Photocopy and Use Authorization

In presenting this dissertation in partial fulfillment of the requirements for an advanced degree at Idaho State University, I agree that the Library shall make it freely available for inspection. I further state that permission for extensive copying of my dissertation for scholarly purposes may be granted by the Dean of the Graduate School, Dean of my academic division, or by the University Librarian. It is understood that any copying or publication of this dissertation for financial gain shall not be allowed without my written permission.

Signature _____

Date _____

Relations among maternal perinatal obsessive-compulsive symptomatology, depressive symptomatology, and infant behavior at 6 months

by

Jason M. Gibbs

A dissertation

submitted in partial fulfillment

of the requirements for the degree of

Doctor of Philosophy in the Department of Psychology

Idaho State University

Summer 2018

To the Graduate Faculty:

The members of the committee appointed to examine the dissertation of Jason M. Gibbs find it satisfactory and recommend that it be accepted.

_____

Nicki L. Aubuchon-Endsley, Ph.D.,
Major Advisor

_____

Michele R. Brumley, Ph.D.,
Committee Member

_____

Steven R. Lawyer, Ph.D.,
Committee Member

_____

Robert D. Rieske, Ph.D.,
Committee Member

_____

Heather L. Ramsdell-Ludock, Ph.D.,
Graduate Faculty Representative

Idaho State
UNIVERSITY
Office for Research - Research Outreach & Compliance
921 S. 8th Avenue, Stop 8046 • Pocatello, Idaho 83209-8046

February 9, 2018

Nicki Aubuchon-Endsley, PhD
Stop 8112
Psychology
Pocatello, ID 83209

RE: Your application dated 2/5/2018 regarding protocol number 4191: Infant Development and Healthy Outcomes in Mothers (Idaho Mom Study)

Dear Dr. Aubuchon-Endsley:

Your request for renewal of the protocol listed above was reviewed 4/12/2016 meeting of the Idaho State University Human Subjects Committee.

This is to confirm that your request for renewal is approved. Your request to modify the protocol by modifying data storage procedures and adding assistants Joe Neal, Jessica Riedstra, Jason Gibbs, Taylor Ramos, Anika Lovgren, Nicole Douthit, Abby Prow, Hailey Wilcox, Sierra Clayson, Reilly Sasaki, and Jennifer Hambleton has been approved via Expedited Review.

You are free to proceed with your protocol as described effective immediately. The protocol is next subject to renewal on or before 2/9/2019, unless closed before that date.

As with the initial approval, changes to the study must be promptly reported and approved. Contact Tom Bailey (208-282-2179; fax 208-282-4723; email: humsubj@isu.edu) if you have any questions or require further information.

Sincerely,

Ralph Baergen, PhD, MPH, CIP
Human Subjects Chair

Phone: (208) 282-1336 • Fax: (208) 282-4723 • isu.edu/research

Acknowledgments

I would like to thank my dissertation committee for their invaluable contributions to my project. I also would like to thank all of the research assistants and participants involved in the IDAHO Mom Study. Their hard work and support made this research possible.

TABLE OF CONTENTS

List of Tables

Relations among maternal perinatal obsessive-compulsive symptomatology, depressive symptomatology, and infant behavior at 6 months

Dissertation Abstract—Idaho State University (2018)

It is well established that perinatal maternal depression, anxiety, and stress are associated with a number of differences in infant outcomes. However, no known study had investigated the effects of perinatal obsessive-compulsive symptomatology on infant outcomes. Due to the widespread chronicity and potentially different underlying neurological substrates of obsessive-compulsive disorder as compared to other disorders, this represents an understudied area of research. Therefore, we investigated the effects of prenatal and postnatal obsessive-compulsive symptomatology on infant behavioral reactivity, beyond the effects of postnatal depressive symptomatology, at 6 months of age. It was expected that socioeconomic status would moderate this relationship. We recruited one hundred twenty-five pregnant women from southeastern Idaho and interviewed them at approximately 34 weeks gestation and again at 6 months postnatally. They were administered questionnaires at both time points measuring levels of obsessive compulsive symptoms and depressive symptoms, and infant behavioral reactivity was gathered during the 6-month follow-up session through both behavioral observation coding and self-report modalities. Greater severity of depressive symptomatology was related to greater of infant reactivity at 6 months as assessed via self-report, but not when assessed through behavioral observation. Greater levels of maternal obsessive-compulsive symptomatology and socioeconomic status were not found to be related to infant reactivity via either self-report or behavioral observation. Practical implications and future areas of research are discussed.

Key Words: perinatal, obsessive-compulsive, depressive symptom, infant behavior

CHAPTER I

The perinatal period is often a joyous time for families as they prepare to welcome a baby into their lives. However, it is also a period consisting of many biopsychosocial changes for an expectant mother, which may be associated with heightened levels of stress and negative affectivity, as well as exacerbation of any pre-existing mental health conditions (Furber et al., 2009). Many biological changes occur over the course of pregnancy, such as increasing levels of maternal plasma corticotropin releasing hormone (CRH), a peptide hormone and neurotransmitter involved in the human stress response (Davis et al., 2005). A heightened level of negative affectivity and stress may influence mothers and their unborn children (Mulder et al., 2002). These outcomes for children may be mediated through the processes of prenatal programming. Prenatal programming refers to the process through which fetal, metabolic, and physiological processes can be permanently altered by in utero exposures during critical periods of development. This is because the structure of the brain is still being formed during this time (Bertram & Hanson, 2002; Davis et al., 2007; de Weerth, van Hees, & Buitelaar, 2003). The biological systems of the developing organism are especially vulnerable to biological and environmental teratogens during this period, such as elevated levels of prenatal maternal glucocorticoids (Buss, Davis, Muftuler, Head, & Sandman, 2010; Seckl & Meaney, 2004).

Many animal studies have demonstrated that maternal stress during pregnancy can alter the behavior and long-term hypothalamic-pituitary-adrenal (HPA) axis or stress responsiveness of offspring (Austin & Leader, 2000). This is due to the fact that development is not simply the gradual construal of a pre-determined plan, but rather is a dynamic interplay of environmental and genetic effects, wherein genetic and epigenetic processes are co-occurring in the organism (Wadhwa, 2002). Indeed, although there is some evidence that mild levels of

depression, anxiety, and stress experienced during pregnancy can enhance fetal maturation in healthy populations (DiPietro, Novak, Costigan, Atella, & Reusing, 2006), most evidence suggests that increases in these prenatal symptoms are associated with poorer mental and physical wellbeing for offspring throughout the lifespan (Gutteling, de Weerth, & Buitelaar, 2005a). Additionally, some of these effects begin in utero (Sjöström, Valentin, Thelin, & Maršál, 1997). While there has been a plethora of research on the relationship of maternal perinatal anxiety and depression to offspring outcomes (both via experimental studies on animals and correlational studies on humans), no known literature to date has evaluated the specific relationship of perinatal obsessive-compulsive (OC) symptomatology to infant outcomes. Due to the widespread chronicity (approximately 25% of the general population) of obsessions and compulsions (Fullana et al., 2009), along with the onset and exacerbation of OC symptomatology during the perinatal period (Agrati et al., 2005; Forray, Focseneanu, Pittman, McDougle, & Epperson, 2010; Labad et al., 2005; Neziroglu, Anemone, & Yaryura-Tobias, 1992; Uguz, Akman, Kaya, & Cilli, 2007; Vulink, Denys, Bus, & Westenberg, 2006; Wenzel, Haugen, Jackson, & Brendle, 2005; Zambaldi et al., 2009), these issues merit closer investigation and are the focus of the current dissertation.

Indeed, incidence rates of Obsessive-Compulsive Disorder (OCD) in postpartum women are approximately 4% (Uguz et al., 2007; Wenzel, Gorman, O'Hara, & Stuart, 2001), which is higher than general community lifetime estimates of 1.2% (American Psychiatric Association , 2013) to 3% (Kolada, Bland, & Newman, 1994). Therefore, the purpose of this study is to evaluate whether maternal OC symptomatology is related to infant behavior at 6 months, and to see if this differs from mothers with symptoms of other disorders with known onset and exacerbation during the perinatal period (i.e., depression symptoms). We also sought to replicate

previous research findings regarding positive relationships with maternal perinatal depressive symptomatology and infant reactivity. These findings may assist with understanding how to best help prenatal women experiencing psychological difficulties, as well as their infants.

Perinatal Anxiety, Depression, and Stress and Infants

Depression

Depression is a state in which an individual evidences symptoms of sadness, emptiness, or irritable mood, along with cognitive and somatic changes that significantly impact functional capability (American Psychiatric Association, 2013). The DSM-5 lists eight depressive disorders: Disruptive Mood Dysregulation Disorder, Major Depressive Disorder, Persistent Depressive Disorder, Premenstrual Dysphoric Disorder, Substance/Medication-Induced Depressive Disorder, Depressive Disorder due to Another Medical Condition, Other Specified Depressive Disorder, and Unspecified Depressive Disorder (American Psychiatric Association, 2013). Most research on the relationship between perinatal psychological adversity and infant outcomes has focused on depression. Depression is common during the perinatal period, with approximately 7-13% of women exhibiting clinically significant symptoms of depression during pregnancy and/or postpartum (Bennett, Einarson, Taddio, Koren, & Einarson, 2004). However, point prevalence rates of 23% at 14 weeks postpartum and 19% at 30 weeks postpartum, and incidence rates during the 14 to 30 weeks postpartum period of 7%, have also been documented (Stuart, Couser, Schilder, O'Hara, & Gorman, 1998).

Subthreshold symptoms can be severe and impairing as well. For example, significantly impairing symptoms of depression may occur in as many as 20% of women during the prenatal period (Marcus, Flynn, Blow, & Barry, 2004). Higher levels of depression experienced by mothers during pregnancy have been linked to stunted fetal growth and shorter birth length

among infants of mothers with low socioeconomic status (SES; Hoffman & Hatch, 2000). Mothers with prenatal depression are also at elevated risk for developing preeclampsia (Mulder et al., 2002), going into premature birth (Field, Diego, & Hernandez-Reif, 2006), and having infants with lower birth weight (Field et al., 2006; Mulder et al., 2002). Furthermore, maternal prenatal depression has been linked to greater infant behavioral reactivity to novelty at 2 months (Davis et al., 2007) and 4 months (Davis et al., 2004), as well as more indeterminate sleep, time fussing/crying, and stress behaviors in infants up to 2 weeks of age (Diego, Field, & Hernandez-Reif, 2005; Zuckerman, Bauchner, Parker, & Cabral, 1990). Depressed mothers are also more likely to have children with externalizing problems during childhood (Luoma et al., 2001).

Newborns of depressed mothers also evidence neurologically worse outcomes, such as greater relative right frontal electroencephalogram (EEG) asymmetry, which is associated with depression, inhibition, and a lack of empathy within a developmental context (Field et al., 2004; Field et al., 2006; Jones, Field, Fox, Lundy, & Davalos, 1997; Jones et al., 1998). Additionally, newborns of depressed mothers evidence lower vagal tone, which is associated with autonomic nervous system dysregulation and less optimal performance on attention and learning tasks (DiPietro et al., 2006; Field et al., 2004; Field et al., 2006; Jones et al., 1998). Moreover, in infancy, offspring have faster heart rates with greater exposure to prenatal depressive symptomatology pregnancy-specific stress (DiPietro et al., 2006).

Infants of mothers with prenatal depression also evidence significantly different levels of hormones and neurotransmitters when compared to mothers without prenatal depression. Specifically, newborns of depressed mothers evidence higher levels of cortisol, a physiological indicator of stress (Field et al., 2004; Field et al., 2006; Lundy et al., 1999), and higher levels of norepinephrine (NE; Lundy et al., 1999). Lower levels of dopamine (DA; Field et al., 2004; Field

et al., 2006; Lundy et al., 1999) are found in newborns of prenatally depressed mothers as well. This association of elevated NE and lowered DA support the model placed forth by Weiss, Demetrikopoulos, West, and Bonsall (1995), wherein high levels of NE interact with low levels of DA to cause depressive symptoms. This suggests that the neurotransmitter levels associated with depressive symptoms in mothers are also present in their newborns. Newborns also evidence lower levels of serotonin (5-HT; Field et al., 2004; Field et al., 2006), and higher levels of adrenocorticotropic hormone (ACTH; Marcus et al., 2011), biochemical markers also associated with the onset and maintenance of depressive symptomatology.

Anxiety

Anxiety can be conceptualized as the psychological consequence of exposure to imagined or real stressors, with individuals higher in trait anxiety experiencing adverse physical and psychological consequences from environmental stressors (Austin & Leader, 2000; Ruiz & Avant, 2005). Specifically, anxiety refers to the anticipation of future threats, for which individuals may respond with excessive levels of distress (American Psychiatric Association, 2013). It differs from fear, which is an immediate distress reaction to an external stimulus which is occurring in the moment (Pavuluri, Henry, & Allen, 2002). When such anxiety is experienced, an anxiety disorder may develop. The DSM-V lists 11 anxiety disorders: Separation Anxiety Disorder, Selective Mutism, Specific Phobia, Social Anxiety Disorder, Panic Disorder, Agoraphobia, Generalized Anxiety Disorder, Substance/Medication-Induced Anxiety Disorder, Anxiety Disorder Due to Another Medical Condition, Other Specified Anxiety Disorder, and Unspecified Anxiety Disorder. Furthermore, approximately 90% of individuals with anxiety disorders will suffer from comorbid depressive symptoms, and these individuals are typically more distressed than individuals with a single disorder (Gorman, 1996). In general, women

experience anxiety disorders approximately 1.5 to twice as often as men, and experience greater rates of comorbid depression and anxiety (American Psychiatric Association, 2013; McLean, Asnaani, Litz, & Hofmann, 2011).

Anxiety, like depression, is also common during the perinatal period. Point prevalence rates include approximately 9% at 14 weeks postpartum and 17% at 30 weeks postpartum, with incidence rates in the period of 14 to 30 weeks postpartum of 10% (Stuart et al., 1998). While not studied to the extent that perinatal depression has been, maternal perinatal anxiety has begun to garner increasing research attention and is related to several offspring outcomes. As assessed by a range of measurements (e.g., retrospective self-report, prospective self-report, DSM-IV diagnosis, etc.), high levels of anxiety experienced during pregnancy have been linked to lower birth weight (Glover & O'Connor, 2002; Mulder et al., 2002), maternal pre-eclampsia (Mulder et al., 2002), and preterm birth (Glover & O'Connor, 2002; Mancuso, Schetter, Rini, Roesch, & Hobel, 2004; Sandman, Wadhwa, Chicz-Demet, Dunkel-Schetter, & Porto, 1997).

In infants, maternal prenatal anxiety has been linked to higher levels of behavioral reactivity to novelty at 4 months (Davis et al., 2004), increased colic risk (Canivet, Ostergren, Rosen, Jakobsson, & Hagander, 2005), difficult temperament at 4 and 6 months (Austin, Hadzi-Pavlovic, Leader, Saint, & Parker, 2005), and higher risk for excessive crying through the first 16 months of life (Petzoldt et al., 2014). Maternal prenatal anxiety is also related to lower mental and psychomotor developmental scores at 8 months (Huizink, de Medina, Mulder, Visser, & Buitelaar, 2003; Buitelaar, Huizink, Mulder, de Medina, & Visser, 2003), lower levels of focused attention at 18 months (Plamondon et al., 2015), and poorer general cognitive outcomes (see Tarabulsy et al., 2014 for a review). Biological changes have been noted as well. For example, prenatal maternal anxiety has been linked to lower levels of DA and 5-HT in newborns and

infants who present with lower vagal tone, more postnatal complications, less time spent in active- and quiet-alert states, a greater number of state changes, and greater relative right frontal EEG activation (Field et al., 2003).

In later childhood, maternal prenatal anxiety has been linked to behavioral/emotional problems at age 4 (O'Connor, Heron, & Glover, 2002a), lower levels of attention focusing at age 4 (Plamondon et al., 2015), higher levels of salivary cortisol in 5-year-old children during their first day of school (Gutteling et al., 2005a), poorer general cognitive outcomes up to 5 years of age (see Tarabulsy et al., 2014 for a review), impaired executive functioning among 6- to 9-year-old offspring (Buss, Davis, Hobel, & Sandman, 2011), generally higher levels of anxiety at 8 to 9 years of age (van den Bergh & Marcoen, 2004), and symptoms of Attention Deficit Hyperactivity Disorder (ADHD; as assessed by the Attention Problems scale of the Child Behavior Checklist) and externalizing problems in 8- to 9-year-old boys and girls (van den Bergh & Marcoen, 2004). Higher levels of prenatal maternal anxiety exposure have been linked to less efficient decision making on a gambling paradigm and associated cognitive control task in 17-year-old boys (Mennes, Bergh, Lagae, & Stiers, 2009), and ADHD symptoms (as assessed by a continuous performance task) in adolescent boys (van den Bergh et al., 2006).
Anatomical changes are noted as well. For example, maternal prenatal anxiety has been linked to reductions in gray matter volume in 6- to 9-year-old children in the prefrontal, lateral temporal, and premotor cortices, the medial temporal lobe, the postcentral gyrus, and the cerebellum extending to the middle occipital and fusiform gyri (Buss et al., 2010).

Similar outcomes have been found in population-wide studies as well. For example, in a prospective, longitudinal study utilizing 85% - 90% of the eligible pregnant population from April 1991 to December 31, 1992 in the Avon, UK area, O'Connor, Heron, Golding, Beveridge,

and Glover (2002b) found that antenatal anxiety predicted children's behavioral and emotional problems at 4 years of age. These group differences were found across boys and girls, with the associations between antenatal anxiety and the majority of children's behavioral and emotional problems remaining significant even when controlling for obstetric and sociodemographic risks. This suggests that associations among antenatal anxiety and poor offspring outcomes are not necessarily mediated by maternal health and socioeconomic factors.

Generalized Stress

It is also important to examine the literature to evaluate the relationship of maternal stress and fetal and infant outcomes, regardless of any specific diagnoses. Stress can be conceptualized as any psychological or physical challenge that threatens or is believed to have the potential to threaten the internal stability of an organism (Wadhwa et al., 2002), or as a psychophysiological consequence of an event that challenges an organism's coping capacity (Austin & Leader, 2000). In general, stress leads to activation of the HPA axis and sympathetic adrenal medullary (SAM) system, with chronic levels of stress being associated with global immunosuppression (Segerstrom & Miller, 2004).

Stress can be measured in a multitude of different ways. Psychological questionnaires (e.g., Perceived Stress Scale), physiological measurements (e.g., cortisol), and autonomic measurements (e.g., blood pressure) are commonly used modalities (Lupien, Sonia, & Seguin, 2007). Different studies suggest that a diverse range of measures of neuroendocrine activity are more sensitive indicators of stress than one another. This suggests that it may be important to utilize multiple measures of neuroendocrine activity rather than relying on a single biomarker or variable related to that biomarker (e.g., cortisol awakening response; Wadhwa, Dunkel-Schetter, Chicz-DeMet, Porto, & Sandman, 1996). Nonetheless, cortisol, the primary human stress

hormone, is a frequently utilized way of assessing neuroendocrine activity. Elevated levels of maternal prenatal cortisol are related to potentially harmful effects on the developing fetus. For example, higher levels have been linked to intrauterine growth restriction, lower infant birth weight (Sandman et al., 1997), preterm birth (Austin & Leader, 2000; Copper et al., 1996; Hedegaard, Henriksen, Sabroe, & Secher, 1993; Mulder et al., 2002), maternal preeclampsia, and spontaneous abortion (Mulder et al., 2002).

Similar to the physiological outcomes evidenced in infants of mothers with elevated cortisol levels, behavioral differences are found as well. Specifically, higher levels of prenatal maternal cortisol have been linked to infant colic (Rautava, Helenius, & Lehtonen, 1993; Sondergaard et al., 2003), higher levels of fearfulness (Bergman, Sarkar, O'Connor, Modi, & Glover, 2007), lower affective reactivity to novelty at 4 months of age (Mohler, Parzer, Brunner, Wiebel, & Resch, 2006), and more fussing, crying, and negative facial expressions in infants, with higher levels of fussing still evident at 4 to 5 months of age (de Weerth et al., 2003). Prenatal anxiety exposure is also associated with worse infant mental development at 3 and 8 months and worse psychomotor development at 8 and 9 months (Huizink et al., 2003), poorer cognitive development from 14 to 19 months (Bergman et al., 2007), lower general intellectual and productive language abilities at 2 years (Laplante et al., 2004), autistic traits in 2-year-old boys (Ronald, Pennell, & Whitehouse, 2011), and ADHD behaviors in 2-year-old boys and girls (Ronald et al., 2011).

Relationships between maternal prenatal stress and offspring outcomes have been identified in later childhood as well. For example, higher levels of cortisol in pregnant mothers have been found to correlate with higher levels of cortisol in offspring up to 5.5 years of age (Gitau, Cameron, Fisk, & Glover, 1998; Gutteling et al., 2005a). Additionally, as assessed by

non-biological methodologies (e.g., self-reports and observations), severe levels of stress experienced by mothers during the prenatal period have been linked to generalized externalizing problems in 27-month-old-toddlers (Gutteling et al., 2005b), poorer generalized cognitive outcomes up to 5 years of age (see Tarabulsy et al., 2014 for a review), worse school marks in children at 6 years of age (Niederhofer & Reiter, 2004), and ADHD behaviors in 7-year-old boys (Rodriguez & Bohlin, 2005).

Mediating Factors

It is important to note that many fetal and offspring outcomes are associated with perinatal depression, anxiety, and stress. Individuals suffering from these mental health difficulties present with additional sociodemographic and psychological factors that could hypothetically be related to neuroendocrine functioning and parent/child interaction factors (e.g., premature birth, maternal personality traits, perinatal/medical complications, etc.). However, relations between perinatal depression, anxiety, and stress retain significance even after controlling for these sociodemographic and psychological factors. These control variables include parental age (Canivet et al., 2005; Copper et al., 1996; Huizink et al., 2003; Sjöström et al., 1997), educational level (Bergman et al., 2007; Canivet et al., 2005; Copper et al., 1996; Mancuso et al., 2004; Rodriguez & Bohlin, 2005; van den Bergh & Marcoen, 2004; van den Bergh et al., 2006), marital status (Copper et al., 1996), parity (Canivet et al., 2005; Mancuso et al., 2004) , cohabitation status (Canivet et al., 2005), gestational age (Huizink et al., 2003), maternal personality traits (Mohler et al., 2006), maternal weight, weight increase, and height (Sjöström et al., 1997), attitude toward risk of spoiling (Canivet et al., 2005), parental intelligence (van den Bergh et al., 2006), biomedical risks (Mancuso et al., 2004; Huizink et al., 2003), perinatal/medical complications (Huizink et al., 2003; Mohler et al., 2006), obstetric risks

(Bergman et al., 2007; O'Connor et al., 2002b), income (Mancuso et al., 2004; Rodriguez & Bohlin, 2005; Zuckerman et al., 1990), child's gender (van den Bergh & Marcoen, 2004), discrete episodes of major depression (Brouwers, van Baar, & Pop, 2001; Petzoldt et al., 2014), child's birth weight (Huizink et al., 2003; van den Bergh & Marcoen, 2004; Zuckerman et al., 1990), maternal marital status (Sjöström et al., 1997), postnatal depressive/anxious/stress symptomatology (Bergman et al., 2007; Brouwers et al., 2001; Buss et al., 2010; Copper et al., 1996; Davis et al., 2004; Huizink et al., 2003; Mohler et al., 2006; O'Connor et al., 2002a; van den Bergh & Marcoen, 2004; van den Bergh et al., 2006), and maternal smoking, alcohol, marijuana, cocaine, and other drug use (Bergman et al., 2007; Sjöström et al., 1997; van den Bergh & Marcoen, 2004; Zuckerman et al., 1990).

However, it should be noted that certain factors are associated with mediating/moderating the relationship between maternal anxiety, depression, stress, and infant outcome. For example, high SES has been found to moderate the impacts of adverse perinatal stressors, providing a buffering effect (Blumenshine, Egerter, Barclay, Cubbin, & Braveman, 2010). However, it is likely that many of the relationships between anxiety, depression, and fetal outcomes are moderated by other factors. For example, Hoffman & Hatch, 2000 found that higher ratings of depression were associated with the delivery of smaller infants, although only among women characterized by low SES, suggesting that these women are at an increased risk of negative outcomes (or, alternatively, that protective factors found among higher SES women offer a buffering effect). Plamondon et al. (2015) found that, for male offspring who received poor postnatal maternal care, negative life events experienced by mothers prenatally were associated with greater working memory errors on spatial tasks. However, this association was not present for male offspring who received high levels of maternal postnatal care. It is therefore possible

that many of the detrimental neurobiological consequences of maternal depression, anxiety, and stress can be mitigated by a positive rearing environment.

Similar and Differential Relationships

It is important to understand that the relationships of perinatal depression, anxiety, and stress to infant outcome do not exist in isolation; rather, they are interrelated. For example, Sutter-Dallay, Giaconne-Marcesche, Glatigny-Dallay, and Verdoux (2004) found that the presence of an anxiety disorder during pregnancy predicted the later occurrence of intense postnatal depressive symptoms, which were found to exist independently of prenatal maternal depression and other confounding factors. Heron, O'Connor, Evans, Golding, and Glover (2004) found that anxiety during the prenatal period was associated with a substantially increased risk of postnatal depression, even after controlling for prenatal depressive symptoms. Additionally, Field et al. (2003) discovered that of 132 prenatal women assessed at 20 weeks' gestation, those with high anxiety experienced high levels of both anger and depressive symptomatology. Therefore, while it is plausible that the relationship of prenatal distress symptoms may differ depending on type (i.e., anxiety vs. depression vs. generalized stress), they are also likely to overlap substantially and to contribute to the pathogenesis of one another.

Biopsychosocial Mechanisms

Maternal Relationships

As described earlier, reactions to stress lead to activation of the HPA axis and SAM systems, leading to the release of various hormones such as increases in production of corticotropin-releasing hormone (CRH), adrenocorticotropic hormone (ACTH), β-endorphins (BE), (nor)adrenaline, and cortisol (Lundy et al., 1999; Mulder et al., 2002; Ruiz, Fullerton, Brown, & Dudley, 2002; Ruiz & Avant, 2005; Sandman et al., 1994; Sandman et al., 1997;

Segerstrom & Miller, 2004; Wadhwa et al., 1996). It is important to note that there is a gradual increase in these hormones throughout pregnancy, even in the absence of additional stressors (Sandman et al., 1994). Higher levels of stress hormones, and altered levels of neurotransmitters, have been found in mothers suffering from depression. For example, compared to non-pregnant women, depressed women during the prenatal period have been found to have elevated levels of cortisol (Field et al., 2004; Lundy et al., 1999), and NE (Lundy et al., 1999), and lower levels of 5-HT (Field et al., 2004) and DA (Field et al., 2004; Lundy et al., 1999). Maternal neurotransmitter levels seem to be correlated with infant levels (Lundy et al., 1999). Furthermore, higher levels of stress hormones have been found in mothers experiencing elevated levels of psychosocial stress (Davis et al., 2007), suggesting that physiological stress systems are impacted by depression, anxiety, and perceived psychosocial stress, which may all interact. This research suggests that altered emotional states may be related to dysregulation in hormonal and neurotransmitter levels, which may be passed onto offspring.

Physiological dysregulation impacts one of the most important predictors of maternal and infant mortality, pregnancy length. Preterm birth is associated with premature activation of parturition (functional and structural changes occurring in the cervix, myometrium, and fetal membranes which lead to labor and delivery) via pathological processes occurring late in a normal pregnancy (McLean et al., 1995). Additional research suggests that early patterns of plasma CRH established by the end of the first trimester influence the subsequent timing of parturition and length of birth, referring to it as a "placental clock," and likening it to other biological clocks such as puberty (McLean et al., 1995). It is possible that elevated hormone levels of psychologically distressed mothers could impact either of these factors, leading to the

previously stated findings of premature births, and thus could potentially account for these "pathological processes."

Offspring Relationships

Both animal and human studies have found converging evidence that offspring factors (e.g., genetic vulnerability) play a role in the relationship between maternal perinatal distress/psychological symptoms and offspring outcome.

Animal studies. Animal research has indicated that prenatal stress is associated with a host of different outcomes on offspring. These include impediments in the acquiring of discriminative learning, shifting problem-solving strategies, and the demonstration of operant response skills, all of which are indicative of maladaptive learning abilities (Smith, Wills, & Naylor, 1981). Additionally, early maternal separation can alter 5-HT signaling and metabolism later in adulthood (Curley, Jensen, Mashoodh, & Champagne, 2011), and CRH injected directly into pregnant female rats has been found to lead to offspring who weigh less and have more ultrasonic vocalizations during isolation tests (Williams, Hennessy, & Davis, 1995), which is often considered an indicator of distress. However, it should be noted that some authors have found rat vocalizations as deriving from origins other than distress, such as a biological mechanism to increase intra-abdominal and venous pressure (IAP; Blumberg & Sokoloff, 2011). Furthermore, maladaptive thymus functioning (e.g., lighter thymuses, lower thymic cell numbers, and greater percentages of necrotic cells, all of which can affect the proper functioning of the immune system), deficient hippocampal development, and deficient cytokine responsivity in the immune system of infants of mothers experiencing chronic stress has also been noted (see Ruiz & Avant, 2005 for a review).

The design of most human studies does not allow one to estimate the relative biological and statistical contributions of environmental versus genetic effects on infant outcomes, nor their interaction effects. This is because genetic vulnerability factors passed from mother to child and chronic environmental stressors may both influence maternal stress and long-term outcomes, making them difficult to separate (Mulder et al., 2002). Additionally, animal studies allow researchers to control for extraneous variables, to perform more invasive experimental procedures than could be conducted in humans, and to increase our knowledge of mechanisms underlying the modalities of specific antidepressants on specific symptoms (Darnaudéry & Maccari, 2008). All of the major brain structures found in humans are also found in rodents, and serve in similar functional capacities (Rice & Barone, 2000).

Unfortunately, animal research does not necessarily translate into human research due to a variety of structural, functional, and biological issues, such as differential importance of the prefrontal cortex in humans, differential placentation, differential pregnancy lengths, etc. (Barry & Anthony, 2008; Mak, Evaniew, & Ghert, 2014; Martić-Kehl, Schibli, & Schubiger, 2012; Rice & Barone, 2000; Sasaki, Shinkawa, & Yoshinaga, 1990). Additionally, animal models may approximate human models in different ways, depending on the specific model utilized (Barry & Anthony, 2008; Carter, 2007). For example, mice tend to have immunological and endocrine differences from humans. Guinea pigs have less accelerated mutation rates compared to rats, yet occupy more space and have longer gestation times, making study difficult. Sheep have a longer in utero development, have fetuses of approximately the same weight as human fetuses, and are easy to handle. Conversely, they are phylogenetically removed from primates and have a different placentation compared to humans (Barry & Anthony, 2008; Carter, 2007). We can use animal experiments to inform hypotheses and follow-up research. For example, prenatal restraint

stress (PNRS) in rats leads to long-term deficits in multiple biological systems (e.g., structural abnormalities in hippocampus formation). These biological system deficits can lead to an increased stress response and abnormal sleep and circadian functions, which are similar to the biological correlates of depressed human (Darnaudéry & Maccari, 2008; Maccari et al., 2003).

Human studies. In humans, as in animals, there are likely complex interactions with specific genetic vulnerabilities of children increasing susceptibility to the experience of adverse effects of prenatal stress, although more studies are needed in this area (Bergman et al., 2007). For example, a child with a predisposed genetic vulnerability to poor stress regulation may develop adverse outcomes only if exposed to prenatal stressors; without this exposure, they would develop along the same trajectory as a child without a predisposed genetic vulnerability to stress. Specifically, oxytocin pathways have been shown to be influenced by variations in genetics, and can be programmed by exposure to hormones and social experiences (Carter, 2014). Sjöström et al. (1997) demonstrated that higher levels of maternal prenatal trait anxiety significantly affect fetal cerebral circulation, physiological consequences that could theoretically persist after birth and affect cognitive and emotional functioning. In a review of the human and animal literature, Sandman et al. (1994) discuss many of the effects of maternal prenatal biological correlates and their relation to infant outcomes. For example, rats exposed as fetuses to BEs, opioid peptides that contribute to the inhibitory feedback loop of CRH release after a stressor in humans (Huizink, 2001), experienced detriments such as decreases in striatal DA (D2) receptors, as well as in opiate receptor density.

Mechanisms of Action

While the relationship of perinatal depression, anxiety, and distress to infant outcome has been well researched, currently the methods in which they affect offspring is under investigation.

Despite no consensus, there are many theories describing the effects of maternal hormones on the fetus. There are no direct connections between the mother and fetus, with all communication being mediated through the placenta, which is fetal in nature (Wadhwa et al., 2002). It has been suggested that elevated levels of maternal hormones may cross the placenta thereby directly affecting the hormone levels of fetuses (Field et al., 2006; Gitau et al., 1998; Huizink et al., 2003; Lundy et al., 1999; Mennes et al., 2009; Mulder et al., 2002). Indeed, studies suggest that maternal cortisol may account for approximately 40% of the variance in fetal concentrations of cortisol during high stress conditions (Gitau et al., 1998), as the placenta is not entirely protected against direct exposure to high concentrations of cortisol (Mulder et al., 2002).

NE, although not yet shown to cross the placenta, could also affect the prenatal environment through its impact upon the cardiovascular system, with NE infusions being associated with increased arterial pressure and uterine artery resistance, as well as decreased fetal oxygenation and blood flow (Field et al., 2006). When the hormones described earlier are released following activation of the HPA axis and SAM system, cytokine production is increased, immune functioning is compromised, and a predisposition to premature labor, preterm delivery, and low birthweight is formed (Lundy et al., 1999; Mulder et al., 2002; Ruiz et al., 2002; Ruiz & Avant, 2005; Sandman et al., 1994; Sandman et al., 1997; Wadhwa et al., 1996). Increased BEs during pregnancy are associated with altered fetal heart rate (a measure sensitive to fetal central nervous system maturity) and habituation slopes to novel stimuli presented ex utero (Sandman et al., 1994). This suggests that the BEs affect the child prior to birth. Additionally, higher levels of maternal cortisol during pregnancy have been linked to infant reactivity and worse mental and motor development at 3 and 8 months (Buitelaar et al., 2003; Davis et al., 2007). Sandman et al. (1994) likened many of these effects on human offspring to

similar permanent changes observed in the offspring of animal studies. These show that offspring of stressed mothers experience enhanced behavioral reactivity to stressors later in life, decreased μ receptors, irritable temperament, increased reactivity of the HPA axis, and decreased immune functioning; changes in offspring brain and behavior that appear to last longer and occur in greater severity than similar exposures occurring to the individual during the neonatal or adult period.

Additionally, physiological responses to high levels of allostatic load produce significant changes in maternal immune responses, affecting pro-inflammatory cytokines (regulators of host responses to stressors that exacerbate disease; Dinarello, 2000). This could lead to differential effects on the fetus upon their crossing of the placenta or due to downstream effects of persistent inflammation, wherein activation of the stress system leads to a suppressed immunological response and decrease in production of specific cytokines and inflammatory mediators. This leads to the production of CRH, which is then used to regulate physiological stress and inflammatory responses (Ruiz & Avant, 2005). It is also plausible that high levels of prenatal maternal psychopathology and associated hormonal changes may increase uterine artery resistance and subsequently reduce uterine blood flow, thereby indirectly affecting behavioral development and infant outcomes such as birth weight (Huizink et al., 2003; Lundy et al., 1999; Mulder et al., 2002; Teixeira, Fisk, & Glover, 1999), or result in vasoconstriction and subsequent calorie and oxygen reduction to the fetus (Copper et al., 1996; Field et al., 2006).

A large component of the stress response consists of CRH, which regulates peripheral activities of the HPA axis (Ruiz & Avant, 2005). CRH has been found to be correlated with higher levels of anxiety in perinatal populations (Mancuso et al., 2004). At approximately 8-10 weeks' gestation, CRH is produced by the placenta. Placental CRH is identical to hypothalamic

CRH in structure, bioactivity, and immunoreactivity, and increases throughout gestation (Mulder et al., 2002; Ruiz & Avant, 2005; Sandman et al., 1997). Maternal anxiety, depression, and stress could affect the fetus through stress-induced release of placental hypothalamic CRH into the intrauterine environment, which further activates the fetal HPA axis, leading to dysfunction (Huizink et al., 2003; Mulder et al., 2002).

It is important to consider indirect relationships as well. For example, pregnant mothers experiencing higher levels of depression, anxiety, and stress may engage in more maladaptive health behaviors (e.g., smoking and drug use; Copper et al., 1996), which could thereby impact the developing fetus. Additional factors to consider include the indirect relationship of social support to birth outcome by reducing stress or anxiety, maternal anxiety on infant temperament problems mediated through its relationship to fetal behavior (Huizink, 2001), and maternal education/utilization of prenatal care on reducing infant mortality (Desai & Alva, 1998).

These biological mechanisms are particularly relevant for mothers suffering from OC symptoms. Specifically, women experiencing symptoms of OCD have been found to exhibit heightened endocrine stress responses, suggesting that higher levels of detrimental biological correlates of OCD could affect fetal exposure and associated infant outcomes (Lord, Steiner, Soares, Carew, & Hall, 2012). Therefore, although not specifically studied, it is plausible that many of the same detrimental effects caused by the biological correlates of symptoms of depression, anxiety, and stress may occur in infants of mothers suffering from OC symptoms as well, and perhaps to a greater degree.

Meta-Analytic Findings

Tarabulsy et al. (2014) found that studies reporting on more objective life events (i.e., the presence or absence of a stressful event occurring, such as trauma exposure and giving birth) for

the assessment of prenatal stress and anxiety yielded greater effect sizes than those related to more subjective measurements of prenatal stress and anxiety (e.g., asking mothers to retrospectively rate their overall levels of stress and anxiety during pregnancy on a self-report measure). Furthermore, measurements conducted after the birth of a child led to significantly greater effect sizes than measures assessed prenatally. This suggests that either postnatal assessments may inflate the relation between prenatal stress and anxiety and cognitive outcome and should be regarded with caution, or pregnant mothers may use cognitive coping strategies to help modify appraisals of stress and may be more objectively able to appraise their stress levels during the postnatal period. Additionally, it may also be a temporal effect, due to the fact that more robust relations are typically seen between measures taken at similar points in time. Unfortunately, a close examination of the relationship between pre- and postnatal measures of prenatal stress and anxiety has not yet been assessed, so these hypotheses remain conjecture at this time.

Timing of Effects

The timing of prenatal maternal distress may impact children in different ways. For example, van den Bergh and Marcoen (2004) found that maternal anxiety at 12 to 22 weeks' gestation was significantly associated with ADHD symptoms, externalizing problems, and anxiety in 8- and 9-year-olds, whereas maternal anxiety at 32 to 40 weeks was not. Furthermore, Hoffman and Hatch (2000) found that higher levels of depressive symptomatology overall were not directly related to fetal growth. However, higher levels of maternal depressive symptoms experienced during the second trimester were directly related to worse fetal growth in women with lower SES. Additionally, Mancuso et al.. (2004) found that higher levels of maternal

prenatal anxiety and CRH levels at 28 to 30 weeks' gestation were associated with earlier gestational age at birth, but not at 18 to 20 weeks.

While the aforementioned studies suggest that exposure in the 2nd or 3rd trimesters is associated with poorer outcomes in offspring, particularly stunting and preterm birth, Rodriguez and Bohlin (2005) found that higher levels of stress and smoking experienced by mothers during the first half of pregnancy, but not the second half, were linked to ADHD symptoms in boys. Laplante et al. (2004) found that, for mothers exposed to a traumatic environmental stressor, prenatal maternal stress during the first or second trimesters accounted for 28% and 41% of the variance of Bayley Mental Development Index (MDI) scores in their 2-year-old offspring, but only 1% when exposed during the third trimester. This suggests that the impact of maternal stress on child mental development may be greater in the first or second trimesters, in comparison to the third trimester. However, timing of the stressor in this study was not related to toddlers' language abilities. Further, Hedegaard et al. (1993) found that psychological distress in the 30th week of pregnancy was associated with risk of preterm delivery, but not during the 16th week. Finally, Buss et al. (2010) reported that prenatal anxiety measured at 19 weeks' gestation, but not 25 and 31 weeks' gestation, was associated with reduced gray matter volume in certain brain regions in 6- to 9-year-old children. It is possible that these differential findings may be due to biological stressors differing in the gestational period during which they will have their maximum impact upon the fetus. For example, stressors early during pregnancy, (approximately 6-18 weeks of gestation), during the major period of neurogenesis of cerebral cortical neurons, may cause more neural developmental damage than stressors later in pregnancy, which may be associated with more physical growth outcomes (Uylings, 2006). However, more studies need to be conducted to support this.

Infant Neurobehavior

Neurobehavior is a term used to encompass the specific interaction between physiology and temperamental behaviors, and includes different neurophysiological mechanisms that underlie psychological and behavioral processes (Lester & Tronick, 2004). The study of infant neurobehavior can be useful in identifying the effects of prenatal teratogens on newborn infants (Smith et al., 2008), and is critical in providing parents and clinicians with information about an infant's long-term health and behavioral outcomes (Liu et al., 2010). Scales of neonatal neurobehavior are often utilized to measure correlates of infant neurobehavior. The Brazelton Neonatal Behavioral Assessment Scale (NBAS) is one such measurement. Scores on the NBAS are factored into seven clusters: Habituation, Orientation, Motor, Range of State, Regulation of State, Autonomic Stability, and Reflexes (Brazelton & Nugent, 1995; Brouwers et al., 2001). On the NBAS, infants of prenatally anxious and depressed mothers have been found to have less optimal scores on the orientation (Abrams, Field, Scafidi, & Prodromidis, 2006; Brouwers et al., 2001; Diego et al., 2005; Field et al., 2004; Jones et al., 1998; Lundy, Field, & Pickens, 1996; Lundy et al., 1999), regulation of state (Jones et al., 1998), habituation (Diego et al., 2005; Field et al., 2004), motor (Diego et al., 2005; Field et al., 2003; Field et al., 2004), range of state (Field et al., 2004), autonomic stability (Field et al., 2003; Field et al., 2004), and reflex (Lundy et al., 1999) clusters. Poorer scores on these subscales are associated with many detrimental outcomes further on in an infant's life, including medical (e.g., higher levels of neurological and brain disease) and behavioral (e.g., higher levels of hypertonicity and excitability) outcomes at 4.5 years of age (Liu et al., 2010) and motor outcome (e.g., worse quality of movement and higher levels of lethargy) in 2 year olds (Stephens et al., 2010), and so represent a critical and efficient early measure of future life functioning.

The NICU Network Neurobehavioral Scale (NNNS) is another measurement of neonatal neurobehavior. The NNNS was developed as an assessment for at-risk infants and assesses neurological integrity, behavioral functioning, and signs of abstinence/stress (Lester, Andreozzi-Fontaine, Tronick, & Bigsby, 2014). The NNNS is administered in packages, with every package starting with a change in focus or position of the infant, with alternative orders of administration being allowed depending on infant characteristics (e.g., if the infant is not in an alert state, or cannot be brought to one when placed in a supine position with the examiner; Lester & Tronick, 2004). In infants, poor performance on the NNNS has been related to prenatal maternal substance use. Additionally, infants who score poorly on the NNNS are more likely to have medical problems such as congenital heart disease and to be born prematurely. Finally, in infants, poor NNNS scores predict difficult temperament at 3 months; poor mental and motor outcomes at 18 months; impaired Bayley Mental Development Index (MDI) scores at 1 and 2 years of age; poor motor status at 2 years; behavior problems at 3 years of age; chronic neurological abnormalities and brain-related illnesses by 3 years of age; motor, concept, and language school readiness difficulties at 4 years of age; and lower IQ at 4.5 years of age (Tronick & Lester, 2013; Lester et al., 2014).

Maternal-Child Interactions

In addition to the biological consequences of maternal psychopathology during pregnancy, environmental rearing consequences need to be described as well. In a review of the research conducted utilizing rat models, Meaney (2001) suggested that the behavior of mothers toward offspring can “program’ neuroendocrine and behavioral responses to adulthood. This thereby contributes to the effects of environment on infant outcomes, and these effects can alter the developmental trajectory of offspring into the next generation as well. Research suggests that

maternal care may have such long-lasting effects as increasing hippocampal N-methyl-D-aspartate (NMDA) receptor levels, which could lead to enhanced spatial learning in adulthood. It could also lead to the dampening of neonatal HPA activity, which can serve a protective function from catabolic effects of adrenal glucocorticoids during rapid development (Meaney, 2001).

In general, difficult mother-infant interactions are associated with a range of different outcomes. For example, infants who cry frequently have been found to have more distressed relationships and to evidence higher levels of interactional failures during face-to-face encounters with their mothers as compared to infants who do not cry frequently (Papousek & von Hofacker, 1998). Additionally, taking measurements during the first 10 days of life, Crockenberg and Smith (1982) discovered that maternal attitudes of inflexibility and unresponsiveness, along with the length of time it took them to respond to fuss/cry signals, predicted the amount of time an infant cried and fussed during measurement at 3 months. Mothers experiencing psychiatric illnesses experience more difficult mother/infant interactions that can lead to impaired infant development (Brandes, Soares, & Cohen, 2004).

While maternal perinatal psychological distress may impact maternal-child interactions, it is important to discuss the opposite aspect as well. Indeed, environmental factors have proven to negate many potentially threatening genetic and behavioral predispositions present in children. For example, in his longitudinal study of infant reactivity from 4 months to 4.5 years of age, Kagan (1997) found that only a modest proportion of infants originally deemed highly reactive or under-reactive at 4 months maintained extreme forms of their theoretically expected profile to 4.5 years of age. Kagan suggested that this was due to the influence of intervening familial experiences.

Finally, the effects of gene-environment interactions (GxE) are of importance. A GxE occurs when exposure to environmental pathogens are conditional on an individuals' genotype, or when they moderate the effects of genes on health (Moffitt, Caspi, & Rutter, 2005). It is plausible that many of the detrimental biological effects upon a fetus during pregnancy may not cause damage in-and-of themselves. However, by placing a child at higher risk via a genetic vulnerability, impaired maternal-child interactions may be just the environmental stressor needed to lead to different outcomes for infants later in life. These interactions may not affect an infant without the same type of genetic predisposition. Existing studies have focused on GxE regarding maternal perinatal depression. For instance, higher levels of maternal postnatal depression are correlated with children's behavioral/emotional problems at 4 years of age, independent of antenatal mood (O'Connor et al., 2002a), and depressed mothers tend to exhibit withdrawn or intrusive interaction profiles, which are associated with infant outcomes due to inadequate arousal modulation and stimulation (Field, 1998). Even after birth, postnatal maternal depression has been found to be associated with certain properties, such as poorer cognitive outcome of children at age 4 (Cogill, Caplan, Alexandra, Robson, & Kumar, 1986). This is of concern due to high levels of prenatal diagnostic comorbidity, and could provide additive effects on an infant, above and beyond the effects of depressive or anxiety symptoms on parental rearing style. Currently, no study has investigated the interplay between neurobiological and environmental rearing correlates comparing mothers suffering from elevated symptom levels of depression, anxiety, and stress. Therefore, this represents a fruitful area of research that could have significant implications for the prevention and treatment of at-risk families.

Obsessive-Compulsive Disorder

Obsessive-Compulsive Disorder (OCD) is a constellation of symptoms characterized by

recurrent obsessions and/or compulsions that are severe enough to be time consuming or to cause marked distress in critical areas of functioning, such as occupational or social domains (American Psychiatric Association, 2013). Specifically, obsessions are persistent and intrusive thoughts or images that an individual is unable to remove from one's head, while compulsions are repetitive behaviors (including repetitive thoughts) that are enacted to neutralize the intrusive thoughts. Lifetime diagnostic prevalence rates of OCD have been found to be around 1% to 3% (American Psychiatric Association, 2013; Kolada et al., 1994). These symptoms are highly disabling (Pollitt, 1957) and comorbid with other disorders, particularly affective and anxiety disorders (Toro, Cervera, Osejo, & Salamero, 1992). In general, OCD is considered to be affected by the serotonergic system. This is evidenced by pharmacologic challenges (i.e., administration of 5-HT agonists that occasionally lead to increased OC symptoms), the pharmacological actions of medications (i.e., prolonged administration of 5-HT agonists being associated with therapeutic improvement in OC symptoms), and genetic studies (i.e., the gene that codes for 5-HT receptors may be preferentially transmitted to individuals diagnosed with OCD), although further studies are need to identify specific 5-HT receptors associated with OCD (Zohar, Kennedy, Hollander, & Koran, 2004).

However, much of the research on OCD does not take into account subthreshold OCD symptoms, nor does it consider other OC-spectrum disorders that share characteristics with OCD (McElroy, Phillips, & Keck, 1994). Indeed, past literature has suggested that obsessive-compulsive (OC) symptoms may be widespread and bothersome (Rachman & de Silva, 1978; Salkovskis & Harrison, 1984; Frost, Sher, & Geen, 1986), with more recent research confirming these data by examining larger community samples. Specifically, Fullana et al. (2009) followed 1,037 individuals from 3 to 32 years of age, with a majority (96%) being available for study at

long-term follow-up. When interviewed at 26 and 32 years of age, obsessions and compulsions were frequently found among individuals with mental health diagnoses other than OCD (31% to 49%), as well as among individuals without mental health diagnoses (13% to 17%), indicating a much higher prevalence rate of OC symptoms than expected based upon reported OCD prevalence rates. Fullana et al. (2009) estimate that approximately 21% to 25% of the population has clinically significant obsessions and compulsions. While not meeting diagnostic thresholds for diagnosis, these symptoms are nonetheless impairing. Indeed, Spinella (2005) found a significant positive relationship between the severity of subclinical OC symptomatology and participants' negative emotional states, suggesting that even subthreshold OC symptoms are associated with different socioemotional outcomes.

It is interesting to note that gender can account for discrepancies in the expression of OC symptoms as well. In a study utilizing 186 outpatients with DSM-IV diagnoses of OCD, Labad et al. (2008) found that contamination/cleaning subtypes of OCD were higher in women, and sexual/religious obsessions were higher in men, with all other subtypes being equal across gender. Importantly, the authors held variables such as age of onset, history of tic disorders, and severity of depression and OC symptoms constant, as some of these variables have been demonstrated to influence phenotypic expression of OC symptoms. Due to the current study's exclusive focus on OC symptoms in perinatal women, gender differences are important to understand and conceptualize.

There is evidence to support the idea that different subtypes of OCD are modulated by distinct but partially overlapping neural systems. For example, in a symptom provocation study, Mataix-Cols et al. (2004) found that patients with OCD evidenced significantly greater activation than individuals without OCD in the bilateral ventromedial prefrontal regions and the right

caudate nucleus during washing symptom provocation. However, during the checking symptom provocation, individuals with OCD evidenced significantly greater activation in the putamen/globus pallidus, thalamus, and dorsal cortical areas than individuals without OCD. This suggests that OCD may best be conceptualized as a spectrum of several different, potentially overlapping areas, rather than a single specific dysfunction. Taken together, it is therefore plausible that the different subtypes of OCD in men versus women may be caused by different neural processes and biological vulnerabilities.

Perinatal OC Symptoms and Infants

It is common for mothers suffering from OC symptoms to experience potential dysfunction in their maternal roles (Arnold, 1999). Specifically, new mothers experiencing recurrent and intrusive thoughts of causing harm to their baby may avoid or minimize contact with them (Chelmow & Halfin, 1997; Ross & McLean, 2006; Sichel, Cohen, Dimmock, & Rosenbaum, 1993). Additionally, these mothers experience excessive levels of suffering and dysfunction that can lead to interruption of typical mother-infant bonding (Brandes et al., 2004), thereby impairing offspring development. For example, Weinberg and Tronick (1998) found that mothers in treatment for OCD were more disengaged with their infants, talked less to their infants, displayed fewer facial expressions of interest, were less likely to share joint focus with an infant on their objects of attention, and touched them less than mothers without OCD.

Furthermore, the relationship of OC symptomatology to infant outcomes may differ from the relationship of perinatal depression, anxiety, and stress to infant outcomes. Although research on the differential impact of diverse perinatal disorders is limited, some researchers have begun to explore this area. For example, Field and Diego (2008) investigated 33 mothers with dysthymia and 39 with major depression, and found that the dysthymia group had infants with

lower birthweight, lower birth length, lower gestational age, more obstetric complications, and more postnatal complications. These infants performed worse on the orientation, motor, and depression modules of the NBAS than did those with mothers with major depression, suggesting that length of depression may be a risk factor for altered fetal development and neonatal outcomes. Further, Buss et al. (2011) found that pregnancy-specific anxiety (i.e., anxiety surrounding potential problematic occurrences during pregnancy, such as medical complications, as assessed with the pregnancy related anxiety scale) was a stronger predictor of impaired executive functioning in offspring than prenatal depression or state anxiety [i.e., anxiety experienced at that moment in time, as assessed with the State Trait Anxiety Inventory (STAI)]. This suggests that pregnant mothers with different disorders may have differentially functioning HPA axes and stress responses.

This could impact the fetus in diverse ways. In an investigation of anxiety disorders, Vreeburg et al. (2010) found that, independently of the presence of comorbid major depressive disorder, panic disorder with agoraphobia was associated with an increase in cortisol awakening response (CAR). CAR is a measure of early-morning cortisol level peak linked to greater psychophysiological stress and associated health outcomes in perinatal women (Fries, Dettenborn, & Kirschbaum, 2009). However, panic disorder, GAD, and social phobia were less associated with CAR. In fact, when comorbid major depressive disorder was controlled for, individuals with GAD and social phobia showed no difference in CAR than individuals without a psychological disorder. As the focus of OC symptoms during pregnancy and afterwards are often directed toward the child (e.g., “I’m going to push my child out into the stress”), it is possible that OC symptoms related directly to pregnancy could signal more detrimental infant outcomes. However, no study to date has investigated the differential impact of OC symptoms on infant

outcomes as compared to the symptoms of depression, other anxiety, and stress. Despite this, there are unique aspects of OCD which may result in differential outcomes on infants than other disorders.

Unique Aspects of OCD

OCD symptomatology is thought to have a stronger neurobiological basis than many other disorders, being closely associated with other neurological conditions (e.g., Tourette's syndrome) and cortico-striatal-thalamic-cortical circuits, and with symptoms varying little across age, culture, time, and gender (Ross & McLean, 2006). OC symptoms may have a differential presentation from that of depressive and anxious symptoms. For example, utilizing 80 case and 73 control probands, and 343 case and 300 control first-degree relatives of these probands, Nestadt et al. (2001) found that individuals experiencing symptoms of OCD were more likely to have relatives with GAD, agoraphobia, social anxiety disorder, panic disorder, and recurrent major depression. Furthermore, they were more likely to have a relative with either GAD or agoraphobia regardless of the presence of OCD in these relatives. However, they were not more likely to have a relative with social anxiety disorder, panic disorder, separation anxiety disorder, specific phobia, schizophrenia, alcohol dependence, substance dependence, bipolar disorder, dysthymia, recurrent brief depressive disorder, or recurrent major depression if these relatives did not also present with a comorbid OCD diagnosis. This could potentially suggest a common familial etiology, possibly genetic, for OCD, GAD, and agoraphobia, but not for the others.

Although a role for the HPA axis in OCD has been suggested, the exact nature of this relationship is not currently clear. Some studies, utilizing lumbar punctures and hypertonic saline infusions, have found higher levels of cerebrospinal fluid (CSF) CRH (which has been linked to infant outcomes as described earlier) in individuals with OCD symptoms compared to those with

other disorders (Altemus et al., 1992). However, others have not found this difference (Chappell et al., 1996). Delineating this relationship is important due to the infant outcomes described earlier that are associated with heighted levels of CRH. If the HPA axis is over-active or under-active in OCD compared to other psychological disorders, it could lead to differential effects on the infant in utero via various levels of CRH. Additionally, higher levels of nocturnal ACTH have been found in individuals with OCD compared to individuals without OCD (Kluge et al., 2007), and higher levels of cortisol have been found in individuals with OCD compared to individuals without OCD (Gehris, Kathol, Black, & Noyes Jr., 1990; Gustafsson, Gustafsson, Ivarsson, & Nelson, 2008; Kluge et al., 2007; Monteleone, Catapano, Del Buono, & Maj, 1994; Monteleone, Catapano, Tortorella, & Maj, 1997). This suggests that OCD symptoms may be associated with altered activity of the HPA axis.

Additionally, altered circadian profiles of melatonin and prolactin have been found to be directly related to the severity of OC, but not depressive, symptoms (Monteleone et al., 1994). Similar melatonin and cortisol circadian patterns have been found among individuals with OCD as compared to individuals without OCD, although at respectively lower and higher levels (Catapano, Monteleone, Fuschino, Maj, & Kemali, 1992). Catapano et al. (1992) suggested that this lowered secretion of melatonin may be considered an effect of disturbed serotonergic functioning in the brain, which has been postulated as a mechanism in the development of OCD symptoms. These findings could suggest different underlying biological processes between individuals with OCD and other psychiatric disorders, and individuals with OCD and those without a psychological disorder. Due to the associations with melatonin and potential abnormal brain and ocular development (Gitto, Pellegrino, Gitto, Barberi, & Reiter, 2009), this suggests that mothers experiencing symptoms of OCD during the perinatal period may experience

different underlying biological processes than individuals with other psychiatric disorders or individuals without a psychiatric disorder. This could lead these mothers to have infants with slightly different outcomes than healthy mothers or mothers experiencing symptoms of other disorders.

Of interest, Gustafsson et al. (2008) found decreases in cortisol (near-significant) among children with OCD symptoms in response to a psychological stressor, as compared to an increase in cortisol among healthy children. Although they indicate that this could be due to possible confounding of the data, it may be an example of HPA dysregulation in children with OCD, as other studies with samples of situationally and socially phobic patients have found hyperresponsiveness of cortisol secretion in response to stressors (Alpers, Abelson, Wilhelm, & Roth, 2003; Condren, O'Neill, Ryan, Barrett, & Thakore, 2002). Gustafsson et al. (2008) also found higher early-morning cortisol values in individuals with OCD as compared to individuals without OCD, with no differences between evening and late-morning values. These results contrast with the lower nighttime cortisol secretions found between individuals with and without OCD symptoms, yet similar levels during the day (Feder et al., 2004). This suggests that individuals with OCD may have different neurobiological correlates than those with other disorders, which could be associated with differential infant outcomes.

It has been suggested that the abnormalities of HPA functioning in OCD may be influenced by factors such as hospitalization, comorbidity, severity, and subtype (Gustafsson et al., 2008). This could possibly account for some of the discrepant findings, particularly given gender differences in OCD prevalence and distribution of subtype. It is possible that these elevated physiological and endocrine responses may arise from stressful experiences encountered by individuals with OCD. This is due to the fact that physical and psychological stress

experiences can initiate endocrine responses via activation of the HPA axis, and individuals with OCD have been shown to significantly suffer more from daily life stressors than individuals without OCD (Kluge et al., 2007). Although more research needs to be conducted, it appears that the HPA axis may partially account for the pathophysiology of OCD symptoms.

Oxytocin (OT), a cyclic polypeptide released from the pituitary gland and responsible for uterine contractions during labor and lactation, is also emerging as a possible correlate with OCD, and especially a hormone-related subtype of OCD. Specifically, there may be a subgroup of women who experience a distinct sensitivity to reproductive hormones, thus leading reproductive events to exacerbate or induce OC symptoms in this subgroup of women (Forray et al., 2010). This is due to the potential effects of OT on cognitive, affiliative, grooming, and sexual behaviors and how they are disrupted in certain presentations of OCD symptoms (Forray et al., 2010; Labad et al., 2005; Leckman et al., 1994a; Leckman et al., 1994b). OT has been demonstrated to affect the excitability of neurons in several regions of the brain, and the range of symptomatology seen in patients with OCD symptoms is similar to the range of behavioral effects evidenced in individuals after the central administration of OT (Leckman et al., 1994a). For example, centrally administered OT leads to increased grooming behaviors in animals, particularly in the head and anogenital region, which have been associated with OC symptomatology (Leckman et al., 1994a).

Additionally, OT is associated with the initiation of maternal behavior (Leckman et al., 1994a). For example, injection of OT has been found to lead to induction of maternal behaviors (e.g., grooming and arched-back nursing) in rats, with most of those displaying these behaviors in the last day of diestrus, or in proestrus or estrus. This suggests that elevated estrogen levels may be necessary for induction of maternal behavior by OT (Meaney, 2001; Pedersen & Prange,

1979). Leckman et al. (1994b), investigated levels of OT in 29 individuals with OCD, 23 with Tourette's syndrome, and 31 without a psychological disorder, and found that levels of OT were increased in a subset of patients (*n*=22) with OCD who indicated being without a personal or familial history of tic disorders. The CSF OT levels of this subset of 22 individuals with OCD were correlated with their current OC symptom severity. The lack of distinct findings of OT's role with regard to OCD symptoms up to this point may be related to the heterogeneity of the OCD diagnostic category. If OCD symptoms are indeed etiologically heterogeneous, then failure to group individuals with OCD accordingly (e.g., tic-related OCD vs OT-related OCD, perinatal-onset vs non-perinatal-onset, etc.) may lead to ambiguous results (Leckman et al., 1994a).

Finally, OCD may present in a behaviorally distinct manner from other disorders. For example, mothers suffering from postnatal depression experience a range of interactional impairments with their infants, often in the form of avoidance or impaired responsiveness to infant cues (Murray & Cooper, 1997). While these interactions may occur among mothers with OCD symptoms, additional interactions are likely to occur as well, depending upon the specific OC symptomatology. For example, a mother obsessively concerned over her baby's safety may check on them excessively, and therefore be over-engaged with the infant compared to mothers with depressive symptoms or no psychological symptoms. This may potentially be associated with different infant outcomes.

In summation, there is preliminary evidence to suggest that OC symptoms may have a different underlying neurobiological basis than other disorders. This difference could potentially be associated with variations in prenatal factors that have been shown to affect infant outcomes (e.g., CRH, ACTH, etc.). Therefore, while it is possible that mothers experiencing severe OC symptoms may have infants with greater levels of reactivity than mothers without OC symptoms,

it is also plausible that these infants may appear different from infants born to mothers experiencing symptoms of psychological disorders other than OCD. These variations among infants could be reflective of different in-utero biological effects they experienced. It is also plausible that different interaction styles between mother and infant after birth could be associated with different longitudinal outcomes. Particularly, OC symptoms have been associated with less confidence in parenting abilities, enjoyment of parenting, and maternal sensitivity (Challacombe et al., 2016), the last of which is related to infant emotional reactivity (Spinrad & Stifter, 2002). While this may be due to depression that is commonly associated with OC symptoms in this period, the current study sought to determine whether there was a direct relationship with OC symptoms and infant reactivity, while controlling for the supported effect of postpartum depression.

Maternal Perinatal-Onset OCD

Although far less researched than perinatal depression, anxiety, and stress, the understanding that the perinatal period is associated with OCD has been around since at least the 1950's (Pollitt, 1957). However, it has only recently begun getting more attention from researchers. A discussion regarding the relationship of maternal perinatal-onset OCD and infant outcomes would not be complete without a discussion of perinatal-onset OCD in general, due to the perinatal period being associated with an increased risk of onset of OCD and OC symptoms (Abramowitz, Schwartz, Moore, & Luenzmann, 2003b; Abramowitz, Schwartz, & Moore, 2003a; Agrati et al., 2005; Arnold, 1999; Fairbrother & Abramowitz, 2007; Forray et al., 2010; Labad et al., 2005; Neziroglu et al., 1992; Ross & McLean, 2006; Uguz et al., 2007; Wenzel et al., 2005; Williams & Koran, 1997; Zambaldi et al., 2009), and an increase in the exacerbation of pre-existing OC symptoms (Agrati et al., 2005; Arnold, 1999; Brandes et al., 2004; Forray et al.,

2010; Labad et al., 2005; Vulink et al., 2006; Wenzel et al., 2005; Williams & Koran, 1997; Wisner, Peindl, Gigliotti, & Hanusa, 1999). Prior research suggests that the prevalence of OCD during pregnancy is around 0.2%-1.2%, and 2.7%-3.9% during the postpartum period (Ross & McLean, 2006).

Maternal perinatal-onset subtypes. Maternal perinatal-onset OCD may differ in clinical presentation than non-perinatal-onset OCD (Labad et al., 2010). For example, contamination/cleaning subtypes of OCD are more prevalent with perinatal onset (Labad et al., 2010; Real et al., 2011). Labad et al. (2010) likened this to the fact that OCD symptom dimensions may be mediated by different neural circuits, which differ by gender and the levels of sex steroid hormone receptors present. The authors indicated that individuals with different subtypes of OCD may differ in their susceptibility to having pregnancy-induced OCD. Further, OCD symptom onset may be linked to the occurrence of miscarriages, more so than panic disorders, phobic disorders, or agoraphobia (Geller, Klier, & Neugebauer, 2001).

Elevated levels of aggressive obsessions have also been documented among mothers in the postpartum period (e.g., putting the baby in the microwave, stabbing them, throwing them over a railing, etc.; Arnold, 1999; Ross & McLean, 2006; Wisner et al., 1999). However, these intrusive aggressive thoughts in mothers are not associated with an increased risk of harming the infant (Fairbrother & Abramowitz, 2007). Some authors have even found a higher prevalence rate of aggressive obsessions among women with perinatal OCD than women with non-perinatal OCD (Uguz et al., 2007). It is important to note that aggressive obsessions may be difficult for the mother to discuss with others, which may lead to underreporting (Jennings, Ross, Popper, & Elmore, 1999; Pedersen & Prange, 1979). However, there does not appear to be a difference between perinatal and non-perinatal OCD and degree of insight (Uguz et al., 2007). Others have

found greater levels of contamination obsessions in the perinatal period as compared to non-perinatal onset OCD (Pedersen & Prange, 1979).

Comorbidity. It is common for individuals with a diagnosis of OCD to experience depressive symptoms (Abramowitz et al., 2003a; Arnold, 1999; Sichel et al., 1993; Williams & Koran, 1997; Zambaldi et al., 2009), which could be associated with additional detrimental outcomes, as described previously. It should also be noted that, even without a diagnosis of OCD, OC thoughts and behaviors are likely to occur among mothers experiencing depressive symptoms, with a prevalence rate of approximately 40-60% (Abramowitz et al., 2003b; Brandes et al., 2004; Fairbrother & Abramowitz, 2007; Ross & McLean, 2006; Wenzel et al., 2001; Wisner et al., 1999). This is typically higher than the levels of OC symptomatology seen in those mothers without a depressive disorder diagnosis (Jennings et al., 1999), and in those whose depression occurs outside of childbearing (Wisner et al., 1999). Additionally, individuals with comorbid depression/OCD may be at higher risk of perinatal onset and worsening of OC symptoms compared to non-depressed individuals with OCD (Labad et al., 2005; Vulink et al., 2006). In sum, the comorbidity between depression and OCD and associated complications place individuals with OC symptoms in the perinatal period at increased odds of detrimental consequences to mothers and offspring.

Mechanisms/mediators. It has been suggested that the perinatal period may be associated with an increased risk of OC behaviors due to changes in gonadal steroids (Brandes et al., 2004; Uguz et al., 2007; Williams & Koran, 1997). For example, estrogen has a modulatory effect on the serotonergic system, which has been implicated in the pathogenesis of OCD (Rubinow, Schmidt, & Roca, 1998). Furthermore, Vulink et al. (2006) suggested that gonadal steroids may indirectly influence OCD symptom severity via their effect on DA functioning.

Additionally, perseverant behavior induced in rats via 8-hydroxy-2-(di-*n*-propylamino)tetralin (8-OH-DPAT; a 5-HT agonist that induces perseverative behaviors in rats) induced different levels of perseverant behavior in female rats depending on their reproductive stage This suggests that, as described prior with regard to the concept of the timing of maternal prenatal distress, OC symptoms may present/manifest themselves in different ways depending on the stage of pregnancy(Agrati et al., 2005; Fernandez-Guasti, Agrati, Reyes, & Ferreira, 2006). It is possible that this may be a "hormone related" subtype of OCD, with a subtype of women who have a different level of sensitivity to reproductive hormones being more likely to develop OC symptoms or experience exacerbation of OC symptoms (Forray et al., 2010).

Leckman et al. (1999) suggests that dysregulation of early parental preoccupations and behaviors (EPPB; i.e., caregiving, relationship building, and anxious intrusive thoughts and harm avoidant behaviors) may be associated with some forms of OCD. The authors indicated several lines of evidence to support this. Namely, OCD symptoms are more likely to emerge and exacerbate during the last weeks of pregnancy and the postpartum period, there are similarly operating neurobiological circuits and pathways that are activated during both maternal behavior and symptoms of OCD, there is a similarity between symptoms of OCD and EPPB, and there is notable similarity between adjustments made to accommodate newly born children and family and the pathological accommodations made to accommodate close family members with OCD. Indeed, the authors found that the birth of a new child is associated with altered mental states in both parents, and includes heightened sensitivity, preoccupation, and responsibility. Therefore, while these thoughts appear normal during pregnancy, should they begin to generate distress, suppression attempts, and ideas that they should not have such thoughts, OC symptomatology could emerge.

Another reason why there may be a higher rate of OC symptoms during the perinatal period is due to the stressful nature of pregnancy, labor, delivery, and familial adjustment, given the correlation of OCD onset during periods of stressful life events (SLE; Cromer, Schmidt, & Murphy, 2007; McKeon, Roa, & Mann, 1984; Gothelf, Aharonovsky, Horesh, Carty, & Apter, 2004; Toro et al., 1992). SLEs have also been related to more severe clinical expression of OC symptoms (Cromer et al., 2007). In general, individuals with OCD report more SLEs, perhaps even more so than those with other anxiety disorders (Gothelf et al., 2004). Interestingly, some authors have indicated no relationship between OCD onset and a higher number or severity of general SLEs. However, when specific SLEs were evaluated, a relationship between a specific SLE (birth of a live child) and OCD emerged (Albert, Maina, & Bogetto, 2000; Maina, Albert, Bogetto, Vaschetto, & Ravizza, 1999).

Postpartum model. Fairbrother & Abramowitz (2007) proposed a cognitive-behavioral model of postpartum OCD that maintains that individuals who have recently become parents are vulnerable to the development of a subset of OC symptoms due to aspects of new parenthood that confer vulnerability to these symptoms. Specifically, they propose the following: the perinatal period lowers the threshold for development/exacerbation of OCD by bringing about a dramatic increase in responsibility for the infant. This increased responsibility primes the mother for misinterpretations of normally occurring, intrusive infant-related thoughts as being threatening, significant, and requiring attention, and leads to behavioral patterns developed in response to the intrusive thoughts (e.g., checking repeatedly on the child) that contribute to the maintenance of distress. Support for this model can be seen in previous research that demonstrates propositions of exaggerated estimates of responsibility for harm prevention to be important in the development and maintenance of OCD symptomatology. Support can also be

seen from cognitive models that propose the tendency to overestimate the likelihood and severity of threat as underlying the development of OCD symptoms, and higher rates of intrusive, unwanted thoughts that occur during times of excessive life stress.

Importance of Studying Infant Temperament

Longitudinal studies over the course of decades can be costly and unwieldy, making them difficult to conduct. As discussed previously, the study of neurobehavior can provide information regarding long-term infant outcomes. Although similar, temperament and neurobehavior refer to slightly different processes. Temperament can be described as an initial state of personality development characterized by individual differences in reactivity of emotional, motor, and attentional responses. These individual differences are conceptualized as being linked to underlying neural networks, and grow into a more complex personality profile which comes to encompass higher order cognitions, values, and attitudes later in life (Rothbart, 2007). As described previously, neurobehavior encompasses the specific interaction between physiology and these temperamental behaviors, and includes different neurophysiological mechanisms that underlie psychological and behavioral processes (Lester & Tronick, 2004). Infant neurobehavior and toddler temperament are associated with outcomes many years after initial assessment. For example, individuals classified as inhibited as toddlers displayed higher levels of inhibition and shyness when assessed at 4 and 7 years of age (Pfeifer, Goldsmith, Davidson, & Rickman, 2002). Additionally, higher levels of infant reactivity (defined as those infants who become distressed and active in response to external stimuli such as moving brightly colored toys back and forth in front of their faces) at 4 months has been linked to higher levels of fearfulness at 14 and 21 months (i.e., inhibited children) and lower levels of spontaneity and sociability at 5 years of age (Kagan, 1997; Kagan, Snidman, & Arcus, 1998). Via fathers' self-reports, higher levels of

activity in infancy predict high levels of anger and frustration, and low levels of soothability and inhibitory control in middle childhood. Additionally, higher levels of smiling and laughter in infancy predict smiling, laughter, higher levels of attentional focusing, low-intensity pleasure, and soothability in middle childhood as well (Komsi et al., 2008). On the other hand, anger/frustration, fear, and low levels of soothability and inhibitory control in middle childhood were predicted by high levels of distress to limitation in infancy. Additionally, perceptual sensitivity, low-intensity pleasure, smiling, and laughter in middle childhood were predicted by higher levels of fear in infancy (Komsi et al., 2008).

Many of these measurements of inhibition are subsequently associated with the development of anxiety disorders. Specifically, early adolescents who had been classified in infancy as inhibited were more likely to have social phobia than those infants classified as uninhibited, although separation anxiety, compulsive symptoms, and specific phobias were not differentiated among adolescents who were classified as either inhibited or uninhibited during infancy (Kagan, 1997; Kagan, Reznick, & Snidman, 1988). Furthermore, infant reactivity at 6 months is associated with children's school marks at 6 years of age (Niederhofer & Reiter, 2004), and inhibition in 2-year-olds is related to generalized social anxiety during adolescence (Schwartz, Snidman, & Kagan, 1999). In addition, infant irritability assessed during the first 10 days of life is associated with lengthier period of time to calm and irritability at 1 and 3 months (Crockenberg & Smith, 1982). Two-year-old, highly restrained infants are quieter and more socially avoidant with unfamiliar individuals at 7 years of age, as compared to higher levels of talkativeness and interaction of 7-year-olds classified as more spontaneous at 2 years of age (Kagan et al., 1988). To date, no known study has investigated the impact of maternal perinatal OC symptoms on infant behavior, despite its relation to long-term developmental outcomes.

Current Study

The current study adds to the existing literature in several ways. There is a dearth of research into perinatal OC symptomatology despite its associated harmful relationships. This research is needed due to the likelihood that distinct environmental and biological correlates may underlie OC symptomatology that are not present in anxious and depressive disorders. It has been well established that certain environmental and biological characteristics of pregnant mothers are linked to infant behavior. Therefore, mothers with OC symptoms may be at risk for experiencing differential psychological and biological stressors, and may have infants who present with different behavioral profiles than those of mothers with anxiety and depression. Due to the link between behavior and later outcome, understanding this link between maternal OC symptoms and infant behavior is critical in order to provide psychoeducation for and to assist expectant mothers, identify appropriate treatments and interventions, and to improve long-term infant outcome. Accordingly, it is hypothesized that maternal prenatal OC symptomatology, while controlling for maternal depressive symptomatology, will be related to infant behavior at 6 months. In particular, mothers with higher levels of prenatal OC symptomatology will have infants with higher levels of behavioral reactivity. Additionally, it is hypothesized that SES will moderate this relation, due to the findings that higher levels of SES operate as a buffering agent against early developmental stressors (Blumenshine et al., 2010). In other words, higher parental SES levels are expected to prevent against many of the prenatal biological stress reactivity changes that occur and lead to different infant outcomes. Therefore, it is hypothesized that the correlation of infant behavior and maternal OC symptomatology will be weaker for mother/infant pairs from higher SES backgrounds than for mother/infant pairs from lower SES backgrounds. Similarly, maternal postnatal OC symptomatology, while controlling for maternal

depressive symptomatology, will be related to infant behavior at 6 months. Particularly, mothers with higher levels of postnatal OC symptomatology will have infants with higher levels of behavioral reactivity. Additionally, it is hypothesized that SES will also moderate this relation, such that the correlation of infant behavior and maternal OC symptomatology will be weaker for mother/infant pairs from higher SES backgrounds than for mother/infant pairs from lower SES backgrounds.

CHAPTER II

Methodology

Procedure

The Idaho State University (ISU) Human Subjects Committee approved this study for use with the perinatal population in and around the Pocatello, Idaho area (See Appendix A for the original approval letter). We recruited participants through the efforts of a larger, longitudinal study of the influence of maternal perinatal health and behavior on infant development, the Infant Development and Healthy Outcomes in Moms (IDAHO MOM) Study. For my dissertation, I wrote a modification to incorporate the Prenatal Obsessive-Compulsive Scale (POCS-Pre), Postnatal Obsessive-Compulsive Scale (POCS-Post), and Yale-Brown Obsessive-Compulsive Scale – Self-Report Version (Y-BOCS) into the study. Specifically, we recruited participants from local news advertisements, medical centers, family service/product providers, OB/GYN providers, Idaho State University (ISU) women's resource centers, Facebook, ISU's Sona system, prenatal and family courses, and ISU health/research centers. The recruitment materials utilized involved research flyers, Laboratory Business Cards, and trifold brochures. Following a prospective participant's confirmed interest in the study, they received a brief phone call from a trained research assistant (RA) in order to provide them with more information about

participation in the study, and to determine their eligibility for the study using a semi-structured interview. The interviewer provided the potential participants with an overview of the study's purpose, informed consent, benefits of participation, potential risks, and ability to withdraw without penalty and to skip interview questions.

Prenatal session. As part of the larger IDAHO MOM study, during the prenatal session, participants first completed an interview regarding their current and past pregnancy-related information, a brief health history, as well as questions surrounding sociodemographic characteristics. Furthermore, they answered examiner questions surrounding psychological symptoms on the Mini International Neuropsychiatric Inventory Version 6.0 (M.I.N.I. 6.0), and questions surrounding substance use on the Timeline Followback (TLFB; i.e., caffeine, alcohol, nicotine, second-hand smoke, other drugs, and other tobacco products). This took approximately 40 minutes. Mothers then completed anthropometric measurements (i.e., weight, height, and abdominal circumference), which took approximately 10 minutes. Subsequently, mothers completed several electronic or paper-and-pencil self-report questionnaires, including the Perceived Stress Scale (PSS), Dietary Screening Questionnaire (DSQ), Eating Behavior Questionnaire (EBQ), Perinatal Anxiety Screening Scale (PASS), POCS-Pre, Y-BOCS, Severity of Violence Against Women Scale (SVAWS), Trauma History Questionnaire (THQ), Domain-Specific Risk-Taking Scale (DOSPERT), and the Infant Crying Questionnaire-Prenatal Version (ICQ-PN), taking approximately 40 minutes. We provided participants who endorsed critical items with a mental health resource list which we reviewed with them. Suicidality or other imminent risk disclosure was addressed via consultation with the larger study's Principal Investigator, Dr. Nicki Aubuchon-Endsley. Finally, we provided expectant mothers instructions for collecting saliva samples over 3 days for further study, as well as $30 for their participation.

Specifically, we utilized the POCS-Pre for this study, and a copy of this measure can be found in Appendix B.

Postnatal session. As part of the larger study, mothers came back to the laboratory at approximately 6 months (±2 weeks) postnatally, along with their infants. They first completed brief videotaped standardized behavioral tasks, including: (1) a caregiving task, (2) a free-play interaction, (3) a multiple object free play task, and (4) a gentle infant arm restraint by the RA. These behavioral tasks lasted approximately 20 minutes. During the caregiving task, mothers completed portions of the Infant Diet and Breastfeeding Questionnaire. The behavioral tasks were discontinued by the experimenter if the infant displayed an excessive level of distress (as evidenced by a red, flushed face and continual crying) for 30 seconds or longer. Afterwards, the mother and infant completed anthropometric measurements (i.e., weight, height/length, and abdominal circumference), which took approximately 10 minutes. Next, mothers completed interviews regarding both their and their baby's health since the last session. Then, similar to the prenatal session, they once again completed the M.I.N.I., TLFB, PSS, DSQ, EBQ, PASS, POCS-Post, and Y-BOCS. They also completed the Infant Crying Questionnaire-Postpartum Version (ICQ-PP), the Edinburgh Postnatal Depression Scale (EPDS), and the Infant Behavior Questionnaire-Revised Short Form (IBQ-R SF). These questionnaires took approximately 60 minutes to complete. Mothers were provided $30 for their participation. We utilized the POCS-Post, EPDS, and IBQ-R SF for this study and copies of these measures can be found in Appendices C, D, and E, respectively.

Participants

Data were collected from adult mothers during their third trimester (33-37 weeks gestation) by a team of undergraduate and graduate students for the prenatal session beginning

04/21/2015. I began conducting prenatal interviews on 07/09/2015. Data collection and my involvement for the postnatal sessions began on 11/16/2015. The last postnatal session was completed on 7/7/2017. Overall, I conducted 33% of the prenatal sessions, and 28% of the postnatal sessions. Of the 506 participants who were contacted to participate in the study, 71 were ruled ineligible (see Table 1 for exclusion criteria).

Participants who did not meet exclusion criteria were invited to their initial prenatal session. One hundred and thirty-one participants declined to participate. Reasons for declining included difficulty making the commute to Idaho State University (*n*=53), the time commitment (*n*=19), scheduling conflicts (*n*=15), moving out of the area prior to study completion (*n*=7), lack of energy (*n*=1), would not give reason/not interested (*n*=27) *worried* about going into labor (*n*=2), not having custody of the baby (*n*=1), or bedrest (*n*=6). One hundred and seventy-nine prospective participants were unreachable by telephone, text, and e-mail communication attempts.

Measures

To test hypotheses within the current dissertation, only a subset of measures utilized in the larger IDAHO MOM Study were necessary and they are described in detail below.

Perinatal Obsessive-Compulsive Scale (POCS). The POCS is a self-report measure used to assess OC symptoms in the perinatal period. It consists of both a prenatal (POCS-Pre) version, which assesses for OC symptomatology during the participant's most recent pregnancy, and the postnatal (POCS-Post) version, which assesses for OC symptomatology following the participant's most recent pregnancy. It was specifically created and normed for the perinatal population, due to the differential presentation of certain OC symptoms in the perinatal period compared to OC symptoms occurring outside the context of pregnancy (Labad et al., 2010). The

POCS-Pre has 2 scales: a 10-item Likert rating scale of severity that assesses amount of time spent, interference, resistance, and control of the indicated obsessive thoughts and compulsive behaviors (e.g., "How much time do or did you spend being bothered by these thoughts described above?") and a 6-item Likert rating scale of interference that assesses how much the reported symptoms interfered with the individual's life (e.g., "How often do or did these thoughts interfere(d) with your relationships with your family?"). The POCS-Post also has 2 scales: an 8-item Likert rating scale of severity that assesses amount of time spent, interference, resistance, and control of the indicated thoughts and behaviors (e.g., "How much time do or did you spend being bothered by these thoughts described above?") and a 14-item Likert rating scale of interference that assesses how much the reported symptoms interference with the individual's life (e.g., "How often do or did these thoughts interfer(ed) with your relationships with your family?"). Responses on the severity scale range from 0 to 4, with higher scores being indicative of greater OC symptomatology (Lord, Rieder, Hall, Soares, & Steiner, 2011).

The POCS prenatal and postnatal versions have demonstrated average Cronbach's α reliability coefficients of .95 for the severity scale and .92 for the interference scale, and evidence a concurrent validity coefficient of .81 with the Y-BOCS (Lord et al., 2011). It has a sensitivity for identifying those with an OCD diagnosis of .62 and .64 on the severity and interference scales, respectively, and .92 and .94 specificity for identifying those without an OCD diagnosis on the severity and interference scales, respectively (Lord et al., 2011). Finally, utilizing area under the curve (AUC) analyses (a statistical technique plotting the true-positive versus the false-positive rate of a binary classifier; Hanley & McNeil, 1982) it demonstrated an AUC of .81 on both the severity and interference scales, and was found to consist of a single factor (Lord et al., 2011). For this study, the POCS severity scale from both the POCS-Pre and

POCS-Post was utilized as an additional index of OC symptom severity. Within the current study sample, the internal consistency reliability or Cronbach's α for the POCS-Pre and the POCS-Post was .89.

Edinburgh Postnatal Depression Scale (EPDS). The EPDS is a brief, 10-item self-report measure created for the assessment of perinatal depression in the past 7 days (Cox, Holden, & Sagovsky, 1987). Specifically, the participant responds to 10 separate statements (e.g., I have felt happy) on a Likert rating scale of 0-3, indicating the degree to which the statement describes how they have felt over the past 7 days. Many symptoms experienced during pregnancy, such as changes in appetite and loss of energy, overlap with vegetative depressive symptoms. Indeed, it has been found that the Beck Depression Inventory (BDI) has poorer validity than the EPDS when applied to postpartum women (Eberhard-Gran, Eskild, Tambs, Opjordsmoen, & Samuelsen, 2001; Harris, Huckle, Thomas, Johns, & Fung, 1989). In a review of validation studies on the EPDS, Eberhard-Gran et al. (2001) indicated sensitivity ranges between 65% - 100%, and specificity ranges between 49% - 100%, and stated that the measure is best utilized as a screening instrument. Generally, scores of 13 and higher are indicative of individuals suffering from Major Depressive Disorder (Cox et al., 1987; Matthey, Henshaw, Elliott, & Barnett, 2006). For this study, the EPDS total score was calculated by summing the responses on the 10 items, with higher scores more indicative of severe depressive symptomatology. Items 3, 5, 6, 7, 8, 9, and 10 were reversed scored. The current sample Cronbach's α for this measure was .82.

Infant Behavior Questionnaire – Revised Short Form (IBQ-R SF). The IBQ-R SF is a parent-rated measure, wherein parents rate specific infant behaviors during the previous 2 weeks on a 7-point Likert scale, from "never" to "always." The utilization of a self-report allows for a

longer period of assessment of infant reactivity than what could be feasibly observed by a researcher. It consists of 14 scales, including: (1) a 12-item Approach scale (e.g., "when given a new toy, how often did the baby get very excited about getting it?"), (2) a 12-item Vocal Reactivity scale (e.g., "when being dressed undressed during the last week, how often did the baby coo or vocalize?"), (3) an 11-item High Intensity Pleasure scale (e.g., "during a peek-a-boo game, how often did the baby smile?"), (4) a 10-item Smile and Laughter scale (e.g., "how often during the last week did the baby smile or laugh when given a toy?"), (5) a 15-item Activity Level scale (e.g., "when put into the bath water, how often did the baby splash or kick?"), (6) a 12-item Perceptual Sensitivity scale (e.g., "how often did the baby notice fabrics with scratchy texture?"), (7) a 14-item Sadness scale (e.g., "Did the baby seem sad when the caregiver was gone for an unusually long period of time?"), (8) a 16-item Distress to Limitations scale (e.g., "when placed on his/her back, how often did the baby fuss or protest?"), (9) a 16-item Fear scale (e.g., "how often during the last week did the baby startle to a sudden or loud noise?"), (10) a 13-item Falling Reactivity/rate of recovery from distress scale (e.g., "when frustrated with something, how often did the baby calm down within 5 minutes?"), (11) a 13-item Low Intensity Pleasure scale (e.g., "when playing quietly with one of his/her favorite toys, how often did the baby show pleasure?"), (12) a 17-item Cuddliness scale (e.g., "when rocked or hugged, during the last week, how often did the baby seem to enjoy him/herself?"), (13) a 12-item Duration of Orienting scale (e.g., "how often during the last week did the baby stare at a mobile, and (14) an 11-item Soothability scale (e.g., "When patting or gently rubbing some part of the baby's body, how often did s/he soothe immediately?").

Utilizing Principal Axis extraction (iterating to communalities) and obliquely rotating the extracted factors utilizing the Oblimin algorithm (Norusis, 1994), a 3-factor solution

(Surgency/Extraversion, Negative Affectivity, and Orienting/Regulation Factor) has been established in a previous study (Gartstein & Rothbart, 2003). Surgency/Extraversion consists of the Activity Level, Smiling and Laughter, High-intensity Pleasure, Perceptual Sensitivity, Approach/Positive Anticipation, and Vocal Reactivity scales. Negative Affectivity consists of the Distress to Limitations, Fear, Sadness, and Falling Reactivity scales. The Orienting/Regulation factor consists of the Orienting, Low-intensity Pleasure, Soothability, and Cuddliness scales (Gartstein & Rothbart, 2003; Gartstein & Bateman, 2008). Factor loadings ranged from .031 (Fear scale) to 0.79 (Sadness scale). Gartstein and Rothbart (2003) found all 14 subscales to have internal consistency reliability ranging from α=.70 to .90, while Parade and Leerkes (2008) found α's for the 14 subscales to range from .71 to .88 in both mothers and fathers. Additionally, correlational analyses between parallel mother and father reports were all found to occur in the positive direction, and 11 of the 14 scale comparisons between mothers and fathers were significantly correlated (range of 0.26 to 0.46; Parade & Leerkes, 2008) Additionally, Gartstein and Bateman (2008) found that the Fear and Smiling and Laughter scales, utilized in isolation, had Cronbach's αs of .81 and .80, respectively, while Gartstein, Slobodskaya, and Kinsht (2003) found Cronbach's αs of .81 to .96 for a Russian sample. In addition to the three factors, a total score can be established that serves as an overall index of infant reactivity. In this study, we utilized the Negative Affectivity scale as an index of infant reactivity. Cronbach's α for the Negative Affectivity scale was .67.

Hollingshead's Four Factor Index of Social Status (Hollingshead). The Hollingshead is a measure of SES originally created in 1958, but was updated in 1975 to take into account an individuals' education, occupation, gender, and marital status (Hollingshead, 1975). Occupation is assessed based off of the approximate 450 occupational titles and codes of the 1970 United

States Census, and is assessed on a 9-point scale. Education is based upon the number of years of schooling and is assessed on a 7-point scale. Social status is assessed by calculating marital status and multiplying this by factor weights (Gottfried, 1985; Hollingshead, 1975). A total score ranging from 1-5 is calculated, with higher scores being associated with higher levels of SES. It has a statistically significant correlation of .41 with the Bayley Mental Development Index when individuals are assessed at 24 months, and .39 with the McCarthy General Cognitive Index when assessed at 42 months. Additionally, it has a statistically significant correlation of .79 with the Revised Duncan Socioeconomic Index, and .73 with the Siegel Prestige Scale (Gottfried, 1985). When assessing two-wage-earner families, one-wage-earner families, one-male-wage-earner families, and one-female-wage-earner families, interrater reliability for the Hollingshead is significant and high, with kappa coefficients ranging from .73 to .95 (Cirino et al., 2002). It is also significantly correlated with IQ (r=.43), reading achievement (r=.27), spelling achievement (r=.17), and mathematics achievement (r=.17) scores (Cirino et al., 2002). The current study Cronbach's α for the Hollingshead was .55.

Behavioral observation coding. In addition to the self-report methodology for assessing infant reactivity, we recorded infant reactivity from the 6-month postnatal sessions utilizing a 7-point coding system. Specifically, we utilized frequency of negative affect during the gentle infant arm restraint portion of the standardized behavioral tasks. Specifically, we recorded and coded every instance of infant reactivity in one of the following categories: Positive Reactivity (e.g., infant displays an open mouth, smiles, displays positive vocalizations, etc.), Neutral Reactivity (infant displays no negative or positive affect in vocalizations, facial expressions, and body movements), and Negative Reactivity (e.g., infant cries, displays fussiness, evidences body tension, etc.). This behavioral task was added due to the differences observed between self-

reported and actual behavior (Baumeister, R. F., Vohs, K. D., & Funder, D. C., 2007). Additionally, infant reactivity (as assessed by behavioral coding) at 6-months has been linked to a multitude of long-term outcomes in infants, such as anxious behavior at 2.5 years (Crockenberg & Leerkes, 2006) and aggressive behavior at 2.5 years (Crockenberg, Leerkes, & BArrig JO, 2008), and thus provides additional valuable information.

Behavioral coding was conducted by a team of 2 RAs using Mangold INTERACT Software. The two RAs engaged in the coding process were trained by a senior graduate RA. Reliability was established by the creation of a standard file based on well-established and defined behavioral codes. Behaviors were coded by using keystrokes to define the behavior as well as onset of behavior. A different keystroke or second keystroke determined offset of behavior. Each behavior was assigned a letter or number on the keyboard. Inter- and intra-rater reliability was calculated using the Kappa Reliability analysis option in INTERACT with the criterion set for 80% matches within 5 seconds for onset and offset time codes. INTERACT then automatically calculates frequency and durations of the codes for infant reactivity. The behavioral task and coding procedures were discontinued if the infant displayed volatile levels of reactivity for a minimum of 30 seconds straight. Within the current study, intra-rater coding reliability for this measure was .96, and inter-rater coding reliability was .88.

Hypotheses

Based on a review of the literature and existing IDAHO MOM Study procedures, the following four hypotheses were generated independently from the larger study utilizing novel measures of perinatal OCD. Maternal *prenatal* OC symptomatology (as measured with the *POCS*), while controlling for maternal depressive symptomatology, would be related to infant reactivity (as measured with the *IBQ-R* [**Hypothesis 1**] and *behavioral observation coding*

[**Hypothesis 2**]) at 6 months. Specifically, mothers with higher levels of prenatal OC symptomatology would have infants with higher levels of reactivity. Additionally, it was hypothesized that socioeconomic status (SES) would moderate this relation, due to the findings that higher SES operates as a buffering agent against early developmental stressors (Blumenshine et al., 2010). In other words, higher parental SES was expected to prevent against many of the prenatal biological stress reactivity changes that occur and lead to different infant outcomes. Therefore, it was hypothesized that the correlation of infant reactivity and maternal OC symptomatology would be weaker for mother/infant pairs from higher SES backgrounds than for mother/infant pairs from lower SES backgrounds.

Similarly, maternal *postnatal* OC symptomatology (as measured with the *POCS*), while controlling for maternal depressive symptomatology, would be related to infant reactivity (as measured with the *IBQ-R* [**Hypothesis 3**] and *behavioral observation coding* [**Hypothesis 4**]) at 6 months. Specifically, mothers with higher levels of postnatal OC symptomatology would have infants with higher levels of reactivity. Additionally, it was hypothesized that SES would moderate this relation, given that access to greater resources for mothers and infants may reduce the impact of maternal postnatal mental health difficulties on infant developmental outcomes (Plamondon et al., 2015). Therefore, it was hypothesized that the correlation of infant reactivity and maternal OC symptomatology would be weaker for mother/infant pairs from higher SES backgrounds than for mother/infant pairs from lower SES backgrounds. Maternal anxiety levels were not controlled for in these analyses, as the PASS overlaps with the POCS (i.e., has questions about OCD) and would therefore decrease the probability of finding an effect due to multicollinearity.

Statistical Analyses

A priori power analyses using SamplePower3 revealed that with a medium effect size (R^2=0.09; Cohen, 1992) at each step of the model for (1) the covariate, (2) two main effect predictors, and (3) the interaction; two-tailed α=0.05 (i.e., 5% probability of Type I error or false positive); and power=0.80, a sample size of 84 is required for hierarchical regression analyses. The medium effect size was conservatively chosen based upon previous research which demonstrated medium to large effect sizes for the effects of both maternal anxiety and depression symptoms on infant reactivity levels (Davis et al., 2004; Field et al., 2004). Utilizing the aforementioned enrollment strategy, both our prenatal sample size of 125 and postnatal sample size of 95 are adequate for the research design.

In addition to testing regression assumptions prior to testing each model, descriptive statistics were used to characterize the sociodemographic and primary study variables in the current sample. Each hypothesis was evaluated using hierarchical multiple regression analyses in which the covariate was entered into the first step, the primary predictor and moderator main effects were entered into the next step, and the primary predictor and moderator interaction were entered into the third step. This allowed us to control for perinatal depression while testing main effects of maternal perinatal OCD symptoms and SES (i.e., R^2_{change} between model 1 and 2) on infant behavior, as well as the interactive relation between OC symptoms and SES (i.e., R^2_{change} between model 2 and 3) on infant behavior. Prior to completing these analyses, maternal depression, maternal perinatal OC symptoms, and SES were mean centered and an interactive term was computed via multiplication. This was done in order to avoid problems associated with multicollinearity. The regression equations for each hypothesis took the following form: (Step 1) infant reactivity (y)=maternal perinatal depressive symptoms (x1), (Step 2) infant reactivity

(y)=maternal perinatal OC symptoms (x2) + SES (x3), and (Step 3) infant reactivity (y)=maternal perinatal OC symptoms x SES (x4).

We examined effect sizes utilizing standardized regression coefficients given the easier comparison across multiple scales of measurement regarding variables in the model. However, we investigated significance from unstandardized coefficients given that the standard errors of standardized coefficients with the interaction term cannot be estimated accurately with existing statistical software. Follow-up analyses were completed with all significant interactions via simple slopes analyses (Aiken & West, 1991) using Preacher, Curran, and Bauer's (2006) online interactive calculation tools. This allowed us to model relations between maternal perinatal OCD symptoms and infant reactivity at each level of SES in addition to exploring regions of significance to determine upper and lower SES thresholds for which significant relations exist between maternal perinatal OCD symptoms and infant reactivity, while also considering important covariates (i.e., maternal perinatal depressive symptoms; Preacher et al., 2006).

CHAPTER III

Results

Regression Assumptions

All regression assumptions were tested prior to conducting data analyses. These included casewise diagnostics for outliers (±3 SD), scatterplots to ensure linearity and to rule out range restriction, frequency histograms to test for normality of variables of interest, intercorrelations and variance inflation factors to rule out multicollinearity, residuals plots to test for homoscedasticity and normality of residuals, and the Durbin-Watson Statistic to investigate independence of error.

Observation of frequency histograms suggested a positive skew for the behavioral observation reactivity outcome variable and several independent variables (i.e., EPDS, POCS-Pre, and POCS-Post). Square-root transformations were performed on the EPDS, POCS-Pre, and POCS-Post variables, resulting in a linear relationship between all dependent and independent variables, as determined via residuals plots. This also resulted in skewness and kurtosis statistics for each dependent and independent variable within the acceptable range (i.e., all values were less than $\left|1\right|$).

To test for the presence of multicollinearity, a cutoff tolerance value below .10 and/or a VIF value above 5 were used (Menard, 1995). In this data set, the lowest tolerance score was .86 and the highest VIF score was 1.17, suggesting that independent variables within each of the four Hypotheses/models were not too highly correlated with one another. This is also supported via an intercorrelations table (see Table 2), in which the only two variables that were correlated above .30 were prenatal and postnatal OC symptoms, which theoretically should be highly correlated and were entered into separate models. Additionally, postnatal depression and OC symptoms were significantly correlated (r=.30, p=.003), which again should be the case given comorbidity between postnatal anxiety and depression. That is why postnatal depression was entered into step 1 of the hierarchical regression in order to determine whether OC symptoms explain a significant amount of variability in infant reactivity outcome variables above and beyond variance accounted for by postnatal depression.

The residuals for all four hypotheses were homoscedastic in that the residuals plots were approximately the same width for all values of the predicted dependent variables. Utilizing behavioral observation coding as the dependent variable, the values of the residuals for Hypotheses 2 and 4 were not normally distributed. Therefore, a log (y+1) transformation was

performed on the behavioral observation coding data, resulting in a normal distribution of residuals and behavioral observation data upon inspection of frequency histograms. Additionally, skewness and kurtosis statistics for this outcome variable were all within acceptable limits (i.e., less than $\left|1\right|$).

The Durbin-Watson statistic ranged from 1.58 to 1.95. Scores between 1 and 3 are typically considered acceptable (Field, 2009), suggesting that the errors of adjacent observations in this study were not significantly correlated. Therefore, autocorrelation of the errors of the regression models and associated Type 1 error were not thought to be concerns within current study models.

Descriptive Statistics

The frequencies of sociodemographic variables (see Table 3) and means and standard deviations for primary study variables (i.e., maternal perinatal depression and OCD symptoms, SES, and infant reactivity) and maternal age (see Table 4) were computed to describe the current sample. During the prenatal session, mothers had a mean age of 26.82 years (*SD*=4.40 years), while the mean maternal age at approximately 6 months postpartum was 28.32 years (*SD*=3.96 years). With regard to ethnicity, 87% identified as White/Caucasian, 1% as Black or African American, 1% as Asian, 2% as White and Native Hawaiian or Other Pacific Islander, 2% as White and American Indian/Alaska Native, 1% as White and Black or African American, 1% as White and Other, and 6% as other (categories were not mutually exclusive). With regard to marital status, 8% identified as single/never married, 79% as married, 2% as divorced, 7% as in a committed relationship, and 3% as engaged. With regard to total annual familial income, 2% reported earning less than $5,000, 2% between $5,000 and $9,000, 15% between $10,000 and $19,999, 19% between $20,000 and $29,999, 12% between $30,000 and $39,000, 10% between

$40,000 and $49,000, 25% between $50,000 and $74,999, 7% between $75,000 and $99,999, and 8% earning $100,000 or greater.

The mean number of seconds an infant engaged in affectively negative behavior was 77.73 (*SD*=4.79) seconds out of a total session period of 25 – 35 minutes. The mean SES score obtained (range 1-5) on the Hollingshead was 3.45 (*SD*=0.97), approximately halfway between skilled craftsmen, clerical, and sales workers (3), and medium business, minor professionals, and technical workers (4). The mean score on infant reactivity as measured by the IBQ-R was 2.96 (*SD*=0.62). Scores on the IBQ-R (specifically, the Negative Affect subscale) in our sample were significantly lower than five of six other normative samples evaluated (Putnam et al., 2014; see Table 5). The mean level of OC symptomatology as measured by the POCS-Pre was 3.07 (range 0-20; *SD*=3.87), and 3.56 (range 0-16; *SD*=4.64) as measured on the POCS-Post. Comparative scores from additional normative and clinical samples were not available for this measure. The mean level of depressive symptomatology as measured by the EPDS was 4.79 (*SD*=3.96). Scores on the EPDS in our sample were not significantly different from a normative sample (Austin et al., 2005; see Table 6) and were significantly below the recommended cut-off point of 13 for depressive disorder diagnoses (Matthey et al., 2006), suggesting that, on average, our sample was representative of the typically subthreshold symptoms observed in community samples.

Hypothesis 1: Maternal *prenatal* OC symptomatology (as measured with the *POCS*), while controlling for maternal postnatal depressive symptomatology, will be related to infant reactivity (as measured with the *IBQ-R*) at 6 months

In the first analysis, infant reactivity was regressed on maternal depressive symptomatology, *prenatal* OC symptomatology, and the interaction of SES and *prenatal* OC symptomatology (see Table 7). Overall, the full model did not account for significant variance in

infant reactivity [$F(4, 90)=1.69$, $p=.159$]. In the first step of the hierarchical multiple regression, maternal depressive symptomatology accounted for a significant amount of variance in infant reactivity [$F(1, 93)=4.930$, $p=.029$]. Specifically, for each one point increase in EPDS Score, there was a .02 increase in infant reactivity [IBQ-R=.02(EPDS) + e]. In the second step, the inclusion of maternal *prenatal* OC symptomatology and SES main effects did not significantly add to the model [$F_{change}(2, 91)=.422$, $p=.657$]. In the third step, the interaction between prenatal OC symptomatology and SES did not significantly add to the model [$F_{change}(1, 90)=.903$, $p=.345$]. Overall, maternal prenatal OC symptoms were unrelated to infant 6-month reactivity, as measured by maternal report, while controlling for depressive symptomatology. SES was not significantly related to infant reactivity, nor was the interaction between OC symptoms and SES. However, a greater number of maternal depressive symptoms were related to higher levels of infant 6-month reactivity, as measured by maternal report.

Hypothesis 2: Maternal *prenatal* OC symptomatology (as measured with the *POCS*), while controlling for maternal postnatal depressive symptomatology, will be related to infant reactivity (as measured with *behavioral observation coding*) at 6 months

In the second analysis, infant reactivity was regressed on maternal depressive symptomatology, *prenatal* OC symptomatology, and the interaction of SES and *prenatal* OC symptomatology (see Table 8). Overall, the full model did not account for significant variance in infant reactivity [$F(4, 87)=.20$, $p=.937$]. In the first step of the hierarchical multiple regression, maternal depressive symptomatology did not account for significant variance in infant reactivity [$F(1, 90)=.01$, $p=.924$]. In the second step, inclusion of maternal *prenatal* OC symptomatology and SES main effects did not significantly add to the model [$F_{change}(2, 88)=.40$, $p=.672$]. In the third step, the interaction between prenatal OC symptomatology and SES did not significantly

add to the model [$F_{change}(1, 87)=.01$, $p=.916$]. Overall, the main and interactive effects of prenatal OC symptomatology and SES on infant reactivity were not statistically significant, as measured by standardized behavioral observation.

Hypothesis 3: Maternal *postnatal* OC symptomatology (as measured with the *POCS*), while controlling for maternal postnatal depressive symptomatology, will be related to infant reactivity (as measured with the *IBQ-R*) at 6 months

In the third analysis, infant reactivity was regressed on maternal depressive symptomatology, *postnatal* OC symptomatology, and the interaction of SES and *postnatal* OC symptomatology (see Table 9). Overall, the full model did not account for significant variance in infant reactivity [$F(4, 90)=2.163$, $p=.079$]. In the first step of the hierarchical multiple regression, maternal depressive symptomatology accounted for significant variance in infant reactivity [$F(1,93)=4.930$, $p=.029$]. Specifically, for each one point increase in EPDS Score, there was a .02 increase in infant reactivity [IBQ-R=.02(EPDS) + e]. In the second step, the inclusion of maternal *postnatal* OC symptomatology and SES main effects did not significantly add to the model [$F_{change}(2, 91)=1.075$, $p=.346$]. In the third step, the interaction between postnatal OC symptomatology and SES did not significantly add to the model [$F_{change}(1, 90)=1.437$, $p=.234$]. Overall, maternal postnatal OC symptoms were unrelated to higher levels of infant 6-month reactivity, as measured by maternal report, while controlling for depressive symptomatology. SES was not significantly related to infant reactivity, nor was the interaction between OC symptoms and SES. However, a greater number of maternal depressive symptoms were related to higher levels of infant 6-month reactivity, as measured by maternal report.

Hypothesis 4: Maternal *postnatal* OC symptomatology (as measured with the *POCS*), while controlling for maternal postnatal depressive symptomatology, will be related to infant reactivity (as measured with *behavioral observation coding*) at 6 months

In the fourth analysis, infant reactivity was regressed on maternal depressive symptomatology, *postnatal* OC symptomatology, and the interaction of SES and *postnatal* OC symptomatology (see Table 10). Overall, the full model did not account for significant variance in infant reactivity [$F(4, 87)=.40$, $p=.806$]. In the first step of the hierarchical multiple regression, maternal depressive symptomatology did not account for significant variance in infant reactivity [$F_{change}(1, 90)=.01$, $p=.924$]. In the second step, the inclusion of maternal *postnatal* OC symptomatology and SES main effects did not significantly add to the model [$F_{change}(2, 88)=.81$, $p=.450$]. In the third step, the interaction between postnatal OC symptomatology and SES did not significantly add to the model [$F_{change}(1, 87)=.01$, $p=.920$]. Overall, main and interactive effects of postnatal OC symptomatology and SES on infant reactivity were not statistically significant, as measured by standardized behavioral observation.

CHAPTER IV

Discussion

Study Strengths

This methodology of this study contained several strengths. First, its longitudinal nature allowed us to track changes in symptomatology over approximately a 6-month period. Second, the IBQ-R, EPDS, POCS-Pre, POCS-Post, and Hollingshead had strong theoretical backing and moderate to sound psychometric properties. Third, the statistical analyses that we utilized allowed us to investigate complex, multivariate relationships. Finally, our sample size for a longitudinal study of this scope was robust and sound. Finally, we investigated the effects of

perinatal maternal OC symptomatology on infant outcomes, an important area of research that is understudied in the field of psychology.

Current Study Findings

Research has demonstrated that maternal perinatal depression, anxiety, and stress have consequences for the unborn fetus later in life (Petzoldt et al., 2014; Tarabulsy et al., 2014). However, no known study has investigated the relationship between maternal perinatal OC symptomatology and infant outcomes. The purpose of this study was to investigate the relationship between OC symptoms and infant reactivity. Four hypotheses were tested to investigate this relationship. Hypothesis 1 predicted that maternal prenatal OC symptomatology would be related to infant reactivity (as measured with the IBQ-R). Hypothesis 2 predicted that maternal prenatal OC symptomatology would be related to infant reactivity (as measured with behavioral observation coding. Hypothesis 3 predicted that maternal postnatal OC symptomatology would be related to infant reactivity (as measured with the IBQ-R). Hypothesis 4 predicted that maternal postnatal OC symptomatology would be related to infant reactivity (as measured with behavioral observation coding). It was expected that these relationships would occur after controlling for the effects of maternal postnatal depression. It was also expected that prenatal SES would moderate these relationships.

With regard to Hypotheses 1 and 3, the full model did not account for a significant amount of variance in infant reactivity utilizing the IBQ-R; however, maternal postnatal depressive symptomatology was significantly related to higher levels of infant reactivity in the first step, a discovery in line with previous research findings. There are several aspects of this study that were unique from others. First of all, fewer studies have found robust relationships between postnatal depression and infant behavior at the 6-month time point. Prior studies

primarily evaluated neurological and hormonal outcomes immediately following birth (DiPietro et al., 2006; Field et al., 2006; Marcus et al., 2011), or behavioral reactivity observed within several weeks to a couple months following birth (Davis et al., 2007, Diego et al., 2005). Additionally, prior studies have been conducted primarily on urban populations. This study's utilization of a rural sample suggests that the effects of postnatal maternal depression are widespread across a diverse range of mothers. In contrast, maternal OC symptomatology did not account for a significant amount of variance beyond that of depressive symptomatology. SES was not found to moderate the relationship between maternal OC symptomatology and infant reactivity.

With regard to Hypotheses 2 and 4, the full model did not account for a significant amount of variance in infant reactivity utilizing standardized behavioral coding of infant behavior. There were no significant relationships between maternal depressive symptomatology, prenatal or postnatal maternal OC symptomatology, and infant reactivity. SES was not found to moderate the relationship between maternal prenatal or postnatal OC symptomatology and infant reactivity.

It is unclear as to the reason for no significant relationship among primary study variables utilizing behavioral observation coding of infant reactivity. One possibility is that the length (approximately 25 to 35 minutes) of the behavioral observation coding did not allow for a thorough assessment of general infant reactivity. For example, the short period of time assessed may not be indicative of the infant's typical reactivity. The IBQ-R, although a self-report measure, allows for an assessment of infant reactivity over a longer period of time and across various settings, thereby perhaps allowing for a more accurate overall assessment. Furthermore, the behavioral observation coding was specifically assessing for infant affect, while the Negative

Affect subscale of the IBQ-R assesses a range of infant behaviors that are associated with negative reactivity (contentedness, protest, wants not being met, clinging, not warming up to adults, etc.). An additional possibility is that prior research has demonstrated that mothers experiencing elevated levels of psychopathology, especially depression, may be biased in their self-reports of their infants' behaviors (Muller, Achtergarde, & Furniss, 2011; Youngstrom, Izard, & Ackerman, 1999). Therefore, it is plausible that maternal ratings of infant reactivity evaluated in our study may not be as accurate as those due to behavioral observation modalities. One way that this could be investigated in the IDAHO MOM Study would be to compare maternal ratings and behavioral observation findings to cortisol levels provided by the mothers in our study. Finally, it has been demonstrated that the interactions between mothers and their infants are occasionally different depending on whether behaviors are being observed in a naturalistic or laboratory setting. For example, Belsky (1980) found that, while infant functioning was not greatly affected by context, mothers talked to, attended to, responded too, and stimulated their infants more frequently in laboratory settings than at home. Stevenson, Leavitt, Roach, Chapman, & Miller (1986) found that, while there was no statistically significant difference between mothers in the home and laboratory setting on how many words their infants spoke, complexity of maternal speech, and infant vocalization rate, mothers tended to speak at a faster rate in the laboratory than when at home. Therefore, future studies may be able to attain more accurate behavioral observation recordings if the observations are obtained in the participants' home. Additionally, future studies are encouraged to compare self-report data and behavioral observation data to biological measures of infant reactivity such as cortisol and hormone concentrations.

It is also unclear as to why maternal OC symptomatology was not significantly related to infant reactivity when either the IBQ-R or behavioral observation coding was being used to quantify the outcome variable. The scores on the IBQ-R were lower than in other studies; therefore, it is plausible that the restricted range of observed infant reactivity may have masked any associations with OC symptomatology. It is also plausible that this has to do with the sample in which OC symptomatology was assessed. Due to the fact that the sample utilized in this study was a community sample and therefore evidenced primarily subthreshold OC symptoms, the prevalence of full-threshold OC symptoms was rare. Indeed, only 6 prenatal (out of 125) and 2 postnatal (out of 94) clients met criteria for OCD via the Mini International Neuropsychiatric Interview. Therefore, it is likely that we may have experienced difficulties with restricted range and limited variability in the OC symptoms of our sample. Another possibility is that mild maternal OC symptoms may not be related to infant outcomes. In fact, low levels of OC symptoms may actually be beneficial for some individuals, leading to behaviors which could be adaptive for the survival of the group. For instance, checking to make sure that your baby's crib is safely secured may lead to the identification that it is not secure, and then this can be corrected (Gonda, Jekkel, Varga, Miklosi, & Forintos, 2008). This could theoretically mask some of the associations between maternal OC symptoms and infant behavioral reactivity that were hypothesized.

A final possibility has to do with the nature of OC symptomatology. The specific subtypes of obsessions (e.g., scrupulosity, harm, cleanliness, etc.) and compulsions (e.g., excessive praying for forgiveness, checking on the infant, excessive cleaning the home and the infant, etc.) were not assessed. It is possible that some subtypes may be more highly associated with infant reactivity than others. For example, constantly checking on the infant to make sure

they are safe may not necessarily exacerbate underlying problematic symptomatology (and could theoretically increase contact between mother and infant leading to beneficial outcomes), whereas avoiding engaging with the infant due to intense fears of harming them may be more detrimental in nature. These differences may have masked associations between maternal OC symptomatology and infant reactivity on both the IBQ-R and during behavioral observations of mother and infant interactions.

Further, it is unclear as to why SES did not have significant main or interactive effects with perinatal OC symptomatology on infant reactivity. It is likely that the low level of observed internal consistency affected the validity of the construct and the associated null findings. Additionally, identifying the processes through which SES influences behavioral reactivity can be difficult. For example, low SES frequently co-occurs with other situations that can affect infant outcomes, such as: single parenthood, immigrant and minority status, exposure to teratogens, and health conditions (Baum, Garofalo, & Yali, 1999; Hoffman & Hatch, 2000; Kivimaki, et al., 2007), of which the majority of our sample did not present with. Additionally, our sample had several resiliency factors based upon exclusion criteria and regional characteristics, including low rate of health concerns, limited perinatal complications, positive birth outcomes, and support from their religious community. The positive impact of community support on mental health, along with its buffering effects against stress, have been well-documented in the literature (Moreira-Almeida, Lotufo Neto, & Koenig, 2006). Taken together, our sample's higher-than-average SES levels [our sample's mean SES score of 3.45 fell approximately halfway between skilled craftsmen, clerical, and sales workers (3), and medium business, minor professionals, and technical workers (4)], high levels of community support, and relative lack of ethnic and cultural diversity, likely masked any potential moderating effects of

SES. Indeed, there is a great deal of debate in the literature on how best to measure SES (Bradley & Corwyn, 2002), and the Hollingshead does not take the aforementioned ethnic and cultural factors into account in its calculation of SES. It is also possible that, due to the rapidly changing sociodemographic makeup of the U.S. population, newer and more up-to-date modalities of measuring SES are needed. It is further plausible that SES may simply not have a direct or indirect relationship with infant reactivity or that the interaction effects were not significant due to the aforementioned methodological limitations regarding the POCS and our community sample.

Regarding other significant intercorrelations that were found in the current study, the persistence of perinatal OC symptoms through birth replicates other findings in the field suggesting that, without treatment, a large percentage of women will fail to experience a natural remittance of OC symptomatology (Miller, Chu, Gollan, & Gossett, 2013). In addition to the observed high correlation between maternal prenatal and postnatal OC symptoms, maternal postnatal depression was significantly positively correlated with postnatal OC symptoms. The relationship between postnatal depression and postnatal OC symptoms parallels previous research demonstrating an overlap between perinatal depression and anxiety (Field et al., 2003; Heron et al., 2004; Sutter-Dallay et al., 2004). It is possible that depression and OC symptoms substantially overlap and contribute to the pathogenesis of each other in a similar manner to depression and anxiety disorders. Maternal postnatal depression was also significantly negatively correlated with SES, paralleling other findings in the literature describing the interplay between both constructs (Beck, 2001). It is possible that women at risk for postpartum depression may experience a range of stressors directly related to their lower SES, as is the possibility that

women in lower SES levels may not have the monetary opportunities at hand to manage the stressors of early motherhood.

Practical Implications

This study adds to the current literature regarding perinatal depression and infant behavior in important ways. It is well established that there is a relationship between greater maternal depressive symptomatology and infant outcomes, including premature birth (Field et al., 2006), lower infant birth weight (Field et al., 2005; Mulder et al., 2002), greater behavioral reactivity (Davis et al., 2004; Davis et al., 2007), and greater occurrence of externalizing behaviors in childhood (Luoma et al., 2001). However, a large number of these studies have been conducted using mothers experiencing full-threshold symptoms of depression. In addition, they often originate from higher SES, more urban backgrounds. This study suggests that even within a sample of low risk mothers from more rural areas of the country, this population may be at risk of having their subthreshold depressive symptoms impacting their offspring. A 1-point increase in maternal depressive symptomatology (EPDS total score ranges from 0 to 30) is associated with a .02-point increase in mean infant behavioral reactivity as measured with the IBQ-R (IBQ-R total mean score ranges from 0-6). Although statistically significant, this is a small effect size. However, the observed association with early infant reactivity, coupled with previous literature supporting long-term outcome differences for infants of mothers with perinatal depression [such as greater occurrence of externalizing behaviors (Luoma et al., 2001) and cognitive impairments (Grace, Evindar, & Stewart, 2003) in childhood], these findings may be important. This is especially true given the widespread chronicity of maternal depressive symptomatology observed in the perinatal population. Overall, this study suggests that the actual diagnosis of depression is not as important for the potential detrimental impact on infants as is the mothers' overall level of

depression. The greater number of symptoms of depression that mothers experience during the postnatal period, the more likely they are to require early intervention, regardless of depressive disorder diagnosis.

In addition, expectant mothers can take comfort in this study's findings, as it suggests that small increases in depressive symptoms are unlikely to be associated with large differences in infant outcomes. Findings from this study did not support the hypothesis that maternal perinatal OC symptomatology and SES level would be related to greater levels of infant reactivity, nor did they support the hypothesis that SES level would moderate the relationship between maternal perinatal OC symptomatology and infant reactivity.

Current Study Limitations and Future Directions

There were several limitations to this study. First of all, given the correlational nature of this study, causation cannot be established between maternal perinatal mental health symptomatology and infant behavioral reactivity. While this would be difficult to attain in human populations due to ethical and moral constraints, causation could be better established utilizing animal populations. Future studies should induce OC symptoms in maternal animal populations in order to ascertain if this leads to increased infant behavioral reactivity via modalities discussed earlier (e.g., heightened levels of cortisol associated with OC symptomatology directly affecting the fetus). There has been discussion in the literature surrounding whether all aspects OCD symptoms seen in humans, especially obsessions (e.g., being responsible for harm to others), can be replicated in animal models (Alonso, Lopez-Sola, Real, Segalas, & Menchon, 2015); however, compulsivity, preservation, and stereotypy can likely be induced. For example, injection of quinpirole, a dopaminergic agonist, has been shown to induce compulsive checking behavior in rats, as identified by excessively returning to one or

two objects, excessively shorter time to return to these objects, excessively fewer places visited between returns to these objects, a characteristic set of acts being performed at the preferred place/object, and the rats' activity being altered when environmental properties associated with the places/objects were changed (Szechtman, Sulis, & Eilam, 1998). In future studies, it would be important to control for certain factors known to influence human infants like perinatal weight, free access to food and water, handling by the experimenter for several days prior to start of treatment, and treatment administrations during daylight hours. What's more, clomipramine, an SSRI utilized to treat OC symptoms, postponed (although did not prevent) the development of the quinpirole effect, further suggesting that these behaviors may reflect an animal model of OCD.

Another limitation of this study was the measurement of OC symptoms as a broad and heterogeneous construct, as opposed to measuring different symptom presentation. Indeed, OCD has been described as constituting a broad range of dimensions, and it may be more appropriate to consider OCD as a general term that encompass a diverse range of more specific disorders (Mataix-Cols et al., 2004). It is plausible that these differential presentations may be associated with differential outcomes in infants. Future studies should assess for the impacts of specific subtypes of OC symptomatology on infant reactivity and other infant outcomes to identify how these symptom subtypes may affect the direction and/or size of effects on infant reactivity.

Another limitation of this study was the use of infant reactivity as an indicator of later functioning. Although infant reactivity and worse scores on infant neurobehavioral scales are associated with longer-term life outcomes [higher levels of observed infant fearfulness and lower sociability at 5 years of age (Kagan, 1997; Kagan, 1998), worse grades in school at 6 years of

age (Niederhofer & Reiter, 2004), generalized social anxiety during adolescence (Schwartz et al., 1999), and worse motor outcomes in 2-year-olds (Stephens et al., 2010)] , the correlations are not one-to-one in nature. Future studies should utilize longer-term follow-up methodology in order to assess for the association between maternal perinatal psychopathology, particularly OC symptoms, and the long-term outcome of the infant in social cognitive, and physical health domains. This can be assessed via self-report, parental-report, teacher-report, and behavioral observation modalities in naturalistic and laboratory settings. Given current study findings and the limitations surrounding different modes of measurement, a multi-method, multi-informant modality of assessment is recommended, which may include using parental- and teacher-reports along with behavioral observation in naturalistic settings. Individuals enrolled in the IDAHO MOM Study are currently undergoing further, long-term follow-up analyses at 10, 14, and 18 months. This longer-term follow-up will allow us to analyze the relationship between maternal perinatal psychopathology and a broad range of infant outcomes, including: language, motor, sensory, affective, functional behavior, and physical growth when infants are older.

Higher levels of depressive symptomatology were associated with higher levels of infant reactivity regardless of depressive disorder diagnosis. Therefore, future studies should assess for the effectiveness of psychotherapeutic methods to reduce depressive symptomatology in perinatal populations regardless of depressive disorder diagnosis. This would involve initial self-report using measure like the EPDS or clinician-administered assessments of depression using a measure like the Hamilton Rating Scale for Depression (HRSD) or the Quick Inventory of Depressive Symptomatology (QIDS) at the beginning of treatment, several times during treatment progression, at the end of treatment, and at follow-up time points.

A further limitation of this study was that OC symptoms and infant reactivity were measured in a non-clinical, community sample. Future studies should assess for the relationship between maternal OC symptoms and infant reactivity in populations meeting full diagnostic threshold criteria for OCD, and then compare these individuals to a population not experiencing a psychological disorder. This would potentially allow for clearer relationships to be observed and would help to elucidate the potentially complex relationship that maternal OC symptomatology has with infant outcomes. To qualify for the OCD group, individuals would have to meet OCD symptomatology on the M.I.N.I. and score at or above 16 on the Y-BOCS (commonly used as a cutoff point for OCD diagnosis; Baer, Brown-Beasley, Sorce, & Henriques, 1993). To qualify for the group without a psychological disorder, individuals would have to not meet OCD diagnostic criteria on the M.I.N.I. and would need to score below a 16 on the Y-BOCS. The Y-BOCS would be a better screening measure to use than the POCS for potential OCD diagnosis due to no currently developed clinical cutoff scores currently delineated for the POCS. Utilizing similar recruitment methodology to the IDAHO MOM Study (e.g., monetary compensation, exclusion criteria, etc.), a Mixed Design Analysis of Variance (ANOVA) could compare individuals with OCD vs. individuals without a psychological disorder during the third trimester and at least one point during the postnatal period like the 6-month visit. Due to the relatively low prevalence rate of OCD and pregnant women in the community population, as well as the relative lack of pregnant women participating in intensive treatment programs for OCD, recruitment for this would likely take several years. However, due to its location as a hub where most women give birth, this process could be accelerated by integrating recruitment flyers primarily into labor/delivery rooms, as well as hospital waiting rooms.

Due to the potential beneficial effects of very low subthreshold OC symptoms (Gonda et al., 2008) the relationship between OC symptomatology and infant reactivity may not be directly linear. It may be modified by the severity level of symptoms (low vs. medium vs. high). Future studies should evaluate the effects of diverse levels of OC symptomatology and their relation to infant outcomes. This would help to separate out the complex relationship between OC symptoms and infant outcomes.

The lack of moderation observed with SES also should be evaluated further, as effects of SES may not be as readily apparent on homogenous samples of middle-to-upper class White/Caucasian populations. Future studies should evaluate a wider range of SES, and these samples should consist of a diverse range of ethnic and cultural groups. Additionally, due to the aforementioned methodological limitations of the POCS, Hollingshead, and infant reactivity measures, future studies are encouraged to assess for the relation between maternal perinatal OC symptoms on infant reactivity (and SES's moderation of this relationship) utilizing additional measures like the Y-BOCS for OC symptomatology, behavioral observation of infants over multiple time points and/or in naturalistic settings, and The Duncan Socioeconomic Index (Duncan, 1961) for SES. However, additional studies would need to be conducted to confirm this hypothesis.

Due to the relationships observed between postnatal depression and infant reactivity, it would be prudent to assess for the impact of maternal postnatal depression on areas of offspring functioning evaluated at longer follow-up periods through toddlerhood and early childhood. Infant behavioral reactivity, temperament, and neurobehavior have been linked to a wide range of outcomes, such as higher levels of inhibition and shyness at 4 and 7 years of age (Pfeifer et al., 2002), lower levels of spontaneity and sociability at 5 years of age (Kagan, 1997; Kagan et al.,

1998), and higher levels of anger and frustration during middle childhood (Komsi et al., 2008). Discovering additional links between infant behavioral reactivity and long-term functioning would help to better elucidate long-term outcomes of maternal perinatal depression, due to the relationship between the two constructs. It would also be useful to further investigate the potential relationship between *prenatal* maternal depression and infant outcomes. While much research has already been conducted in this area (Davis et al., 2007; Field et al., 2006; Mulder et al., 2002) as discussed earlier, most of it has been conducted with urban samples. Replication of these studies' results on more rural, community samples is necessary in order to assess for generalizability. This would be conducted to ascertain if this represents a distinct construct with a potentially different relationship with infant outcomes, as compared to postnatal maternal depression, within an understudies and underserved population.

Finally, an important addition of this study was the utilization of the IBQ-R. While not widely utilized, it appears to be sensitive to capturing differences in infant reactivity based upon maternal mental health. It would be prudent for future studies to use this measure and compare it among multiple raters (e.g., mothers, fathers, teachers, etc.) in order to better understand the validity of the measure.

Summary/Conclusion

In summation, we utilized a longitudinal study to evaluate relationships among perinatal maternal depressive and OC symptomatology, SES and infant behavioral reactivity. We found that maternal postnatal depressive symptomatology was significantly correlated with higher levels of infant behavioral reactivity as measured by the IBQ-R. Maternal perinatal OC symptomatology and SES levels were not related to infant behavioral reactivity.

While the positive relationship of higher levels of perinatal maternal depressive symptomatology to worse infant outcomes has already been established, these findings were most often conducted utilizing urban samples with full threshold symptoms of depression. This study expands upon these findings and suggests that similar results are obtained in individuals from rural populations experiencing subthreshold symptoms. This is important as it suggests that the different effects of postpartum depression on infant outcomes are wide-ranging across urban and rural mothers, and that even mothers experiencing subthreshold postpartum symptoms may have infants with impairments in emotional reactivity, which represents a risk factor for developmental difficulties in a variety of domains. This holds important implications for the implementation of nation-wide screening for perinatal depressive symptomatology in hospitals across the nation. We hope that these findings will provide additional knowledge and understanding for expectant mothers about perinatal depression and infants.

Overall, a wealth of study areas remains open for investigation. This includes inducing OC symptoms in animal populations, investigating infant outcomes utilizing longer-term follow-up methods, assessing the effectiveness of psychotherapeutic methods to reduce depression in perinatal populations, and focus on investigating infant outcomes in samples meeting full diagnostic threshold criteria for OCD. Additionally, future studies should use a wider range of SES measurement with diverse cultural and ethnic groups, confirm findings using different measures, such as the Y-BOCS for OC symptomatology to establish convergent validity, and establish confirmatory studies to validate the use of the IBQ-R in perinatal populations.

References

Abramowitz, J. S., Schwartz, S. A., & Moore, K. M. (2003a). Obsessional Thoughts in Postpartum Females and Their Partners: Content, Severity, and Relationship with Depression. *Journal of Clinical Psychology in Medical Settings, 10*(3), 157-164. doi:10.1023/a:1025454627242

Abramowitz, J. S., Schwartz, S. A., Moore, K. M., & Luenzmann, K. R. (2003b). Obsessive-compulsive symptoms in pregnancy and the puerperium: a review of the literature. *J Anxiety Disord, 17*(4), 461-478.

Abrams, S. M., Field, T., Scafidi, F., & Prodromidis, M. (2006). Newborns of depressed mothers. *Infnat Mental Health Journal, 16*(3), 233-239.

Agrati, D., Fernández-Guasti, A., Zuluaga, M. J., Uriarte, N., Pereira, M., & Ferreira, A. (2005). Compulsive-like behaviour according to the sex and the reproductive stage of female rats. *Behavioural Brain Research, 161*(2), 313-319. doi:http://dx.doi.org/10.1016/j.bbr.2005.02.017

Ahuja, J. K. A., Montville, J. B., Omolewa-Tomobi, G., Heendeniya, K. Y., Martin, C. L., Steinfeldt, L. C., . . .Moshfegh, A. J. (2012). USDA Food and Nutrient Database for Dietary Studies, 5.0. U.S. Department of Agriculture, Agricultural Research Service, Food Surveys Research Group, Beltsville, MD.

Aiken, L. S., West, S. G., & Reno, R. R. (1991). *Multiple regression: Testing and interpreting interactions*. Sage.

Albert, U., Maina, G., & Bogetto, F. (2000). Obsessive-compulsive disorder (OCD) and triggering life events. *The European Journal of psychiatry, 14*(3), 180-188.

Alexander, G. R., & Cornely, D. A. (1987). Racial disparities in pregnancy outcomes: The role of prenatal care utilization and maternal risk status. *American Journal of Preventive*

Medicine, 3(5), 254-261.

Alonso, P., Lopex-Sola, C., Real, E., Segalas, C., & Menchon, J. M. (2015). Animal models of obsessive-compulsive disorder: Utility and limitations. *Neuropsychiatric Disease and Treatment, 11,* 1939-1955.

Alpers, G. W., Abelson, J. L., Wilhelm, F. H., & Roth, W. T. (2003). Salivary Cortisol Response During Exposure Treatment in Driving Phobics. *Psychosomatic Medicine, 65*(4), 679-687. doi:10.1097/01.psy.0000073872.85623.0c

Altemus, M., Pigott, T., Kalogeras, K. T., Demitrack, M., Dubbert, B., Murphy, D. L., & Gold, P. (1992). Abnormalities in the regulation of vasopressin and corticotropin releasing factor secretion in obsessive-compulsive disorder. *Archives of General Psychiatry, 49*(1), 9-20. doi:10.1001/archpsyc.1992.01820010009002

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.

Arnold, L. M. (1999). A Case Series of Women With Postpartum-Onset Obsessive-Compulsive Disorder. *Primary Care Companion to The Journal of Clinical Psychiatry, 1*(4), 103-108. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC181073/

Aubuchon–Endsley, N. L., Thomas, D. G., Kennedy, T. S., Grant, S. L., & Valtr, T. (2012). Interactive relations among maternal depressive symptomatology, nutrition, and parenting. *Women & Health, 52*(3), 197-213.

Austin, M. P., Hadzi-Pavlovic, D., Leader, L., Saint, K., & Parker, G. (2005). Maternal trait anxiety, depression and life event stress in pregnancy: relationships with temperament. *Early Human Development, 81*, 183-190.

Austin, M.P., & Leader, L. (2000). Maternal stress and obstetric and infant outcomes:

epidemiological findings nad neuroendrocrine mechanisms. *American and New Zealand Journal of Obstetrics and Gynaecology, 40*(3), 331-337.

Azizi-Egrari, R., Neumann, C. G., Bourque, L. B., Harrison, G. G., & Sigman, M. D. (2004). Maternal anemia and postpartum weight change associated with decreased maternal-infnat interaction in a rural Kenyan population. *Ecology of Food and Nutrition, 43*(5), 355-373. doi: 10.1080/03670240490500253

Baer, L., Brown-Beasley, M. W., Sorce, J., & Henriques, A. I. (1993). Computer-assisted of a structured interview for obsessive-compulsive disorder. *American Journal of Psychiatry, 150*, 1737-1738.

Barry, J. S., & Anthony, R. V. (2008). The Pregnant Sheep as a Model for Human Pregnancy. *Theriogenology, 69*(1), 55-67. doi:10.1016/j.theriogenology.2007.09.021

Baum, A., Garofalo, J. P., Yali, A. M. (2006). Socioeconomic status and chronic stress: Does stress account for SES effects on health? *Annals of the New York Academy of Sciences, 896*(1), 131-144.

Baumeister, R. F., Vohs, K. D., & Funder, D. C. (2007). Psychology as the science of self-reports and finger movements: Whatever happened to actual behavior? *Perspectives on Psychological Science, 2*(4), 396-403.

Beck, C. T. (2001). Predictors of postpartum depression: An update. *Nursing Research, 50*(5), 275-285.

Belsky, J. (1980). Mother-infant interaction at home and in the laboratory: A comparative study. *The Journal of Genetic Psychology, 137*, 37-47.

Bennett, H. A., Einarson, A., Taddio, A., Koren, G., & Einarson, T. R. (2004). Prevalence of Depression During Pregnancy: Systematic Review. *Obstetrics & Gynecology, 103*(4),

698-709. doi:10.1097/01.AOG.0000116689.75396.5f

Bergman, K., Sarkar, P., O'Connor, T. G., Modi, N., & Glover, V. (2007). Maternal Stress During Pregnancy Predicts Cognitive Ability and Fearfulness in Infancy. *Journal of the American Academy of Child & Adolescent Psychiatry, 46*(11), 1454-1463. doi:http://dx.doi.org/10.1097/chi.0b013e31814a62f6

Bertram, C. E., & Hanson, M. A. (2002). Prenatal programming of postnatal endocrine responses by glucocorticoids. *Reproduction, 124*(4), 459-467.

Blais, A. R., & Weber, E. U. (2006). A domain-specific risk-taking (DOSPERT) scale for adult populations. *Judgment and Decision Making, 1*(1), 33-47.

Blumberg, M. S. & Sokoloff, G. (2001). Do infant rats cry? *Psychological Review, 108*(1), 83-95.

Blumenshine, P., Egerter, S., Barclay, C. J., Cubbin, C., & Braveman, P. A. (2010). Socioeconomic Disparities in Adverse Birth Outcomes: A Systematic Review. *American Journal of Preventive Medicine, 39*(3), 263-272. doi:http://dx.doi.org/10.1016/j.amepre.2010.05.012

Bodnar, L. M., & Wisner, K. L. (2005). Nutrition and depression: Implications for improving mental health among childbearing-aged women. *Biological Psychiatry, 58*(9), 679-685. doi: http://dx.doi.org/10.1016/j.biopsych.2005.05.009

Bornstein, S. R., Schuppenies, A., Wong, M. L., & Licinio, J. (2006). Approaching the shared biology of obesity and depression: The stress axis as the locus of gene-environment interactions. *Molecular Psychiatry, 11*(10), 892-902.

Bradley, R. H., & Corwyn, R. F. (2002). Socioeconomic status and child development. *Annual Review of Psychology, 53*(1), 371-399.

Brandes, M., Soares, C. N., & Cohen, L. S. (2004). Postpartum onset obsessive-compulsive disorder: diagnosis and management. *Archives of Women's Mental Health, 7*(2), 99-110. doi:10.1007/s00737-003-0035-3

Braungart-Rieker J. M., Hill-Soderlund, A. L., & Karrass, J. (2010). Fear and anger reactivity trajectories from 4 to 16 months: The roles of temperament, regulation, and maternal sensitivity. *Developmental Psychology, 46*, 791-804.

Braveman, P., Cubbin, C., Marchi, K., Egerter, S., & Chavez, G. (2001). Measuring socioeconomic status/position in studies of racial/ethnic disparities: Maternal and infant health. *Public Health Reports*, *116*(5), 449–463.

Brazelton, T. B., & Nugent, J. K. (1995). *Neonatal behavioral assessment scale*: Cambridge University Press.

Brouwers, E. P. M., van Baar, A. L., & Pop, V. J. M. (2001). Maternal anxiety during pregnancy and subsequent infant development. *Infant Behavior & Development, 24*, 95-106.

Buitelaar, J. K., Huizink, A. C., Mulder, E. J., de Medina, P. G., & Visser, G. H. A. (2003). Prenatal stress and cognitive development and temperament in infants. *Neurobiology of Aging, 24, Supplement 1*, S53-S60.

Buss, C., Davis, E. P., Hobel, C. J., & Sandman, C. A. (2011). Maternal pregnancy-specific anxiety is associated with child executive function at 6-9 years age. *Stress, 14*(6), 665-676.

Buss, C., Davis, E. P., Muftuler, L. T., Head, K., & Sandman, C. A. (2010). High pregnancy aniety during mid-gestation is associated with decreased gray matter density in 6-9 year-old children. *Psychoneuroendocrinology, 35*(1), 141-153.

Canivet, C. A., Ostergren, P. O., Rosen, A. S., Jakobsson, I. L., & Hagander, B. M. (2005).

Infantile colic and the role of trait anxiety during pregnancy in relation to psychosocial and socioeconomic factors. *Scandinavian Journal of Public Health, 33*(1), 26-34. doi:10.1080/14034940410028316

Carter, A. M. (2007). Animal Models of Human Placentation – A Review. *Placenta, 28, Supplement,* S41-S47. doi:http://dx.doi.org/10.1016/j.placenta.2006.11.002

Carter, A. S., Baker, C. W., & Brownell, K. D. (2000). Body mass index, eating attitudes, and symptoms of depression and anxiety in pregnancy and the postpartum period. *Psychosomatic Medicine, 62*(2), 264-270.

Carter, A. S., Garrity-Rokous, F. E., Chazan-Cohen, R., Little, C., & Briggs-Gowan, M. J. (2001). Maternal depression and comorbidity: Predicting early parenting, attachment security, and toddler social-emotional problems and competencies. *Journal of the American Academy of Child & Adolescent Psychiatry, 40*(1), 18-26. doi:http://dx.doi.org/10.1097/00004583-200101000-00012

Carter, C. S. (2014). Oxytocin Pathways and the Evolution of Human Behavior. *Annual Review of Psychology, 65*(1), 17-39. doi:doi:10.1146/annurev-psych-010213-115110

Catapano, F., Monteleone, P., Fuschino, A., Maj, M., & Kemali, D. (1992). Melatonin and cortisol secretion in patients with primary obsessive-compulsive disorder. *Psychiatry Research, 44*(3), 217-225. doi:http://dx.doi.org/10.1016/0165-1781(92)90025-X

Challacombe, F. L., Salkovskis, P. M., Woolgar, M., Wilkinson, E. L., Read, J., & Acheson, R. (2016). Parenting and mother-infant interactions in the context of maternal postpartum obsessive-compulsive disorder: Effects of obsessional symptoms and mood. *Infant Behavior and Development, 44*, 11-20. doi:https://doi.org/10.1016/j.infbeh.2016.04.003

Chappell, P., Leckman, J., Goodman, W., Bissette, G., Pauls, D., Anderson, G., . . . Cohen, D.

(1996). Elevated cerebrospinal fluid corticotropin-releasing ractor in Tourette's syndrome: Comparison to obsessive compulsive disorder and normal controls. *Biological Psychiatry, 39*(9), 776-783. doi:http://dx.doi.org/10.1016/0006-3223(95)00221-9

Chelmow, D., & Halfin, V. P. (1997). Pregnancy complicated by Obsessive-Compulsive Disorder. *Journal of Maternal-Fetal Medicine, 6*(1), 31-34. doi:10.3109/14767059709161948

Cirino, P. T., Chin, C. E., Sevcik, R. A., Wolf, M., Lovett, M., & Morris, R. D. (2002). Measuring socioeconomic status reliability and preliminary validity for different approaches. *Assessment, 9*(2), 145-155.

Cogill, S. R., Caplan, H. L., Alexandra, H., Robson, K. M., & Kumar, R. (1986). Impact of maternal postnatal depression on cognitive development of young children. *British Medical Journal, 292*(6529), 1165-1167. Retrieved from http://www.bmj.com/content/292/6529/1165.abstract

Condren, R. M., O'Neill, A., Ryan, M. C. M., Barrett, P., & Thakore, J. H. (2002). HPA axis response to a psychological stressor in generalised social phobia. *Psychoneuroendocrinology, 27*(6), 693-703. doi:10.1016/S0306-4530(01)00070-1

Conradt, E., Lester, B. M., Appleton, A. A., Armstrong, D. A., & Marsit, C. J. (2013). The roles of DNA methylation of NR3C1 and 11β-HSD2 and exposure to maternal mood disorder in utero on newborn neurobehavior. *Epigenetics, 8*(12), 1321-1329.

Copper, R. L., Goldenberg, R. L., Das, A., Elder, N., Swain, M., Norman, G., . . . Meier, A. (1996). The preterm prediction study: Maternal stress is associated with spontaneous preterm birth at less than thirty-five weeks' gestation. *American Journal of Obstetrics and Gynecology, 175*(5), 1286-1292. doi:http://dx.doi.org/10.1016/S0002-9378(96)70042-X

Cox, J. L., Holden, J. M., & Sagovsky, R. (1987). Detection of postnatal depression. Development of the 10-item Edinburgh Postnatal Depression Scale. *The British Journal of Psychiatry, 150*(6), 782-786.

Crockenberg, S. C., & Leerkes, E. M. (2006). Infant and maternal behavior moderate reactivity to novelty to predict anxious behavior at 2.5 years. *Development and Psychopathology, 18*(1), 17-34.

Crockenberg, S. C., Leerkes, E. M., & BArrig JO, P. S. (2008). Predicting aggressive behavior in the third year from infant reactivity and regulation as moderated by maternal behavior. *Development and Psychopathology, 20*(1), 37-54.

Crockenberg, S. B., & Smith, P. (1982). Antecedents of mother–infant interaction and infant irritability in the first three months of life. *Infant Behavior & Development, 5*(2), 105-119. doi:10.1016/S0163-6383(82)80021-0

Cromer, K. R., Schmidt, N. B., & Murphy, D. L. (2007). An investigation of traumatic life events and obsessive-compulsive disorder. *Behaviour Research and Therapy, 45*(7), 1683-1691. doi:http://dx.doi.org/10.1016/j.brat.2006.08.018

Curley, J. P., Jensen, C. L., Mashoodh, R., & Champagne, F. A. (2011). Social influences on neurobiology and behavior: Epigenetic effects during development. *Psychoneuroendocrinology, 36*(3), 352-371.

Darnaudéry, M., & Maccari, S. (2008). Epigenetic programming of the stress response in male and female rats by prenatal restraint stress. *Brain Research Reviews, 57*(2), 571-585. doi:http://dx.doi.org/10.1016/j.brainresrev.2007.11.004

Davis, E. P., Glynn, L. M., Schetter, C. D., Hobel, C., Chicz-Demet, A., & Sandman, C. A. (2005). Corticotropin-releasing hormone during pregnancy is associated with infant

temperament. *Developmental Neuroscience, 27*, 299-305.

Davis, E. P., Glynn, L. M., Schetter, C. D., Hobel, C., Chicz-Demet, A., & Sandman, C. A. (2007). Prenatal Exposure to Maternal Depression and Cortisol Influences Infant Temperament. *Journal of the American Academy of Child & Adolescent Psychiatry, 46*(6), 737-746. doi:http://dx.doi.org/10.1097/chi.0b013e318047b775

Davis, E. P., Snidman, N., Wadhwa, P. D., Glynn, L. M., Schetter, C. D., & Sandman, C. A. (2004). Prenatal Maternal Anxiety and Depression Predict Negative Behavioral Reactivity in Infancy. *Infancy, 6*(3), 319-331. doi:10.1207/s15327078in0603_1

de Weerth, C., van Hees, Y., & Buitelaar, J. K. (2003). Prenatal maternal cortisol levels and infant behavior during the first 5 months. *Early Human Development, 74*, 139-151.

Desai, S., & Alva, S. (1998). Maternal Education and Child Health: Is There a Strong Causal Relationship? *Demography, 35*(1), 71-81. Retrieved from http://www.jstor.org/stable/3004028

Diego, M. A., Field, T., & Hernandez-Reif, M. (2005). Prepartum, postpartum and chronic depression effects on neonatal behavior. *Infant Behavior and Development, 28*(2), 155-164. doi:http://dx.doi.org/10.1016/j.infbeh.2005.02.002

Dinarello, C. A. (2000). Proinflammatory cytokines. *Chest, 118*(2), 503-508.

DiPietro, J. A., Novak, M. F. S. X., Costigan, K. A., Atella, L. D., & Reusing, S. P. (2006). Maternal psychological distress during pregnancy in relation to child development at age two. *Child Development, 77*(3), 573-587.

Duncan, O. D. (1961). A socioeconomic index for all occupations. In A. J. Reiss (Ed.), *Occupations and social status*. New York: Free Press.

Eberhard-Gran, M., Eskild, A., Tambs, K., Opjordsmoen, S., & Samuelsen, S. O. (2001). Review

of validation studies of the Edinburgh Postnatal Depression Scale. *Acta Psychiatric Scandinavia, 104*(4), 243-249.

Faden, V. B., & Graubard, B. I. (2000). Maternal substance use during pregnancy and developmental outcome at age three. *Journal of Substance Abuse, 12*(4), 329-340. doi: http://dx.doi.org/10.1016/S0899-3289(01)00052-9

Fairbrother, N., & Abramowitz, J. S. (2007). New parenthood as a risk factor for the development of obsessional problems. *Behaviour Research and Therapy, 45*(9), 2155-2163. doi:10.1016/j.brat.2006.09.019

Fernandez-Guasti, A., Agrati, D., Reyes, R., & Ferreira, A. (2006). Ovarian steroids counteract serotonergic drugs actions in an animal model of obsessive-compulsive disorder. *Psychoneuroendocrinology, 31*(8), 924-934.

Feder, A., Coplan, J. D., Goetz, R. R., Mathew, S. J., Pine, D. S., Dahl, R. E., . . . Weissman, M. M. (2004). Twenty-four-hour cortisol secretion patterns in prepubertal children with anxiety or depressive disorders. *Biological Psychiatry, 56*(3), 198-204. doi:10.1016/j.biopsych.2004.05.005

Field, A. P. (2009). *Discovering statistics using SPSS.* London: Sage

Field, T. (1998). Maternal Depression Effects on Infants and Early Interventions. *Preventive Medicine, 27*(2), 200-203. doi:http://dx.doi.org/10.1006/pmed.1998.0293

Field, T., & Diego, M. (2008). Prenatal dysthymia versus major depression effects on the neonate. *Infant Behavior & Development, 31*(2), 190-193.

Field, T., Diego, M., Dieter, J., Hernandez-Reif, M., Schanberg, S., Kuhn, C., . . . Bendell, D. (2004). Prenatal depression effects on the fetus and the newborn. *Infant Behavior & Development, 27*, 216-229.

Field, T., Diego, M., & Hernandez-Reif, M. (2006). Prenatal depression effects on the fetus and newborn: a review. *Infant Behavior and Development, 29*(3), 445-455. doi:http://dx.doi.org/10.1016/j.infbeh.2006.03.003

Field, T., Diego, M., Hernandez-Reif, M., Schanberg, S., Kuhn, C., Yando, R., & Bendell, D. (2003). Pregnancy anxiety and comorbid depression and anger: Effects on the fetus and neonate. *Depression and Anxiety, 17*(3), 140-151. doi:10.1002/da.10071

Forray, A., Focseneanu, M., Pittman, B., McDougle, C. J., & Epperson, C. N. (2010). Onset and exacerbation of Obsessive-Compulsive Disorder in pregnancy and the postpartum period. *The Journal of Clinical Psychiatry, 71*(8), 1061-1068. doi:10.4088/JCP.09m05381blu

Fries, E., Dettenborn, L., & Kirschbaum, C. (2009). The cortisol awakening response (CAR): Facts and future directions. *International Journal of Psychophysiology, 72*(1), 67-73. doi:http://dx.doi.org/10.1016/j.ijpsycho.2008.03.014

Frost, R. O., Sher, K. J., & Geen, T. (1986). Psychopathology and personality characteristics of nonclinical compulsive checkers. *Behaviour Research and Therapy, 24*(2), 133-143. doi:http://dx.doi.org/10.1016/0005-7967(86)90084-7

Fullana, M. A., Mataiz-Cols, D., Caspi, A, Harrington, H., Grisham, J. R., Moffitt, T. E., & Poulton, R. (2009). Obsessions and compulsions in the community: prevalence, interference, help-seeking, developmental stability, and co-occurring psychiatric conditions. *American Journal of Psychiatry, 166*(3), 329-336.

Furber, C. M., Garrod, D., Maloney, Eileen, Lovell, K., & McGowan, L. (2009). A qualitative study of mild to moderate psychological distress during pregnancy. *International Journal of Nursing Studies, 46*(5), 669-677.

Garstein M. A., & Bateman, A. E. (2008). Early manifestations of childhood depression:

influences of infant temperament and parental depressive symptoms. *Infant and Child Development, 17*(3), 223-248.

Garstein, M. A., & Marmion, J. (2008). Fear and positive affectivity in infancy: Convergence/discrepancy between parent-report and laboratory-based indicators. *Infant Behavior & Development, 31*, 227-238.

Garstein, M. A., & Rothbart, M. K. (2003). Studying infant temperament via the Revised Infant Behavior Questionnaire. *Infant Behavior & Development, 26*, 64-86.

Garstein, M. A., Slobodskaya, H. R., & Kinsht, I. A. (2003). Cross-cultural differences in temperament in the first year of life: United States of America (US) and Russia. *International Journal of Behavioral Development, 27*(4), 316-328.

Gehris, T. L., Kathol, R. G., Black, D. W., & Noyes Jr, R. (1990). Urinary free cortisol levels in obsessive-compulsive disorder. *Psychiatry Research, 32*(2), 151-158. doi:http://dx.doi.org/10.1016/0165-1781(90)90081-F

Geller, P. A., Klier, C. M., & Neugebauer, R. (2001). Anxiety disorders following miscarriage. *Journal of Clinical Psychiatry, 62*(6), 432-438. doi:10.4088/JCP.v62n0606

Gitau, R., Cameron, A., Fisk, N. M., & Glover, V. (1998). Fetal exposure to maternal cortisol. *The Lancet, 352*, 707-708.

Gitto, E., Pellegrino, S., Gitto, P., Barberi, I., & Reiter, R. J. (2009). Oxidative stress of the newborn in the pre-and postnatal period and the clinical utility of melatonin. *Journal of Pineal Research, 46*(2), 128-139.

Glover, V., & O'Connor, T. G. (2002). Effects of antenatal stress and anxiety. *Implications for development and psychiatry, 180*(5), 389-391. doi:10.1192/bjp.180.5.389

Godfrey, K. M., & Barker, D. J. (2000). Fetal nutrition and adult disease. *The American Journal*

of Clinical Nutrition, 71(5), 1344s-1352s.

Goldsmith, H. H., & Rothbart, M. K. (1988). The Laboratory Temperament Assessment Battery (Lab-TAB): Locomotor Version (No. 88-01). Oregon Center for the Study of Emotion.

Gonda, X., Jekkel, E., Varga, A., Miklosi, M., & Forintos, D. P. (2008). Advantage of obsessive-compulsive symptoms from the aspect of individual selection and group selection: An evolutionary psychological approach to obsessive-compulsive disorder. *Neuropsychopharmacologia Hungarica, 10*(4), 225-232.

Gorman, J. M. (1996). Comorbid depression and anxiety spectrum disorders. *Depression and Anxiety, 4*(4), 160-168. doi:10.1002/(SICI)1520-6394(1996)4:4<160::AID-DA2>3.0.CO;2-J

Gothelf, D., Aharonovsky, O., Horesh, N., Carty, T., & Apter, A. (2004). Life events and personality factors in children and adolescents with obsessive-compulsive disorder and other anxiety disorders. *Comprehensive Psychiatry, 45*(3), 192-198. doi:http://dx.doi.org/10.1016/j.comppsych.2004.02.010

Gottfried, A. W. (1985). Measures of socioeconomic status in child development research: Data and recommendations. *Merrill-Palmer Quarterly, 31*(1), 85-92.

Grace, S. L., Evindar, A., & Stewart, D. E. (2003). The effect of postpartum depression on child cognitive development and behavior: A review and critical analysis of the literature. *Archives of Women's Mental Health, 6*(4), 263-274.

Gustafsson, P. E., Gustafsson, P. A., Ivarsson, T., & Nelson, N. (2008). Diurnal cortisol levels and cortisol response in youths with Obsessive-Compulsive Disorder. *Neuropsychobiology, 57*(1-2), 14-21. Retrieved from http://www.karger.com/DOI/10.1159/000123117

Gutteling, B. M., de Weerth, C., & Buitelaar, J. K. (2005a). Prenatal stress and children's cortisol reaction to the first day of school. *Psychoneuroendocrinology, 30*(6), 541-549. doi:http://dx.doi.org/10.1016/j.psyneuen.2005.01.002

Gutteling, B. M., de Weerth, C., Willemsen-Swinkels, S. H. N., Huizink, A. C., Mulder, E. J. H., Visser, G. H. A., & Buitelaar, J. K. (2005b). The effects of prenatal stress on temperament and problem behavior of 27-month-old toddlers. *European Child & Adolescent Psychiatry, 14*, 41-51.

Hanley, J. A., & McNeil, B. J. (1982). The meaning and use of the area under a receiver operating characteristic (ROC) curve. *Radiology, 148*(1), 29-36.

Harris, B., Huckle, P., Thomas, R., Johns, S., & Fung, H. (1989). The use of rating scales to identify post-natal depression. *The British Journal of Psychiatry, 154*, 813-817.

Hedegaard, M., Henriksen, T. B., Sabroe, S., & Secher, N. J. (1993). Psychological distress in pregnancy and preterm delivery. *British Medical Journal, 307*, 234-239.

Heron, J., O'Connor, T. G., Evans, J., Golding, J., & Glover, V. (2004). The course of anxiety and depression through pregnancy and the postpartum in a community sample. *Journal of Affective Disorders, 80*(1), 65-73.

Hoffman, S., & Hatch, M. C. (2000). Depressive symptomatology during pregnancy: evidence for an association with decreased fetal growth in pregnancies of lower social class women. *Health Psychology : Official Journal of the Division of Health Psychology, American Psychological Association, 19*(6), 535-543. doi:10.1037//0278-6133.19.6.535

Hollingshead, A. A. (1975). Four-factor index of social status. Unpublished manuscript, Yale University, New Haven, CT.

Hooper, L., Stockton, P., Krupnick, J., & Green, B., (2011). Development, use, and psychometric

properties of the Trauma History Questionnaire. *Journal of Loss and Trauma, 16*, 258-283. doi: 10.1080/15325024.2011.572035

Huizink, A. C. (2001). *Prenatal stress and its effect on infant development.* (Doctorate), University of Utrecht, The Netherlands.

Huizink, A. C., Robles de Medina, P. G., Mulder, E. J. H., Visser, G. H. A., & Buitelaar, J. K. (2003). Stress during pregnancy is associated with developmental outcome in infancy. *Journal of Child Psychology and Psychiatry, 44*(6), 810-818. doi:10.1111/1469-7610.00166

Jennings, K. D., Ross, S., Popper, S., & Elmore, M. (1999). Thoughts of harming infants in depressed and nondepressed mothers. *Journal of Affective Disorders, 54*(1-2), 21-28.

Jones, N. A., Field, T., Fox, N. A., Davalos, M., Lundy, B., & Hart, S. (1998). Newborns of mothers with depressive symptoms are physiologically less developed. *Infant Behavior & Development, 21*(3), 537-541.

Jones, N. A., Field, T., Fox, N. A., Lundy, B., & Davalos, M. (1997). EEG activation in 1-month-old infants of depressed mothers. *Development & Psychopathology, 9*(3), 491-505.

Kagan, J. (1997). Temperament and the reactions to unfamiliarity. *Child Development, 68*(1), 139-143. doi:10.2307/1131931

Kagan, J., Reznick, J., & Snidman, N. (1988). Biological bases of childhood shyness. *Science, 240*(4849), 167-171. doi:10.1126/science.3353713

Kagan, J., Snidman, N., & Arcus, D. (1998). Childhood derivatives of high and low reactivity in infancy. *Child Development, 69*(6), 1483-1493. doi:10.2307/1132126

Kingston, D., Tough, S., & Whitfield, H. (2012). Prenatal and postpartum maternal

psychological distress and infant development: a systematic review. *Child Psychiatry & & Human Development, 43*(5), 683-714. doi: 10.1007/s10578-012-0291-4

Kivimaki, M., Lawlor, D. A., Smith, G. D., Kouvonen, A., Virtanen, M., Elovainio, M., & Vahtera, J. (2007). Socioeconomic position, co-occurrence of behavior-related risk factors, and coronary heart disease: the Finnish Publich Sectory Study. *American Journal of Public Health, 97*(5), 874-879.

Kluge, M., Schüssler, P., Künzel, H. E., Dresler, M., Yassouridis, A., & Steiger, A. (2007). Increased nocturnal secretion of ACTH and cortisol in obsessive compulsive disorder. *Journal of Psychiatric Research, 41*(11), 928-933. doi:http://dx.doi.org/10.1016/j.jpsychires.2006.08.005

Kolada, J. L., Bland, R. C., & Newman, S. C. (1994). Obsessive-Compulsive Disorder. *Acta Psychiatrica Scandinavica, 89*, 24-35. doi:10.1111/j.1600-0447.1994.tb05788.x

Komsi, N., Räikkönen, K., Heinonen, K., Pesonen, A.-K., Keskivaara, P., Järvenpää, A. L., & Strandberg, T. E. (2008). Continuity of father-rated temperament from infancy to middle childhood. *Infant Behavior and Development, 31*(2), 239-254. doi:http://dx.doi.org/10.1016/j.infbeh.2007.10.002

Labad, J., Alonso, P., Segalas, C., Real, E., Jimenez, S., Bueno, B., . . . Menchon, J. M. (2010). Distinct correlates of hoarding and cleaning symptom dimensions in relation to onset of obsessive-compulsive disorder at menarche or the perinatal period. *Archives of Women's Mental Health, 13*(1), 75-81. doi:10.1007/s00737-009-0098-x

Labad, J., Menchon, J. M., Alonso, P., Segalas, C., Jimenez, S., Jaurrieta, N., . . . Vallejo, J. (2008). Gender differences in obsessive–compulsive symptom dimensions. *Depression and Anxiety, 25*(10), 832-838. doi:10.1002/da.20332

Labad, J., Menchon, J. M., Alonso, P., Segalas, C., Jimenez, S., & Vallejo, J. (2005). Female reproductive cycle and obsessive-compulsive disorder. *Journal of Clinical Psychiatry, 66*(4), 428-435; quiz 546.

LaCoursiere, D. Y., Barrett-Connor, E., O'Hara, M. W., Hutton, A., & Varner, M. W. (2010). The association between prepregnancy obesity and screening positive for postpartum depression. *BJOG: An International Journal of Obstetrics & Gynaecology, 117*(8), 1011-1018. doi: 10.1111/j.1471-0528.2010.02569.x

Lanius, R. A., Vermetten, E., & Pain, C. (2010). *The impact of early life trauma on health and disease: The hidden epidemic.* New York, NY: Cambridge University Press.

Laplante, D. P., Barr, R. G., Brunet, A., du Fort, G. G., Meaney, M. L., Saucier, J.-F., . . . King, S. (2004). Stress during pregnancy affects general intellectual and language functioning in human toddlers. *Pediatric Research, 56*(3), 400-410.

Leckman, J. F., Goodman, W. K., North, W. G., Chappell, P. B., Price, L. H., Pauls, D. L., . . . Cohen, D. J. (1994a). The role of central oxytocin in obsessive compulsive disorder and related normal behavior. *Psychoneuroendocrinology, 19*(8), 723-749. doi:http://dx.doi.org/10.1016/0306-4530(94)90021-3

Leckman, J. F., Goodman, W. K., North, W. G., Chappell, P. B., Price, L. H., Pauls, D. L., . . . Cohen, D. J. (1994b). Elevated cerebrospinal fluid levels of oxytocin in obsessive-compulsive disorder: Comparison with tourette's syndrome and healthy controls. *Archives of General Psychiatry, 51*(10), 782-792. doi:10.1001/archpsyc.1994.03950100030003

Leckman, J. F., Mayes, L. C., Feldman, R., Evans, D. W., King, R. A., & Cohen, D. J. (1999). Early parental preoccupations and behaviors and their possible relationship to the

symptoms of obsessive-compulsive disorder. *Acta Psychiatrica Scandinavica, 100*, 1-26. doi:10.1111/j.1600-0447.1999.tb10951.x

Leerkes, E. M., Supple, A. J., O'Brien, M., Calkins, S. D., Haltigan, J. D., Wong, M. S., & Fortuna, K. (2014). Antecedents of maternal sensitivity during distressing tasks: Integrating attachment, social information processing, and psychobiological perspectives. *Child Development, 0*, 1-18. doi: 10.1111/cdev.12288

Lester, B. M., Andreozzi-Fontaine, L., Tronick, E., & Bigsby, R. (2014). Assessment and evaluation of the high risk neonate: the NICU Network Neurobehavioral Scale. *J Vis Exp*(90). doi:10.3791/3368

Lester, B. M., & Tronick, E. Z. (2004). History and description of the Neonatal Intensive Care Unit Network Neurobehavioral Scale. *Pediatrics, 113*(3), 634-640.

Liu, J., Bann, C., Lester, B., Tronick, E., Abhik, D., Lagasse, L., . . . Bada, H. (2010). Neonatal neurobehavior predicts medical and behavioral outcome. *Pediatrics, 125*(1), e90-e98. doi:10.1542/peds.2009-0204

Lord, C., Rieder, A., Hall, G. B., Soares, C. N., & Steiner, M. (2011). Piloting the Perinatal Obsessive-Compulsive Scale (POCS): Development and validation. *Journal of Anxiety Disorders, 25*(8), 1079-1084. doi: 10.1016/j.janxdis.2011.07.005

Lord, C., Steiner, M., Soares, C. N., Carew, C. L., & Hall, G. B. (2012). Stress response in postpartum women with and without obsessive–compulsive symptoms: an fMRI study. *Journal of Psychiatry & Neuroscience : JPN, 37*(2), 78-86. doi:10.1503/jpn.110005

Lundy, B., Field, T., & Pickens, J. (1996). Newborns of mothers with depressive symptoms are less expressive. *Infant Behavior & Development, 19*(4), 419-424.

Lundy, B., Jones, N. A., Field, T., Nearing, G., Davalos, M., Pietro, P. A., . . . Kuhn, C. (1999).

Prenatal depression effects on neonates. *Infant Behavior & Development, 22*(1), 119-129.

Luoma, I., Tamminen, T., Kaukonen, P., Laippala, P., Puura, K., Salmelin, R., & Almqvist, F. (2001). Longitudinal study of maternal depressive symptoms and child well-being. *Journal of the American Academy of Child & Adolescent Psychiatry, 40*(12), 1367-1374.

Lupien, S. J. & Seguin, F. (2007). *How to Measure Stress in Humans*. Retrieved from http://www.humanstress.ca/documents/pdf/SalivaLab/HOW%20TO%20MEASURE%20STRESS_CSHS.pdf

Maccari, S., Darnaudery, M., Morley-Fletcher, S., Zuena, A. R., Cinque, C., & Van Reeth, O. (2003). Prenatal stress and long-term consequences: implications of glucocorticoid hormones. *Neuroscience & Biobehavioral Reviews, 27*(1–2), 119-127. doi:http://dx.doi.org/10.1016/S0149-7634(03)00014-9

Maina, G., Albert, U., Bogetto, F., Vaschetto, P., & Ravizza, L. (1999). Recent life events and obsessive–compulsive disorder (OCD): the role of pregnancy/delivery. *Psychiatry Research, 89*(1), 49-58. doi:http://dx.doi.org/10.1016/S0165-1781(99)00090-6

Mak, I. W. Y., Evaniew, N., & Ghert, M. (2014). Lost in translation: animal models and clinical trials in cancer treatment. *American Journal of Translational Research, 6*(2), 114-118. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3902221/

Mancuso, R. A., Schetter, C. D., Rini, C. M., Roesch, S. C., & Hobel, C. J. (2004). Maternal prenatal anxiety and corticotropin-releasing hormone associated with timing of delivery. *Psychosomatic Medicine, 66*, 762-769.

Mangelsdorf, S. C., Schoppe, S. J., & Buur, H. (2014). The meaning of parental reports: A contextual approach to the study of temperament and behavior problems in childhood. In V. J. Molfese & D. L. Molfese (Eds.), *Temperament and Personality Development*

Across the Life Span (121-134). New York, NY: Routledge.

Marcus, S., Lopez, J. F., McDonough, S., MacKenzie, M. J., Flynn, H., Neal Jr, C. R., . . . Vazquez, D. M. (2011). Depressive symptoms during pregnancy: Impact on neuroendocrine and neonatal outcomes. *Infant Behavior and Development, 34*(1), 26-34. doi:http://dx.doi.org/10.1016/j.infbeh.2010.07.002

Marcus, S. M., Flynn, H. A., Blow, F. C., & Barry, K. L. (2004). Depressive symptoms among pregnant women screened in obstetrics settings. *Journal of Women's Health, 12*(4), 373-380.

Marshall, L. (1992). Development of the severity of violence against women scales. *Journal of Family Violence, 7*(2), 103-121. doi: 10.1007/BF00978700

Matthey, S., Henshaw, C., Elliott, S., & Barnett, B. (2006). Variability in use of cut-off scores and formats on the Edinburgh Postnatal Depression Scale: Implications for clinical and research practice. *Archives of Women's Mental Health, 9*(6), 309-315.

Martić-Kehl, M. I., Schibli, R., & Schubiger, P. A. (2012). Can animal data predict human outcome? Problems and pitfalls of translational animal research. *European Journal of Nuclear Medicine and Molecular Imaging, 39*(9), 1492-1496. doi:10.1007/s00259-012-2175-z

Mataix-Cols, D., Wooderson, S., Lawrence, N., Brammer, M. J., Speckens, A., & Phillips, M. L. (2004). Distinct neural correlates of washing, checking, and hoarding symptom dimensions in obsessive-compulsive disorder. *Archives of General Psychiatry, 61*(6), 564-576. doi:10.1001/archpsyc.61.6.564

McElroy, S. L., Phillips, K. A., & Keck, P. E. (1994). Obsessive compulsive spectrum disorder. *The Journal of Clinical Psychiatry, 55 Suppl*, 33-51; discussion 52-33. Retrieved from

http://europepmc.org/abstract/MED/7961531

McKeon, J., Roa, B., & Mann, A. (1984). Life events and personality traits in obsessive-compulsive neurosis. *The British Journal of Psychiatry, 144*(2), 185-189. doi:10.1192/bjp.144.2.185

McLean, C. P., Asnaani, A., Litz, B. T., & Hofmann, S. G. (2011). Gender differences in anxiety disorders: Prevalence, course of illness, comorbidity and burden of illness. *Journal of Psychiatric Research, 45*(8), 1027-1035. doi:10.1016/j.jpsychires.2011.03.006

McLean, M., Bisits, A., Davies, J., Woods, R., Lowry, P., & Smith, R. (1995). A placental clock controlling the length of human pregnancy. *Nature Medicine, 1*(5), 460-463. Retrieved from http://dx.doi.org/10.1038/nm0595-460

Meaney, M. J. (2001). Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. *Annual Review of Neuroscience, 24*(1), 1161-1192. doi:doi:10.1146/annurev.neuro.24.1.1161

Menard, S. (1995). *Applied Logistic Regression Analysis: Sage University Series on Qunatitative Applications in the Social Sciences*. Thousand Oaks, CA: Sage.

Mennes, M., Bergh, B. V. d., Lagae, L., & Stiers, P. (2009). Developmental brain alterations in 17 year old boys are related to antenatal maternal anxiety. *Clinical Neurophysiology, 120*(6), 1116-1122. doi:http://dx.doi.org/10.1016/j.clinph.2009.04.003

Miller, E. S., Chu, C., Gollan, J., & Gossett, D. R. (2013). Obsessive-compulsive symptoms during the postpartum period. *The Journal of Reproductive Medicine, 58*(3-4), 115.

Moffitt, T. E., Caspi, A., & Rutter, M. (2005). Strategy for investigating interactions between measured genes and measured environments. *Archives of General Psychiatry, 62*(5), 473-481. doi:10.1001/archpsyc.62.5.473

Mohler, E., Parzer, P., Brunner, R., Wiebel, A., & Resch, F. (2006). Emotional stress in pregnancy predicts human infant reactivity. *Early Human Development, 82,* 731-737.

Monk, C., Georgieff, M. K., & Osterholm, E. A. (2013). Research review: Maternal prenatal distress and poor nutrition – mutually influencing risk factors affecting infant neurocognitive development. *Journal of Child Psychology and Psychiatry, 54*(2), 115-130. doi: 10.1111/jcpp.12000

Monteleone, P., Catapano, F., Del Buono, G., & Maj, M. (1994). Circadian rhythms of melatonin, cortisol and prolactin in patients with obsessive-compulsive disorder. *Acta Psychiatrica Scandinavica, 89*(6), 411-415. doi:10.1111/j.1600-0447.1994.tb01538.x

Monteleone, P., Catapano, F., Tortorella, A., & Maj, M. (1997). Cortisol Response to *d*-fenfluramine in patients with obsessive-compulsive disorder and in healthy subjects: Evidence for a gender-related effect. *Neuropsychobiology, 36*(1), 8-12. Retrieved from http://www.karger.com/DOI/10.1159/000119352

Mulder, E. J. H., Robles de Medina, P. G., Huizink, A. C., van den Bergh, B. R. H., Buitelaar, J. K., & Visser, G. H. A. (2002). Prenatal maternal stress: Effects on pregnancy and the (unborn) child. *Early Human Development, 70*(1–2), 3-14. doi:http://dx.doi.org/10.1016/S0378-3782(02)00075-0

Muller, J. M., Achtergarde, S., & Furniss, T. (2011). The influence of maternal psychopathology on ratings of child psychiatric symptoms: an SEM analysis on cross-informant agreement. *European Child & Adolescent, Psychiatry, 20*(5), 241-252.

Murray, L., & Cooper, P. J. (1997). Effects of postnatal depression on infant development. *Archives of Disease in Childhood, 77*(2), 99-101.

National Heart Lung and Blood Institute. (2011). Early Adult Reduction of Weight through

Lifestyle Intervention (EARLY) Trials. Retrieved September 27, 2012, from http://www.nhlbi.nih.gov/resources/obesity/trials/early.htm

Nestadt, G., Samuels, J., Riddle, M. A., Liang, K.-Y., Bienvenu, O. J., Hoehn-Saris, R., . . . Cullen, B. (2001). The relationship between obsessive-compulsive disorder and anxiety and affective disorders: results from the John Hopkins OCD family study. *Psychological Medicine, 31*, 481-487.

Neziroglu, F., Anemone, R., & Yaryura-Tobias, J. A. (1992). Onset of obsessive-compulsive disorder in pregnancy. *American Journal of Psychiatry, 149*(7), 947-950. doi:doi:10.1176/ajp.149.7.947

Niederhofer, H., & Reiter, A. (2004). Prenatal Maternal Stress, Prenatal Fetal Movements and Perinatal Temperament Factors Influence Behavior and School Marks at the Age of 6 Years. *Fetal Diagnosis and Therapy, 19*(2), 160-162. Retrieved from http://www.karger.com/DOI/10.1159/000075142

Norusis, M. J. , and SPSS Inc. (1994). *SPSS for Windows Professional Statistics. Release 6.1*. Englewood Cliffs: Prentice Hall.

O'Connor, T. G., Heron, J., & Glover, V. (2002a). Antenatal anxiety predicts child behavioral/emotional problems independently of postnatal depression. *Journal of the American Academy of Child & Adolesc Psychiatry, 41*(12), 1470-1477. doi:10.1097/00004583-200212000-00019

O'Connor, T. G., Heron, J., Golding, J., Beveridge, M., & Glover, V. (2002b). Maternal antenatal anxiety and children's behavioural/emotional problems at 4 years. *British Journal of Psychiatry, 180*, 502-508.

Papousek, M., & von Hofacker, N. (1998). Persistent crying in early infancy: a non-trivial

condition of risk for the developing mother-infant relationship. *Child: Care, Health, and Development, 24*(5), 395-424.

Parade, S. H., & Leerkes, E. M. (2008). The reliability and validity of the Infant Behavior Questionnaire-Revised. *Infant Behavior and Development, 31*(4), 637-646.

Pavuluri, M. N., Henry, D., & Allen, K. (2002). Anxiety and fear. *European Child & Adolescent Psychiatry, 11*, 273-280.

Pedersen, C. A., & Prange, A. J. (1979). Induction of maternal behavior in virgin rats after intracerebroventricular administration of oxytocin. *Proceedings of the National Academy of Sciences, 76*(12), 6661-6665. Retrieved from http://www.pnas.org/content/76/12/6661.abstract

Perez, E. M., Hendricks, M. K., Beard, J. L., Murray-Kolb, L. E., Berg, A., Tomlinson, M., . . . Vernon-Feagans, L. (2005). Mother-infant interactions and infant development are altered by maternal iron deficiency anemia. *Journal of Nutrition, 135*(4), 850-855.

Petzoldt, J., Wittchen, H.-U., Wittich, J., Einsle, F., Höfler, M., & Martini, J. (2014). Maternal anxiety disorders predict excessive infant crying: a prospective longitudinal study. *Archives of Disease in Childhood*. doi:10.1136/archdischild-2013-305562

Pfeifer, M., Goldsmith, H. H., Davidson, R. J., & Rickman, M. (2002). Continuity and change in inhibited and uninhibited children. *Child Development, 73*(5), 1474-1485.

Plamondon, A., Akbari, E., Atkinson, L., Steiner, M., Meaney, M. J., & Fleming, A. S. (2015). Spatial working memory and attention skills are predicted by maternal stress during pregnancy. *Early Human Development, 91*(1), 23-29. doi:http://dx.doi.org/10.1016/j.earlhumdev.2014.11.004

Pollitt, J. (1957). Natural history of obsessional states; a study of 150 cases. *British Medical*

Journal, 1(5012), 194-198.

Preacher, K. J., Curran, P. J., & Bauer, D. J. (2006). Computational tools for probing interactions in multiple linear regression, multilevel modeling, and latent curve analysis. *Journal of Educational and Behavioral Statistics, 31*(4), 437-448.

Putnam, S. P., Helbig, A. L., Gartstein, M. A., Rothbart, M. K., & Leerkes, E. (2014). Development and assessment of short and very short forms of the infant behavior questionnaire-revised. *Journal of Personality Assessment, 96*(4), 445-458. doi: 10.1080/00223891.2013.841171

Rachman, S., & de Silva, P. (1978). Abnormal and normal obsessions. *Behaviour Research and Therapy, 16*(4), 233-248.

Ramakrishnan, U., Grant, F., Goldenberg, T., Zongrone, A., & Martorell, R. (2012). Effect of women's nutrition before and during early pregnancy on maternal and infant outcomes: A systematic review. *Paediatric and Perinatal Epidemiology, 26*, 285-301. doi: 10.1111/j.1365-3016.2012.01281.x

Rautava, P., Helenius, H., & Lehtonen, L. (1993). Psychosocial predisposing factors for infantile colic. *The British Medical Journal, 307*, 600-604.

Real, E., Labad, J., Alonso, P., Segalàs, C., Jiménez-Murcia, S., Bueno, B., . . . Menchón, J. M. (2011). Stressful life events at onset of obsessive–compulsive disorder are associated with a distinct clinical pattern. *Depression and Anxiety, 28*(5), 367-376. doi:10.1002/da.20792

Rice, D., & Barone, S., Jr. (2000). Critical periods of vulnerability for the developing nervous system: evidence from humans and animal models. *Environmental Health Perspectives, 108*(Suppl 3), 511-533.

Rodriguez, A., & Bohlin, G. (2005). Are maternal smoking and stress during preganncy related to ADHD symptoms in children? *Journal of Child Psychology and Psychiatry, 46*(3), 246-254.

Ronald, A., Pennell, C. E., & Whitehouse, A. J. O. (2011). Prenatal maternal stress associated with ADHD and autistic traits in early childhood. *Frontiers in Psychology, 1*(223), 1-8.

Ross, L. E., & McLean, L. M. (2006). Anxiety disorders during pregnancy and the postpartum period: A systematic review. *Journal of Clinical Psychiatry, 67*(8), 1285-1298.

Rothbart, M. K. (2007). Temperament, development, and personality. *Current Directions in Psychological Science, 16*(4), 207-212. doi:10.1111/j.1467-8721.2007.00505.x

Rubinow, D. R., Schmidt, P. J., & Roca, C. A. (1998). Estrogen–serotonin interactions: implications for affective regulation. *Biological Psychiatry, 44*(9), 839-850. doi:http://dx.doi.org/10.1016/S0006-3223(98)00162-0

Ruiz, R. J., & Avant, K. C. (2005). Effects of maternal prenatal stress on infant outcomes: A synthesis of the literature. *Advances in Nursing Science, 28*(4), 345-355. Retrieved from http://journals.lww.com/advancesinnursingscience/Fulltext/2005/10000/Effects_of_Maternal_Prenatal_Stress_on_Infant.6.aspx

Ruiz, R. J., Fullerton, J., Brown, C. E., & Dudley, D. J. (2002). Predicting risk of preterm birth: the roles of stress, clinical risk factors, and corticotropin-releasing hormone. *Biological Research for Nursing, 4*(1), 54-64.

Salkovskis, P. M., & Harrison, J. (1984). Abnormal and normal obsessions—A replication. *Behaviour Research and Therapy, 22*(5), 549-552. doi:http://dx.doi.org/10.1016/0005-7967(84)90057-3

Salisbury, A. L., Lester, B. M., Seifer, R., Lagasse, L., Bauer, C. R., Shankaran, S., . . . Poole, K.

(2007). Prenatal cocaine use and maternal depression: Effects on infant neurobehavior. *Neurotoxicology & Teratology, 29*(3), 331-340. doi: 10.1016/j.ntt.2006.12.001

Sandman, C. A., Wadhwa, P. D., Chicz-Demet, A., Dunkel-Schetter, C., & Porto, M. (1997). Maternal stress, HPA activity, and fetal/infant outcome. *Annals of the New York Academy of Sciences, 814,* 266-275.

Sandman, C. A., Wadhwa, P. D., Dunkel-Schetter, C., Chicz-DeMet, A., Belman, J., Porto, M., . . . Crinella, F. M. (1994). Psychobiological influences of stress and HPA regulation on the human fetus and infant birth outcomes. *Annals of the New York Academy of Sciences, 739*(1), 198-210. doi:10.1111/j.1749-6632.1994.tb19822.x

Sasaki, A., Shinkawa, O., & Yoshinaga, K. (1990). Immunoreactive corticotropin-releasing hormone in amniotic fluid. *American Journal of Obstetrics and Gynecology, 162*(1), 194-198.

Schwartz, C. E., Snidman, N., & Kagan, J. (1999). Adolescent social anxiety as an outcome of inhibited temperament in childhood. *Journal of the American Academy of Child & Adolescent Psychiatry, 38*(8), 1008-1015. doi:http://dx.doi.org/10.1097/00004583-199908000-00017

Seckl, J. R. & Meaney, M. J. (2004). Glucocorticoid programming. *Annals of the New York Academy of Sciences, 1032,* 63-84.

Segerstrom, S. C. & Miller, G. E. (2004). Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin, 130*(4), 601-630.

Sen, S., Carpenter, A. H., Hochstadt, J., Huddleston, J. Y., Kustanovich, V., Reynolds, A. A., & Roberts, S. (2012). Nutrition, weight gain and eating behavior in pregnancy: A review of experimental evidence for long-term effects on the risk of obesity in offspring.

Physiology & Behavior, 107(1), 138-145. doi:http://dx.doi.org/10.1016/j.physbeh.2012.04.014

Sharps, P. W., Laughon, K., & Giangrande, S. K. (2007). Intimate partner violence and the childbearing year: Maternal and infant health consequences. *Trauma, Violence, & Abuse, 8*(2), 105-116. doi: 10.1177/1524838007302594.

Sheldon, C., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24*(4), 385-396. doi: 10.2307/2136404

Sichel, D. A., Cohen, L. S., Dimmock, J. A., & Rosenbaum, J. F. (1993). Postpartum obsessive compulsive disorder: A case series. *Journal of Clinical Psychiatry, 54*(4), 156-159.

Sjöström, K., Valentin, L., Thelin, T., & Maršál, K. (1997). Maternal anxiety in late pregnancy and fetal hemodynamics. *European Journal of Obstetrics & Gynecology and Reproductive Biology, 74*(2), 149-155. doi:http://dx.doi.org/10.1016/S0301-2115(97)00100-0

Smith, B. L., Wills, G., & Naylor, D. (1981). The effects of prenatal stress on rat offsprings' learning ability. *The Journal of Psychology, 107*(1), 45-51. doi:10.1080/00223980.1981.9915203

Smith, L. M., LaGasse, L. L., Derauf, C., Grant, P., Shah, R., Arria, A., . . . Lester, B. M. (2008). Prenatal methamphetamine use and neonatal neurobehavioral outcome. *Neurotoxicology and Teratology, 30*(1), 20-28. doi:10.1016/j.ntt.2007.09.005

Sondergaard, C., Olsen, J., Friis-Hasche, E., Dirdal, M., Thrane, N., & Sorensen, H. T. (2003). Psychosocial distress during pregnancy and the risk of intantile colic: a follow-up study. *Acta Paediatrica, 92*(7), 811-816.

Spinella, M. (2005). Mood in relation to sublinical obsessive-compulsive symptoms. *International Journal of Neuroscience, 115*(4), 433-443.

doi:10.1080/00207450590522838

Spinrad, T. L., & Stifter, C. A. (2002). Maternal sensitivity and infant emotional reactivity. *Marriage & Family Review, 34*(3-4), 243-263. doi:10.1300/J002v34n03_03

Stanley, C., Murray, L., & Stein, A. (2004). The effect of postnatal depression on mother–infant interaction, infant response to the Still-face perturbation, and performance on an Instrumental Learning task. *Development and Psychopathology, 16*(01), 1-18. doi:doi:10.1017/S0954579404044384

Stephens, B. E., Liu, J., Lester, B., Lagasse, L., Shankaran, S., Bada, H., . . . Higgins, R. (2010). Neurobehavioral assessment predicts motor outcome in preterm infants. *The Journal of Pediatrics, 156*(3), 366-371. doi:http://dx.doi.org/10.1016/j.jpeds.2009.09.042

Stevenson, M. B., Leavitt, L. A., Roach, M. A., Chapman, R. S., & Miller, J. F. (1986). Mothers' speech to their 1-year-old infants in home and laboratory settings. *Journal of Psycholinguistic Research, 15*(5), 451-461.

Stewart, R. C. (2007). Maternal depression and infant growth: A review of recent evidence. *Maternal & Child Nutrition, 3*(2), 94-107. doi: 10.1111/j.1740-8709.2007.00088.x

Stuart, S., Couser, G., Schilder, K., O'Hara, M. W., & Gorman, L. (1998). Postpartum anxiety and depression: Onset and comorbidity in a community sample. *The Journal of Nervous and Mental Disease, 186*(7). Retrieved from http://journals.lww.com/jonmd/Fulltext/1998/07000/Postpartum_Anxiety_and_Depression__Onset_and.6.aspx

Stunkard, A. J., Faith, M. S., & Allison, K. C. (2003). Depression and obesity. *Biological Psychiatry, 54*(3), 330-337. doi: http://dx.doi.org/10.1016/S0006-3223(03)00608-5

Sutter-Dallay, A. L., Giaconne-Marcesche, V., Glatigny-Dallay, E., & Verdoux, H. (2004).

Women with anxiety disorders during pregnancy are at increased risk of intense postnatal depressive symptoms: a prospective survey of the MATQUID cohort. *European Psychiatry, 19*, 459-463.

Swain, J. E., Lorberbaum, J. P., Kose, S., & Strathearn, L. (2007). Brain basis of early parent–infant interactions: psychology, physiology, and in vivo functional neuroimaging studies. *Journal of Child Psychology and Psychiatry, 48*(3-4), 262-287. doi:10.1111/j.1469-7610.2007.01731.x

Szechtman, H., Sulis, W., & Eilam, D. (1998). Quinpirole induces compulsive checking behavior in rats: A potential animal model of obsessive-compulsive disorder (OCD). *Behavioral Neuroscience, 112*(6), 1475-1485.

Tarabulsy, G. M., Pearson, J., Vaillancourt-Morel, M. P., Bussieres, E. L., Madigan, S., Lemelin, J. P., . . . Royer, F. (2014). Meta-analytic findings of the relation between maternal prenatal stress and anxiety and child cognitive otucome. *Journal of Developmental & Behavioral Pediatrics, 35*, 38-43.

Teixeira, J. M. A., Fisk, N. M., & Glover, V. (1999). Association between maternal anxiety in pregnancy and increased uterine artery resistance index: cohort based study. *British Medical Journal, 318*(7177), 153-157. doi:10.1136/bmj.318.7177.153

Thompson, J. F., Roberts, C. L., Currie, M., & Ellwood, D. A. (2002). Prevalence and persistence of health problems after childbirth: Associations with parity and method of birth. *Birth, 29*(2), 83-94. doi: 10.1046/j.1523-536X.2002.00167.x

Toro, J., Cervera, M., Osejo, E., & Salamero, M. (1992). Obsessive-compulsive disorder in childhood and adolescence: A clinical study. *Journal of Child Psychology and Psychiatry, 33*(6), 1025-1037. doi:10.1111/j.1469-7610.1992.tb00923.x

Tronick, E., & Lester, B. M. (2013). Grandchild of the NBAS: The NICU Network Neurobehavioral Scale (NNNS). *Journal of Child and Adolescent Psychiatric Nursing, 26*(3), 193-203. doi:10.1111/jcap.12042

Uguz, F., Akman, C., Kaya, N., & Cilli, A. S. (2007). Postpartum-onset obsessive-compulsive disorder: incidence, clinical features, and related factors. *The Journal of Clinical Psychiatry, 68*(1), 132-138. Retrieved from http://europepmc.org/abstract/MED/17284141

Uylings, H. B. M. (2006). Development of the human cortex and the concept of "critical" or "sensitive" periods. *Language Learning, 56*, 59-90. doi:10.1111/j.1467-9922.2006.00355.x

van den Bergh, B. R. H., & Marcoen, A. (2004). High antenatal maternal anxie3ty is related to ADHD symptoms, externalizing problems, and anxiety in 8- and 9-year-olds. *Child Development, 75*(4), 1085-1097.

van den Bergh, B. R. H., Mennes, M., Stevens, V., van der Meere, J., Borger, N., Stiers, P., . . . Lagae, L. (2006). ADHD deficit as measured in adolescent boys with a continuous performance task is related to antenatal maternal anxiety. *Pediatric Research, 59*(1), 78-82. Retrieved from http://dx.doi.org/10.1203/01.pdr.0000191143.75673.52

Vasudevan, C., Renfrew, M., & McGuire, W. (2011). Fetal and perinatal consequences of maternal obesity. *Archives of Disease in Childhood - Fetal and Neonatal Edition, 96*(5), F378-F382. doi: 10.1136/adc.2009.170928

Vreeburg, S. A., Zitman, F. G., van Pelt, J., DeRijk, R. H., Verhagen, J. C. M., van Dyck, R., . . . Penninx, B. W. J. H. (2010). Salivary cortisol levels in persons with and without different anxiety disorders. *Psychosomatic Medicine, 72*(4), 340-347.

doi:10.1097/PSY.0b013e3181d2f0c8

Vulink, N. C. C., Denys, D., Bus, L., & Westenberg, H. G. M. (2006). Female hormones affect symptom severity in obsessive–compulsive disorder. *International Clinical Psychopharmacology, 21*(3), 171-175. doi:10.1097/01.yic.0000199454.62423.99

Wachs, T. D. (2009). Models linking nutritional deficiencies to maternal and child mental health. *The American Journal of Clinical Nutrition, 89*(3), 935S-939S.

Wadhwa, P. D., Dunkel-Schetter, C., Chicz-DeMet, A., Porto, M., & Sandman, C. A. (1996). Prenatal psychosocial factors and the neuroendocrine axis in human pregnancy. *Psychosomatic Medicine, 58*(5), 432-446. Retrieved from http://journals.lww.com/psychosomaticmedicine/Fulltext/1996/09000/Prenatal_Psychosocial_Factors_and_the.6.aspx

Wadhwa, P. D., Glynn, L. M., Hobel, C. J., Garite, T. J., Porto, M., Chicz-DeMet, A., . . . Sandman, C. A. (2002). Behavioral perinatology: Biobehavioral processes in human fetal development. *Regulatory Peptides, 108*(2), 149-157.

Weinberg, M. K., & Tronick, E. Z. (1998). The impact of maternal psychiatric illness on infant development. *Journal of Clinical Psychiatry,* 59(Suppl 2), 53-61.

Weiss, J. M., Demetrikopoulos, M. K., West, C. H. K., & Bonsall, R. W. (1995). Hypothesis linking the noradrenergic and dopaminergic systems in depression. *Depression, 3*(5), 225-245. doi:10.1002/depr.3050030503

Wenzel, A., Gorman, L. L., O'Hara, M. W., & Stuart, S. (2001). The occurrence of panic and obsessive compulsive symptoms in women with postpartum dysphoria: a prospective study. *Archives of Women's Mental Health, 4*(1), 5-12. doi:10.1007/s007370170002

Wenzel, A., Haugen, E. N., Jackson, L. C., & Brendle, J. R. (2005). Anxiety symptoms and

disorders at eight weeks postpartum. *Journal of Anxiety Disorders, 19*(3), 295-311. doi:10.1016/j.janxdis.2004.04.001

Williams, K. E., & Koran, L. M. (1997). Obsessive-compulsive disorder in pregnancy, the puerperium, and the premenstruum. *Journal of Clinical Psychiatry, 58*(7), 330-334; quiz 335-336.

Williams, M. T., Hennessy, M. B., & Davis, H. N. (1995). CRF administered to pregnant rats alters offspring behavior and morphology. *Pharmacology Biochemistry and Behavior, 52*(1), 161-167. doi:http://dx.doi.org/10.1016/0091-3057(95)00082-8

Wisner, K. L., Peindl, K. S., Gigliotti, T., & Hanusa, B. H. (1999). Obsessions and compulsions in women with postpartum depression. *Journal of Clinical Psychiatry, 60*(3), 176-180.

Youngstrom, E., Izard, C., & Ackerman, B. (1999). Dysphoria-related bias in maternal ratings of children. *Journal of Consulting and Clinical Psychology, 67*(6), 905-916.

Zambaldi, C. F., Cantilino, A., Montenegro, A. C., Paes, J. A., de Albuquerque, T. L. C., & Sougey, E. B. (2009). Postpartum obsessive-compulsive disorder: prevalence and clinical characteristics. *Comprehensive Psychiatry, 50*(6), 503-509. doi:http://dx.doi.org/10.1016/j.comppsych.2008.11.014

Zohar, J., Kennedy, J. L., Hollander, E., & Koran, L. M. (2004). Serotonin-1D hypothesis of obsessive-compulsive disorder: an update. *Journal of Clinical Psychiatry, 65 Suppl 14*, 18-21.

Zuckerman, B., Bauchner, H., Parker, S., & Cabral, H. (1990). Maternal depressive symptoms during pregnancy, and newborn irritability. *Jornal of Developmental & Behavioral Pediatricts, 11*(4), 190-194.

Table 1

List of Exclusion Criteria for the IDAHO MOM Study

Mothers who induced early/delivered preterm	Mothers pregnant with multiple births
Mothers past the age and/or gestation criteria	Mothers who miscarried/were not pregnant
Mothers with health conditions that could potentially impact endocrine functioning	Mothers deemed a high-risk pregnancy
Surrogate mothers	Mothers with severe behavioral or physical health diagnoses/symptoms
Mothers who chronically consumed recreational substances	Mothers taking medications from FDA categories D and X
Mothers who consumed excessive amounts of alcohol during pregnancy	Mothers younger than 18 and older than 35

Table 2

Primary Study Variables Intercorrelation Table

Variable	1	2	3	4	5	6
1. Maternal Postnatal Depression	-					
2. Maternal Prenatal OC	.178	-				
3. Maternal Postnatal OC	.300**	.365**	-			
4. SES	-.221*	.134	-0.76	-		
5. IBQ-R	.219*	.009	.178	-.138	-	
6. Behavioral Observation Coding	.010	.043	.108	-0.81	.082	-

**** Correlation is significant at the *p*=.01 level (2-tailed)**

*** Correlation is significant at the *p*=.05 level (2-tailed)**

Table 3

Sample Sociodemographic Descriptives (n=125)

Race/Ethnicity	***N***	***%***
White	109	87
Black or African American	1	1
Asian	1	1
White and Native Hawaiian or Other Pacific Islander	2	2
White and American Indian/Alaska Native	3	2
White and Black or African American	1	1
White and Other	1	1
Other	7	6
Hispanic (Not exclusive from other groups)	16	13
Income		
<$5,000	2	2
$5,000-$9,999	3	2
$10,000-$19,999	19	15
$20,000-$29,999	24	19
$30,000-$39,999	15	12
$40,000-$49,999	12	10
$50,000-$74,999	31	25
$75,000-$99,999	9	7
More than or equal to $100,000	10	8
Infant Sex		
Male	48	51
Female	47	50
Maternal Education Level		
Junior high school	1	1
Partial high school	4	3
High school degree (including GED)	18	14
Partial college or other specialized/technical training	45	36
Standard college or university degree	46	37
Graduate training with degree	11	9
Marital Status		
Single/Never Married	10	8
Married	99	79
Divorced	3	2
Committed Relationship	9	7
Engaged	4	3

Table 4

Maternal Age and Primary Study Variable Descriptives

Measure	*Mean*	*SD*
Maternal Prenatal Age (Years)	26.82	4.39
Maternal Postnatal Age (Years)	28.32	3.96
Infant Prenatal Age (Weeks Gestation)	34.41	1.35
Infant Postnatal Age (Months)	6.08	0.30
POCS-Pre Square-Root Transformation	1.31	1.17
POCS-Post Square-Root Transformation	1.42	1.25
EPDS Square-Root Transformation	1.96	0.98
Behavioral Observation Log+1 Transformation	1.33	0.76
Hollingshead	3.45	0.97
IBQ-R	2.96	0.62

Table 5

Comparative Studies of IBQ-R-SF Sample Descriptives

	IBQ-R-SF Sadness	IBQ-R-SF Distress	IBQ-R-SF Fear	IBQ-R-SF Falling	IBQ-R-SF NEG	Comparison to IDAHO MOM data
IDAHO MOM Mean (SD)	3.34 (.88)	3.55 (.88)	2.73 (1.08)	5.27 (.94)	3.09 (.64)	
Braungart-Rieker Mean (SD)[a]	3.55 (.96)	3.96 (.98)	2.51 (.89)	5.30 (.96)	3.68 (.79)	t=7.31, p<.0001
Calkins Mean (SD)[b]	3.63 (1.08)	3.89 (1.06)	2.42 (.99)	5.08 (1.00)	3.62 (.96)	t=7.12, p<.0001
Davis Mean (SD)[c]	3.43 (1.01)	3.66 (1.02)	2.16 (.96)	5.17 (.97)	3.31 (.82)	t=3.73, p<.05
Gartstein Mean (SD)[d]	3.71 (1.16)	3.87 (1.14)	2.73 (1.11)	5.29 (1.07)	3.88 (.94)	t=7.53, p<.0001
Ross-Sheehy Mean (SD)[e]	3.50 (.97)	3.57 (.99)	2.01 (.67)	5.28 (.91)	3.30 (.84)	t=2.82, p=.055
Porges Mean (SD)[f]	3.36 (.94)	3.83 (1.12)	2.83 (1.23)	5.24 (1.05)	3.72 (.97)	t=6.63, p<.0001

**Note.* [a]n=131, [b]n=191, [c]n=223, [d]n=68, [e]n=54, [f]n=119.

IBQ-R-SF= Infant Behavior Questionnaire-Revised-Short Form.

Table 6

Comparative Studies of EPDS Sample Descriptives

	EPDS Mean	EPDS SD
IDAHO MOM Study	4.79	3.96
Austin[a]	4.70	4.30
t-test	*t*=0.19, *p*=.847	

**Note.* [a]*n*=700.

EPDS= Edinburgh Postnatal Depression Scale.

Table 7

Hypothesis 1 Hierarchical Regression Results

DV=Infant Reactivity (IBQ-R)	*b*	*SE*	*F*	*p*
Model 1			4.930	.029
Intercept	2.960	0.063	6.870	.000
Maternal Depression	0.139	0.064	1.471	.033
Model 2			1.944	.128
Intercept	2.960	0.063	6.849	.000
Maternal Depression	0.129	0.068	1.379	.060
Maternal Prenatal OC	-0.011	0.056	0.446	.843
SES	-0.060	0.069	.932	.387
Model 3			1.691	.159
Intercept	2.956	0.063	6.832	.000
Maternal Depression	0.133	0.068	1.402	.052
Maternal Prenatal OC	-0.007	0.057	0.358	.898
SES	-0.074	0.070	1.024	.297
OC x SES Interaction	0.059	0.062	0.975	.345

Table 8

Hypothesis 2 Hierarchical Regression Results

DV=Infant Reactivity (Behavioral Observation Coding)	*b*	*SE*	*F*	*p*
Model 1			0.009	.924
Intercept	1.335	.080	4.089	.000
Maternal Depression	.008	.080	0.310	.924
Model 2			0.269	.848
Intercept	1.335	.080	4.076	.000
Maternal Depression	-.015	.085	0.416	.863
Maternal Prenatal OC	.034	.072	0.686	.639
SES	-.070	.087	0.894	.426
Model 3			0.202	.937
Intercept	1.335	.081	4.063	.000
Maternal Depression	-.015	.086	0.423	.858
Maternal Prenatal OC	.033	.072	0.678	.647
SES	-.068	.091	0.863	.459
OC x SES Interaction	-.008	.080	0.324	.916

Table 9

Hypothesis 3 Hierarchical Regression Results

DV=Infant Reactivity (IBQ-R)	*b*	*SE*	*F*	*p*
Model 1			4.930	.029
Intercept	2.960	0.063	6.870	.000
Maternal Depression	0.139	0.064	1.471	.033
Model 2			2.354	.077
Intercept	2.960	0.063	6.873	.000
Maternal Depression	0.103	0.069	1.222	.139
Maternal Postnatal OC	0.061	0.053	1.075	.251
SES	-0.060	0.068	0.944	.375
Model 3			2.163	.079
Intercept	2.966	0.063	6.877	.000
Maternal Depression	0.113	0.069	1.279	.105
Maternal Postnatal OC	0.070	0.053	1.150	.190
SES	-0.058	0.068	0.926	.394
OC x SES Interaction	0.066	0.055	1.095	.234

Table 10

Hypothesis 4 Hierarchical Regression Results

DV=Infant Reactivity (Behavioral Observation Coding)	*b*	*SE*	*F*	*p*
Model 1			0.009	.924
Intercept	1.335	.080	4.090	.000
Maternal Depression	.008	.080	0.310	.924
Model 2			0.540	.656
Intercept	1.335	.080	4.086	.000
Maternal Depression	-.032	.087	0.609	.712
Maternal Postnatal OC	.068	.067	1.007	.313
SES	-.063	.087	0.853	.469
Model 3			0.403	.806
Intercept	1.334	.081	4.062	.000
Maternal Depression	-.033	.088	0.615	.706
Maternal Postnatal OC	.067	.068	0.991	.329
SES	-.063	.087	0.851	.471
OC x SES Interaction	-.007	.071	0.316	.920

Appendix A: Initial Human Subjects Committee Approval for IDAHO MOM Study

Idaho State
UNIVERSITY

Office for Research Integrity
921 South 8th Avenue, Stop 8046 • Pocatello, Idaho 83209-8046

February 13, 2015

Nicki Aubuchon-Endsley, PhD
Stop 8112
Psychology
Pocatello, ID 83209

RE: Your application dated 2/2/2015 regarding study number 4191: Infant Development and Healthy Outcomes in Mothers (Idaho Mom Study)

Dear Dr. Aubuchon-Endsley:

Thank you for your response to requests from a prior review of your application for the new study listed above. Your study is eligible for expedited review under FDA and DHHS (OHRP) designation.

This is to confirm that your application is now fully approved. The protocol is approved through 2/13/2016.

You are granted permission to conduct your study as most recently described effective immediately. The study is subject to continuing review on or before 2/13/2016, unless closed before that date.

Please note that any changes to the study as approved must be promptly reported and approved. Some changes may be approved by expedited review; others require full board review. Contact Tom Bailey (208-282-2179; fax 208-282-4723; email: humsubj@isu.edu) if you have any questions or require further information.

Sincerely,

Ralph Baergen, PhD, MPH, CIP
Human Subjects Chair

Phone: (208) 282-2179 • Fax: (208) 282-4723 • www.isu.edu/research

Appendix B: Prenatal Obsessive-Compulsive Scale (POCS)

Tel: (905) 522-1155 ext.33605
Fax: (905) 521-6098

Date: **Delivery Date:** **ID:**

Prenatal Obsessive-Compulsive scale (PreOCS)

Date: _____**(dd/mm/yy)** **Due Date:**_____**(dd/mm/yy)** **ID:**_____

This questionnaire investigates thoughts/images and behaviours, which may have occurred **during your most recent pregnancy**. The following thoughts and behaviours may seem disturbing to you but are fairly common in expectant mothers. Your answers will be kept confidential.

Please circle YES or NO on the following questions.

Section A.

Have you ever worried a lot, or had repeated thoughts or pictures in your head about:		
1. Being criticized and/or judged as a mother?	YES	NO
2. Your baby being contaminated (for example by germs)?	YES	NO
3. Your baby being unwell at birth?	YES	NO
4. Excessive worry about the birth, delivery, episiotomy, c-section or vacuum/forcep extraction	YES	NO
5. Accidentally harming your baby?	YES	NO

Have any of the following thoughts or pictures in your head repeatedly entered your mind, without implying that you would act on it?		
6. Stabbing your baby with a sharp object/knife?	YES	NO
7. Your baby being spiritually possessed (for example by a negative force)?	YES	NO
8. Other thoughts/images? Describe:	YES	NO

If you answered **NO** to all of the above please skip to Section B.

If you answered **YES** to any of the above questions:

How much time do you spend being bothered by these thoughts/images described above?

☐ Not at all	☐ Less than 1 hour/day	☐ 1-3h/day	☐ 3-8h/day	☐ More than 8h/day

How much do the thoughts/images above interfere with your ability to think of other things?

☐ Not at all	☐ Minimal interference	☐ Moderate interference	☐ A lot of interference	☐ Severe interference affecting your ability to function day to day

Are the thoughts/images above distressing?

☐ Not at all	☐ A little	☐ Moderately	☐ Severe	☐ Extreme, disabling

How difficult is it for you to dismiss the thoughts/images above?

☐ Not difficult at all	☐ Minimally difficult	☐ Moderately difficult	☐ Very difficult	☐ Unable to dismiss the thoughts

How much control do you have over the thoughts/images above?
(i.e. when you have these thoughts, are you able to change them to create more desirable thoughts?)

☐ Complete control	☐ Much control	☐ Some control	☐ Little control	☐ No control

When did these thoughts start? Check all that apply:

☐ While planning for a pregnancy	☐ During my first trimester (weeks)
☐ During a previous pregnancy	☐ During my second trimester (weeks)
☐ During the year postpartum of a previous pregnancy	☐ During my third trimester (weeks)
☐ When I found out I was pregnant with this child	☐ Specific memorable trigger (hearing, watching or reading about something, seeing somebody, etc) Please specify:

Did you have worries or unpleasant thoughts/images (in general, not necessarily about pregnancy or the baby) prior to this pregnancy? **YES NO (If NO, skip to section B)**

If so, when did they begin:_____

Would you say that your thoughts/images changed when you became pregnant? Please circle:

☐ They were better	☐ No change	☐ They were worse
☐ Less frequent	☐ No change	☐ More frequent

Section B.

Have you ever engaged in the following behaviours:		
9. Repeatedly washing or cleaning your hands?	YES	NO
10. Strong urge to count or add?	YES	NO
11. Repeatedly checking the door, locks, or oven, etc.?	YES	NO
12. Repeatedly lining up and/or putting things in order?	YES	NO
13. Repeatedly checking that you did not make a mistake?	YES	NO
14. Excessive researching (internet, doctors appointments, books) about pregnancy, childbirth and babies?	YES	NO
15. Repeatedly asking for reassurance?	YES	NO
16. Perform a combination of behaviours to prevent something bad from happening or to reduce your feelings of anxiety (routines, mental rituals, superstitious rituals, etc.)?	YES	NO
17. Other behaviours? Describe:	YES	NO

If you answered **NO** to all of the above please skip to Section C.

If you answered **YES** to any of the above questions:

How much time do you spend being bothered by these behaviours described above?

☐ Not at all	☐ Less than 1 hour/day	☐ 1-3h/day	☐ 3-8h/day	☐ More than 8h/day

How much do the behaviours above interfere with your activities?

☐ Not at all	☐ Minimal interference	☐ Moderate interference	☐ A lot of interference	☐ Severe interference affecting your ability to function day to day

Are the behaviours above distressing?

☐ Not at all	☐ A little	☐ Moderately	☐ Severe	☐ Extreme, disabling

How difficult is it for you to resist engaging in the behaviours above?

☐ Not difficult at all	☐ Minimally difficult	☐ Moderately difficult	☐ Very difficult	☐ Unable to resist

How much control do you have over the behaviours above?

☐ Complete control	☐ Much control	☐ Some control	☐ Little control	☐ No control

When did this behaviour start? Check all that apply:

☐ While planning for a pregnancy	☐ During my first trimester (weeks)
☐ During a previous pregnancy	☐ During my second trimester (weeks)
☐ During the year postpartum of a previous pregnancy	☐ During my third trimester (weeks)
☐ When I found out I was pregnant with this child	☐ Specific memorable trigger (hearing, watching or reading about something, seeing somebody, etc) Please specify:

Did you experience behaviour like this (in general, not necessarily about pregnancy or the baby) prior to this pregnancy? **YES NO (if NO, skip to section C)**

If so, when did they begin:_____

Would you say that your behaviours changed when you became pregnant? Please circle:

☐ Got better	☐ No change	☐ Got worse
☐ Less frequent	☐ No change	☐ More frequent

Section C.

Thinking of all the thoughts/images/behaviours you circled in the previous sections, please circle the appropriate answer:

How often have the thoughts or behaviour interfered with:	Never	Sometimes	Moderately	A lot	All the time	Not Applicable
Your relationships with your family	0	1	2	3	4	x
Your relationship with your significant other	0	1	2	3	4	x
Your relationships with your older child(ren)	0	1	2	3	4	x
Your social activities	0	1	2	3	4	x
Your home responsibilities/housework	0	1	2	3	4	x
Your job or work responsibilities	0	1	2	3	4	x

Thank you

Appendix C: Postnatal Obsessive-Compulsive Scale (POCS)

Postnatal Obsessive-Compulsive scale (POCS)

Date: _____ **Delivery Date:** _____ **ID:** _____

This questionnaire investigates thoughts and behaviors which may have occurred **following your most recent pregnancy**. The following thoughts and behaviours may seem disturbing to you but are fairly common in mothers of newborn babies. Please mark an X in the appropriate box.

Have you **ever worried** or had **thoughts** about:				
1. Being criticized and/or judged as a mother?	☐NO	☐YES	☐ Current	When did it start?
			☐ Past	When did it stop?
2. Shaking your baby?	☐NO	☐YES	☐ Current	When did it start?
			☐ Past	When did it stop?
3. Screaming at your baby?	☐NO	☐YES	☐ Current	When did it start?
			☐ Past	When did it stop?
4. Your baby being contaminated (for example by germs)?	☐NO	☐YES	☐ Current	When did it start?
			☐ Past	When did it stop?
5. Harming your baby during bath time?	☐NO	☐YES	☐ Current	When did it start?
			☐ Past	When did it stop?
6. Somebody taking your baby away?	☐NO	☐YES	☐ Current	When did it start?
			☐ Past	When did it stop?
7. Dropping your baby?	☐NO	☐YES	☐ Current	When did it start?
			☐ Past	When did it stop?

8. Your baby dying in her/his sleep?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
9. Your baby being harmed or dying in an accident?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
10. Harming your baby while he/she is asleep?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
11. Your baby acquiring a head injury?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
12. Your baby bleeding?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
13. Throwing your baby?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
14. Accidentally harming your baby with a sharp object/knife?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
15. Stabbing your baby with a sharp object/knife?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
16. Having inappropriate sexual contact with your baby?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?

17. Someone else having inappropriate sexual contact with your baby?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
18. Your baby being spiritually possessed (for example by negative force)?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
19. Other thoughts? Describe:	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?

If you answered **NO** to all of the above thoughts please skip to p.4, third table (behaviours).

If you answered **YES** to any of the above questions:

How much time do or did you spend being bothered by these thoughts describe above?

☐ Not at all	☐ Less than 1 hour/day	☐ 1-3h/day	☐ 3-8h/day	☐ More than 8h/day

How much do or did the thoughts above interfere with your ability to think of other things?

☐ Not at all	☐ Minimal interference	☐ Moderate interference	☐ A lot of interference	☐ Severe interference affecting your ability to function day to day

How difficult is or was it for you to dismiss the thoughts above?

☐ Not difficult at all	☐ Minimally difficult	☐ Moderately difficult	☐ Very difficult	☐ Unable to dismiss the thoughts

How much control do or did you have over the thoughts above?
(i.e. when you have these thoughts are you able to change the imaged scenario to create more desirable thoughts?)

☐ Complete control	☐ Much control	☐ Some control	☐ Little control	☐ No control

If you have ever had any of the above thoughts in the **PAST** and indicated that they have stopped, **was it because you received treatment (medication, therapy)?** Please specify.

☐ NO	☐ YES	Type of treatment:
		When:

Please circle the appropriate answer Are or were your thoughts:	Not at all	A little	Moderately	Very	Extremely
Irrational	0	1	2	3	4
Unwanted	0	1	2	3	4
Depressing/Upsetting	0	1	2	3	4
Consistent with your personality or usual behavior	0	1	2	3	4
Invading	0	1	2	3	4
Pleasant	0	1	2	3	4
Stressful	0	1	2	3	4
Shameful/Embarrassing	0	1	2	3	4
Distressing	0	1	2	3	4
Disturbing	0	1	2	3	4

Please circle the appropriate answer How often do or did these thoughts interfer(ed) with:	Never	Sometimes	Moderately	A lot	All the time	Not Applicable
Your relationships with your family	0	1	2	3	4	x
Your relationship with your significant other	0	1	2	3	4	x
Your relationships with your older child(ren)	0	1	2	3	4	x
Your relationship with your newborn baby	0	1	2	3	4	x
Your social activities	0	1	2	3	4	x
Your home responsibilities/housework	0	1	2	3	4	x
Your job or work responsibilities	0	1	2	3	4	x

Have you **ever** engaged in the following **behaviours**:				
20. Repeatedly washing or cleaning your hands?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
21. Strong urge to count or add?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
22. Making sure that you are not alone with your baby?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?

23. Repeatedly checking the door, locks, or oven, etc.?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
24. Repeatedly lining up and/or putting things in order?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
25. Repeatedly checking that you did not make a mistake (for example verifying that your baby drank enough)?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
26. Collecting useless items (not including hobbies)?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
27. Repeatedly asking for reassurance?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
28. Avoiding your baby?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
29. Repeatedly washing and cleaning your baby's environment?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
30. Repeatedly washing and cleaning your newborn?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
31. Repeatedly checking the baby while she/he is asleep?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?

32. Perform a combination of behaviours to prevent something bad from happening or to reduce your feelings of anxiety (routines, mental rituals, superstitious rituals, etc.)?	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?
33. Others behaviours? Describe:	☐NO ↓	☐YES →	☐ Current	When did it start?
			☐ Past	When did it stop?

If you answered **NO** to all of the above behaviours you have reached the end of this questionnaire.
Thank you.

If you answered **YES** to any of the above questions:

How much time do or did you spend being bothered by these behaviours describe above?

☐ Not at all	☐ Less than 1 hour/day	☐ 1-3h/day	☐ 3-8h/day	☐ More than 8h/day

How much do or did the behaviours above interfere with your activities?

☐ Not at all	☐ Minimal interference	☐ Moderate interference	☐ A lot of interference	☐ Severe interference affecting your ability to function day to day

How difficult is or was it for you to resist engaging in the behaviours above?

☐ Not difficult at all	☐ Minimally difficult	☐ Moderately difficult	☐ Very difficult	☐ Unable to dismiss the thoughts

How much control do or did you have over the behaviours above?
(I.e. when you have these thoughts are you able to change the imaged scenario to create more desirable thoughts?)

☐ Complete control	☐ Much control	☐ Some control	☐ Little control	☐ No control

If you have ever had any of the above behaviours in the **PAST** and indicated that they have stopped, **was it because you received treatment (medication, therapy)?** Please specify.

☐ NO	☐ YES	Type of treatment:
		When:

Please circle the appropriate answer Are or were your behaviors:	Not at all	A little	Moderately	Very	Extremely
Irrational	0	1	2	3	4
Unwanted	0	1	2	3	4
Depressing/Upsetting	0	1	2	3	4
Consistent with your personality or usual behavior	0	1	2	3	4
Invading	0	1	2	3	4
Pleasant	0	1	2	3	4
Stressful	0	1	2	3	4
Shameful/Embarrassing	0	1	2	3	4
Distressing	0	1	2	3	4
Disturbing	0	1	2	3	4

Please circle the appropriate answer How often do or did these behaviours interfer(ed) with:	Never	Sometimes	Moderately	A lot	All the time	Not Applicable
Your relationships with your family	0	1	2	3	4	x
Your relationship with your significant other	0	1	2	3	4	x
Your relationships with your older child(ren)	0	1	2	3	4	x
Your relationship with your newborn baby	0	1	2	3	4	x
Your social activities	0	1	2	3	4	x
Your home responsibilities/housework	0	1	2	3	4	x
Your job or work responsibilities	0	1	2	3	4	x

Thank you

If you have any questions or concerns with any of the questions asked in this questionnaire please feel free to speak with someone from the clinic or a member of the research team.

Appendix D: Edinburgh Postnatal Depression Scale (EPDS)

Edinburgh Postnatal Depression Scale[1] (EPDS)

Name: _____ Address: _____

Your Date of Birth: _____ _____

Baby's Date of Birth: _____ Phone: _____

As you are pregnant or have recently had a baby, we would like to know how you are feeling. Please check the answer that comes closest to how you have felt **IN THE PAST 7 DAYS**, not just how you feel today.
Here is an example, already completed.
I have felt happy:
☐ Yes, all the time
☒ Yes, most of the time This would mean: "I have felt happy most of the time" during the past week.
☐ No, not very often Please complete the other questions in the same way.
☐ No, not at all

In the past 7 days:

1. I have been able to laugh and see the funny side of things
   - ☐ As much as I always could
   - ☐ Not quite so much now
   - ☐ Definitely not so much now
   - ☐ Not at all

2. I have looked forward with enjoyment to things
   - ☐ As much as I ever did
   - ☐ Rather less than I used to
   - ☐ Definitely less than I used to
   - ☐ Hardly at all

*3. I have blamed myself unnecessarily when things went wrong
   - ☐ Yes, most of the time
   - ☐ Yes, some of the time
   - ☐ Not very often
   - ☐ No, never

4. I have been anxious or worried for no good reason
   - ☐ No, not at all
   - ☐ Hardly ever
   - ☐ Yes, sometimes
   - ☐ Yes, very often

*5 I have felt scared or panicky for no very good reason
   - ☐ Yes, quite a lot
   - ☐ Yes, sometimes
   - ☐ No, not much
   - ☐ No, not at all
   - ☐

*6. Things have been getting on top of me
   - ☐ Yes, most of the time I haven't been able to cope at all
   - ☐ Yes, sometimes I haven't been coping as well as usual
   - ☐ No, most of the time I have coped quite well
   - ☐ No, I have been coping as well as ever

*7 I have been so unhappy that I have had difficulty sleeping
   - ☐ Yes, most of the time
   - ☐ Yes, sometimes
   - ☐ Not very often
   - ☐ No, not at all

*8 I have felt sad or miserable
   - ☐ Yes, most of the time
   - ☐ Yes, quite often
   - ☐ Not very often
   - ☐ No, not at all

*9 I have been so unhappy that I have been crying
   - ☐ Yes, most of the time
   - ☐ Yes, quite often
   - ☐ Only occasionally
   - ☐ No, never

*10 The thought of harming myself has occurred to me
   - ☐ Yes, quite often
   - ☐ Sometimes
   - ☐ Hardly ever
   - ☐ Never

Administered/Reviewed by _____ Date _____

[1]Source: Cox, J.L., Holden, J.M., and Sagovsky, R. 1987. Detection of postnatal depression: Development of the 10-item Edinburgh Postnatal Depression Scale. *British Journal of Psychiatry* 150:782-786.

[2]Source: K. L. Wisner, B. L. Parry, C. M. Piontek, Postpartum Depression N Engl J Med vol 347, No 3, July 18, 2002, 194-199

Appendix E: Infant Behavior Questionnaire – Revised Short Form (IBQ-R SF)

Infant Behavior Questionnaire - Revised

Subject No. _____ Date of Baby's Birth _____ ____ _____
month. . day year

Today' s Date_____ Age of Child _____ _____
mos. . weeks

Sex of Child _____

INSTRUCTIONS:

Please read carefully before starting:

As you read each description of the baby' s behavior below, please indicate how often the baby did this during the LAST WEEK (the past seven days) by circling one of the numbers in the left column. . These numbers indicate how often you observed the behavior described during the last week.

(1) Never	(2) Very Rarely	(3) Less Than Half the Time	(4) About Half the Time	(5) More Than Half the Time	(6) Almost Always	(7) Always	(X) Does Not Apply

The "Does Not Apply" (X) column is used when you did not see the baby in the situation described during the last week. . For example, if the situation mentions the baby having to wait for food or liquids and there was no time during the last week when the baby had to wait, circle the (X) column. "Does Not Apply" is different from "Never" (1). . "Never" is used when you saw the baby in the situation but the baby never engaged in the behavior listed during the last week. . For example, if the baby did have to wait for food or liquids at least once but never cried loudly while waiting, circle the (1) column.

Please be sure to circle a number for <u>every</u> item.

(1) Never	(2) Very Rarely	(3) Less Than Half the Time	(4) About Half the Time	(5) More Than Half the Time	(6) Almost Always	(7) Always	(X) Does Not Apply

<u>One Week Time Span</u>

<u>How often did your baby:</u>

1 2 3 4 5 6 7 X . . . . (1) make talking sounds when s/he was ready

for more food?
1 2 3 4 5 6 7 X . . . . (2) seem angry (crying and fussing) when you left
her/him in the crib?
1 2 3 4 5 6 7 X . . . . (3) seem contented when left in the crib?
1 2 3 4 5 6 7 X . . . . (4) cry or fuss before going to sleep for naps?

(1) Never	(2) Very Rarely	(3) Less Than Half the Time	(4) About Half the Time	(5) More Than Half the Time	(6) Almost Always	(7) Always	(X) Does Not Apply

1 2 3 4 5 6 7 X . . . . (5) look at pictures in books and/or magazines for
5 minutes or longer at a time?
1 2 3 4 5 6 7 X . . . . (6) stare at a mobile, crib bumper or picture for
5 minutes or longer?
1 2 3 4 5 6 7 X . . . . (7) play with one toy or object for 5-10 minutes?
1 2 3 4 5 6 7 X . . . . (8) play with one toy or object for 10 minutes or longer?
1 2 3 4 5 6 7 X . . . . (9) laugh aloud in play?
1 2 3 4 5 6 7 X . . . . (10) repeat the same movement with an object for 2
minutes or longer (e.g., putting a block in a cup, kicking or hitting a mobile)?
1 2 3 4 5 6 7 X . . . . (11) smile or laugh after accomplishing something (e.g., stacking blocks, etc.)?
1 2 3 4 5 6 7 X . . . . (12) smile or laugh when given a toy?
1 2 3 4 5 6 7 X . . . . (13) enjoy being read to?
1 2 3 4 5 6 7 X . . . . (14) enjoy hearing the sound of words, as in nursery rhymes?
1 2 3 4 5 6 7 X . . . . (15) enjoy gentle rhythmic activities, such as rocking or swaying?
1 2 3 4 5 6 7 X . . . . (16) enjoy being tickled by you or someone else in your family?
1 2 3 4 5 6 7 X . . . . (17) enjoy the feel of soft blankets?
1 2 3 4 5 6 7 X . . . . (18) enjoy being rolled up in a warm blanket?
1 2 3 4 5 6 7 X . . . . (19) enjoy listening to a musical toy in a crib?
1 2 3 4 5 6 7 X . . . . (20) look up from playing when the telephone rang?
1 2 3 4 5 6 7 X . . . . (21) protest being placed in a confining place (infant seat, play pen, car seat, etc)?

1 2 3 4 5 6 7 X . . . . (22) startle at a sudden change in body position (for example, when moved suddenly)?
1 2 3 4 5 6 7 X . . . . (23) move quickly toward new objects?
1 2 3 4 5 6 7 X . . . . (24) show a strong desire for something s/he wanted?
1 2 3 4 5 6 7 X . . . . (25) watch adults performing household activities (e.g., cooking, etc.) for more than 5 minutes?
1 2 3 4 5 6 7 X . . . . (26) squeal or shout when excited?
1 2 3 4 5 6 7 X . . . . (27) notice low-pitched noises (e.g. air conditioner, heating system, or refrigerator running or starting up)?
1 2 3 4 5 6 7 X . . . . (28) notice a change in light when a cloud passed over the sun?
1 2 3 4 5 6 7 X . . . . (29) notice the sound of an airplane passing overhead?
1 2 3 4 5 6 7 X . . . . (30) notice a bird or a squirrel up in a tree?
1 2 3 4 5 6 7 X . . . . (31) notice fabrics with scratchy texture (e.g., wool)?
1 2 3 4 5 6 7 X . . . . (32) appear sad for no apparent reason?

(1) Never	(2) Very Rarely	(3) Less Than Half the Time	(4) About Half the Time	(5) More Than Half the Time	(6) Almost Always	(7) Always	(X) Does Not Apply

<u>During feeding, how often did the baby:</u>
1 2 3 4 5 6 7 X . . . . (33) lie or sit quietly?
1 2 3 4 5 6 7 X . . . . (34) squirm or kick?
1 2 3 4 5 6 7 X . . . . (35) wave his/her arms?

<u>When going to sleep at night, how often did your baby:</u>
1 2 3 4 5 6 7 X . . . . (36) fall asleep within 10 minutes?
1 2 3 4 5 6 7 X . . . . (37) have a hard time settling down to sleep?
1 2 3 4 5 6 7 X . . . . (38) settle down to sleep easily?

<u>When being dressed or undressed during the last week, how often did the baby:</u>
1 2 3 4 5 6 7 X . . . . (39) squirm and/or try to roll away?
1 2 3 4 5 6 7 X . . . . (40) smile or laugh?
1 2 3 4 5 6 7 X . . . . (41) coo or vocalize?

<u>When put into the bath water, how often did the baby:</u>
1 2 3 4 5 6 7 X . . . . (42) smile?
1 2 3 4 5 6 7 X . . . . (43) laugh?

When tossed around playfully how often did the baby:
1 2 3 4 5 6 7 X . . . . (44) smile?
1 2 3 4 5 6 7 X . . . . (45) laugh?

During a peekaboo game, how often did the baby:
1 2 3 4 5 6 7 X . . . . (46) smile?
1 2 3 4 5 6 7 X . . . . (47) laugh?

How often did your baby enjoy bouncing up and down:
1 2 3 4 5 6 7 X . . . . (48) while on your lap?
1 2 3 4 5 6 7 X . . . . (49) on an object, such as a bed, bouncer chair, or toy?

When being held, how often did the baby:
1 2 3 4 5 6 7 X . . . . (50) pull away or kick?
1 2 3 4 5 6 7 X . . . . (51) seem to enjoy him/herself?

When the baby wanted something, how often did s/he:
1 2 3 4 5 6 7 X . . . . (52) become upset when s/he could not get what s/he wanted?
1 2 3 4 5 6 7 X . . . . (53) have tantrums (crying, screaming, face red, etc.) when s/he did not get what s/he wanted?

(1) Never	(2) Very Rarely	(3) Less Than Half the Time	(4) About Half the Time	(5) More Than Half the Time	(6) Almost Always	(7) Always	(X) Does Not Apply

When placed in an infant seat or car seat, how often did the baby:
1 2 3 4 5 6 7 X . . . . (54) wave arms and kick?
1 2 3 4 5 6 7 X . . . . (55) squirm and turn body?

How often did your baby make talking sounds when:
1 2 3 4 5 6 7 X . . . . (56) riding in a car?
1 2 3 4 5 6 7 X . . . . (57) riding in a shopping cart?
1 2 3 4 5 6 7 X . . . . (58) you talked to her/him?

When rocked or hugged, in the last week, how often did your baby:
1 2 3 4 5 6 7 X . . . . (59) seem to enjoy her/himself?
1 2 3 4 5 6 7 X . . . . (60) seem eager to get away?
1 2 3 4 5 6 7 X . . . . (61) While being fed in your lap, how often did the baby seem eager to get away as soon as the feeding was over?
1 2 3 4 5 6 7 X . . . . (62) After sleeping, how often did the baby cry if someone didn't come within a few

minutes?

1 2 3 4 5 6 7 X . . . . (63) When put down for a nap, how often did your baby settle down quickly?

1 2 3 4 5 6 7 X . . . . (64) When it was time for bed or a nap and your baby did not want to go, how often did s/he whimper or sob?

1 2 3 4 5 6 7 X . . . . (65) When face was washed, how often did the baby smile or laugh?

1 2 3 4 5 6 7 X . . . . (66) When hair was washed, how often did the baby vocalize?

1 2 3 4 5 6 7 X . . . . (67) When playing quietly with one of her/his favorite toys, how often did your baby enjoy lying in the crib for more than 5 minutes?

1 2 3 4 5 6 7 X . . . . (68) When your baby saw a toy s/he wanted, how often did s/he get very excited about getting it?

1 2 3 4 5 6 7 X . . . . (69) When given a new toy, how often did your baby immediately go after it?

1 2 3 4 5 6 7 X . . . . (70) When placed on his/her back, how often did the baby squirm and/or turn body?

1 2 3 4 5 6 7 X . . . . (71) When frustrated with something, how often did your baby calm down within 5 minutes?

1 2 3 4 5 6 7 X . . . . (72) When your baby was upset about something, how often did s/he stay upset for up to 20 minutes or longer?

1 2 3 4 5 6 7 X . . . . (73) When being carried, how often did your baby push against you until put down?

1 2 3 4 5 6 7 X . . . . (74) When tired, how often did your baby show distress?

(1) Never	(2) Very Rarely	(3) Less Than Half the Time	(4) About Half the Time	(5) More Than Half the Time	(6) Almost Always	(7) Always	(X) Does Not Apply

1 2 3 4 5 6 7 X . . . . (75) At the end of an exciting day, how often did your baby become tearful?

Two Week Time Span

When introduced to an unfamiliar adult, how often did the baby:

1 2 3 4 5 6 7 X . . . . (76) cling to a parent?

1 2 3 4 5 6 7 X . . . . (77) refuse to go to the unfamiliar person?

1 2 3 4 5 6 7 X . . . . (78) never “warm up” to the unfamiliar adult?

When you were busy with another activity and your baby was not able to get

your attention, how often did s/he:

1 2 3 4 5 6 7 X . . . . (79) become sad?
1 2 3 4 5 6 7 X . . . . (80) cry?

When singing or talking to your baby, how often did s/he:

1 2 3 4 5 6 7 X . . . . (81) soothe immediately?
1 2 3 4 5 6 7 X . . . . (82) take more than 10 minutes to soothe?

When showing the baby something to look at, how often did s/he:

1 2 3 4 5 6 7 X . . . . (83) soothe immediately?
1 2 3 4 5 6 7 X . . . . (84) take more than 10 minutes to soothe?

When patting or gently rubbing some part of the baby's body, how often did s/he:

1 2 3 4 5 6 7 X . . . . (85) soothe immediately?
1 2 3 4 5 6 7 X . . . . (86) take more than 10 minutes to soothe?
1 2 3 4 5 6 7 X . . . . (87) When in the presence of several unfamiliar adults, how often did the baby continue to be upset for 10 minutes or longer?
1 2 3 4 5 6 7 X . . . . (88) When visiting a new place, how often did the baby get excited about exploring new surroundings?
1 2 3 4 5 6 7 X . . . . (89) When an unfamiliar adult came to your home or apartment, how often did your baby cry when the visitor attempted to pick her/him up?
1 2 3 4 5 6 7 X . . . . (90) When familiar relatives/friends came to visit, how often did your baby get excited?
1 2 3 4 5 6 7 X . . . . (91) When rocking your baby, how often did s/he take more than 10 minutes to soothe?

Appendix F: Recruitment Materials

Are You Pregnant?

Sign up for the Idaho Mom Study!

What is it all about?

Conducted within: The Idaho State University Department of Psychology.

Purpose: To examine women's experiences during pregnancy and how they may relate to the growth and behavior of their babies.

Payment: You will receive up to **$75** and students may receive 1 credit for each half hour of participation toward relevant ISU courses.

IDAHO Mom

*I*nfant *D*evelopment *A*nd *H*ealthy *O*utcomes in Mothers

Contact Us!

Dr. Nicki Aubuchon-Endsley

Telephone: 208·380·1140

Email: idahomom@isu.edu

Idaho Mom Study 208-380-1140 idahomom@isu.edu

Idaho Mom Study 208-380-1140 idahomom@isu.edu
Idaho Mom Study 208-380-1140 idahomom@isu.edu
Idaho Mom Study 208-380-1140 idahomom@isu.edu
Idaho Mom Study 208-380-1140 idahomom@isu.edu
Idaho Mom Study 208-380-1140 idahomom@isu.edu
Idaho Mom Study 208-380-1140 idahomom@isu.edu
Idaho Mom Study 208-380-1140 idahomom@isu.edu
Idaho Mom Study 208-380-1140 idahomom@isu.edu
Idaho Mom Study 208-380-1140 idahomom@isu.edu
Idaho Mom Study 208-380-1140 idahomom@isu.edu
Idaho Mom Study 208-380-1140 idahomom@isu.edu

Are You Pregnant?

Sign up for the Idaho Mom Study!

What is it all about?

Purpose: To examine women's experiences during pregnancy and how they may relate to the growth and behavior of their babies.

Payment: You will receive up to $75 and students may receive 1 credit for each half hour of participation toward relevant ISU courses.

IDAHO Mom

Infant Development And Healthy Outcomes in Mothers

Contact Us!

Dr. Nicki Aubuchon-Endsley
Telephone: 208.282.2574
E-mail: idahomom@isu.edu

Idaho Mom Study	Phone: (208) 282-2574	E-mail: idahomom@isu.edu	Idaho Mom Study	Phone: (208) 282-2574	E-mail: idahomom@isu.edu	Idaho Mom Study	Phone: (208) 282-2574	E-mail: idahomom@isu.edu	Idaho Mom Study	Phone: (208) 282-2574	E-mail: idahomom@isu.edu	Idaho Mom Study	Phone: (208) 282-2574	E-mail: idahomom@isu.edu	Idaho Mom Study	Phone: (208) 282-2574	E-mail: idahomom@isu.edu	Idaho Mom Study	Phone: (208) 282-2574	E-mail: idahomom@isu.edu

Are You Pregnant?

Sign up for the Idaho Mom Study!

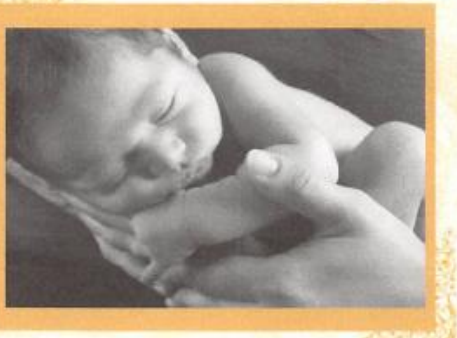
What is it all about?

Purpose: To examine women's experiences during pregnancy and how they may relate to the growth and behavior of their babies.

Payment: You will receive up to $75 and students may receive 1 credit for each half hour of participation toward relevant ISU courses.

IDAHO Mom

Infant Development And Healthy Outcomes in Mothers



Contact Us!

Dr. Nicki Aubuchon-Endsley
Telephone: 208.282.2574
E-mail: idahomom@isu.edu

Perinatal Psychobiology Laboratory

Dr. Nicki Aubuchon-Endsley

1440 E. Terry St.
Garrison Hall, Bldg. 63, Room 527
Pocatello, ID 83209-8120

PH: (208) 282-2574
idahomom@isu.edu

You Have an Appointment!

With: Perinatal Psychobiology Laboratory

Date: Time:

Location: 1440 E Terry St. Garrison Hall, Bldg. 63, Rm. 527

Pocatello, ID, 83209-8120

If you are unable to keep your appointment, please contact us as soon as possible via email or phone. Contact information is listed on the front of this card.

Study Purpose

To examine how maternal physical (anthropometry, nutrition) and behavioral (depression, anxiety, and stress) health during pregnancy influence infant growth and behavior at 6 months postpartum.

We are also interested in examining mechanisms that may explain these relationships, including prenatal glucocorticoid exposure and maternal-child interactions.

We will also take into account potential moderators to these relationships, including: sociodemographic variables, pregnancy history and complications, breastfeeding, and infant health.

Study Details

We plan to enroll 60-80 women in their third trimester and follow-up with them and their infants at 6 months postpartum.

Participants must be adult (18+), pregnant women, 33-37 weeks gestation who are fluent in English.

Participants will be excluded with multiple births, severe and persistent mental or physical health concerns, or substance misuse during pregnancy.

Participants will receive up to $75 and students may receive 1 credit for each half hour of participation toward relevant ISU courses.

Study Benefits

Many participants report increased insight into their thoughts, feelings, and behavior following participation in such research.

Given limited longitudinal research into biopsychosocial pathways to offspring health, this research will inform prenatal education and interventions.

You are being contacted because you are a health provider and potential collaborator in southeastern Idaho.

As a licensed Psychologist, Dr. Nicki Aubuchon-Endsley would like to enlist your help in participant recruitment, in exchange for providing educational or other resources to those you serve.

Who We Are

About Us

The Idaho Mom Study seeks to examine women's experiences during pregnancy and how they may relate to the growth and behavior of their infants.

The study is directed by Dr. Nicki Aubuchon-Endsley within the Perinatal Psychobiology Laboratory at Idaho State University.

Contact Us

Phone: (208) 282-2574
Email: idahomom@isu.edu

IDAHO MOM STUDY
1440 E. Terry St.
Garrison Hall, Room 527
Pocatello, ID 83209

IDAHO MOM STUDY

Infant Development And Healthy Outcomes in Mothers

Study Purpose

A woman's experiences during pregnancy may impact her infant's development as well as her adjustment into motherhood, making an understanding of this time period important.

Therefore, the study is designed to examine how your experiences during pregnancy influence the growth and behavior of your baby.

These experiences include pregnancy-related changes such as weight, body size, diet, mood, medical regimen, and health in addition to your experiences during childbirth.

Study Details

We plan to enroll 60-80 women in their third trimester and follow-up with them and their infants at 6 months postpartum.

Participants must be 18 years of age or older, before 37 weeks gestation, and fluent in English.

To learn more about the study, please contact us today!

Study Benefits

Participants will receive up to $75 and students may receive 1 credit for each half hour of participation toward relevant ISU courses.

Many participants report increased insight into their thoughts, feelings, and behavior following participation in such research as well as learning more about their babies.

This research will have the potential to inform prenatal education and interventions, leading to improvements in maternal and offspring health.

Who We Are

About Us

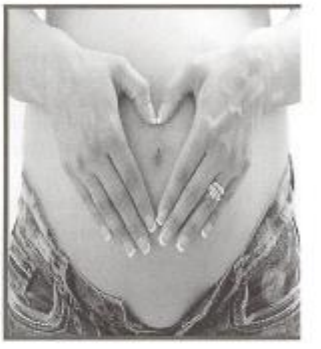
The Idaho Mom Study seeks to examine women's experiences during pregnancy and how they may relate to the growth and behavior of their infants.

The study is directed by Dr. Nicki Aubuchon-Endsley within the Perinatal Psychobiology Laboratory at Idaho State University.

Contact Us

Phone: (208) 282-2574
Email: idahomom@isu.edu

IDAHO MOM STUDY
1440 E. Terry St.
Garrison Hall, Room 527
Pocatello, ID 83209



IDAHO MOM STUDY

Infant Development And Healthy Outcomes in Mothers

Are You Pregnant?

Sign up for the Idaho Mom Study!

Thank you for taking the time to hear about this important study taking place at Idaho State University! If you'd like to learn more about the Idaho Mom Study, please list your contact information below and one of our experienced Research Assistants will contact you at a time that is more convenient for you and your family.

1. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
2. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
3. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
4. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
5. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
6. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
7. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
8. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
9. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
10. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
11. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
12. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
13. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
14. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
15. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
16. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
17. Name:_____ Phone:_____ E-mail:_____ Best Time:_____
18. Name:_____ Phone:_____ E-mail:_____ Best Time:_____

Electronic, print, or telephone recruitment content:

Greetings from the Idaho Mom Study conducted within the Perinatal Psychobiology Laboratory at Idaho State University. You are being contacted because you are a health provider in southeastern Idaho. The Principal Investigator of the study, Dr. Nicki Aubuchon-Endsley, licensed Psychologist, would like to enlist your help in participant recruitment, in exchange for providing educational or other resources to those you serve. Specifically, the Idaho Mom Study seeks to examine women's experiences during pregnancy and how they may relate to the growth and behavior of their infants. For more information on this potentially fruitful collaboration, please contact us at (208) 282-2574 or idahomom@isu.edu. Thank you very much for your time and consideration.

Electronic or print advertisement:

Seeking Paid Research Participants for Idaho Mom Study: The Perinatal Psychobiology Laboratory at Idaho State University (ISU) is conducting a study with pregnant women in southeastern Idaho. If you are prior to your 37th week of gestation, you may be eligible to participate. Participation will include reimbursement of up to $75 and 1 extra credit unit per ½ hour of study participation toward eligible ISU courses. We would love to tell you more about our important research, which may improve prenatal education and interventions to enhance positive outcomes in babies. So, please contact us at (208) 282-2574 or idahomom@isu.edu.

Appendix G: Coding Room Set-Up Instructions