases, with involvement increasing slightly at each phase. Mothers' depressive symptoms and anxiety symptoms averaged below the clinical cutoffs at all time points. Mothers' depressive symptoms met the clinical cutoff among 26.5% of participants at 1-month postpartum, 30.2% at 4-months postpartum, and 26.4% at 12-months postpartum. Overall, 47.8% of mothers had depressive symptoms that met the clinical cutoff at one or more of these time points, and 36.8% of mothers had anxiety

symptoms that met the clinical cutoff for anxiety at one or more of these time points. Compared to other studies, mothers reported clinical levels of depression and anxiety symptoms at higher rates than in the general population, but at similar levels found in studies looking at low-income populations (Early Head Start Research and Evaluation Project, 2006; Miller, 2002). In terms of comorbidity, 14.4% of mothers met the clinical cutoff for both depressive and anxiety symptoms at 1-month, 23.7% at 4-months, and 19.0% at 12-months postpartum. Just over a third (36.5%) of couples worked opposite shifts at 12-months postpartum.

Inferential Analyses

Timing and Duration of Depression

Question 1: As a point of clarification, I will refer to depression clinical cutoffs as clinical levels of depressive symptoms, and in analyses that look at change in sum scores I will use the term change in depressive symptoms. Hierarchical regression models supported my hypothesis that clinical levels of maternal depressive symptoms would be associated with higher levels of concurrent father involvement at 1-month postpartum (See Figure 2 for hypothesized model). As seen in Table 4, when mothers had clinical levels of depressive symptoms, fathers reported being more involved with childcare ($B = 0.172$, $SE = 0.071$, $p = .016$). In addition, fathers with clinical levels of depressive symptoms and older fathers reported being less involved in childcare. The distribution for fathers' depressive symptoms had low variability, so the clinical cutoff was used instead. No other controls were significant, so they were trimmed from the model. The effect size (change in R^2) for mothers' depression was small, explaining an estimated 3 percent of

the variance in fathers' involvement on average across the 10 imputed data sets.

Temperament and child gender were not significant covariates in analyses for any of the research question, and were thus omitted from this and all of the following models.

Question 1a: A multilevel SEM model (Table A1) was fit in Mplus to test Hypothesis 1a, that presence of maternal clinical levels of PPD at 1-month will be related to a decline in father involvement from 1- to 12-months postpartum if mothers' PPD symptoms increase or are maintained from 4- to 12-months postpartum, but will be stable if mothers' PPD symptoms decrease (See Figure 3 for hypothesized model). Latent growth curves were simultaneously fit for changes in father involvement and changes in maternal depressive symptoms. The growth curve for father involvement was positive ($B = 0.156$, $SE = 0.036$, $p < .001$) with marginally significant variance (variance = 0.054, $SE = 0.030$, $p = .074$). The growth curve for maternal depression was not significantly different from zero ($B = -0.002$, $SE = 0.056$, $p = .967$), and its variance did not approach significance (variance = 0.027, $SE = 0.081$, $p = .328$), so it could not be used as a predictor to test Hypothesis 1a.

Question 1b: A multilevel model was fit in Mplus to test Hypothesis 1b, that presence of maternal clinical levels of PPD at 1-month will be related to lower subsequent levels of father involvement at 12-months postpartum and a decline in father involvement from 1- to 12-months postpartum if mothers maintain clinical levels of PPD at 4-months postpartum, but will be higher and steady or increasing if mothers no longer have clinical levels of PPD at 4-months postpartum (See Figure 4 for hypothesized model). As seen in Table 5, the final trimmed model revealed a significant interaction predicting fathers' levels of involvement ($B = -2.917$, $SE = 1.241$, $p = .019$). Simple

slopes for the association between mothers' depression at 1-month postpartum and father involvement at 12-month postpartum were tested for sub-clinical and clinical levels of depressive symptoms at 4-months postpartum as seen in Figure 8. Mothers' 1-month postpartum depression was positively related to 12-months father involvement when mothers did not have clinical levels of depressive symptoms at 4-months postpartum (solid line, $B = 2.420$, $SE = 0.808$, $p = .003$), and was not significantly related to father involvement when mothers had clinical levels of depressive symptoms at 4-months postpartum (dotted line, $B = -0.497$, $SE = 0.961$, $p > .50$). In addition, when mothers were not depressed at 1-month PP, mothers' depression at 4-months postpartum was positively related to 12-months father involvement ($B = 1.886$, $SE = 0.773$, $p = .015$), but mothers' depression at 4-months postpartum was not significantly related to father involvement when mothers did have clinical levels of depression at 1-month postpartum ($B = -1.031$, $SE = 1.013$, $p = .309$). In other words, fathers compensated for mothers' depression by doing more childcare when mothers were depressed at either 1-month or 4-months postpartum, compared to when mothers were never depressed. Fathers performed the least childcare when mothers did not meet the clinical cutoff for depression at either time point. Thus, as seen in Figure 8, fathers compensated with more involvement if mothers were depressed at one time point, but less so if they remained depressed at two time points. In addition, mothers' age, fathers' anxiety, and fathers' work hours were negatively related to fathers' levels of involvement, whereas mothers' work hours were positively related to fathers' levels of involvement. As seen in Table 5, fathers with greater anxiety symptoms and those working more hours experienced less of an increase in involvement across the postpartum year. I calculated the proportion of variance

explained, often called a pseudo- R^2 , to obtain an effect size; this showed that addition of the interaction in the final trimmed model explained an additional 5.8% of variance in fathers' levels of involvement when compared to the control only model, suggesting a small to medium effect size of early and later depression and their interaction.

Question 1c: A hierarchical linear regression was performed in SPSS to test the hypothesis that the presence of clinical levels of maternal depression at 1-month postpartum would predict greater father involvement at 12-months postpartum if mothers no longer have clinical levels of PPD at 12-months postpartum, and lower father involvement at 12-months postpartum if mothers still had clinical levels of depressive symptoms at 12-months postpartum (See Figure 5 for hypothesized model, and Table A2 for coefficients). Significant covariates were entered as control variables. Analyses did not support Hypothesis 1c, as no significant interaction was found ($B = -0.252$, $SE = 0.177$, $p = .153$).

Comorbid Anxiety

Questions 2: Hierarchical regression analyses were used to test Hypothesis 2 (See Figure 6 for hypothesized model), which was that when mothers had clinical levels of PPD at 1-month postpartum, fathers would have more concurrent involvement if mothers had low anxiety symptoms and less involvement if mothers had high anxiety symptoms (Table 6). Results supported this hypothesis, with a significant interaction between depression and anxiety symptoms ($B = -0.018$, $SE = 0.009$, $p = .047$). As seen in Figure 9, clinical levels of maternal depression were related to greater father involvement when mothers had low levels of anxiety, and less father involvement when mothers had high

anxiety, whereas anxiety had little relation to father involvement when mothers' depression was below the clinical cutoff. Simple slopes analysis revealed a significant negative association between mothers' anxiety symptoms and father involvement when mothers had clinical levels of depression (Figure 9 dotted line, $B = -0.167$, $t = -24.744$, $p < .001$). There was a small but significant positive association between anxiety symptoms and father involvement when mothers did not have clinical levels of depressive symptoms (Figure 9 solid line, $B = .013$, $t = 2.291$, $p = .023$). In addition, fathers' clinical levels of depression and greater age were related to lower father involvement in childcare. The effect size (change in R^2) for the interaction was small, explaining an estimated 2.2 percent of the variance in fathers' involvement on average across the 10 imputed data sets.

Question 2a: Hierarchical regression was used to address Hypotheses 2a (see Figure 7 for hypothesized model and Tables A3 – A6 for coefficients), which was that maternal initial PPD would be related to subsequent father involvement differently depending on mothers' subsequent depression and initial and subsequent comorbid anxiety symptoms (a 4-way interaction). Given that comorbid anxiety in Q2 was a better predictor when used as a sum score, rather than clinical cutoff, I proceeded to use hierarchical regression analysis to test a 4-way interaction using depression clinical cutoffs and anxiety sum scores. Results found no significant interaction. Given the power requirements of a 4-way interaction, I proceeded to run four 3-way interaction models, looking at Time 1 and Time 2 clinical levels of depression with comorbid anxiety symptom scores first at Time 1 then at Time 2, and repeated this with Time 1 and Time 3 depression with comorbid anxiety at Time 1 and then Time 3. These models resulted in

no significant findings, thus hypothesis 2a was not supported.

Couple Work Shift

Question 3a: I fit a multigroup multilevel model in Mplus to address Hypothesis 3a, which was that the interaction between initial PPD at 1-month postpartum and change in maternal depressive symptoms from 4- to 12-months postpartum predicting levels of father involvement at 12-months postpartum and change in father involvement (as described in Hypothesis 1a) would be stronger for couples who work the same shift than for those who work opposite shifts. The fit of an SEM model in which parameters are constrained to be the same for the two groups was compared to a model in which the parameters are allowed to differ to determine whether the associations in question differ depending on couple shift. As with Question 1a, there was no significant variability for changes in depression for either same shift or opposite shift couples, so I did not proceed to fit models using change in depression as a moderator. Thus, hypothesis 3a was not supported (Table A7).

Question 3b: Multigroup multilevel modeling in Mplus was also used to address Hypothesis 3b, which was that the moderated effects of clinical levels of PPD at 1-month and 4-months postpartum on levels of father involvement at 12-months postpartum and change in involvement (described in Hypothesis 1b) would be stronger for same shift couples than for opposite couples. Similarly to Question 1b, mothers' depression did not significantly predict the slope for change in father involvement for either group, but was a significant predictor for levels of father involvement at 12-months PP.

The model supported different associations for same- versus opposite-shift couples, but in the opposite direction from what I expected. The final trimmed model omitted the nonsignificant predictors, in this case the depression predictors from the equation for change in involvement, and can be seen in Table 7. This model found a significant interaction between early and later depressive symptoms for opposite-shift couples ($B = -5.532$, $SE = 1.640$, $p = .001$), but no interaction for same-shift couples ($B = -0.100$, $SE = 1.868$, $p = .957$). A Wald Test of Parameter Constraints comparing the interaction coefficients between the groups found a significant difference ($Wald\ value = 4.689$, $df = 1$, $p = .03$), suggesting that the interaction occurs for opposite-shift couples only. The interaction, depicted in Figure 10, suggests a positive association between Time 1 clinical levels of depression and father involvement when mothers are not depressed at Time 2, and a negative relationship when mother are depressed at Time 2. The interaction suggests that fathers are most involved when mothers are depressed early on and then recover, followed by when mothers are depressed for the first time later on, and are the least involved when mothers are never depressed. Follow-up analyses comparing simple slopes and group differences showed that fathers whose partners were depressed at one time point were significantly more involved than those whose partners were never depressed, but the difference between father involvement when mothers were depressed at both time points was not significant compared to other groups. Maternal clinical levels of depressive symptoms at 4-months postpartum was marginally positively related to father involvement when mothers did not have clinical levels of depression at 1-month postpartum ($B = 1.833$, $SE = 1.088$, $p = .092$), whereas mothers depressive symptoms at 4-months postpartum was negatively related to father involvement at 12-

months postpartum when mothers had clinical levels of depression at 1-month postpartum ($B = -3.698$, $SE = 1.249$, $p = .003$). The interaction is depicted in Figure 10.

In addition, fathers' levels of anxiety symptoms was negatively associated with father involvement for same-shift, but not opposite-shift couples, and fathers' greater work hours were marginally related to less involvement for same-shift but not opposite-shift fathers. Greater maternal age and fathers' work hours were significantly related, and fathers' anxiety marginally related, to less of an increase in father involvement across the postpartum year for same-shift, but not opposite-shift couples.

Question 3c: Hierarchical regression was also used to address Hypothesis 3c, which was that the moderated effects of PPD at 1-month and 12-months postpartum on levels of father involvement at 12-months postpartum and change in involvement would be stronger for same shift couples than for opposite couples. As in Question 1c, no significant interaction between maternal PPD at 1-month and 12-months in predicting father involvement at 12-months postpartum was found (Table A8).

CHAPTER 4

DISCUSSION

While research on postpartum mental health has focused primarily on how father involvement influences mothers' depressive symptoms, the current project examined the reverse direction of effects, namely how maternal depression predicts father involvement. There is a bidirectional relationship between the two that is important in understanding how parents cope with the arrival of a new baby. Given that father involvement buffers children from the negative effects of mothers' PPD, and is linked to a better prognosis for mothers' postpartum depression (Ross, Evans, Sellers, & Romach, 2003), it is important to understand what factors might enable fathers to compensate, and what might lead to a spillover effect and fathers' withdrawing from parenting. The aim of the present study was to test competing hypotheses in the literature. Some research points to a spillover effect of mothers' PPD on father involvement, whereby mothers' depression predicts less father involvement. In contrast, other findings support a compensation effect, whereby fathers become more involved when mothers are depressed (e.g. Cabrera et al., 2011; Goodman, et al., 2014; Jia et al., 2016; Lewin et al., 2014; Paulson et al., 2006, 2011; Séjourné et al., 2012). Several findings from this study support a compensation hypothesis, whereas others support a spillover hypothesis. Specifically, timing and duration of maternal PPD, as well as the presence of comorbid anxiety symptoms, are important factors influencing whether fathers compensate for maternal PPD with greater involvement.

Spillover vs. Compensation

Supporting the compensation hypothesis, I found that fathers performed more concurrent childcare at 1-month postpartum when mothers had clinical levels of depression (Question 1). This finding for the early postpartum year fits with prior literature, which has typically supported compensation when measures were taken early in the postpartum year (de Mendonça et al., 2012; Edhborg et al., 2003; Goodman et al., 2014), and spillover later in the postpartum year (Goodman et al., 2014; Paulson et al., 2006, 2011). Fathers also compensated for mothers' clinical levels of depression with higher levels of subsequent involvement in childcare at 12-months postpartum when mothers were depressed at either 1-month or 4-months postpartum, but not if they were depressed at both phases (Question 1b). Compared to families where mothers were never depressed, fathers were more involved with childcare in families where mothers were depressed at one time point, but not when they were depressed at two. When I looked at the role of comorbid anxiety (Question 2), I found that fathers compensated when mothers had clinical levels of depression and low levels of anxiety symptoms, but not when they had higher levels of anxiety.

In support of the spillover hypothesis, I found that fathers performed less concurrent childcare when mothers had clinical levels of depression and higher levels of comorbid anxiety symptoms. Though no prior literature has examined comorbidity, this finding does fit with prior research support for a spillover effect of maternal postpartum anxiety itself on father involvement (Beestin, Hugh-Jones, & Gough, 2014; Jia et al., 2016). Given the high level of comorbid anxiety symptoms among mothers with postpartum depression, this is an important yet little examined factor in the literature.

This finding suggests that different presentations of depression may pull for different responses from partners in terms of childcare involvement, and that both research and clinical practice needs to account for comorbidity of PPD and PPA.

Timing and Duration of Depression

My findings concerning the timing and duration of maternal postpartum depression, partially fit with a proposed explanation by Goodman and colleagues' (2014) for spillover later in the postpartum year, which is that the research was capturing fathers whose spouses had been depressed for longer. I found a compensation effect when mothers were depressed at one time point, but compensation no longer occurred if mothers were depression at two time points. I did not find a true spillover effect when mothers remained depressed, however, because fathers did not perform significantly less childcare when mothers remained depressed than when mothers were never depressed. Thus fathers were no longer compensating if mothers remained depressed, but they did not become so uninvolved as to produce a spillover effect. It is clear, however, that fathers did not compensate further if mothers remained depressed, and did not perform significantly more childcare than when mothers were never depressed.

Findings concerning the timing and duration of clinical levels of depression have implications for both research and practice. Given the mixed findings in the literature on father involvement and maternal depression, depending on when during the postpartum year they were measured, more longitudinal research on these associations during the perinatal period is needed. In this study, there was an interaction between maternal depression at 1- and 4-months postpartum predicting father involvement at 12-months

postpartum, but maternal depression at 12-months postpartum did not result in such an interaction, adding to the complexity of looking at timing and duration.

No significant findings emerged linking mothers' postpartum depression and change in father involvement over time. While this could be attributed to methodological limitations, further discussed below, this lack of finding could also be due to father involvement being largely established early on and stable over time (Brown, 2009; Hwang & Lamb, 1997). In addition, given that the second time point was one month after mothers had returned to work, it is possible that most of the change in father involvement would have occurred already at that point, with couples already having adjusted to having both parents working, with little room for continued change at the third time point. The use of dual-earner couples also limits the expected variability in change in fathers' involvement, as childcare involvement would be expected to become more equal when mothers return to work, resulting in the majority of fathers changing their involvement in similar ways to each other.

Comorbidity

Placing the comorbidity finding in the context of Coyne's theory on the interpersonal nature of depression, which would point to a spillover hypothesis, the question arises as to whether different presentations of depression might pull for different responses from partners. Coyne's theory would posit that depressed mothers' express high levels of negative affectivity and seek frequent reassurance from their partners, and thus drive their partners away. This study's findings bring up a question as to whether the reassurance-seeking behaviors might be driven, in part, by anxiety and, without the

activating effect of anxiety, partners may not pull away in the same manner. One can imagine a mother with the feelings of sadness, hopelessness, guilt, lack of pleasure, trouble getting out of bed, and lack of motivation might invite a very different response from her partner than a mother whose depression takes on a form that includes anxiety, which typically is accompanied by insomnia, rumination, constant worry, and resultant reassurance-seeking behaviors (Nolen-Hoeksema, 2000). These findings have important implications for both research and practice. In terms of research, these findings suggest that it is important for studies to examine comorbidity, because it is not only common, but may alter the effects of depression on others. Given that depression and anxiety are highly comorbid, only looking at one or the other may not capture what occurs for a significant segment of depressed mothers.

The comorbidity results fit with Jia and colleagues' (2016) findings that maternal anxiety was related to lower levels of father involvement, although that study did not examine comorbid anxiety and depression. Findings by Planalp and Braungart-Reiker (2016) provide a different context for understanding the comorbidity results. Planalp and Braungart-Reiker found an interaction between maternal depression and marital conflict, such that fathers compensated for mothers' depression when conflict was low, but depression had no effect on father involvement when conflict was high. Given that anxiety has been associated with marital conflict (McCormick, Hsueh, Merrilees, Chou, & Mark Cummings, 2017; Whisman, 2007), I performed exploratory analyses to see if anxiety and marital conflict were correlated in my study and found a significant correlation, where higher levels of marital conflict at 12-months-postpartum was related to higher levels of maternal anxiety at 1-month ($r = .385, p < .001$), 4-months ($r = .377, p$

< .001), and 12-months postpartum ($r = .551, p < .001$). One future direction I intend to investigate is whether and how marital conflict is involved in linking comorbid maternal depression and anxiety to father involvement. Marital conflict could be a potential mediator of the association, with maternal comorbid anxiety and depression causing greater marital conflict, or marital conflict itself could be causal factor in mothers' comorbid depression and anxiety symptoms. Whisman, Davila, and Goodman (2011) explored similar associations with relationship distress, rather than conflict specifically, among couples during the perinatal period. They found relationship distress preceded anxiety, whereas depression preceded relationship distress, so looking at comorbid depression and anxiety would add greater complexity to these associations. One can imagine mothers with high conflict relationships experiencing greater distress and less support when they experience postpartum depressive symptoms, which could increase their anxiety. On the other hand, it is possible that the presentation of postpartum depression with comorbid anxiety could lead to marital conflict about parenting if mothers engage in gatekeeping behaviors. These possibilities merit further exploration in future work.

The findings for comorbid anxiety have a number of practical implications for postpartum health and mental healthcare. There has been considerable movement in the medical field toward screening for perinatal depression, but perinatal anxiety has garnered less attention. Most medical providers use tools like the Edinburgh Perinatal Depression Scale, which are designed to screen for perinatal depression, but do not address anxiety. Given that the absence or presence of comorbid anxiety symptoms could

be related to how fathers are able to support mothers and babies by being more involved, it is important to assess for comorbid anxiety symptoms.

Interventions for mothers with depression and comorbid anxiety symptoms could prioritize addressing the barriers to father involvement, such as gatekeeping behaviors or low confidence in fathers' childcare skills, so that fathers can increase their involvement and serve a protective role for children and mothers. Fathers' greater involvement could also allow depressed mothers with anxiety more time to rest and recover in the weeks following birth, which could potentially help them recover sooner. Researchers have hypothesized that greater maternal gatekeeping on the part of anxious mothers could prevent fathers from being more involved in caring for their infants (Jia et al., 2016). If depressed mothers with comorbid anxiety do engage in more gatekeeping, then prevention and intervention efforts could attempt to address gatekeeping preemptively by increasing father childcare skills and knowledge in childbirth education classes, for example. A mother who knows her partner has learned about caring for newborns from reputable sources may feel less of a need to do all of the infant care herself. In addition, making couples aware that mothers' anxiety may make it harder for fathers to be involved may also serve an inoculating function, allowing couples to recognize maternal postpartum anxiety when it happens, helping fathers recognize mothers' gatekeeping as a result of anxiety rather than as an insult to fathers' parenting, and allowing couples to get help sooner.

Couple Work Shift

When I examined how couples' work shifts might influence how fathers respond

to mothers' postpartum depression, I was surprised to find that the findings on the interaction between earlier and later depression were in fact driven by the opposite-shift families rather than the same-shift as expected. I had hypothesized that if couples worked opposite shifts, much of their time spent in childcare would occur when one spouse was at work, leaving little flexibility to adapt to needs presented by maternal depression. The data showed instead that same-shift fathers' childcare involvement was not related to mothers' depression, but opposite-shift fathers' was. The interaction between maternal depression at 1-month postpartum and 4-months postpartum to predict fathers' involvement at 12-months postpartum (Question 1b) was stronger for the opposite-shift families, and was not significant for the same-shift families.

As these findings were the opposite of what I expected, I completed some exploratory analyses to compare same-shift and opposite-shift families on other variables that could explain this group difference. I examined variables related to gender role ideology, which would explain a difference in father involvement based on their beliefs about men's and women's roles in the family. Given that opposite-shift couples rely on both parents to have some amount of solo childcare time, I expected these parents to have more egalitarian gender role ideology. I also looked at economic contributions to the family, since mothers' greater proportion of income has been linked to greater father involvement, and many opposite-shift couples had mothers working shifts with a higher wage. I also examined factors related directly to childcare including gatekeeping, self childcare skills, and partner childcare skills. I expected gatekeeping to be lower and father childcare skills would be higher in the opposite-shift group since parents have no choice but to have fathers doing some solo childcare that would build their parenting

skills early on. In addition, I looked at marital quality, which has been related to childcare involvement, given that the amount of time couples have together could influence their marriage. I performed a series of T-tests to compare the two groups. These exploratory analyses found that opposite-shift couples had more egalitarian gender role ideology in that they reported that the mothers' role of worker and economic provider was more important than same-shift couples did, and felt that their division of housework and childcare was fairer. These couples also reported engaging in significantly more relationship maintenance behaviors than same-shift couples.

These exploratory analyses suggest that opposite-shift families in this study valued women's employment, and perhaps relied on mothers' wages, more than same-shift families did. Research on the division of family labor has found that men perform more housework and childcare when their wives' earnings are higher (Fetterolf & Rudman, 2014) and when those earnings are deemed necessary for the family in part because if a man views making money as his main job for the family and keeping the household running as primarily his spouse's job, he may see his partner's needed financial contribution as a way to help him, and may be more likely to feel that it's fair for him to contribute to housework and childcare in exchange (Deutsch, 1999; Shows & Gerstel, 2009). It is possible then that, opposite-shift families' greater value placed on mothers' economic provider role made these fathers more likely to view performing childcare as part of their role in supporting their partner when their partner is in need. Opposite-shift fathers also have more time at home alone with their babies, so these fathers may also get more experience with childcare earlier on in the postpartum year. This early experience could make them feel more comfortable with performing more

childcare in response to their partners' needs. In terms of the marital relationship, couples who engage in more relationship maintenance behaviors, such as communicating about their needs and checking in on the others' day, may be more attuned to each others' needs (Ogolsky & Bowers, 2013), hence it is possible that opposite-shift fathers are more aware of how mothers are struggling and are therefore more responsive to their partners' depression.

Limitations

One important limitation of this study is the limited variability for change in maternal depression. Given that my research question addressed the interaction of clinical levels of maternal depressive symptoms at 1-month postpartum with subsequent change in depressive symptoms, available time points for measuring change were limited. In addition, prior research has found a curvilinear change in depression in the postpartum year (Perry-Jenkins, Smith, Wadsworth, & Halpern, 2017), which I did not have the power to capture in this model using change in depression as a moderator.

The lack of findings for change in father involvement points to another limitation of this study. As mentioned above, this lack of variability could be the result of the inclusion criteria for this study, with mothers returning to work, one might expect fathers' involvement in this sample to change in similar ways to each other. In addition the childcare involvement scale assessing the proportion of childcare tasks performed by fathers in relation to mothers may have also limited variability. A scale that allowed for more nuanced responses, such as one indicating the percentage of the task, might allow

for more variability. Diary data could also allow for more variability and a more precise measure of involvement.

Though this study has the advantage of longitudinal data, causal inference is limited. The associations between maternal PPD and father involvement are known to be bidirectional, so reverse causation must be considered. This study's findings concerning timing and duration of maternal PPD make a good case of fathers responding to PPD with their involvement, as the literature on father involvement as a predictor of maternal PPD would have predicted associations in the opposite direction of these findings. Fathers in this study were more involved in childcare when mothers had clinical levels of depression at one time point or another, whereas if father involvement were affecting maternal PPD, one would expect mothers to become more depressed if fathers were less involved. The analyses concerning comorbidity were cross-sectional, however, and could plausibly be explained by mothers being more distressed, including developing anxiety symptoms, when fathers are less involved. Further longitudinal exploration of these associations would serve to strengthen the case of causality.

Additional methodological limitations of this study include the depression measure and the statistical power of these analyses. The use of clinical cutoffs for depression, though more robust than using it as a linear scale, poses another limitation for this study. The CES-D can be used as a screener for depressive disorders, but a score above the clinical cutoff does not constitute a diagnosis. The limited sample size and limited number of time points limit the power of this study. Some of the analyses, particularly the multigroup multilevel models and the 3-way interactions, had sub-

optimal power, stretching beyond the recommended number of parameters per participant. The risk for Type II error was, therefore, higher for these analyses.

Future Directions

The literature has examined maternal postpartum depression as both influenced by and an influence on father involvement, making it clear that this is a bidirectional association. In the present study I have looked at the effects of maternal postpartum depression on father involvement, while controlling for fathers' own mental health, but a more complex time-lag model could account for the bidirectional association between these factors. In addition, we know mothers' and fathers' mental health are interrelated and, while I have included fathers' depression and anxiety as covariates in this study, a larger data set with more time points could examine the interrelations between parents' mental health and father involvement to capture these processes in greater detail.

My findings concerning comorbid anxiety symptoms bring up a number of future directions to explore. To test gatekeeping as an explanation for anxiety, a moderated-mediation model could investigate whether the presence of higher levels of gatekeeping is a mechanism for the association between comorbid depression and anxiety and father involvement. Two potential models are conceivable when considering how relationship conflict could be involved in these associations. A first-stage moderated mediation model could be tested looking at relationship conflict as a mediator predicted by the interaction of depression and comorbid anxiety, with comorbid anxiety predicting greater conflict and, thereby, less father involvement. Additionally, a second-stage moderated mediation

is possible, whereby relationship conflict predicts anxiety symptoms, which interact with postpartum depression to predict father involvement.

Finally, there are several additional factors that may be considered when looking at maternal postpartum mental health and father involvement. First, mothers who have C-sections typically have partners who are more involved early on (Parke, 1981) because they have a longer physical recovery period. Having a C-section is also a risk factor for maternal depression (Xu, Ding, Ma, Xin, & Zhang, 2017), so future research could look at C-section status as a covariate or, a path model examining whether maternal depressive symptoms partially mediate links between C-sections and father involvement. Mothers who breastfeed longer have a lower risk for postpartum depression (Chowdhury et al., 2015). Breastfeeding has also been linked to lower father involvement in infant care in terms of feeding the baby and other childcare tasks (Beitel & Parke, 1998), so it is possible that women who breastfeed for longer have fewer depressive symptoms and partners who are less involved. Future research should examine breastfeeding as a potential link between maternal postpartum depressive symptoms and father involvement to explore these associations.

Conclusion

This study has highlighted key factors that can influence whether fathers' respond to maternal PPD by either stepping up and taking on more childcare tasks, or stepping back and becoming less involved in caring for infants. Researchers have posited compensation and spillover hypotheses as to how maternal depression is related to father involvement, and my findings suggest that both hypotheses are supported, depending on

the course and comorbidity of maternal depression. My findings suggest that fathers will step up and compensate for mothers' PPD, but only for so long, and that they may not do so if mothers have comorbid anxiety symptoms. In addition, for working-class parents, whether or not couples work the same or opposite shifts may influence how fathers respond to maternal PPD. Overall, this study has identified three moderating factors, the course of depression, comorbid anxiety, and couple work-shift, that future research should account for when studying links between maternal PPD and father involvement. The conditions that can alter how fathers respond to maternal PPD point to how mothers' personal characteristics influence fathers' parenting, which in turn can either protect infants or leave them more vulnerable. Prevention and interventions efforts for PPD need to enable fathers to support their partners and children through stepping up and being more involved in childcare.

Table 1.
Hypothesized associations with father involvement at 12-months postpartum

		Late PPD		No Late PPD	
		Late PPA	No Late PPA	Late PPA	No Late PPA
1-mo PPD	1-mo PPA	Low	?	Low	?
	No1-month PPA	?	Low	?	Higher
No1-month PPD	1-mo PPA	?	?	Low	?
	No1-month PPA	Low	Higher	Low	?

Table 2.
Descriptive statistics and frequencies for demographics

Variable	<i>N</i>	<i>Mean</i>	<i>SD</i>	<i>Min</i>	<i>Max</i>
Mother Age	182	26.54	4.95	17.65	40.81
Father Age	182	28.41	5.05	18.61	41.27
Family Income (in thousands) T1	181	\$53.41	\$18.52	\$11.00	\$138.93
Father work hours T1	176	45.03	11.72	0.00	71.50
Father work hours T2	161	45.72	12.13	0	72
Father work hours T3	162	44.22	14.34	0	84
Mother work hours T2	171	33.42	13.03	0	68
Mother work hours T3	169	32.82	15.97	0	83
	<i>N</i>	<i>Percent</i>			
Baby's sex					
Girl	95	52.2			
Boy	87	47.8			
Marital status					
Cohabiting	54	29.7			
Married	128	70.3			
Mother race/ethnicity					
Latino	11	6.0			
White	156	85.7			
African-American	10	5.5			
Other	5	2.7			
Father race/ethnicity					
Latino	17	9.3			
White	148	81.3			
African-American	10	5.5			
Other	7	3.8			
Mother Education					
Less than High School	5	2.7			
High School Graduate	31	17.0			
Some college/Vocational	98	53.8			
Associates' Degree	48	26.4			
Father Education					
Less than High School	5	2.7			
High School Graduate	64	35.2			
Some college/Vocational	91	50.0			
Associates Degree	22	12.1			

Note. T1 = 1-month postpartum, T2, = 4-months-postpartum, T3 = 12-months postpartum.

Table 3.
Descriptive statistics for study variables

Variable	N	Mean	SD	Min	Max
Father Instrumental Childcare T1	176	2.33	0.42	1.00	3.18
Father Instrumental Childcare T2	163	2.45	0.46	1.18	4.18
Father Instrumental Childcare T3	159	2.53	0.48	1.00	3.64
Mothers Depression Sum T1	181	12.11	8.47	0	51
Mothers Depression Sum T2	169	12.02	9.25	0	43
Mothers Depression Sum T3	163	11.97	9.19	0	48
Mothers Anxiety Sum T1	181	33.52	8.89	20	63
Mothers Anxiety Sum T2	170	34.78	9.74	20	71
Mothers Anxiety Sum T3	164	34.91	8.95	21	64
Father Depression Sum T1	177	9.30	7.21	0	46
Father Depression Sum T3	159	8.70	7.98	0	50
Father Anxiety Sum T1	176	31.89	7.73	20	67
Father Anxiety Sum T3	159	31.83	8.40	20	71
Infant Negative Temperament T1	181	3.32	0.70	1.71	5.13

	N	Percent
Mother clinical depressive symptoms T1		
Above clinical cutoff	48	26.5
Below clinical cutoff	133	73.5
Mother clinical depressive symptoms T2		
Above clinical cutoff	51	30.2
Below clinical cutoff	118	69.8
Mother clinical depressive symptoms T3		
Above clinical cutoff	43	26.4
Below clinical cutoff	120	73.6
Father clinical depressive symptoms T1		
Above clinical cutoff	25	14.1
Below clinical cutoff	152	85.9
Couple Shift T3		
Same Shift	106	63.5
Opposite Shift	61	36.5

Note. T1 = 1-month postpartum, T2, = 4-months-postpartum, T3 = 12-months postpartum. Instrumental childcare was measured on a scale of 1 (Father performs 0-20% of childcare) to 5 (father performs 80-100% of childcare). The depression measure had possible scores of 0-60, Anxiety had a potential scale of 20-80, and infant negative temperament had a potential scale of 1-7. The clinical cutoff for depressive symptoms was 16.

Table 4.
 Hierarchical regression of father involvement at 1-month postpartum on mothers' clinical levels of depression at 1-month postpartum

	Trimmed Control Model	Main Effect Model
	<i>B</i> (<i>SE</i>)	<i>B</i> (<i>SE</i>)
Intercept	2.741** (0.189)	2.738** (0.187)
T1 Father Clinical Depression	-0.189* (0.094)	-0.234* (0.094)
T1 Father Age	-0.014* (0.006)	-0.015* (0.006)
T1 Mother Clinical Depression		0.172* (0.071)
<i>R</i> ²	.043	.074
<i>F</i> for change in <i>R</i> ²	4.024*	6.065*

Note. SPSS does not pool *R*² statistics, so the *R*² and *F* for change in *R*² reflect the mean values across the 10 imputations. T1 = 1-month postpartum, Mother and Father clinical depression coded as 1 = above clinical cutoff, 0 = below clinical cutoff. Control variables that were not significantly related to father involvement and were thus trimmed from the final control model include infant temperament, infant gender, fathers' work hours, fathers' anxiety symptoms, and mothers' age.

* *p* < .05, ** *p* < .001

Table 5.

Multilevel SEM model for the interaction of T1 and T2 clinical depression predicting levels and change in father involvement multiplied by 10

	Baseline	Control	Main Effects	Interaction	Trimmed
	<i>B(SE)</i>	<i>B(SE)</i>	<i>B(SE)</i>	<i>B(SE)</i>	<i>B(SE)</i>
Level Fixed effects					
Intercept	25.201** (0.384)	25.189**(0.353)	24.635** (0.493)	24.435** (0.507)	24.352** (0.436)
M Age		-0.210** (0.072)	-0.209** (0.071)	-0.215* (0.071)	-0.215* (0.071)
F Anxiety T3		-0.131** (0.042)	-0.141** (0.041)	-0.147** (0.041)	-0.147** (0.041)
M Wrk Hrs T3		0.076**(0.024)	0.082** (0.024)	0.084** (0.024)	0.084** (0.024)
F Wrk Hrs T3		-0.058** (0.022)	-0.053* (0.023)	-0.050* (0.023)	-0.049* (0.022)
M Clinical Dep T1			0.671 (0.845)	1.642 (1.053)	2.420** (0.808)
M Clinical Dep T2			1.228 (0.870)	2.048* (1.007)	1.886* (0.773)
T1×T2 Clinical Dep				-2.350 (1.536)	-2.917* (1.241)
Change Fixed Effects					
Intercept	0.156**(0.036)	0.156** (0.035)	0.162** (0.047)	0.169** (0.049)	0.156** (0.035)
M Age		-0.008 (0.007)	-0.008 (0.007)	-0.008 (0.007)	-0.008 (0.007)
F Anxiety T3		-0.010* (0.004)	-0.010* (0.004)	-0.010* (0.004)	-0.010** (0.004)
M Wrk Hrs T3		0.003 [†] (0.002)	0.003 (0.002)	0.003 (0.002)	0.003 (0.002)
F Wrk Hrs T3		-0.006* (0.002)	-0.006* (0.002)	-0.006* (0.002)	-0.006* (0.002)
M Clinical Dep T1			-0.088 (0.078)	-0.118 (0.096)	
M Clinical Dep T2			0.055 (0.080)	0.026 (0.091)	
T1×T2 Clinical Dep				0.085 (0.153)	
Random Effects					
Tau00	15.514**	11.464**	10.860**	10.632**	10.794**
Tau10	0.055 [†]	0.037	0.034	0.034	0.037
Sigma Sq	8.954**	8.947**	8.964**	8.960**	8.944**
Model Fit					
Deviance	8962.614	8922.856	8912.156	8906.128	8908.924
# Parameters	20	28	32	34	31
χ^2 for Δ		39.758**	10.700*	6.028*	13.932**
df		8	4	2	3
AIC	9002.614	8978.857	8976.156	8974.129	8970.923

Note. Father involvement scores were multiplied by 10 for analyses involving change in involvement, in order to obtain enough meaningful digits in the estimates. M = mother, F = father. Age, work hours, and fathers' anxiety symptoms were grand-mean centered. Deviance comparison found the final trimmed model was a significant improvement in fit over the Control model.

** $p < .001$, * $p < .05$, [†] $p < .10$

Table 6.
Hierarchical regression of father involvement on concurrent maternal depression and anxiety symptoms.

Variable	Model 1	Model 2: T1xT2 Main Effects	Model 3: T1xT2 Interaction
	<i>B(SE)</i>	<i>B(SE)</i>	<i>B(SE)</i>
Intercept	2.741** (0.189)	2.769** (0.187)	2.789** (0.187)
F T1 Clinical Dep	-0.189* (0.094)	-0.249** (0.094)	-0.245** (0.093)
F Age	-0.014* (0.006)	-0.016* (0.006)	-0.016* (0.006)
M T1 Clinical Dep		0.112 (0.090)	0.183 [†] (0.097)
M T1 Anxiety Symptoms		0.005 (0.005)	0.013* (0.006)
M T1 Dep x Anx			-0.018* (0.009)
<i>R</i> ²	.046	.082	.104
<i>F</i> for change in <i>R</i> ²	4.024*	3.720*	4.344*

Note. SPSS does not pool *R*² statistics, so the *R*² and *F* for change in *R*² reflect the mean values across the 10 imputations. F = father, M = mother. T1 = 1-month postpartum, T2 = 4-months-postpartum, T3 = 12-months postpartum. Dep = Depression.

** *p* < .001, * *p* < .05, [†] *p* < .10

Table 7.

Multigroup multilevel SEM model for same-shift and opposite-shift couples and the interaction of T1 and T2 clinical depression predicting levels and change in father involvement multiplied by 10

	Constrained		Free	
	Same-Shift	Opposite-Shift	Same-Shift	Opposite-Shift
Level Fixed effects	<i>B(SE)</i>	<i>B(SE)</i>	<i>B(SE)</i>	<i>B(SE)</i>
Intercept	24.407** (0.426)	24.407** (0.426)	23.824** (0.514)	25.444** (0.638)
M Age	-0.261** (0.071)	-0.261** (0.071)	-0.236** (0.090)	-0.274* (0.117)
F Anxiety T3	-0.134** (0.042)	-0.134** (0.042)	-0.165** (0.050)	-0.106 (0.068)
M Wrk Hrs T3	0.090** (0.023)	0.090** (0.023)	0.079** (0.027)	0.102** (0.039)
F Wrk Hrs T3	-0.036 (0.024)	-0.036 (0.024)	-0.047 [†] (0.026)	-0.050 (0.062)
M Clinical Dep T1	2.742** (0.857)	2.742** (0.857)	0.229 (1.205)	4.213** (1.026)
M Clinical Dep T2	1.938* (0.775)	1.938* (0.775)	1.741 [†] (0.974)	1.833 [†] (1.088)
T1×T2 Clinical Dep	-3.406* (1.322)	-3.406* (1.322)	-0.100 (1.868)	-5.532** (1.640)
Change Fixed Effects				
Intercept	0.157** (0.032)	0.157** (0.032)	0.135** (0.036)	0.206** (0.056)
M Age	-0.012 [†] (0.006)	-0.012 [†] (0.006)	-0.018* (0.008)	0.000 (0.012)
F Anxiety T3	-0.009* (0.004)	-0.009* (0.004)	-0.008 [†] (0.005)	-0.009 (0.007)
M Wrk Hrs T3	0.004 [†] (0.002)	0.004 [†] (0.002)	0.004 (0.002)	0.003 (0.004)
F Wrk Hrs T3	-0.005* (0.002)	-0.005* (0.002)	-0.004* (0.002)	-0.006 (0.007)
Random Effects				
Tau00	12.754**	7.331**	10.533**	7.087*
Tau10	0.026	0.025	0.021	0.031
Sigma Sq	7.195**	11.689**	7.316**	11.369**
Model Fit				
Deviance	8120.550		8095.360	
# Parameters	50		62	
χ^2 for Δ			25.19	
df			12	
p-value			.014	
AIC	8220.550		8219.360	

Note. Father involvement scores were multiplied by 10 for analyses involving change in involvement, in order to obtain enough meaningful digits in the estimates. As a first step, a baseline growth model was first tested to ensure measurement invariance. The fit of an SEM model in which parameters are constrained to be the same for the two groups was compared to a model in which the parameters are allowed to differ to determine whether the associations in question differ depending on couple shift. The deviance comparison showed no significant improvement in fit when parameters were unconstrained, allowing me to proceed with fitting predictors to the model. A comparison of individual paths was used to determine how associations between maternal mental health variables and father involvement differ, depending on whether couples work the same or opposite shifts. A full model was tested and then nonsignificant predictors were trimmed for a more parsimonious model. A comparison of deviance for the fully constrained and unconstrained models using the chi square test found a significant improvement in fit when coefficients for same shift couples were allowed to differ from those of opposite shift couples.

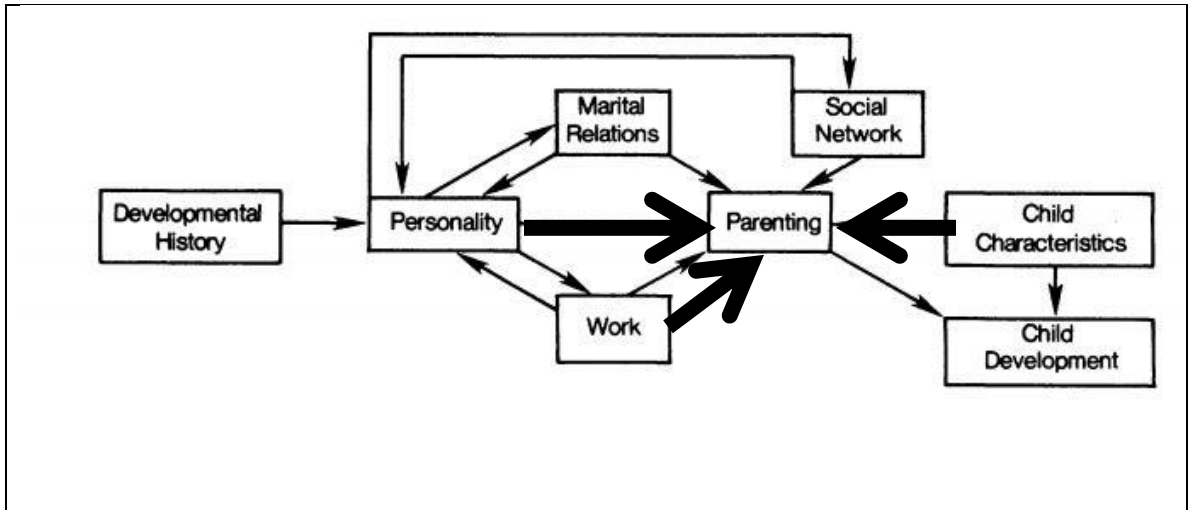


Figure 1. Belsky's (1984) Process Model of Parenting. Paths under investigation in the proposed research are highlighted with large arrows.

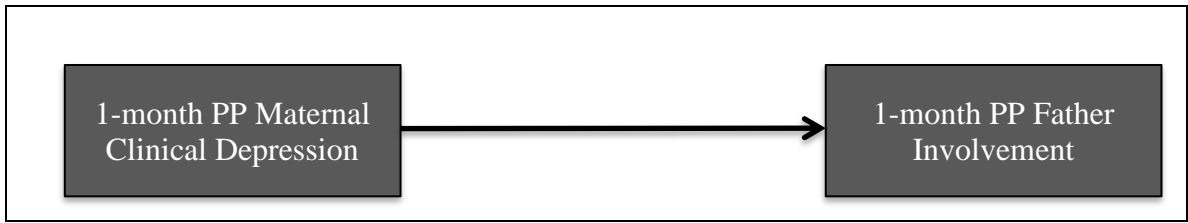


Figure 2. Model for Question 1.

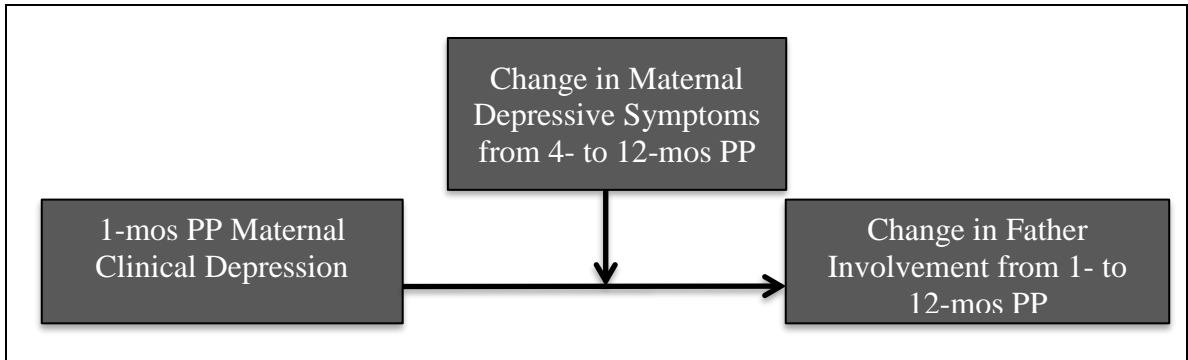


Figure 3. Model for Question 1a.

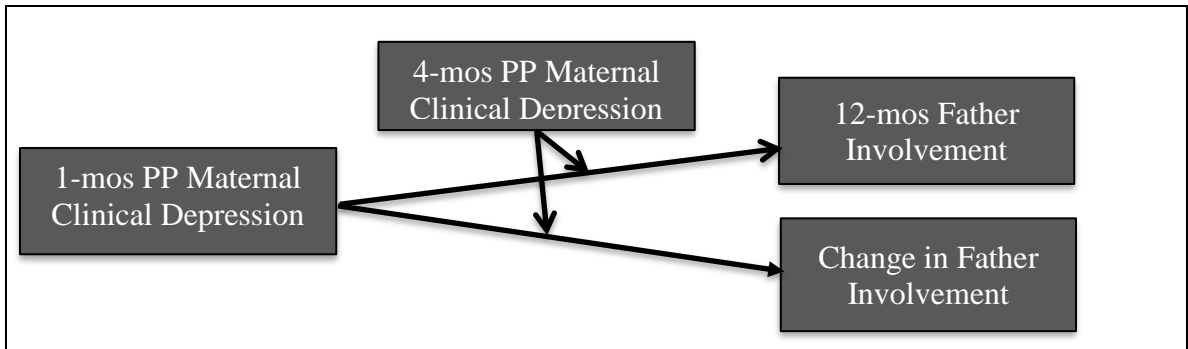


Figure 4. Model for Question 1b.

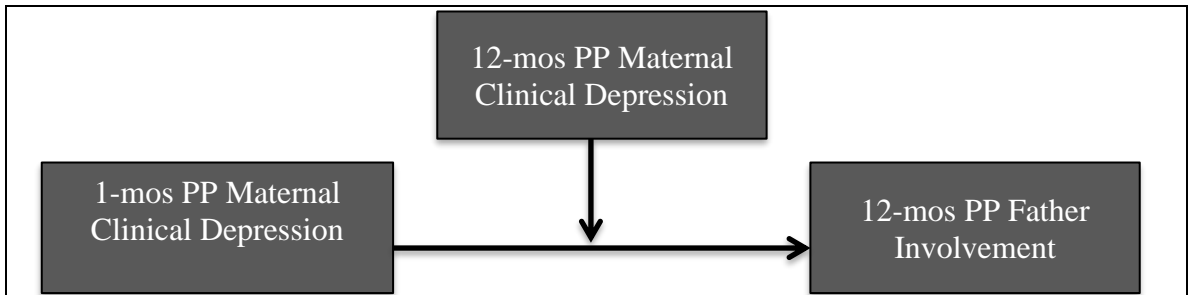


Figure 5. Model for Question 1c.

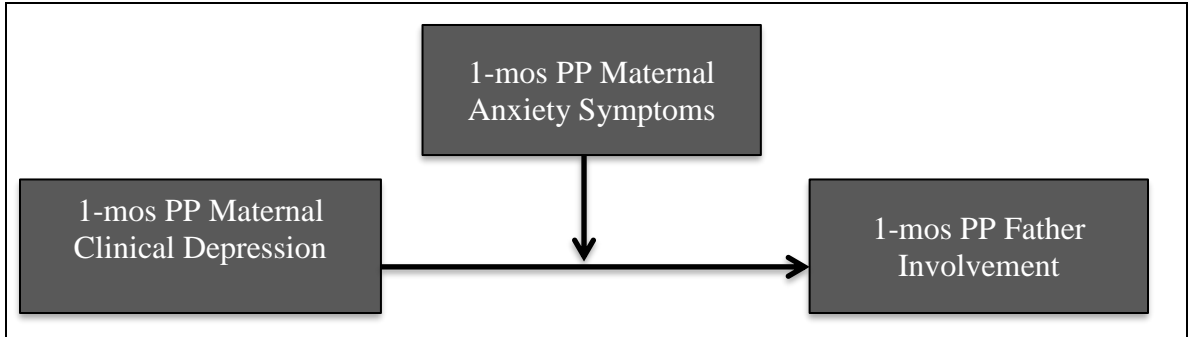


Figure 6. Model for Question 2.

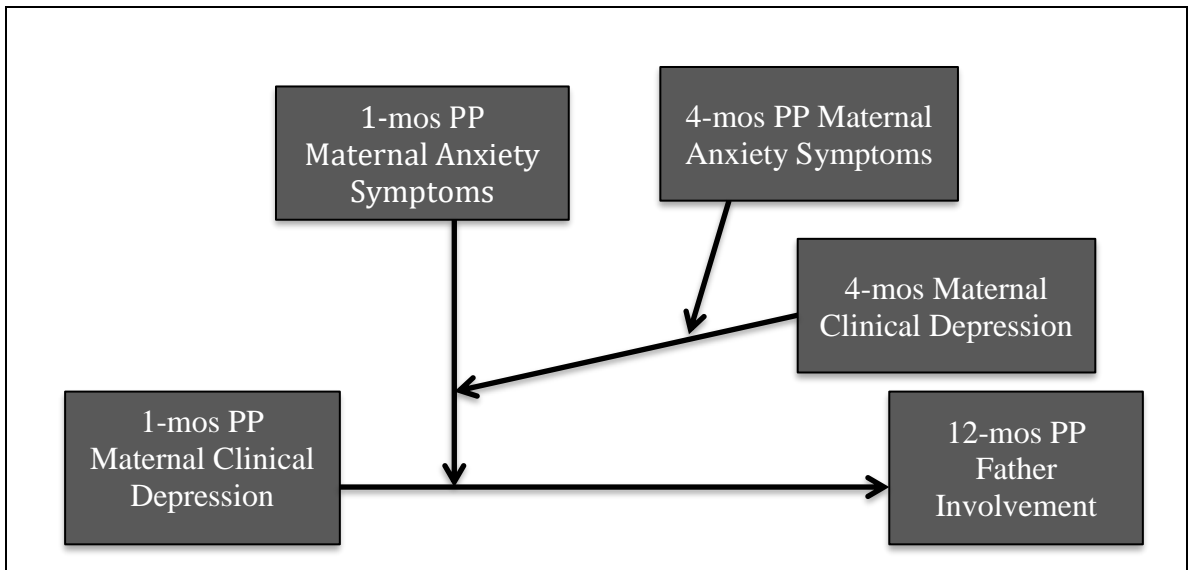


Figure 7. Model for Question 2a.

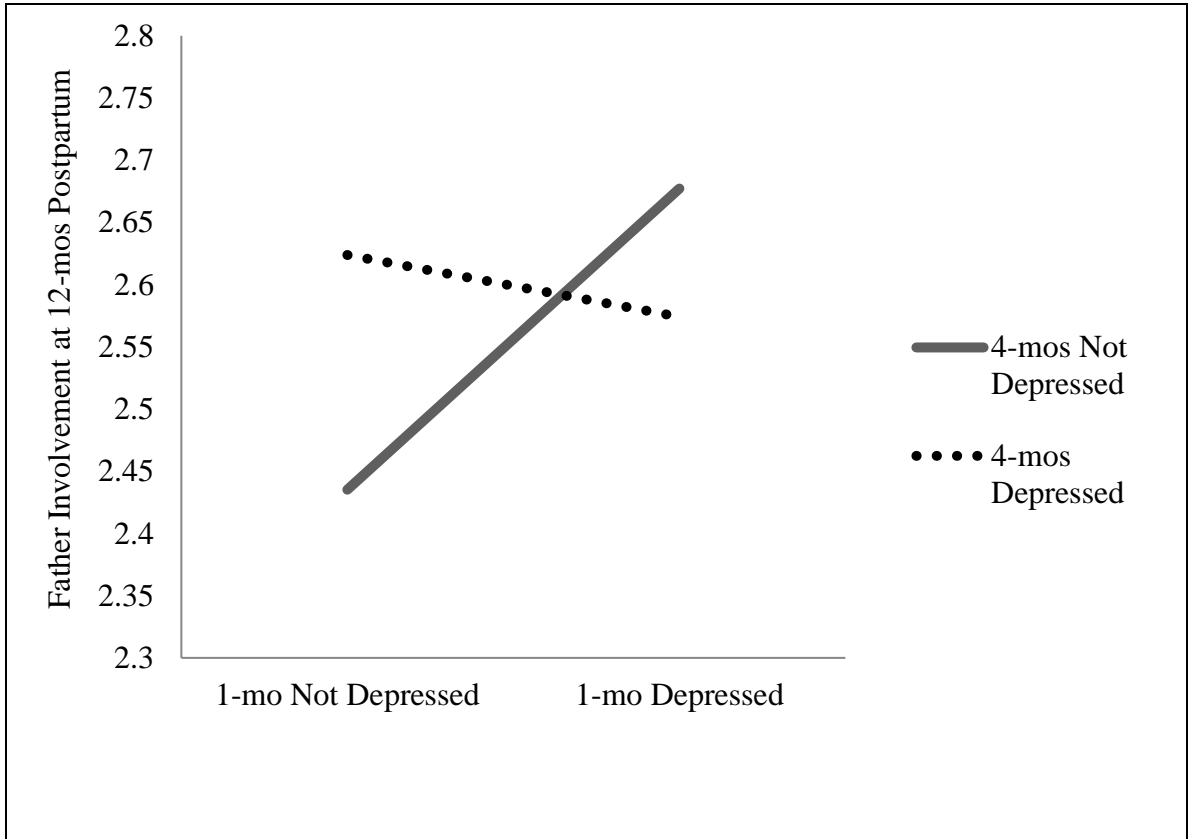


Figure 8. Interaction of Time 1 and Time 2 depression predicting father childcare involvement at Time 3. Father involvement is represented in its original scale to aid in interpretation.

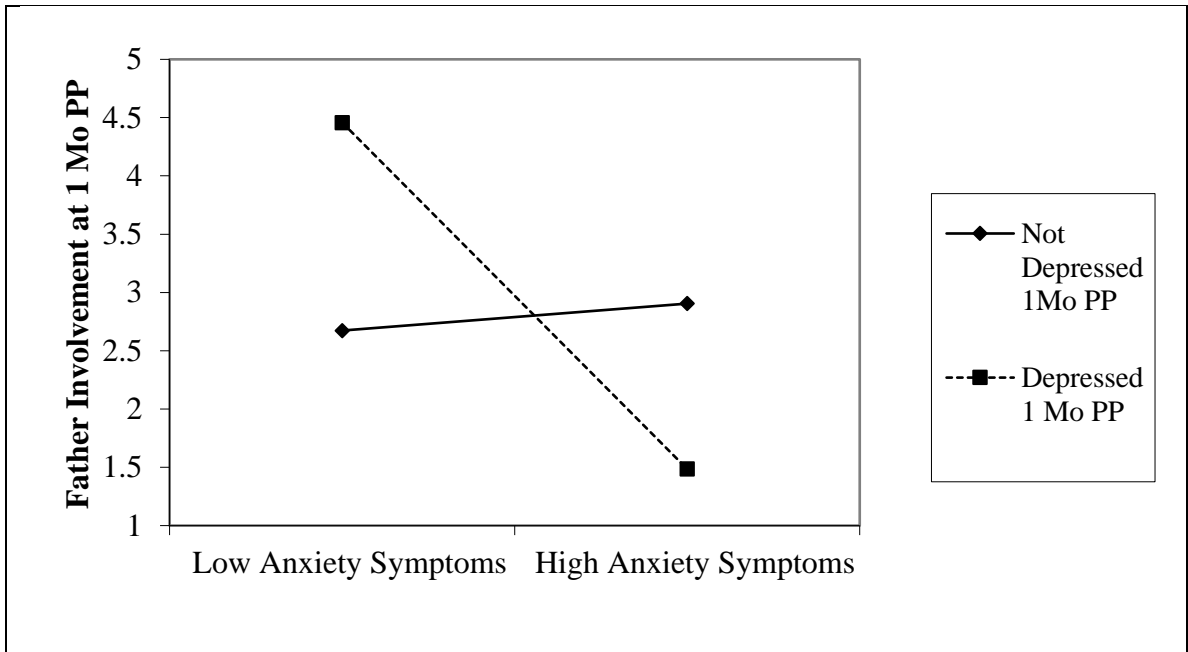


Figure 9. Interaction of anxiety symptoms with clinical levels of depressive symptoms at 1-month postpartum predicting levels of father involvement.

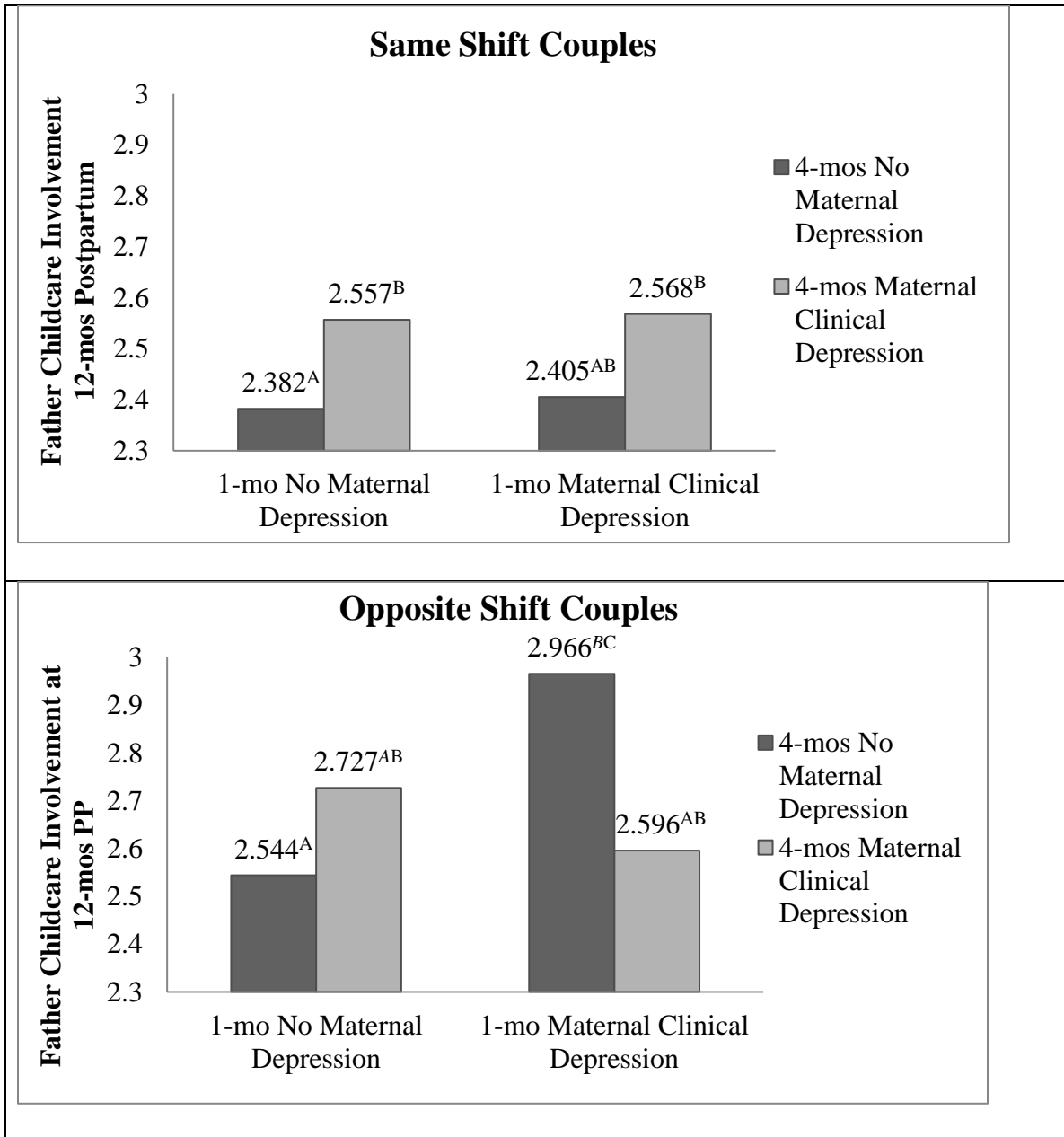


Figure 10. The interaction between T1 and T2 maternal clinical depressive symptoms predicting levels of father involvement at T3 for couples who work same shifts vs. opposite shifts. Father involvement is represented in its original scale to aid in interpretation. Different superscripts within each of the bar graphs denote the estimate involvement was significantly different between groups at the $p < .05$ level.

APPENDIX A

SUPPLEMENTAL TABLES

Table A1.
Multilevel SEM model for Question 1a modeling change in
maternal depression and change in father involvement

	Baseline
	<i>B(SE)</i>
Fixed effects	
Father Involvement Level	
Intercept	25.201** (0.384)
Father Involvement Slope	
Intercept	0.156** (0.038)
Maternal Depression Level	
Intercept	11.969** (0.672)
Maternal Depression Slope	
Intercept	-0.002 (0.056)
Random Effects	
Involvement Level Variance	15.440** (2.713)
Involvement Slope Variance	0.054 [†] (0.036)
Depression Level Variance	49.141** (8.932)
Depression Slope Variance	0.027 (0.081)
Involvement Sigma Sq	9.005** (1.055)
Depression Sigma Sq	36.294** (3.770)

Note. ** $p < .01$, * $p < .05$, [†] $p < .10$

Table A2.

Question 1c: Hierarchical regression of father involvement at 12-months postpartum on the interaction of mothers' clinical levels of depression at 1-month and 12-months postpartum

	Trimmed Control Model	Main Effect Model	Interaction Model
	<i>B</i> (<i>SE</i>)	<i>B</i> (<i>SE</i>)	<i>B</i> (<i>SE</i>)
Intercept	3.645** (0.305)	3.609** (0.314)	3.593** (0.317)
T3 Father Anxiety Symptoms	-0.014** (0.004)	-0.014** (0.005)	-0.014** (0.005)
T1 Mother Age	-0.024** (0.008)	-0.024** (0.008)	-0.023** (0.008)
T3 Father Work Hrs	-0.007** (0.003)	-0.007** (0.003)	-0.007* (0.003)
T3 Mother Work Hrs	0.008** (0.003)	0.008** (0.003)	0.008** (0.003)
T1 Mother Clinical Depression		0.055 (0.101)	0.146 (0.118)
T3 Mother Clinical Depression		0.023 (0.105)	0.112 (0.126)
T1 x T3 Mother Clinical Depression			-0.253 (0.177)
R^2	.188	.195	.206
F for change in R^2	10.283*	0.775	2.415

Note. SPSS does not pool R^2 statistics, so the R^2 and F for change in R^2 reflect the mean values across the 10 imputations. T1 = 1-month postpartum, Mother and Father clinical depression coded as 1 = above clinical cutoff, 0 = below clinical cutoff. Control variables that were not significantly related to father involvement and were thus trimmed from the final control model include infant temperament, infant gender, fathers' depression symptoms, and fathers' age.

* $p < .05$, ** $p < .01$

Table A3.

Q2a: Hierarchical regression of father involvement at 12-months postpartum on the 3-way interactions of mothers' clinical levels of depression at 1- and 4-months and anxiety symptoms at 1-month postpartum

	Trimmed Control Model	Main Effect Model	Interaction Model
	<i>B(SE)</i>	<i>B(SE)</i>	<i>B(SE)</i>
Intercept	3.340** (0.258)	3.304** (0.276)	3.255** (0.276)
T3 Father Depression Symptoms	-0.012* (0.005)	-0.013* (0.005)	-0.013* (0.005)
T1 Mother Age	-0.024** (0.008)	-0.025** (0.008)	-0.025** (0.009)
T3 Father Work Hrs	-0.007** (0.003)	-0.007* (0.003)	-0.006* (0.003)
T3 Mother Work Hrs	0.008** (0.003)	0.008** (0.003)	0.008** (0.003)
T1 Mother Clinical Depression		0.009 (0.109)	0.123 (0.150)
T2 Mother Clinical Depression		0.068 (0.109)	0.151 (0.129)
T1 Mother Anxiety Symptoms		0.089 (0.119)	0.062 (0.177)
T1 x T2 Mother Clinical Depression			-0.195 (0.256)
T1 Depression x T1 Anxiety			0.054 (0.340)
T2 Depression x T1 Anxiety			0.257 (0.351)
T1 Dep x T2 Dep x T1 Anx			-0.387 (0.527)

Note. T1 = 1-month postpartum, T2 = 4-months postpartum. Mother and Father clinical depression coded as 1 = above clinical cutoff, 0 = below clinical cutoff. Anxiety Symptoms were the sum score on the anxiety measure. Control variables that were not significantly related to father involvement and were thus trimmed from the control model were infant temperament, infant gender, fathers' anxiety symptoms, and fathers' age. * $p < .05$, ** $p < .01$

Table A4.

Q2a: Hierarchical regression of father involvement at 12-months postpartum on the 3-way interactions of mothers' clinical levels of depression at 1- and 4-months and anxiety symptoms at 4-months postpartum

	Trimmed Control Model	Main Effect Model	Interaction Model
	<i>B(SE)</i>	<i>B(SE)</i>	<i>B(SE)</i>
Intercept	3.340** (0.258)	3.274** (0.276)	3.219** (0.285)
T3 Father Depression Symptoms	-0.012* (0.005)	-0.013** (0.005)	-0.013* (0.005)
T1 Mother Age	-0.024** (0.008)	-0.024** (0.008)	-0.025** (0.009)
T3 Father Work Hrs	-0.007** (0.003)	-0.007* (0.003)	-0.007* (0.003)
T3 Mother Work Hrs	0.008** (0.003)	0.009** (0.003)	0.009** (0.003)
T1 Mother Clinical Depression		0.060 (0.096)	0.201 (0.131)
T2 Mother Clinical Depression		0.115 (0.134)	0.178 (0.163)
T2 Mother Anxiety Symptoms		-0.024 (0.120)	-0.110 (0.173)
T1 x T2 Mother Clinical Depression			-0.286 (0.259)
T1 Depression x T2 Anxiety			0.115 (0.458)
T2 Depression x T2 Anxiety			0.219 (0.296)
T1 Dep x T2 Dep x T2 Anx			-0.188(0.589)

Note. T1 = 1-month postpartum, T2 = 4-months postpartum. Mother and Father clinical depression coded as 1 = above clinical cutoff, 0 = below clinical cutoff. Anxiety Symptoms were the sum score on the anxiety measure. Control variables that were not significantly related to father involvement and were thus trimmed from the control model were infant temperament, infant gender, fathers' anxiety symptoms, and fathers' age. $p < .05$, ** $p < .01$

Table A5.

Q2a: Hierarchical regression of father involvement at 12-months postpartum on the 3-way interactions of mothers' clinical levels of depression at 1- and 12-months and anxiety symptoms at 1-month postpartum

	Trimmed Control Model	Main Effect Model	Interaction Model
	<i>B(SE)</i>	<i>B(SE)</i>	<i>B(SE)</i>
Intercept	3.340** (0.258)	3.340** (0.264)	3.336** (0.269)
T3 Father Depression Symptoms	-0.012* (0.005)	-0.013* (0.005)	-0.013* (0.005)
T1 Mother Age	-0.024** (0.008)	-0.025** (0.008)	-0.024** (0.009)
T3 Father Work Hrs	-0.007** (0.003)	-0.007* (0.003)	-0.007* (0.003)
T3 Mother Work Hrs	0.008** (0.003)	0.008** (0.003)	0.008** (0.003)
T1 Mother Clinical Depression		0.001 (0.108)	0.092 (0.138)
T3 Mother Clinical Depression		-0.015 (0.114)	0.044 (0.131)
T1 Mother Anxiety Symptoms		0.131 (0.118)	0.291 (0.168)
T1 x T3 Mother Clinical Depression			-0.243 (0.311)
T1 Depression x T1 Anxiety			-0.290 (0.321)
T3 Depression x T1 Anxiety			-0.202 (0.242)
T1 Dep x T3 Dep x T1 Anx			0.283 (0.497)

Note. T1 = 1-month postpartum, T3 = 12-months postpartum. Mother and Father clinical depression coded as 1 = above clinical cutoff, 0 = below clinical cutoff. Anxiety Symptoms were the sum score on the anxiety measure. Control variables that were not significantly related to father involvement and were thus trimmed from the final control model include infant temperament, infant gender, fathers' anxiety symptoms, and fathers' age.

* $p < .05$, ** $p < .01$

Table A6.

Q2a: Hierarchical regression of father involvement at 12-months postpartum on the 3-way interactions of mothers' clinical levels of depression at 1- and 12-months and anxiety symptoms at 12-months postpartum

	Trimmed Control Model	Main Effect Model	Interaction Model
	<i>B</i> (<i>SE</i>)	<i>B</i> (<i>SE</i>)	<i>B</i> (<i>SE</i>)
Intercept	3.340** (0.258)	3.327** (0.266)	3.316** (0.272)
T3 Father Depression Symptoms	-0.012* (0.005)	-0.013* (0.005)	-0.013* (0.005)
T1 Mother Age	-0.024** (0.008)	-0.025** (0.008)	-0.024** (0.008)
T3 Father Work Hrs	-0.007** (0.003)	-0.007* (0.003)	-0.007* (0.003)
T3 Mother Work Hrs	0.008** (0.003)	0.009** (0.003)	0.008** (0.003)
T1 Mother Clinical Depression		0.053 (0.100)	0.127 (0.122)
T3 Mother Clinical Depression		-0.046 (0.128)	0.058 (0.167)
T1 Mother Anxiety Symptoms		0.108 (0.116)	0.207 (0.162)
T1 x T3 Mother Clinical Depression			-0.214 (0.318)
T1 Depression x T1 Anxiety			-0.186 (0.363)
T3 Depression x T1 Anxiety			-0.259 (0.251)
T1 Dep x T3 Dep x T1 Anx			0.277 (0.517)

Note. T1 = 1-month postpartum, T3 = 12-months postpartum. Mother and Father clinical depression coded as 1 = above clinical cutoff, 0 = below clinical cutoff. Anxiety Symptoms were the sum score on the anxiety measure. Control variables that were not significantly related to father involvement and were trimmed from the control model include infant temperament, infant gender, fathers' anxiety symptoms, and fathers' age.

* $p < .05$, ** $p < .01$

Table A7.

Multilevel SEM model for Question 3a modeling change in maternal depression and change in father involvement for same-shift and opposite-shift couples

	Constrained Baseline		Free Baseline	
	Same-Shift <i>B(SE)</i>	Opposite-Shift <i>B(SE)</i>	Same-Shift <i>B(SE)</i>	Opposite-Shift <i>B(SE)</i>
Fixed effects				
Father Involvement Level				
Intercept	25.217** (0.384)	25.217** (0.384)	24.382** (0.460)	26.591** (0.579)
Father Involvement Slope				
Intercept	0.160** (0.033)	0.160** (0.033)	0.140** (0.039)	0.207** (0.058)
Maternal Depression Level				
Intercept	12.054** (0.684)	12.054** (0.684)	11.921** (0.913)	11.878** (1.018)
Maternal Depression Slope				
Intercept	0.009 (0.057)	0.009 (0.057)	0.044 (0.071)	-0.073 (0.091)
Random Effects				
Involvement Level Variance	16.008** (3.147)	12.868** (3.302)	15.241** (3.204)	11.250** (3.830)
Involvement Slope Variance	0.037 (0.027)	0.055 (0.040)	0.036 (0.029)	0.054 (0.052)
Depression Level Variance	55.626** (10.821)	39.933** (10.796)	58.291** (12.694)	34.750** (12.642)
Depression Slope Variance	0.022 (0.091)	0.084 (0.106)	0.030 (0.104)	0.034 (0.137)
Involvement Sigma Sq	7.472** (1.106)	10.841** (1.877)	7.488** (1.123)	10.819** (2.009)
Depression Sigma Sq	36.977** (4.576)	32.889** (5.506)	36.882** (4.698)	34.123** (6.443)

Note. ** $p < .01$, * $p < .05$, † $p < .10$

Table A8.

Q3c: Hierarchical regression of father involvement at 12-months postpartum on the 3-way interactions of mothers' clinical levels of depression at 1- and 12-months postpartum and couple shift

	Trimmed Control Model	Main Effect Model	Interaction Model
	<i>B</i> (<i>SE</i>)	<i>B</i> (<i>SE</i>)	<i>B</i> (<i>SE</i>)
Intercept	3.657** (0.286)	3.560** (0.290)	3.559** (0.295)
T3 Father Anxiety Symptoms	-0.013* (0.005)	-0.013* (0.005)	-0.014* (0.005)
T1 Mother Age	-0.027** (0.008)	-0.026** (0.008)	-0.026** (0.008)
T3 Father Work Hrs	-0.006** (0.003)	-0.006* (0.003)	-0.006* (0.003)
T3 Mother Work Hrs	0.009** (0.002)	0.008** (0.003)	0.008** (0.003)
T1 Mother Clinical Depression		0.053 (0.100)	-0.029 (0.181)
T3 Mother Clinical Depression		-0.046 (0.128)	0.132 (0.136)
T3 Couple Shift		0.156* (0.076)	0.124 (0.097)
T1 x T3 Mother Clinical Depression			-0.060 (0.256)
T1 Depression x T3 Shift			0.277 (0.224)
T3 Depression x T3 Shift			-0.020 (0.239)
T1 Dep x T3 Dep x T3 Shift			-0.398(0.393)

Note. T1 = 1-month postpartum, T3 = 12-months postpartum. Mother and Father clinical depression coded as 1 = above clinical cutoff, 0 = below clinical cutoff. Couple shift was coded as 0 = same shift, 1 = opposite shift. Control variables that were not significantly related to father involvement and were trimmed from the control model include infant temperament, infant gender, fathers' depression symptoms, and fathers' age.

* $p < .05$, ** $p < .01$

APPENDIX B

MEASURES

Child Care Tasks (Barnett and Baruch, 1987)

<u>1</u>	<u>2</u>	<u>3</u>	<u>4</u>	<u>5</u>
0-20% Mostly or always my spouse	20-40% More likely my spouse	40-60% Shared about equally	60-80% More likely me	80-100% Mostly or always me

1.	Feeding the baby	1	2	3	4	5
2.	Changing the baby's diaper	1	2	3	4	5
3.	Soothing the baby	1	2	3	4	5
4.	Getting up at night with the baby	1	2	3	4	5
5.	Putting the baby to sleep	1	2	3	4	5
6.	Giving the baby a bath	1	2	3	4	5
7.	Helping the baby learn new skills	1	2	3	4	5
8.	Dressing the baby	1	2	3	4	5
9.	Planning the baby's activities	1	2	3	4	5
10.	Picking up after the baby	1	2	3	4	5
11.	Playing with the baby	1	2	3	4	5
12.	Reading/singing to the baby	1	2	3	4	5
13.	Taking the baby on an outing	1	2	3	4	5
14.	Taking the baby to a doctor's appointment	1	2	3	4	5
15.	Taking care of the baby when he or she is sick	1	2	3	4	5

Center for Epidemiological Studies-Depression Scale (CES-D; Radloff, 1975)

Below is a list of the ways you might have felt or behaved recently. Please circle the number that indicates how often you have felt this way during the past week.

<u>0</u>	<u>1</u>	<u>2</u>	<u>3</u>
Rarely or none of the time (less than 1 day)	Some or a little of the time (1-2 days)	Occasionally or a moderate amount of time (3-4 days)	Most or all of the time (5-7 days)

- | | | | | |
|--|---|---|---|---|
| 1. I was bothered by things that don't usually bother me. | 0 | 1 | 2 | 3 |
| 2. I did not feel like eating; my appetite was poor | 0 | 1 | 2 | 3 |
| 3. I felt that I could not shake off the blues even with help from my family or friends. | 0 | 1 | 2 | 3 |
| 4. I felt that I was just as good as other people. | 0 | 1 | 2 | 3 |
| 5. I had trouble keeping my mind on what I was doing. | 0 | 1 | 2 | 3 |
| 6. I felt depressed. | 0 | 1 | 2 | 3 |
| 7. I felt that everything was an effort. | 0 | 1 | 2 | 3 |
| 8. I felt hopeful about the future. | 0 | 1 | 2 | 3 |
| 9. I thought my life had been a failure. | 0 | 1 | 2 | 3 |
| 10. I felt fearful. | 0 | 1 | 2 | 3 |
| 11. My sleep was restless. | 0 | 1 | 2 | 3 |
| 12. I was happy. | 0 | 1 | 2 | 3 |
| 13. I talked less than usual. | 0 | 1 | 2 | 3 |
| 14. I felt lonely. | 0 | 1 | 2 | 3 |
| 15. People were unfriendly. | 0 | 1 | 2 | 3 |
| 16. I enjoyed life. | 0 | 1 | 2 | 3 |
| 17. I had crying spells. | 0 | 1 | 2 | 3 |
| 18. I felt sad. | 0 | 1 | 2 | 3 |
| 19. I felt that people dislike me. | 0 | 1 | 2 | 3 |
| 20. I could not get "going." | 0 | 1 | 2 | 3 |

State-Trait Anxiety Inventory (Spielberger, 1972)

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you feel right now, that is, at this moment. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to best describe your present feelings.

<u>1</u>	<u>2</u>	<u>3</u>	<u>4</u>
Not at all	Somewhat	Moderately so	Very much so

- | | | | | | |
|-----|--|---|---|---|---|
| 1. | I feel pleasant. | 1 | 2 | 3 | 4 |
| 2. | I feel nervous and restless. | 1 | 2 | 3 | 4 |
| 3. | I feel satisfied with myself. | 1 | 2 | 3 | 4 |
| 4. | I wish I could be as happy as others seem to be. | 1 | 2 | 3 | 4 |
| 5. | I feel like a failure. | 1 | 2 | 3 | 4 |
| 6. | I feel rested. | 1 | 2 | 3 | 4 |
| 7. | I am "calm, cool and collected." | 1 | 2 | 3 | 4 |
| 8. | I feel that difficulties are piling up so that I cannot overcome them. | 1 | 2 | 3 | 4 |
| 9. | I worry too much over something that really doesn't matter. | 1 | 2 | 3 | 4 |
| 10. | I am happy. | 1 | 2 | 3 | 4 |
| 11. | I have disturbing thoughts. | 1 | 2 | 3 | 4 |
| 12. | I lack self-confidence. | 1 | 2 | 3 | 4 |
| 13. | I feel secure. | 1 | 2 | 3 | 4 |
| 14. | I make decisions easily. | 1 | 2 | 3 | 4 |
| 15. | I feel inadequate. | 1 | 2 | 3 | 4 |
| 16. | I am content. | 1 | 2 | 3 | 4 |
| 17. | Some unimportant thought runs through my mind and bothers me. | 1 | 2 | 3 | 4 |
| 18. | I take disappointments so keenly that I can't put them out of my mind. | 1 | 2 | 3 | 4 |
| 19. | I am a steady person. | 1 | 2 | 3 | 4 |
| 20. | I get in a state of tension or turmoil as I think over my recent concerns and interests. | 1 | 2 | 3 | 4 |

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