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Physiological Mediators of Parenting Behaviors in Mothers with Low to Moderate Depression Symptoms

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An abstract of A dissertation submitted to the Faculty of the James T. Laney School of Graduate Studies of Emory University in partial fulfillment of the requirements for the degree of Doctor of Philosophy in Psychology 2013 Abstract

There is substantial empirical evidence for the association between depression in mothers and poorer parenting behaviors, with small to moderate effect sizes for associations (Lovejoy, Graczyk, O'Hare, & Neuman, 2000). However, little is known about factors that explain the relationship, with no published studies exploring maternal physiological processes as potential mechanisms. Using the hypothesized Social Engagement System (Porges & Carter, 2011) as a framework, two physiological systems, oxytocin (OT) and respiratory sinus arrhythmia (RSA), were tested, based on their known associations with both depression and parenting behaviors, as potential mediators of the association between depression symptoms and parenting behaviors. We studied a community sample of 70 mothers with low to moderate levels of depressive symptoms (assessed by self report) and their 6 through 12 month old infants. In the laboratory, resting baseline RSA and salivary OT were collected from mothers before they engaged their infants in 10 minutes of play, on which indices of parenting behaviors were based. Following the play, mothers were mildly stressed by restraining infants' arms for a maximum of 2 minutes, after which they soothed their infants. RSA was collected throughout, and a salivary OT sample was collected post-stressor. Mediation hypotheses were not supported. Depression symptoms were significantly associated with only one of the parenting indices (withdrawal; r = .35). Certain indices of dynamic physiological functioning (RSA suppression during stressor, OT response following interaction) emerged as potentially important for their associations with parenting behaviors and/or depression. Key words: maternal depression, parenting, infants, oxytocin, respiratory sinus arrhythmia, vagal tone

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Observational studies have noted qualitative differences in depressed mothers' interactions with their children, when they are compared to well mothers. Specifically, as has been reviewed meta-analytically, the parenting of depressed mothers, compared to well mothers, has been found to be characterized by more hostility, higher rates of negative interactions, less sensitive responding to behavioral cues from their children, lower affective synchrony, and fewer positive interactions (Lovejoy et al., 2000). Given that sensitive parenting is well established as being a major contributor to children's healthy social-emotional and cognitive development (Fogel, 1993; Sroufe, Egeland, Carlson, & Collins, 2005; Tamis-LeMonda, Bornstein, Baumwell, & Damast, 1996), it is essential to understand what contributes to depressed mothers' difficulty in sensitively interacting with their children.

Thus far, researchers have examined parenting deficits in depressed mothers from affective (Lovejoy et al., 2000) and cognitive (Gerdes et al., 2007) theoretical perspectives. Lovejoy et al. (2000) conceptualized the parenting behaviors of depressed mothers as correlates of disturbances in positive and negative affect. This approach is consistent with the tripartite model of depression (L. A. Clark & Watson, 1991), which posits that depression is characterized by both excesses of negative affectivity and deficits in positive affectivity. Using meta-analytic techniques, Lovejoy et al. found support for their hypotheses. Specifically, they found small to moderate effects for group differences between depressed and non-depressed mothers, such that depressed mothers were observed to have greater negative parenting behaviors (d = 0.40) and fewer positive parenting behaviors (d = 0.29). Further, positive parenting behaviors were moderated by the age of the child, such that for mothers of infants less than one year of age, the group

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differences were more pronounced, with a mean effect size in the moderate range (d = 0.47). While these findings offered support for their theoretical model, which was that disturbances in positive and negative affect mediate the association between maternal depression and parenting behaviors, the review, being a meta-analysis, was unable to test a mediational model. The authors could only interpret whether the findings were consistent or inconsistent with their hypothesis. Therefore, empirical studies that measure individual differences in positive and negative affect in depressed mothers will be necessary in order to fully understand the role of maternal affect in parenting deficits. However, the Lovejoy et al. study offers evidence that the parenting of depressed mothers is characterized by negative behaviors, and especially so for depressed mothers of infants, is also characterized by fewer positive behaviors.

In a departure from Lovejoy et al.'s examination of parenting in women with depression from an affective perspective, Gerdes et al. (2007) took a cognitive perspective, relying on Beck's cognitive theory of depression (Beck, 1987) to test whether cognitions associated with the onset and maintenance of depression mediated the association between maternal depression symptoms and parenting behaviors. Of the two cognitions they tested – maternal locus of control and self-esteem, they found that only maternal self-esteem, and not locus of control, mediated the association between depression symptoms and two parenting styles: lax and overreactive (Gerdes et al., 2007). Maternal self-esteem explained 14% and 15% of the variance in the association between depression symptoms and the parenting styles, respectively. Caveats to the interpretation of this study: 1) all measures, including measures of maternal depression, cognitions, and parenting behaviors, were obtained via self-report; 2) the sample was comprised of

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mothers with children diagnosed with ADHD, thereby limiting the generalizability to depressed mothers; and 3) the study design was cross-sectional, and not longitudinal, meaning that the direction of the association cannot be determined – it could be that mothers' low self-esteem was attributable to ineffective parenting styles, and not the other way around. Even so, this study represents an important step in the direction of understanding the role of depressotypic cognitions in the parenting deficits associated with depression. Taken together, the studies by Lovejoy et al. and Gerdes et al. suggest that affective and cognitive models of parenting deficits in depressed mothers are useful, but leave significant portions of variance unexplained. Therefore, additional models need to be explored.

Propelled by advances in instrumentation and measurement, integrative models of human behavior have increasingly incorporated physiological indices to augment cognitive, affective, and behavioral indices of behavior. As such, studies of qualities of parenting behavior have begun to examine physiological predictors, finding in general population samples that individual differences in maternal physiology are related to qualities of parenting in mothers. For example, variations in maternal cardiac regulation (Mills-Koonce et al., 2007), stress hormone functioning (Giardino, Gonzalez, Steiner, & Fleming, 2008), neuropeptide functioning (Feldman, Weller, Zagoory-Sharon, & Levine, 2007), and gene expression (Bakermans-Kranenburg & van IJzendoorn, 2008) have all been found to be associated with maternal behavior in general populations. However, to date, explorations of physiological correlates of maternal behavior have not been extended to consider maternal depression. Including maternal depression in physiological models of deficient parenting is indicated on two theoretical bases. First, there is evidence that specific physiological systems are dysregulated in mothers whose parenting is characterized as deficient, both among depressed mothers and general population samples. Therefore, it may be the dysregulation in the physiological systems that contributes to or is a correlate of both maternal depression and of deficient parenting in general populations. Therefore, it was our goal with this study to expand upon previous work by identifying those specific physiological systems, dysregulation of which is associated with both depression and maternal behavior. Second, and somewhat related, it may be that there is a third, unseen variable – maternal depression – in models of physiological correlates of maternal behavior in the general population. That is, the association between physiological indices and parenting behaviors may be explained by a shared relationship with maternal depression symptoms. However, because depression was not reported to have been measured in the published studies of physiological indices of parenting behavior, the influence of depression is unknown. In this study, we aimed to expand models of physiological correlates of maternal behavior by including measures of maternal depression. Specifically, we planned to test physiological mediators of associations between maternal depression and parenting behaviors.

To build on the promising literature on physiology and parenting, we selected physiological indices for this study using two criteria. First, there had to be evidence that individual differences in the physiological index were associated with variability in parenting behaviors in general population samples. Second, there had to be evidence that dysregulation in the physiological index was associated with depression. There were two physiological systems that had evidence to support both criteria: the autonomic nervous system and the oxytocin (OT) system. Together, these two systems comprise the social engagement system, as posited by Porges and Carter (2011).

Theoretical Framework: The Social Engagement System

The social engagement system (Porges & Carter, 2011) is an integrative theoretical model grounded in literatures describing functioning of the mammalian autonomic nervous system and functioning of the mammalian neuropeptide system. The authors posit that these systems share several important characteristics related to social behaviors. First, these systems are shared among all mammalian species, but are not present in reptiles (Porges & Carter, 2011). Second, the systems are regulated within and interact within the mammalian brainstem (Carter, Grippo, Pournajafi-Nazarloo, Ruscio, & Porges, 2008; Landgraf & Neumann, 2004) – the most evolutionarily ancient area in the nervous system. Third, and perhaps most importantly, these systems are essential in the regulation of mammalian social behavior (Porges & Carter, 2011). Therefore, it is likely that the evolution of the social engagement system was also essential in the shift from "self-regulated" organisms, those that require little to no maternal care after birth (e.g. reptiles) to "other-regulated" organisms, those that require maternal nurturance after birth (e.g. mammals). Porges and Carter emphasize these systems' importance in caregiving behaviors, especially those caregiving behaviors that occur when human offspring are completely dependent on care from others – infancy and early childhood.

The autonomic nervous system. Porges' polyvagal theory (2001) describes a connection between the autonomic nervous system, one of the two components within the social engagement system, and social behavior. Polyvagal theory challenges tradition views of the autonomic nervous system being comprised of sympathetic and

parasympathetic responses by positing that the parasympathetic response actually consists of two circuits: an evolutionarily-adapted vagal circuit that regulates social regions above the diaphragm important to social behavior, and an evolutionarily ancient vagal circuit that regulates autonomic states below the diaphragm and facilitates the immobilization "freezing" response to threat (Porges & Carter, 2011). The former is regulated primarily by the myelinated vagus nerve, while the latter is regulated primarily by the unmyelinated vagus nerve.

With regard to cardiovascular functioning, the myelinated vagus is at dynamic odds with the heart's pacemaker, the sinoatrial node, which has an intrinsic rate that is higher than that of the myelinated vagus (Porges, 2001). Consequently, the myelinated vagus acts as a brake, slowing the heart rate against the intrinsic pace of the sinoatrial node. When vagal tone to the sinoatrial node is high, heart rate is lower. When vagal tone is suppressed, thereby reducing the inhibitory influence to the sinoatrial node, heart rate rapidly increases. The myelinated vagus also has a tonic suppressing influence on the unmyelinated vagus, which can slow the heart. Thus, the myelinated vagus protects the unmyelinated vagus from stopping the heart (Porges, 2007). In response to situations of extreme threat, cardiovascular control shifts from the control of the myelinated vagus to the unmyelinated vagus, which may result in a defensive immobilization response like fainting in humans (Porges & Carter, 2011). Inasmuch as the myelinated vagus is the primary stabilizing influence, it is responsible for respiratory sinus arrhythmia (RSA). Typically, heart rate increases with inspiration and decreases with expiration. RSA is an index of the dynamic influence of the myelinated vagus on the rise and fall in heart rate coordinated with respiration (Porges & Carter, 2011).

RSA is related to social engagement in two primary ways. First, the myelinated vagus is part of the vagal circuit that has afferent and efferent connections from the striated muscles of the face and larynx/pharynx to the heart and lungs. Thus, the same system that regulates the expression of emotion via facial movements and vocal intonations also regulates RSA (Porges & Carter, 2011). It is hypothesized that a depressed social engagement system will be associated with both low RSA and low variability in behavioral expressions of emotions (flat affect, lack of vocal prosody). The second way that RSA is related to social engagement is through the management of the internal bodily experience of negative emotions. For example, by maintaining lower heart rate and calming visceral organs, the myelinated vagus facilitates states of regulation and calm, enabling an individual to engage socially (Porges & Carter, 2011). Conversely, in conditions of perceived danger, RSA is reduced and heart rate increases, contributing to the experience of a negative emotion, which thereby compromises social engagement. Because of its properties in regulating these different states, RSA is useful to scientists an index of both regulation and reactivity.

The neuropeptide system. The other component of the social engagement system is the neuropeptide system, particularly the OT system. OT is a nonapeptide unique to mammals that plays dual roles, as a neurotransmitter acting solely in the central nervous system and as a hormone in the periphery. OT is synthesized in large magnocellular neurons and smaller parvocellular neurons in the paraventricular (PVN) and supraoptic nuclei of the hypothalamus. The magnocellular neurons of the PVN project to the posterior pituitary from which OT is released into the periphery, while OT is transported via parvocellular neurons to target brain areas, including the amygdala and nucleus accumbens. The OT system is essential in promoting social behaviors. Therefore, given their common function in promoting social affiliation, it is not surprising that OT plays a role in regulating the autonomic nervous system. For example, OT receptors have been found in the dorsal nucleus of the vagus nerve (Gimpl & Fahrenholz, 2001), which innervates the vagus nerve projections to the lungs and viscera. Also, OT receptors have been discovered in pathways regulating the myelinated vagus itself (Porges & Carter, 2011).

The OT system is hypothesized to promote affiliation through two mechanisms: the activation of rewards systems and the attenuation of stress responsivity (A. Campbell, 2008). Dopaminergic neurons, which modulate reward, run from the ventral tegmental area to the nucleus accumbens, where they are co-localized and interact with oxytocinergic neurons, which drive affiliative motivation (A. Campbell, 2008). The reward and affiliative systems are inter-related such that OT can increase central opiate release threefold (Csiffary, Ruttner, Toth, & Palkovits, 1992).

Similarly, the pathway whereby OT promotes affiliation via attenuation of the stress response is well validated. OT is posited to attenuate HPA axis reactivity response by directly down-regulating the production and secretion of corticotropin releasing factor (CRF) in the paraventricular nucleus of the hypothalamus (Windle, Shanks, Lightman, & Ingram, 1997). As CRF is thought to stimulate OT release from magnocellular neurons, this sequence may work as a negative feedback loop. By down-regulating CRF release, the cortisol release associated with stress is also down regulated, thereby attenuating the stress response and enabling social engagement.

Optimal functioning of the social engagement system. Because both of the components involved in the social engagement system are complex and dynamic, researchers are still in the process of defining "optimal" functioning. For RSA, three scores are relevant: baseline or resting, suppression, and recovery. During low stress times, higher RSA is the optimal resting condition, or the condition that best facilitates social engagement (Beauchaine, 2001), allowing for more flexible and responsive cardiovascular regulation in reaction to environmental stimuli. Therefore, in our study, lower resting RSA represents the risk condition. However, this is only true during situations free of threat or stress. When threatening or stressful stimuli are present, greater vagal suppression, or a decline in RSA relative to baseline, is optimal and allows for the mobilization of the fight/flight response (Beauchaine, 2001). The index of vagal suppression is typically calculated as the change in RSA from baseline to stressor paradigm. The risk condition can be conceptualized as failure to suppress RSA in response to a stressor, as indicated by a low degree of decrease in RSA from baseline. This statement carries with it some caveats however. First, greater suppression of RSA has been noted in adults with panic disorder (Yeragani et al., 1991) and during marital conflict among male batterers (Gottman, Jacobson, Rushe, & Shortt, 1995) relative to controls. Therefore, excessive RSA suppression may be understood as an index of emotional lability (Beauchaine, 2001) and can be differentiated from normative levels of RSA suppression. Second, in conjunction with the law of initial values (Benjamin, 1963), failure to suppress RSA may be a function of low resting RSA, as levels of baseline RSA and RSA suppression are strongly correlated (Katz & Gottman, 1995). Despite this, they have been established as distinct constructs (Rottenberg, Clift, Bolden, & Salomon,

2007), baseline RSA broadly representing trait emotion and RSA suppression broadly representing state emotion (Beauchaine, 2001). Even so, RSA suppression should not be interpreted in the absence of data about resting RSA. Finally, RSA recovery is a construct parallel to RSA suppression. Low RSA recovery, operationalized as a smaller relative increase in RSA following a stressor, represents a failure of the RSA system to quickly and dynamically resume vagal control of cardiovascular functioning (Porges & Carter, 2011). Therefore, low RSA recovery represents the risk condition in our study.

The literature on the OT system is somewhat more difficult to interpret than the RSA literature. Related to social engagement behaviors, higher levels of OT in adults are consistently associated with greater prosocial outcomes, including greater feelings of trust towards another individual, more frequent prosocial behaviors (e.g. eye contact), and improved memory for social stimuli (MacDonald & MacDonald, 2010). However, the pattern of OT functioning in response to stress is more complicated. Because of the dynamic nature of the negative feedback loop that is believed to exist between the OT system and the HPA axis, the relationship between the two systems is hard to parse out in naturalistic studies of humans. Depending on the point in the feedback loop at which measurements are taken, one might expect a positive or negative association between an index of HPA reactivity and OT. For example, if measurements are taken at the beginning of a stress-OT response feedback loop, when OT release is increasing in response to stress, one might find a positive association. However, if measurements are taken at the point in the stress-OT feedback loop when stress has attenuated partially in response to OT, one might find the negative association. In fact, both types of findings are reported in studies of humans. A review of the literature revealed associations

between OT and indices of stress, anxiety, and distress in both the positive (Hoge, Pollack, Kaufman, Zak, & Simon, 2008; Leckman et al., 1994; Marazziti et al., 2006; Taylor et al., 2006; Taylor, Saphire-Bernstein, & Seeman, 2010; Tops, van Peer, Korf, Wijers, & Tucker, 2007; Turner, Altemus, Enos, Cooper, & McGuinness, 1999) and negative (Anderberg & Uvnas-Moberg, 2000; Heim et al., 2009; Light, Grewen, & Amico, 2005; Light et al., 2000; Scantamburlo et al., 2007) directions.

Given these mixed findings, it may be that the relationship between OT and indices of stress is more complex than one that is uni-directional. Therefore, a "dysregulation" framework for the relationship between OT and depression is useful in that it offers a framework for interpreting the seemingly inconsistent findings from the human literature. Support for this notion comes from studies demonstrating that, among clinical populations, peripheral OT levels demonstrate a quality of within-subject variability over time (e.g. Cyranowski et al., 2008). In addition, a dysregulation model is consistent with the findings related to so many other hormone systems, which describe a "U-shaped" function, where excessive elevations or deficits are associated with worse outcomes. For example, such curvilinear relationships have been noted in the association between DHEA levels and depressive symptoms in both sexes (Goodyer et al., 1996; Goodyer, Herbert, Tamplin, & Altham, 2000), and estrogen (estradiol) and negative affect in adolescent girls (Brooks-Gunn & Warren, 1989). With this understanding, we hypothesize that OT dysregulation, conceptualized as excessive elevations or depletions in peripheral levels, represents the risk condition.

The Social Engagement System and Parenting Behaviors

Turning back to our selection criteria for the physiological indices of study, there is emerging evidence that RSA and OT systems are each related to individual differences in maternal caregiving behaviors, the first of our criteria for inclusion in this study. For example, a general population study of mothers interacting with their children during play and still face (stressor) paradigms hypothesized and found support for lower RSA during the reunion episode, during which the mother typically soothes the distressed child, being associated with greater maternal sensitivity (Moore et al., 2009). The same research group published another study based on the same sample, finding that greater RSA suppression, defined as the change in RSA from a baseline condition to a stressor paradigm, was associated with observer-rated sensitivity to child distress among those mothers of insecurely attached infants (Mills-Koonce et al., 2007). Finally, another study found heart rate accelerations, a state consistent with RSA suppression, in adult mothers when presented with infant pain and hunger cries, as compared to teen mothers and non-mothers (Giardino et al., 2008).

Related to OT functioning, general population studies of mothers and their infants have consistently found positive associations between OT and optimal maternal behaviors. Specifically, a research group in Israel has found significant positive correlations between plasma and/or salivary OT and greater expressions of positive affect, affectionate touch, and "motherese" (commonly known as "baby talk") in interactions with their infants (Gordon, Zagoory-Sharon, Leckman, & Feldman, 2010a), greater mother-child affect synchrony (Feldman, Gordon, & Zagoory-Sharon, 2010), greater triadic (mother, father, and infant) affect synchrony (Gordon, Zagoory-Sharon, Leckman, & Feldman, 2010b), more frequent checking behaviors, and higher scores on a "maternal behavior" composite (time spent gazing at the infant, positive affect, affectionate touch, and motherese) (Feldman et al., 2007).

The Social Engagement System and Depression

Following with our second inclusion criterion, there is robust evidence that depression is associated with dysregulation of both RSA and OT systems. Looking first at baseline RSA levels, while the literature is somewhat inconsistent, a meta-analysis of 13 studies having compared RSA levels in participants with Major Depressive Disorder (MDD) to healthy controls found a moderate effect size for the depressed participants having lower RSA levels (Rottenberg, 2007). Similarly, studies have found continuous relationships between depression symptoms and baseline RSA levels. For example, one study found that heart rate variability (i.e. the amplitude of RSA) was lower in women who rated themselves as being in the highest quartile of depression symptoms among a general population sample, as compared to the lowest quartile (Light, Kothandapani, & Allen, 1998). Evidence for differential RSA suppression comes from a study in which adults with MDD not only exhibited lower baseline RSA levels when compared to healthy control participants, they also showed a lack of RSA suppression in response to two different types of stressor tasks (Rottenberg et al., 2007). Finally, a study examining RSA recovery, defined as an increase in RSA levels following a stressor, in depressed versus non-depressed adults found significantly lower increases in RSA, or RSA recovery, following the viewing of a sad film clip (Rottenberg, Wilhelm, Gross, & Gotlib, 2003).

The theoretical literature examining the role of OT in depression seems to suggest that the OT system may be particularly important in the onset and maintenance of depression in women. There are two notable models of female physiology that posit a central role of OT in the development of depression and other disorders: Cyranowski et al. (2003) and Taylor (2000). The models are slightly different in their express purposes and theoretical frameworks. Cyranowski's model is specific to depression and proposes that there is an increased need for social connectivity in females that begins in puberty, due to the surge in estrogen and OT, and continues into adulthood. The increase in affiliative drives interacts with stressors of adolescence to create a "depressogenic diathesis," or a particular vulnerability to depression. Taylor's model focuses on evolutionarily adaptive sex differences in responses to stress. Taylor maintains that the increased levels of OT in females are an indication that women are biologically programmed to affiliate with others socially, especially in response to stress. The OT that is released in response to affiliation leads to a reduction in elevated stress hormones. Taylor asserts that depression and other forms of psychopathology can result from a failure to have positive social contact needs met after a stressor. Despite the difference in frameworks, the theories share an involvement of OT in the emergence of depression in females.

However, the literature examining depression and OT in females is mixed and therefore does not fully support the theoretical models. Some studies of women found significant inverse associations between OT and depression symptoms (Anderberg & Uvnas-Moberg, 2000; Scantamburlo et al., 2007). However, a more methodologically rigorous study found a positive relationship (Parker et al., 2010). This study was fairly unique in that the authors measured OT in clinically depressed women at regular intervals and compared plasma concentrations to those of non-depressed control women. In addition to their finding showing elevated OT concentrations across the experimental paradigm, Cyranowski and colleagues (2008) also found that the depressed women showed significantly greater variability in their OT release relative to nondepressed control women. This finding led the researchers to conclude that OT dysregulation, rather than elevated OT, might be associated with depression.

Summary

Our study sought to explore physiological mediators of associations between maternal depression and parenting behaviors. Using the social engagement system as our theoretical framework, we planned to examine the two components of the social engagement system – autonomic nervous functioning and neuropeptide functioning – as potential mediators of the association between maternal depression and parenting behaviors. Based on a meta-analytic review of studies of maternal depression and parenting behaviors that found greater negative behaviors and fewer positive behaviors (Lovejoy et al., 2000), we focused on these two types of parenting behaviors in our study. In addition to our theoretical framework, justification for our hypotheses comes from three literatures, which found associations among indices of the following constructs: maternal depression and parenting behaviors, physiology and parenting behaviors, and physiology and depression. To our knowledge, our study is the first to integrate these three literatures.

Moderators

In addition to the main effects and mediated effects hypothesized and tested within our study, it was important to consider factors that may influence how the primary variables of interest relate to one another, e.g. moderators. While information about mediation helps us understand the *how* of a relationship, information about moderation elucidates *for whom* or *under what circumstances* a relationship exists. For example, knowing that the predicted mediated relationship is only supported within a subset of participants would have implications for how the findings are interpreted. Further, evidence of moderated relationships has the potential to inform refinement of the hypothesized model, as well as to generate research questions for future inquiry. Thus, we developed a set of theory-based factors that may increase or decrease the strength of the mediated effect.

Infant temperament. Temperament in infancy is important to consider in a study of parenting behaviors because of its potential role in transactional processes (Sameroff, 1975). That is, infants with different temperamental qualities may shape their environments by evoking negative or positive responses from their caregivers. For example, a number of studies have shown that aspects of infant temperament either concurrently are associated with or prospectively predict maternal parenting behaviors. Most of the studies have focused on negative affect and difficulty in infants and their associations with negative maternal parenting behaviors, finding for example that negative temperaments in infants were associated with less contact from mothers during observations (Crockenberg & Acredolo, 1983), less maternal guidance and more control during a clean-up task (Braungart-Rieker, Garwood, & Stifter, 1997), and prospectively predicted less effort in teaching interactions in mothers of boys (Maccoby, Snow, & Jacklin, 1984). In addition, there is also some evidence that infants' positive temperament is associated concurrently and prospectively with positive maternal parenting behaviors. For example, temperament ratings of more infant smiling and laughter were associated with greater smiling and eye contact during an observed interaction (Crockenberg & Acredolo, 1983). Prospectively, positive maternal parenting behaviors, like eye contact, physical contact, soothing, and responsiveness, were both greater in amount and showed a greater positive growth trajectory when the infant was in a non-irritable group (van den Boom & Hoeksma, 1994).

In addition, there are many cross-sectional studies that find significant concurrent associations between negative infant temperament and maternal depression (Austin et al., 2005; for a meta-analysis, see C. T. Beck, 1996; Edhborg, Seimyr, Lundh, & Widstrom, 2000). While the assumption of most of these studies has been that the direction of influence is from mother to child, there is evidence from a prospective epidemiological study that the relationship between infant negative temperament and maternal depression is transactional, with greater negative child temperament at six months predicted elevations in maternal depression at two years, and vice versa (Hanington, Ramchandani, & Stein, 2010). Thus, given potential associations between infant temperament and parenting behaviors, as well as between infant temperament and maternal depression, we included temperament as a potential moderator in testing our hypotheses.

Maternal childhood trauma. Among the many adverse outcomes associated with trauma experienced during childhood, there is evidence that the Social Engagement System is also altered in the presence of childhood trauma, likely in a persistent way. In a study of adopted children who had suffered severe institutional neglect before adoption, the normative elevation in urinary OT after interacting with a primary caregiver was not present, as compared to family-raised children (Alison, Ziegler, Kurian, Jacoris, & Pollak, 2005). Further, Heim and colleagues (2009) found that CSF levels of OT were

significantly lower among adult women who had experienced maltreatment as children. Moreover, CSF OT levels were inversely correlated with the number of trauma categories to which the women had been exposed, as well as inversely correlated with the severity of the trauma overall across categories. This study suggests that the deficits in OT functioning may have their origins in early experience and persist into adulthood. In addition to the relationship between childhood trauma and OT, childhood trauma is also predictive of later depression symptoms (for a review, see Heim, Newport, Mletzko, Miller, & Nemeroff, 2008) and poorer parenting behaviors in mothers (Banyard, Williams, & Siegel, 2003). This evidence informed our decision to include childhood trauma exposure in our set of potential moderating factors.

Based on the theory and empirical evidence presented, we tested the following hypotheses:

Primary Hypotheses

Hypothesis 1: Based on literature showing that baseline RSA is lower among depressed adults, baseline RSA will mediate the association between maternal depression symptoms and parenting behavior, specifically positive and negative behavior.

Hypothesis 2: Based on literature showing that RSA suppression, defined as the change in RSA from resting baseline to stressor, is lower among depressed adults and literature showing that lower RSA suppression is related to less maternal sensitivity, RSA suppression in response to a stressor will mediate the association between maternal depression symptoms and parenting behavior, specifically positive and negative behavior.

Hypothesis 3: Based on literature showing that higher levels of maternal depression are associated with lower baseline OT and literature showing that maternal

parenting behavior is associated with higher baseline OT, OT levels will mediate the association between maternal depression symptoms and parenting behavior, specifically positive and negative behavior.

Exploratory Hypotheses

Hypothesis 4: Based on literature showing that RSA recovery, defined as the increase in RSA following a stressor, is diminished in depressed adults, RSA recovery will mediate the association between maternal depression symptoms and parenting behavior, specifically positive and negative behavior.

Hypothesis 5: Based on literature showing that higher maternal depression levels are associated with greater OT dysregulation, change in peripheral OT in response to a stressor will mediate the association between maternal depression symptoms and parenting behavior, specifically positive and negative behavior.

Moderated Mediation Hypotheses

Hypothesis 6: Negative infant temperament will moderate the mediated relationships predicted in hypotheses 1-5, such that in the presence of higher levels of negative infant temperament, the mediated effects will be larger in magnitude.

Hypothesis 7: Maternal childhood trauma history will moderate the mediated relationships predicted in hypotheses 1-5, such that in the presence of higher levels of childhood trauma history, the mediated effects will be larger in magnitude. While we have stronger evidence to support the role of childhood trauma on the OT system, because the OT system is hypothesized to be part of the Social Engagement System, along with RSA functioning, we extended our moderated mediation hypothesis to include RSA functioning.

Method

Participants

Inclusion/exclusion criteria. Primary inclusion criteria included: being the mother, either through birth or adoption, to a child from six through twelve months of age; being either married, cohabiting with a significant other, or some other stable living situation in which there was another adult living in the home (typically the father of the infant, but in a few instances the mother of the participant); being older than 20 years of age; having completed the 10th grade. Exclusion criteria included: meeting DSM-IV criteria for a serious mental illness other than depression (schizophrenia or other psychotic disorder, the presence of psychotic features, or bipolar disorder) and infant prematurity (<37 weeks gestational age). Justification for the demographic inclusion criteria comes from evidence that maternal behavior is adversely impacted by being a teen mother (Barratt & Roach), being a single mother or being under-educated (Allen, Affleck, McGrade, & McQueeney). As our desire was to have depression be the primary risk factor for impaired parenting behavior in our study, we excluded on these criteria. For the DSM-IV disorders of exclusion, all could be associated with deficits in parenting, potentially obfuscating the association between depression symptoms and parenting behaviors.

Eligibility. Mothers and their infants were recruited from among women who had indicated willingness to be contacted to participate in studies conducted within the Emory University Department of Psychology. Eligibility for all potential participants was determined via a brief screening call. Potential participants were asked questions about demographics (age, marital status, education, gestational age of child at birth) and

presence or history of serious mental illness. A total of 79 mothers and their infants were determined to be eligible for the study. Of those, we enrolled a sample of 70 motherinfant dyads. Of the remaining nine potential participants, eight either canceled or failed to arrive for their scheduled appointment and were unable to be rescheduled within the window of eligibility. One potential participant changed her mind about participating.

Participant characteristics. The mean age of mothers enrolled in the study was 33.1 years (SD = 4.7, range = 20.0 – 43.8), while the mean age for infants was 9.8 months (SD = 1.6, range = 6.4 – 12.2). The mean infant gestational age at birth, as reported by mothers, was 39.3 weeks, and 51.4% of infants (n = 36) were female. At the time of participating, approximately half of mothers were breastfeeding their infants at least once a day (n = 33; 47.1%), while a quarter were taking a hormone-based birth control medication (n = 18; 25.7%). The participating infant was the first child for 30 of the mothers (42.9%). The racial and ethnic identities of the mothers were as follows: 64.2% White/Caucasian, 18.6% Black/African-American, 5.7% Hispanic/Latina, 2.9% Asian/Asian-American, and 8.6% identified with two or more categories. The median annual household income was \$40,000-\$49,999 (range = \$0-\$9,999 – >\$100,000). The median educational attainment for mothers was a bachelor's degree (range = high school/GED – graduate/professional degree).

Procedure

Emory University Institutional Review Board approval was obtained. Once participant eligibility was determined, mothers were asked about their current breastfeeding status. If mothers were currently breastfeeding, appointments were scheduled to fall between feedings, with the appointment scheduled to start at least an hour after the previous feeding and scheduled to end at least an hour before the subsequent feeding. This procedure was informed by findings showing elevated salivary OT within thirty minutes after breastfeeding and anticipatory elevations in salivary OT starting within thirty minutes before the next feeding (White-Traut et al., 2009). Thus, the timing of the laboratory session and OT sampling was set at a time when OT levels were expected to be at baseline levels for nursing mothers. All laboratory sessions took place in a lab in the Psychology Department on the Emory University campus.

Baseline segment. After informed consent was obtained, infant saliva was collected in order to measure baseline salivary cortisol. The mother and infant were then separated within the same room for a period of ten minutes; a research assistant played with the infant while the mother met with the primary investigator. Mothers were instructed not to interact with or touch the infant during this time, as both have been associated with elevations in peripheral OT (Feldman, Gordon, Schneiderman, Weisman, & Zagoory-Sharon, 2010; Uvnas-Moberg, 1996).

The primary investigator assisted the mother with the placement of disposable electrodes on her upper chest and on top of her ribcage to measure cardiac activity. A band was placed around her abdomen to measure respiration. Once the instruments were placed, the mother was instructed to read a magazine for two minutes, during which time baseline RSA data was collected, in accordance with procedures of researchers who have examined RSA and maternal behavior (Moore et al., 2009). At the end of ten minutes of no interaction with the infant, a saliva sample was collected from the mother for the measurement of baseline salivary OT. **Free play segment.** Infants were placed in a play seat with their mothers sitting in a chair opposite them. Mothers were instructed to face their infants and, after the researchers left the room, to play with their infants as they normally would for ten minutes. Two digital video cameras aimed at the mothers and the infants captured the interactions on a split screen for later behavioral coding and rating. Maternal cardiac and respiration data was collected throughout the play interaction.

Infant stressor and reunion segment. The primary investigator re-entered the room and instructed the mothers in an arm restraint protocol as employed by many researchers (e.g. Braungart-Rieker & Stifter, 1996; Fox, 1989). Mothers were instructed to gently restrain their infants by holding their infants' arms down to their sides. Mothers were also instructed to maintain a neutral facial expression and to refrain from verbally interacting with their infants. The principle investigator remained in the room, standing behind the infants outside of their view, in order to remind the mothers to maintain a neutral expression and to remain silent if they failed to do so. The arm restraint continued until either twenty seconds of intense crying from the infant or two minutes of arm restraint, whichever came first. This protocol was selected as a stressor for the motherinfant dyad because 87% of five to twelve month old infants have been found to respond to it with at least moderate levels of vocal distress (Camras, Oster, Campos, Miyake, & Bradshaw, 1992). Following the arm restraint procedure, the mothers were instructed to freely interact with their infants again, which typically included picking up the infants. The principle investigator left the room for this two-minute reunion period. Cardiac and respiration data were collected during both the stressor and reunion periods.

Conclusion. The principle investigator re-entered the room and conducted a structured diagnostic interview to assess for the presence and history of MDD. The mother then completed two self-report measures: one assessing current depression symptoms and the second assessing for traumatic experiences during the mother's childhood. Finally, she completed a demographic questionnaire that collected demographic information, information about breastfeeding patterns, infant sleep patterns, maternal parity, and hormonal birth control use. Specifically related to breastfeeding, mothers were asked the time of day of the most recent nursing or pumping and to estimate, at the time of participation, how many times per 24-hour period on average she nursed or pumped. Regarding infant sleep, the mothers were asked at what time the infant had woken up on the day of participation, if the infant had napped at all prior to participation, and if so, for how long the infant had napped.

Ten minutes following the end of the arm restraint task, a saliva sample was collected from the mother in order to measure OT response to the stressor, in accordance with the developer of the salivary OT assay in order to detect changes in response to the stimulus (Carter et al., 2007). A second saliva sample was also collected from the infant to measure cortisol response to the arm restraint. Mothers were paid \$25 for their time and provided with a pass to cover their cost of parking.

Mother-report Measures

Beck Depression Inventory-Second Edition (BDI-II; Beck, Steer, & Brown, 1997).The BDI-II is a self-report measure with 21 items. Respondents are instructed to base their answers on the past two weeks, paralleling the DSM-IV criteria of duration for a major depressive episode. Each item on this instrument is rated on a 4-point scale,

ranging from 0 to 3. A total score is computed by adding the ratings across items. The BDI-II has been found to be both a valid and reliable measure of depression severity, with an especially high degree of content validity, construct validity, and internal consistency (Beck et al., 1997). The BDI-II has been shown to have good concurrent validity with measures of postpartum depression (Boyd, Le, & Somberg, 2005). The internal consistency for all items of the BDI-II among the sample was good (Cronbach's $\alpha = .92$). Although the BDI-II was treated as a continuous variable in all of the analyses, descriptively we noted that 10% of the women exceeded the standard cut score (>13) for clinically significant levels of depression (Beck et al., 1997), with 7% reporting symptoms in the "mild depression" range and 3% reporting symptoms in the "severe depression" range.

Structured Clinical Interview for DSM-IV Axis I Disorders (SCID; First, Spitzer, Gibbon, & Williams, 1995). The SCID is a semi-structured diagnostic interview designed to assess Axis I disorders of the DSM-IV. The SCID was used to assess for a past history of MDEs, as well as for current MDE diagnostic status. The SCID ultimately yields a diagnosis as to whether or not the women met full criteria for a Major Depressive Disorder, either in the past or currently, according to the criteria outlined in the DSM-IV. The SCID interview was conducted by the principle investigator, a master's level clinician with significant training and experience in the assessment of depression in mothers. Reliability checks were conducted for 13% of the interviews by a bachelors level research coordinator with extensive experience assessing depression in mothers. Only one mother met diagnostic criteria for a current MDE. However, 41% (n = 29) of mothers met criteria for at least one MDE in the past, which was consistent with our goal of recruiting a sample of mothers where approximately half had a past MDE. The median number of past episodes among mothers meeting criteria for a past episode was one (range = 1 - .50). The mother who reported approximately 50 past episodes was also the mother meeting criteria for a current episode. Among the 28 mothers with a past but not current MDE, the mean time elapsed since the most recent episode was 6.64 years (range = .67 - 19 years).

Childhood Trauma Questionnaire (CTQ: Bernstein & Fink, 1998). The CTQ is a 28-item self-report inventory that measures histories of abuse and neglect. Individuals respond to a series of statements about childhood events, with five possible responses ranging from 'Never True' to 'Very Often True.' The CTQ inquires about five types of maltreatment – emotional, physical, and sexual abuse, and emotional and physical neglect. Five items load onto each scale. There are additionally three items that detect false-negative, or minimizing, accounts. Internal consistency reliability coefficients for the validation samples were mostly good to excellent, with median Cronbach's alphas as follows: Emotional Abuse (.89), Physical Abuse (.82), Sexual Abuse (.92), Emotional Neglect (.89), and Physical Neglect (.66). Internal consistency reliability coefficients for the current sample were as follows: Emotional Abuse (.82), Physical Abuse (.63), Sexual Abuse (.74), Emotional Neglect (.90), and Physical Neglect (.57). Since alpha coefficients below .60 indicate 'poor' reliability'(Kline, 1999), scores on Physical Neglect must be interpreted with caution. The three items comprising the Minimization/Denial scale are dichotomized ("never" = 0, all other responses = 1) and summed; a total of one or greater suggests the possible underreporting of maltreatment (Bernstein & Fink, 1998). There were 14 participants (20%) whose scores on the

Minimization/Denial score were greater than or equal to one. However, excluding them from the reliability analysis did not improve the alpha coefficients.

The CTQ manual provides cut-off scores in each category (e.g. \ge 9 Emotional Abuse, \ge 8 Physical Abuse) that indicate the presence of at least low levels of abuse or neglect. Following procedures used by Heim et al. (2009), we calculated a sum for each mother that indicated the total number of categories for which there was evidence of at least low levels of abuse or neglect, ranging from zero through five. For our sample, this number was zero for 51.4% of the women. The distribution of this score was positively skewed (skewness = .98).

The Infant Behavior Questionnaire - Revised (IBQ-R; Gartstein & Rothbart, 2003). The IBQ-R is a 191 item, factor-analytically derived measure of infant temperament, based on the definition of temperament posited by Rothbart and Derryberry (1981). Respondents rate the infant's behavior during the past week in a variety of domains on a seven-point scale, from one (Never) to seven (Always). The questionnaire yields scores on 14 scales, with ten to 18 items per scale and scale scores being the mean of items on that scale. Scales cluster into three overarching factor scores: Orienting/Regulatory Capacity, Surgency/Extraversion, and Negative Affectivity. Internal consistency for Negative Affectivity, the factor used in subsequent analyses, was good (Cronbach's $\alpha = .85$).

Physiology Measures

Respiratory sinus arrhythmia (RSA). Maternal cardiac and respiration data was recorded with a 3-channel ambulatory recorder (3991x/3 BioLog, UFI, Moro Bay, CA, USA) throughout baseline, free play, stressor, and reunion segments. Event markers
separated the resulting file into the respective conditions. Researchers were trained and certified as reliable in the use of CardioEdit software (Brain-Body Center - University of Illinois at Chicago, 2006-2007). The data were edited by these researchers in order to extract fully analyzable data. Following procedures developed by Porges (1985), RSA was computed from each segment using CardioBatch (Brain-Body Center - University of Illinois at Chicago, 2006-2007). This technique uses time-domain filters to extract the RSA and calculated the amplitude (variance) of the pattern (Porges, Doussard-Roosevelt, & Maiti, 1994).

In order to capture the indices of interest in this study, RSA suppression and RSA recovery, RSA was measured in each of the four segments: baseline, play, stressor, and reunion. We conceptualized RSA suppression as suppression relative to baseline, consistent with previous research in vagal regulation (e.g. Bornstein & Pluess, 2000; Rottenberg et al., 2007). This index was calculated as stressor minus baseline, with more negative values representing greater suppression, which is hypothesized to be more optimal (Rottenberg et al., 2007). RSA recovery was calculated as reunion minus stressor. In this case, more positive values indicate greater recovery, which is hypothesized to be more optimal (Porges & Carter, 2011).

Oxytocin. 50 mL polypropylene tubes were chilled in a -80°C freezer for at least 30 minutes prior to the start of the laboratory session. Chilled tubes were kept in an iced container during the laboratory session. For saliva collection, mothers were instructed in passive drool techniques, following validated procedures (Granger et al., 2007), until 2mL were collected at each collection point. Tubes were returned to the ice container until they could be transferred to a -80°C freezer for storage.

Following procedures outlined by Carter (Carter et al., 2007), dried samples were reconstituted in buffer and separated into two aliquots. OT levels were determined using a commercially prepared enzyme immunoassay (EIA) kit produced by Enzo Life Sciences (formerly Assay Designs) (State College, PA). Measurements were performed in duplicate with reported values representing the mean value of the duplicates. Interassay coefficient of variation was 9.47% at 7.50 pg/mL, and the intra-coefficient of variation was 9.43% at 6.99 pg/mL. OT was sent to be assayed in two batches. Because there were significant group differences in baseline OT and post-stressor OT samples by batch, OT assay batch was included as a control variable for all analyses incorporating OT data [(baseline OT: t(68) = 6.12, p < .001, Cohen's d = 1.50) (post-stressor OT: t(67) = 4.20, p < .001, Cohen's d = 1.21)]. OT assay batch explained 36% and 27% of the variance in baseline and post-stressor OT scores, respectively. OT response to a stressor, one of the indices of interest in the study, was calculated as post-stressor OT minus baseline OT, meaning that more positive values indicate a greater increase in OT relative to baseline.

Maternal menstrual cycle phase. In order to determine at what phase in their menstrual cycles were when mothers participated in the study, mothers were requested to make a note of the first day of their next menstruation following the laboratory session. Five weeks following the date of participation, mothers were contacted and asked to provide the date. Data about menstruation has been found to be more reliable when collected prospectively, rather than retrospectively (Wideman, Montgomery, Levine, Beynnon, & Shultz, 2012). There were three mothers on whom we were unable to collect menstrual cycle data due to non-responsiveness. 21 mothers had not yet menstruated by the time they were contacted after the laboratory session. Of the 21, nine were taking some kind of hormonal birth control medication, ten were not taking any medication, and two declined to provide that information. Also of the 21, 14 were actively breastfeeding, which has been associated with delayed resumption of menses following childbirth (Bonnar, Franklin, Nott, & McNeilly, 1975). Therefore, of the 21 non-menstruating women, 18 were either taking hormonal birth control medication, breastfeeding, or both, two were not breastfeeding but their medication statuses were unknown, and one was neither on medication nor breastfeeding. Of the remaining 46 women, 27 were classified as being in the follicular phase at the time of the laboratory session, while 19 were classified as being in the non-follicular phase of the menstrual cycle at the time of data collection.

Observed Parenting Behavior Measures

Maternal interactive quality ratings. A team of research assistants, unaware of other data on the mothers or infants, was trained to rate the digitally video-recorded free play segments using the Maternal Interactive Quality Ratings, 12 rating scales taken from the standardized rating scales of Ainsworth (Ainsworth, Blehar, Waters, & Wall, 1978), Clark (1985), and Campbell (1991). This set of scales was selected to assess the quality of the mother's interactive behavior with her child, and were chosen to reflect parenting characteristics known to be associated with depression in mothers: Insensitive Parenting, Intrusiveness, (Low) Positive Affect, and Negative Affect. (Lovejoy et al., 2000). Insensitive Parenting was measured with two scales from Campbell: *sensitivity/responsiveness to distress* and *sensitivity/responsiveness to nondistress*. Intrusiveness was also measured with two scales: Campbell's *intrusiveness* and

Ainsworth and colleagues' cooperation vs. interference. Positive Affect was measured with positive regard for the child, warmth, and stimulation of development-all from Campbell, as well as quality of verbalizations and structures and mediates environment from Clark. Finally, Negative Affect was measured using three scales from Clark. They included quality and amount of physical contact: negative; angry, hostile mood; and displeasure, disapproval, criticism. Scores for each of the scales are based upon a 4- or 5point Likert scale, and take into consideration both the quality and quantity or intensity of the behavior measured in the scale. For most of the scales, raters assign a score based on whether the behavior is 'characteristic' or 'not characteristic' of the mother (or some gradient in between). Raters were undergraduate research assistants who have been trained to reliability on the rating scales and who were required to maintain high levels of reliability as checked with weekly random checks, when the same segment was assigned to two or more raters without the raters' awareness. Sixteen (23.19%) of the valid mother-infant interaction videos (n = 69; one video was lost due to a technical malfunction) were randomly selected to be rated independently by a second rater. Interrater reliability was in the 'substantial agreement' range, according to guidelines by Landis and Koch (1977), weighted kappa = .68. There was little variability in two of the scales: angry, hostile mood and displeasure, disapproval, criticism. Only three (4%) mothers displayed instances of behavior warranting a rating above "slight" instances of angry, hostile mood, and only five (7%) mother's behaviors were rated above "slight displeasure, disapproval, or criticism." Therefore, these two scales were dropped from subsequent analyses.

The dimensionality of the remaining 12 scales was analyzed using principal components analysis. For one of the scales, *sensitivity/responsiveness to distress*, there were nine mothers (13%) for which there was no opportunity to rate this parenting quality because their infants never showed distress. Therefore, the decision was made to replace the score for this scale with the sample mean for those nine participants. Three factors were rotated using a direct oblimin rotation procedure. The rotated solution yielded three interpretable factors, which we interpreted as follows: Active, engaged parenting (*structures and mediates environment, quality of verbalizations, sensitivity/responsiveness to distress*, and *stimulation of development*); Sensitive, responsive parenting (*intrusiveness* reverse-scored, *quality and amount of physical contact, cooperation vs. interference*, and *sensitivity/responsiveness to nondistress*); and

reverse-scored). Together, these three factors accounted for 79.97% of the scale variance. Factor score coefficients were estimated using the Regression Method in SPSS. In this method, scores have a mean of zero and variance equal to the squared multiple correlation between the estimated factor scores and the true factor values. This method allows scores to be correlated even when factors are orthogonal.

Withdrawal (detachment, flatness of affect, positive regard reverse-scored, and warmth

Maternal behavioral coding. A team of coders was trained in the coding system as outlined in the *Coding Interactive Behavior Manual–Newborn Version* (Feldman, 1998). Once coders achieved a criterion of kappa = .80 or higher on at least five practice segments, coders were allowed to code play segments from this study. In this system, interactions were continuously coded for mutually exclusive behaviors in each of four categories of maternal behavior: gaze, affect, touch, and vocalization. All coding was

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performed on the Mangold Interact coding system (Mangold, 2010). Within each of these categories, the optimal behavioral code was selected and coded as either being present or absent on a moment-by-moment basis. The optimal code selection within each category was guided by previous factor analytic work by Feldman (Feldman & Eidelman, 2003) and included: gaze to infant's face, positive affect (clear signs of joy and exuberance with a clear smile or laughter), affectionate touch (loving touch with the sole purpose of expressing affection), and motherese (infant-directed speech that is high pitched and often includes repetitions and sing-song vocalizations). There were 66 mother-infant interactions that were coded; in addition to the one video lost due to a technical issue, three infants "fussed out" of the play segment before the end. Of the 66 videos, 14 (21.2%) were randomly chosen to be coded independently to determine inter-rater reliability. Kappas in each coding category were acceptable as follows: gaze ($\kappa = .84$), affect ($\kappa = .79$), touch ($\kappa = .83$), and vocalization ($\kappa = .85$). The relative duration, or the percentage of time the mother spent in the optimal behavior across the entire play interaction, was calculated for each category of maternal behavior. Based on the work of Feldman (2003), we expected the correlation coefficients among these four variables to be positive and large. Correlation coefficients among the parenting variables ranged from -.14 to .46. Therefore, considering the inconsistent pattern of correlation, we analyzed the four coded parenting variables separately in hypothesis testing.

Data Analytic Strategy

Analyses were conducted using SPSS 19.0.0. All mediational and moderated mediational analyses were conducted using the PROCESS macro plug-in for SPSS, which follows statistical procedures as described by Hayes (2012). These procedures are

preferable to the traditional Barron and Kenny (1986) method of testing mediation for several reasons. First, the Hayes method is not limited by the prerequisite of the Barron and Kenny method – that the independent and dependent variable must be correlated in order for the analysis to continue. There are instances where significant mediation exists in the absence of a significant direct association (MacKinnon, Fairchild, & Fritz, 2007), which would go undetected by the Barron and Kenny method. Second, the Hayes method allows for an estimate of the magnitude of the mediated effect (Preacher & Kelley, 2011), whereas the Barron and Kenny method only indicates whether or not there is significant mediation. Finally, and perhaps most importantly, the Baron and Kenny method is generally low-powered (MacKinnon et al., 2007). The Hayes method incorporates bootstrapping methods in order to estimate the standard error of the mediated effect. The result is that, depending on the strength of the direct effect, the Barron and Kenny method requires from 74-397 participants, as compared to 71 participants required for the Hayes method to detect a mediated effect of the same magnitude (Fritz & MacKinnon, 2007).

In order to test hypotheses of moderated mediation, we employed methods described by Hayes (2012). There are several different ways in which a moderator might influence the mediated relationship (Figure 1): 1) by moderating the a' path, 2) by moderating the b' path, or c) by moderating both. In order to test for moderated mediation, interaction terms are created and included in regression models. First, a term was created reflecting the interaction of the independent variable X and the proposed moderator in predicting mediating variable M. Next, a term was created reflecting the interaction of the mediating variable M and the proposed moderator in predicting dependent variable Y. Finally, we pursued significant moderated relationships by estimating the conditional indirect effect of X on Y at values of the moderator (mean and ± 1 SD from the mean) to see if the estimate of the mediated effect differed at different values of the moderator.

Results

Confounding variables

We examined the data for potential confounding variables, that is variables that may have been related to either the independent variable (depression symptoms) or the dependent (parenting) variables. Looking first at variables potentially related to depression symptoms, we found that depression symptoms were not significantly associated with mother age, infant age, infant gestational age at birth, infant gender, maternal breastfeeding status, maternal birth control medication status, or phase of maternal menstrual cycle. However, there were significant associations between maternal depression symptom levels and 1) educational attainment, $r_s = -.33$, p < .01) such that lower educational attainment was associated with higher levels of symptoms of depression; 2) household income, $r_s = -.42$, p < .001, such that lower income was associated with greater depression symptoms; 3) primiparous status, t = -2.36, p < .05, such that women with other children at home reported greater symptoms of depression [infant is only child: M = 4.33, SD = 4.24; >1 child: M = 8.33, SD = 8.51; Cohen's d =0.57]; and 4) race ethnicity, F = 2.71, p < .05, with significant differences between White/Caucasian and Black/African-American participants, such that Black/African-American mothers reported significantly greater symptoms of depression [Black/African-American: M = 10.77, SD = 8.76; White/Caucasian: M = 4.64, SD = 4.12; Cohen's d =1.12]. Thus, educational attainment, household income, primiparousness, and

race/ethnicity were included in a standard set of control variables in all hypothesis testing. Next, we examined associations of potential confounds with the seven parenting variables (four coded variables and three rated variables), finding significant associations between parenting and infant gender on two of the parenting variables, such that mothers tended to be more positive and less negative with their male infants [(Motherese: t = -2.29, p < .05; female infants: M = 68.37, SD = 21.75; male infants: M = 79.07, SD = 15.75; Cohen's d = .56)(Gaze at infant face: t = -2.51, p < .05; female infants: M = 72.71, SD = 13.56; male infants: M = 80.04, SD = 9.91; Cohen's d = .62)]. Therefore, we included infant gender in the standard set of control variables. Analyses were run with and without control variables (Von Elm et al.).

Descriptive Data and Preliminary Analyses

Means and standard deviations for mothers' and infants' measures are presented in Table 1. Examination of the intercorrelations among the study variables (Table 2) revealed few significant associations. Depression symptoms in mothers were significantly associated with one of the seven parenting indices, maternal withdrawal, such that higher levels of reported depression were associated with greater rated maternal withdrawal. Maternal depression symptoms were significantly associated with RSA suppression in response to the stressor, such that greater depression was associated with less suppression, which is conceptualized as less optimal. Depression symptoms were not significantly associated with OT indices. Baseline OT was not associated with any of the parenting indices. However, OT response following the stressor was significantly associated with two of the parenting variables: the percentage of time mothers spent affectionately touching their infants during play and the factor score reflecting rated sensitive responsive parenting, such that a greater increase in OT from baseline was associated with more optimal parenting behaviors. In general, RSA indices were not associated with parenting indices, except for the association between RSA suppression from baseline and the percentage of time mothers spent affectionately touching their infants. Greater suppression in response to a stressor, considered to be optimal, was associated with greater percentage time mothers spent affectionately touching their infants. Finally, the indices which were identified as potential moderators were generally not associated with the primary study indices, with two exceptions: depression symptoms and childhood trauma history were positively associated with a moderate effect size, and greater RSA suppression from baseline (more optimal) was associated with less childhood traumatic exposure.

Hypothesis Testing

Mediation. Next, we tested the primary and exploratory hypotheses that indices of RSA functioning and indices of OT functioning would mediate associations between depression symptoms and parenting behaviors. As can be seen in Tables 3-7 the confidence intervals for the estimates of the mediated effect, obtained through bootstrapping, contained zero, indicating that the estimates were not significantly different from zero. Therefore, mediation was not supported.

Moderated mediation. Next, we tested models of moderated mediation, with the idea that the predicted mediated effect would be larger in magnitude among a subgroup of mothers defined by our two proposed moderator variables: childhood trauma history and infant NA. Tables 8-17 show the analyses of moderated mediation. The statistics shown are for the interaction term entered as the final step in a regression model. As can

be seen in Table 13, there were two moderated relationships, both involving moderation by infant NA. Specifically, the associations between baseline RSA and the percentage of time mothers spent gazing at infants' faces and between baseline RSA and the percentage of time mothers spent in positive affect were moderated by infant NA. However, after controlling for income, maternal race, maternal educational attainment, infant gender, whether there were other children at home, neither interaction term estimate remained statistically significant (gaze, p = .19; positive affect, p = .05). Nonetheless, these interaction terms were probed by estimating the magnitude of the mediated effect at values of the moderator (mean, ± 1 *SD* from the mean). For both outcomes, probing indicated that the mediated effect at all three probed values was not significantly different than zero, and therefore, moderated mediation was not supported.

Discussion

This study sought to test a number of theoretically- and empirically-informed hypotheses examining potential mediators of the association between maternal depression symptoms and parenting behaviors, an association that has been well-established in the literature, and shown in a meta-analytic review (Lovejoy et al., 2000). These hypotheses were informed by empirical literatures establishing relationships among the three study constructs: maternal depression, socially relevant autonomic and hormonal functioning, and maternal parenting behaviors. Specifically, the study tested whether physiological indices of the Social Engagement System (Porges & Carter, 2011), RSA and OT functioning, mediated the association between depression symptoms and parenting in mothers. Further, the study tested hypotheses related to several empirically informed moderators of the mediated effect. We tested these hypotheses in a community sample of mothers with minimal to mild levels of depressive symptoms and their infants in the context of a naturalistic lab interaction and a mild stressor.

Our primary mediation hypotheses were not supported by the data. This was true even for the two indices of parenting that showed at least small associations with depression symptoms. That is, there were not significant mediated effects of baseline RSA, RSA suppression, or baseline OT in associations between maternal depression symptoms and any of the indices of parenting. Similarly, our exploratory hypotheses were not supported in that neither RSA recovery nor OT response to a stressor mediated associations between maternal depression symptoms and any of the parenting indices. Finally, our hypotheses that mediated effects would emerge or be greater in magnitude for particular subgroups within our sample (moderated mediation) were not supported by our data. Therefore, we can conclude that mediated effects were not "washed out" in the sample due to moderation by the hypothesized moderators that we studied. Thus, among a community sample of mothers reporting minimal to mild depression symptoms, these physiological indices did not help to explain associations between their low levels of depression symptoms and any of the indices of parenting behaviors, including the two with at least small degrees of association with depression symptoms.

The primary aims of this study were to contribute to the understanding of mechanisms for the association between maternal depression and dysfunction in parenting behaviors. This study is the first, to our knowledge, to examine OT functioning as a potential mediator for the association and represents an important first step in the examination of the relationship among depression symptoms, OT functioning, and parenting behaviors.

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Our study was also among the first to examine RSA functioning in this context. While data for this study was being collected, a study was published (Musser, Ablow, & Measelle, 2012) that explored baseline RSA in a number of models attempting to explain the association between maternal depression symptoms and maternal sensitivity. Specifically, Musser et al. (2012) tested models of mediation, moderation, and independent effects. They did so in a community sample of mothers interacting with their 5 month-old infants. Similar to our participants, Musser et al. found low levels of depression symptoms within their sample of mothers and a small effect size for the association between depression symptoms and maternal sensitivity. The authors found no support for the hypothesis that baseline RSA mediated the association between maternal depression symptoms and ratings of maternal sensitivity, nor was there support for an interaction between depression symptoms and RSA in predicting maternal sensitivity. Further, there was no significant association between depression symptoms and baseline RSA in the study. However, Musser et al. found that symptoms of depression and baseline RSA independently predicted maternal sensitivity, but when included in the same model, only depression significantly predicted maternal sensitivity.

There are important similarities between the Musser et al. (2012) study and ours that make the studies relatively comparable. Specifically, the studies share in common: minimal to low levels of depression reported among mothers, measurement of depression with a continuous self-report measure of symptoms, measurement of parenting through ratings of a mother-infant interaction paradigm, and collection of quiet, resting baseline RSA in mothers. An important difference in study designs is that our parenting measures were based on a play interaction, while Musser et al. rated parenting following an infant stressor. Bearing this in mind, the Musser et al. study corroborates our finding that baseline RSA did not mediate the association between maternal depression and parenting among women with minimal to low levels of depression. Our study extends the Musser et al. findings by also examining indices of RSA functioning, suppression and recovery in response to a stressor, suggesting that neither static nor dynamic indices of RSA explain associations between low levels of maternal depression and parenting.

Our finding that depression symptoms were not associated with baseline RSA is also corroborated by the Musser et al. (2012) study. This finding represents an important contribution to the literature examining depression symptoms and baseline RSA. Almost exclusively, previous studies have employed group designs comparing participants diagnosed with MDD, that is meeting criteria for a Major Depressive Episode using DSM-IV (American Psychiatric Association, 1994) diagnostic criteria, compared to healthy controls (Rottenberg, 2007). Thus, our finding extends this literature by employing a continuous measure of depression symptoms and testing hypotheses among female participants with low levels of depression. The difference between our findings and those of studies that found significant associations between depression and RSA may be attributed to a number of factors. Most obviously, it may be that differences in baseline RSA are more readily detected in the presence of clinically significant levels of depression. Thus, had our participants reported higher overall levels of depression, with more participants endorsing symptoms in the moderate to severe range, perhaps a significant association between depression symptoms and baseline RSA would have been found. In addition, most studies have tested hypotheses among samples that include both males and females. There is some evidence of sex differences in the relationship between

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depression and baseline RSA. In one study, a significant sex by depression group interaction was found, such that depressed males' baseline RSA was lower than nondepressed males, whereas baseline RSA in depressed females was higher compared to non-depressed females (Thayer, Smith, Rossy, Sollers, & Friedman, 1998). Therefore, it may be that the magnitude of the effect size for the difference in baseline RSA between depressed and non-depressed groups is partially attributable to the proportion of males to females in a sample.

Somewhat contrary to our finding, the Musser et al. study found a statistically significant small to moderate effect ($\beta = -.27$) for higher depression symptoms predicting lower levels of their construct of maternal sensitivity, which was comprised of ratings of maternal warmth, responsiveness, and sensitivity in the context of infant distress. While our study did not find a significant association between depression and most of our indices of positive parenting, we did find a similar, albeit not statistically significant, effect size for the association (r = -.24) between depression symptoms and the percentage of time mothers spent in 'motherese.' Therefore, that this association did not achieve statistical significance in our study but did in the Musser et al. paper is due to differences in power due to sample size (n = 66, critical value r = .24 vs. N = 95; critical value r = .20).

Further, our study extended the findings of Musser et al. (2012) by examining mediators of depressive symptoms associated with withdrawn parenting behaviors, in addition to positive behaviors. We found a moderate effect size for the association between depression symptoms and maternal withdrawal in interactions with infants, although no significant mediation. Future studies might benefit from continuing to consider multiple aspects of parenting in the study of depressed mothers.

Finally, Musser et al. (2012) found a small statistically significant effect (β = .21) for maternal baseline RSA independently predicting maternal sensitivity. Again, although our correlational analyses between baseline RSA and parenting behaviors did not reach statistical significance, there are comparable effect sizes. Specifically, we found that baseline RSA was positively associated with greater active, engaged parenting (*r* = .24), a factor that is comprised of the rating, among others, that captures maternal sensitivity/responsiveness to distress. This is interesting to note because the Musser et al. study captures sensitivity and responsiveness to infants following a stressor. Thus, it may be that baseline RSA is especially relevant to maternal sensitivity and responsiveness to infants in distress.

In addition to the similar effect sizes in the associations between baseline RSA and parenting, our study extends the Musser et al. findings by including indices of dynamic RSA in response to a stressor. While many of our effect sizes between RSA suppression/recovery and parenting were negligible, we found a moderate effect size for the association between greater RSA suppression to a stressor (more optimal) and mothers spending a greater percentage of time affectionately touching their infants. This finding suggests that studies of RSA and parenting should include both static and dynamic RSA indices.

In correlational analyses, our study found effect sizes for the associations between maternal depression symptoms and parenting behaviors primarily to be negligible, with two in the small to moderate range and only one correlation attaining statistical significance. Therefore, the question remains as to why, in our participants, symptoms of depression were largely not significantly associated with indices of parenting and how these findings compare to Lovejoy et al. (2000). First, our effect size for the association between depression symptoms and maternal withdrawal (r = .35) was within the range of study effect sizes reported in the meta-analysis (-.12 - .50). Similarly, the range of effect sizes for associations between depression and positive parenting behaviors (-.24 - .10)was within the range of effect sizes for positive parenting behaviors in the meta-analysis (-.69 — .13). However, we expected larger effect sizes in our study for positive parenting behaviors, given that the mean effect size in studies of infants reported by Lovejoy et al. was r = .23. Closer examination of the individual study characteristics in the Lovejoy et al. meta-analysis is illuminating. Specifically, among studies of mothers interacting with their children under a year old, there was a higher proportion of disadvantaged samples contributing to the mean effect size for depression and positive parenting behaviors. At least 71.7% of participants in these studies were from disadvantaged backgrounds (7.1% were unclassified on SES), defined as living below the poverty level. Importantly, SES was also a moderator of mean study effect size for positive parenting behaviors, such that the association with depression was only statistically significant in studies of disadvantaged mothers and children (r = .21) and negligible in studies of nondisadvantaged samples (r = .03). Therefore, it may be that the proportion of studies of disadvantaged samples in the meta-analysis is driving the association between depression symptoms and positive parenting. This possibility also offers an explanation for the small magnitudes of associations between depression symptoms and positive parenting behaviors among our mostly nondisadvantaged mothers.

Our study makes a contribution to research in the functioning of OT in relation to depression and parenting in that we found no significant association between depression symptoms and baseline OT whereas others report finding significant elevations of OT associated with depression and others still finding significantly decreased OT in relation to depression. As with any nonsignificant association, ours is difficult to interpret. However, given the proliferation of theory papers published in recent years implicating OT in depression (e.g. Ishak, Kahloon, & Fakhry, 2011; Meyer-Lindenberg, Domes, Kirsch, & Heinrichs, 2011; Neumann & Landgraf, 2012), it is surprising that more studies that include OT and depression have not been published. Therefore, it may be that the literature suffers from a "file drawer effect," whereby studies with non-significant findings are not published. Further, given that measurement of OT from saliva is less invasive to participants than measurement of OT from plasma and that the assay used for the measurement of OT in saliva has been validated (Carter et al., 2007), it is surprising that only one published study has taken advantage of this technology to explore potential associations between depression symptoms and salivary OT (Holt-Lunstad, Birmingham, & Light, 2011). Replications of our finding of no significant association between OT and depression will allow for a stronger interpretation of this pattern of findings.

Even despite the error variance introduced during the OT assay process, our examination of dynamic OT functioning in mothers after interacting with their infants represents an important contribution of the study. Because each subjects' two saliva samples were assayed together, it may be that relative within-subject changes in OT are more interpretable than absolute between-subject OT values, an idea that is supported by the lack of significant differences in OT response by batch. Our hypotheses regarding OT change were informed by findings that depressed women were found to have significantly greater variability in OT functioning across a number of paradigms (Cyranowski et al., 2008). Our intention with our study design was to extend the Cyranowski et al. study, in which women participated in a traditional speech stress task, to mothers by including an infant-centered stress task. However, we found no relationship between depression symptoms and change in OT. Instead, we found that change in OT was associated with indices of parenting, such that a greater positive change in OT from baseline to post-interaction was associated with greater percentage of time mothers spent affectionately touching their infants and with greater observer-rated sensitive responsiveness. Both of these effect sizes were moderate in magnitude.

It should be noted that, while our intention was to study the OT response to the stressor in mothers, our study design precludes us from definitively determining that measured changes in OT were due to the stressor and not due to the play interaction with their infants. While we found no published studies of parenting and change in OT in response to a stressor, one study found significant group differences when mothers were dichotomized by percentage of time spent affectionately touching their infants. Those mothers high in affectionate touch demonstrated significant increases in peripheral OT, measured 15 minutes following the interaction with their infants, as compared to those mothers low in affectionate touch (Feldman, Gordon, Schneiderman, et al., 2010). The significant association in our own study between affectionate touch and increase in OT suggests that the OT response may be attributable to the play interaction, rather than to the stressor. However, a study would need to be designed to include no positive contact

attribute change in OT to the stressor. One potential way around the ethical challenges of leaving an infant unconsoled for that period of time would be to have fathers or another of the infants' caregivers accompany mothers to the study to console the baby in another room until a post-stressor saliva sample could be collected from mothers.

Finally, an unexpected finding in our study was the tendency for mothers to be more positive and less withdrawn while interacting with their male infants as compared to the female infants. Although this was true for only two of the seven indices of parenting, for those two parenting variables the effect size was moderate. This was particularly interesting given that two separate meta-analytic studies found no effect for mothers behaving differently with their male or female children when rated on the quality of interaction, warmth, or verbal interaction (Lytton & Romney, 1991) or that, where differences were to be found, mothers tended to be more verbal and use more supportive speech with girls than with boys (Leaper, Anderson, & Sanders, 1998). We offer one possible explanation for the gender differences we found. A recent study found that mothers used significantly longer and more frequent vocalizations when interacting with male infants in distressing situations in which the mother made explicit efforts to control or direct the child's behavior (Ahl, Fausto-Sterling, Garcia-Coll, & Seifer, 2013). In our study, 91% of infants became distressed at some point during the interaction. Infant distress during the interaction was observed to be primarily due to being constrained in the playseat, often accompanied by maternal attempts to restrict the infants' efforts to escape the seat. Therefore, it could be that the observed differences were a reflection of infant distress and differences in the way mothers' attempts to control their girls' and boys' behavior.

Limitations

There are limitations to the current study that must be considered when interpreting the findings. First, the error variance introduced into the OT data during the assay process severely limits the interpretability of the OT findings, and they should be interpreted cautiously. Although we attempted to minimize the influence of this error variance in analyses by including OT batch as a covariate, post-hoc statistical corrections cannot take the place of reliable measurement. In an attempt to maintain fidelity to the body of research generated by the Feldman lab, which has found associations between baseline OT and parenting, we utilized their coding system, the Coding Interactive Behavior Manual–Newborn Version (Feldman, 1998). The considerable error variance in our measurement of OT may itself explain why we failed to replicate published findings. Similarly, the problems with measuring OT may explain our failure to find that either of the indices of OT functioning was significantly associated with the RSA indices, as would be expected by the Social Engagement System model (Porges & Carter, 2011).

The experience of having the reliability of our data compromised, while difficult, was also instructive and illuminating. In our conversations with other hormone researchers after the fact, there seems to be a great deal of variability among labs in their protocols, which was concerning. Also concerning was the fact that few of the published papers on OT and depression or OT and parenting behaviors described whether samples were assayed for OT simultaneously or in batches, and whether there was significant error variance introduced in the process. Notable exceptions include Holt-Lunstad et al. (2010) and Feldman et al. (2007), who reported running all assays together. This level of detailed reporting needs to become the standard. The other major limitation of our study was the relatively small sample size. Our sample of 70 participants was the minimum suggested sample size required to detect a mediated effect in which the effect sizes for both the a' and b' paths (Figure 1) are moderate (.39) in magnitude (Fritz & MacKinnon, 2007). In order to detect a mediated effect in which the effect sizes for both paths are small to moderate in magnitude (.26), the minimum suggested sample size is ~140 participants. Although the effect sizes for literature supported the expectation of moderate effect sizes, the effect sizes for associations in our study were smaller in magnitude. Therefore, it may have been that our study was insufficiently powered to detect mediation. However, the required sample size to detect smaller effects was not feasible within the scope of this project.

Future Directions

Having demonstrated that, even for those significant associations between depression symptoms and parenting behaviors, RSA functioning did not mediate those associations, among mothers with low levels of depression, future studies should test this same hypothesis among a sample of mothers with clinically significant levels of depression symptoms or with diagnosed MDD. While our decision to recruit a community sample of mothers and to measure depression symptoms continuously was well-supported by the literature, the studies finding the most substantial differences in RSA functioning have tested hypotheses among clinically-depressed participants. Therefore, it may be that the hypothesized mediated relationship would be more likely to emerge among more severely depressed mothers than in a community sample of women with low levels of depression symptoms.

Our goal of this study was to examine two physiological systems implicated in both depression and parenting behaviors, OT and RSA, and test those physiological systems as potential mechanisms for the association between depression and parenting. Future research might explore other physiological systems for which emerging findings suggest relevance to both depression and parenting. For example, there is some promise in work examining variants in the OT receptor (OTR) gene in humans. From rodent models comparing maternal behavior in OT null mutant mice and OTR knockout mice receptor, we have learned that the OTR gene is as least as important as circulating levels of OT (Insel, 2010; Takayanagi et al., 2005). There is also evidence that OTR variants play a role in maternal behaviors in humans. For example, in a study of well mothers interacting with their toddlers, significant differences in maternal sensitivity were accounted for by polymorphisms in the OTR gene (Bakermans-Kranenburg & van IJzendoorn, 2008). Similarly, a study of pairs of nulliparous twin females found that those women with the presumably more efficient variant of the OTR gene had more pronounced physiological reactivity to infant cry stimuli, as measured by increase in heart rate (Riem, Pieper, Out, Bakermans-Kranenburg, & van IJzendoorn, 2010). There is also preliminary evidence indicating that variants in the OTR gene may be related to depression. In a study of university students and employees, the same aforementioned risk allele of the OTR gene was associated with significantly higher reports of symptoms of depression (Saphire-Bernstein, Way, Kim, Sherman, & Taylor, 2011).

The case for the OTR gene mediating the association between depression and parenting behaviors is not strong. For example, in the aforementioned twin study (Riem et al., 2010), there were no OTR gene group differences in responses to infant cries in the presence of high depression symptoms, which would seem to indicate that depression symptoms moderate the relationship between OTR gene variants and parenting behaviors. Moreover, the study examining OTR gene group differences in maternal sensitivity (Bakermans-Kranenburg & van IJzendoorn, 2008) found a larger effect after controlling for symptoms of depression, which indicates some overlap in the variance in parenting behaviors explained by OTR gene polymorphisms and depression symptoms. Finally, failure to replicate the findings of many single-nucleotide polymorphism studies in genome wide association studies (Bosker et al., 2010) dictate that these findings be interpreted with extreme caution.

We also have several ideas for ways in which we can further examine the data collected for this study. Similar to Musser et al. (2012), we could statistically explore different types of relationships among our constructs. For example, their findings offer empirical support for testing our proposed mediators (OT/RSA) as moderators of the associations between depression and parenting behaviors, although theoretical justification for such a hypothesis is not clear. Further, we could explore relative contributions of depression symptoms and our physiological indices in models predicting parenting behaviors. Another approach to exploring our data is by looking at the direction of the OT response to the infant interaction, in terms of whether it is increasing or decreasing. In the previously-cited study, mothers high in affectionate touch showed increases in OT following an infant interaction (Feldman, Gordon, Schneiderman, et al., 2010). However, the group of mothers low in affectionate touch showed a mean decrease in OT following the interaction, suggesting that the direction of change may be an important index of OT functioning. In our data, could examine whether relationships

among other indices (depression, RSA, parenting) are different for increasers versus decreasers, with the idea that a decrease in OT might represent the risk condition. Finally, the current study does not account for the individual differences in how the infants responded to their mothers in both the play and stressor paradigms. Therefore, measuring infant behaviors during the interactions might add additional explanatory power to our models of parenting behaviors.

Conclusion

In sum, our study found low levels of depression symptoms in a community sample of mothers and little evidence for these low levels of depression symptoms being associated with parenting behaviors. Even where we found the expected association between depression symptoms and parenting behaviors, we did not find support for this association being mediated by RSA functioning among mothers with low levels of depression symptoms. Further, although there were concerns about the reliability of the OT data, the data suggested that OT functioning similarly did not mediate the association between depression and parenting. In addition, there were several interesting findings that contribute to the understanding of depression, parenting, and the functioning of the Social Engagement System. For example, RSA suppression in response a stressor, associated with both depression symptoms and affectionate touch, may be an important index to consider in future studies of maternal depression and parenting behaviors. Further, OT change following an infant interaction may also be an important correlate of positive parenting behaviors. In community samples of mothers with low levels of depressive symptoms, parenting behaviors are only minimally explained by depression symptoms, suggesting the need to explore additional factors to understand individual differences in parenting behaviors.

References

- Ahl, R. E., Fausto-Sterling, A., Garcia-Coll, C., & Seifer, R. (2013). Gender and discipline in 5—12-month-old infants: A longitudinal study. *Infant Behavior and Development*, 36(2), 199-209. doi: <u>http://dx.doi.org/10.1016/j.infbeh.2013.01.005</u>
- Ainsworth, M. D., Blehar, M. C., Waters, E., & Wall, S. (1978). Patterns of Attachment: A Psychological Study of the Strange Situation. Hillsdale: Lawrence Erlbaum Associates.
- Alison, B. W. F., Ziegler, T. E., Kurian, J. R., Jacoris, S., & Pollak, S. D. (2005). Early experience in humans is associated with changes in neuropeptides critical for regulating social behavior. *Proceedings of the National Academy of Sciences of the United States of America*, 102(47), 17237-17240. doi: 10.1073/pnas.0504767102
- Allen, D. A., Affleck, G., McGrade, B. J., & McQueeney, M. (1984). Effects of singleparent status on mothers and their high-risk infants. *Infant Behavior and Development*, 7(3), 347-359. doi: 10.1016/s0163-6383(84)80049-1
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4 ed.). Washington, DC: Author.
- Anderberg, U. M., & Uvnas-Moberg, K. (2000). Plasma oxytocin levels in female
 fibromyalgia syndrome patients. *Zeitschrift fur Rheumatologi*, *59*(6), 373-379.
 doi: 10.1007/s003930070045
- Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2008). Oxytocin receptor (OXTR) and serotonin transporter (5-HTT) genes associated with observed

parenting. *Social Cognitive and Affective Neuroscience*, *3*(2), 128-134. doi: 10.1093/scan/nsn004

- Banyard, V. L., Williams, L. M., & Siegel, J. A. (2003). The impact of complex trauma and depression on parenting: An xxploration of mediating risk and protective factors. *Child Maltreatment*, 8(4), 334-349. doi: 10.1177/1077559503257106
- Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality & Social Psychology December*, *51*(6), 1173-1182. doi: 10.1037/0022-3514.51.6.1173
- Barratt, M. S., & Roach, M. A. (1995). Early interactive processes: Parenting by adolescent and adult single mothers. *Infant Behavior and Development*, 18(1), 97-109. doi: 10.1016/0163-6383(95)90011-x
- Beauchaine, T. (2001). Vagal tone, development, and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology*, 13(02), 183-214.
- Beck, A. T. (1987). Cognitive models of depression. *Journal of Cognitive Psychotherapy*, *1*(1), 5-37.
- Beck, A. T., Steer, R. A., & Brown, G. K. (1997). Beck Depression Inventory: Second edition. San Antonio: Texas: The Psychological Corporation.
- Benjamin, L. S. (1963). Statistical treatment of the law of initial values (LIV) in autonomic research: A review and recommendation. *Psychosomatic Medicine*, 25(6), 556.

- Bernstein, D. P., & Fink, L. (1998). *Childhood Trauma Questionnaire: A Retrospective Self-Report Manual*. San Antonio, TX: Pearson.
- Bonnar, J., Franklin, M., Nott, P. N., & McNeilly, A. S. (1975). Effect of breast-feeding on pituitary-ovarian function after childbirth. *The British Medical Journal*, 4(5988), 82-84. doi: 10.2307/20407117
- Bornstein, M. H., & Pluess, M. (2000). Child and mother cardiac vagal tone: Continuity, stability, and concordance across the first 5 years. *Developmental Psychology*, 36(1), 54-65. doi: 10.1037/0012-1649.36.1.54
- Bosker, F. J., Hartman, C. A., Nolte, I. M., Prins, B. P., Terpstra, P., Posthuma, D., ...
 Nolen, W. A. (2010). Poor replication of candidate genes for major depressive disorder using genome-wide association data. *Molecular psychiatry*, *16*(5), 516-532. doi: 10.1038/mp.2010.38
- Boyd, R. C., Le, H. N., & Somberg, R. (2005). Review of screening instruments for postpartum depression. Archives of Women's Mental Health, 8, 141-153. doi: 10.1007/s00737-005-0096-6
- Brain-Body Center University of Illinois at Chicago. (2006-2007). CardioBatch (Version 3): Brain-Body Center, University of Illinois at Chicago.
- Braungart-Rieker, J. M., Garwood, M. M., & Stifter, C. A. (1997). Compliance and noncompliance: the roles of maternal control and child temperament. *Journal of Applied Developmental Psychology*, 18(3), 411-428. doi: 10.1016/s0193-3973(97)80008-1

- Braungart-Rieker, J. M., & Stifter, C. A. (1996). Infants' responses to frustrating situations: Continuity and change in reactivity and regulation. *Child Development*, 67(4), 1767-1779. doi: 10.1111/j.1467-8624.1996.tb01826.x
- Brooks-Gunn, J., & Warren, M. P. (1989). Biological and social contributions to negative affect in young adolescent girls. *Child Development*, 60(1), 40-55. doi: 10.2307/1131069
- Campbell, A. (2008). Attachment, aggression and affiliation: The role of oxytocin in female social behavior. *Biological Psychology*, 77(1), 1-10. doi: 10.1016/j.biopsycho.2007.09.001
- Campbell, S. B. (1991). Coding manual for videotaped mother-infant interaction. In NICHD Study of Early Child Care. manuscript.
- Camras, L. A., Oster, H., Campos, J. J., Miyake, K., & Bradshaw, D. (1992). Japanese and American infants' responses to arm restraint. *Developmental Psychology*, 28(4), 578. doi: 10.1037/0012-1649.28.4.578
- Carter, C. S., Grippo, A. J., Pournajafi-Nazarloo, H., Ruscio, M. G., & Porges, S. W.
 (2008). Oxytocin, vasopressin and sociality. *Progress in Brain Research*, 170, 331-336. doi: 10.1016/s0079-6123(08)00427-5
- Carter, C. S., Pournajafi-Nazarloo, H., Kramer, K. M., Ziegler, T. E., White-Traut, R., Bello, D., & Schwertz, D. (2007). Oxytocin: behavioral associations and potential as a salivary biomarker. *Annals of the New York Academy of Sciences, 1098*, 312-322. doi: 10.1196/annals.1384.006

- Clark, L. A., & Watson, D. (1991). The tripartite model of anxiety and depression: Psychometric evidence and taxonomix implications. *Journal of Abnormal Psychology*, 100(3), 316-336. doi: 10.1037/0021-843X.100.3.316
- Clark, R. (1985). The Parent-Child Early Relational Assessment: Instrument and Manual. Madison: University of Wisconsin Medical School, Department of Psychiatry.
- Crockenberg, S., & Acredolo, C. (1983). Infant temperament ratings: A function of infants, of mothers, or both? *Infant Behavior and Development*, 6(1), 61-72. doi: 10.1016/s0163-6383(83)80008-3
- Csiffary, A., Ruttner, Z., Toth, Z., & Palkovits, M. (1992). Oxytocin nerve fibers innervate beta-endorphin neurons in the arcuate nucleus of the rat hypothalamus. *Neuroendocrinology*, 56(3), 429-435.
- Cyranowski, J. M., Hofkens, T. L., Frank, E., Seltman, H., Cai, H. M., & Amico, J. A. (2008). Evidence of dysregulated peripheral oxytocin release among depressed women. *Psychosomatic Medicine*, *70*(9), 967-975. doi: 10.1097/PSY.0b013e318188ade4
- Feldman, R. (1998). Coding Interactive Behavior Manual–Newborn Version Tel Aviv, Israel: Bar-Ilan University Publisher.
- Feldman, R., & Eidelman, A. I. (2003). Direct and indirect effects of breast milk on the neurobehavioral and cognitive development of premature infants. *Developmental Psychobiology*, 43(2), 109-119. doi: 10.1002/dev.10126
- Feldman, R., Gordon, I., Schneiderman, I., Weisman, O., & Zagoory-Sharon, O. (2010). Natural variations in maternal and paternal care are associated with systematic

changes in oxytocin following parent-infant contact. *Psychoneuroendocrinology*, *35*(8), 1133-1141. doi: 10.1016/j.psyneuen.2010.01.013

- Feldman, R., Gordon, I., & Zagoory-Sharon, O. (2010). The cross-generation transmission of oxytocin in humans. *Hormones and Behavior*, 58(4), 669-676. doi: 10.1016/j.yhbeh.2010.06.005
- Feldman, R., Weller, A., Zagoory-Sharon, O., & Levine, A. (2007). Evidence for a neuroendocrinological foundation of human affiliation: plasma oxytocin levels across pregnancy and the postpartum period predict mother-infant bonding. *Psychological Science, 18*(11), 965-970. doi: 10.1111/j.1467-9280.2007.02010.x
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1995). Structured Clinical Interview for DSM-IV Axis I Disorders. Washington, D.C.: American Psychiatric Press.
- Fogel, A. (1993). *Developing through relationships*. . London: Harvester Wheatsheaf and University of Chicago Press.
- Fox, N. A. (1989). Psychophysiological correlates of emotional reactivity during the first year of life. *Developmental Psychology*, 25(3), 364. doi: 10.1037/0012-1649.25.3.364
- Fritz, M. S., & MacKinnon, D. P. (2007). Required sample size to detect the mediated effect. *Psychological Science*, 18(3), 233-239. doi: 10.1111/j.1467-9280.2007.01882.x
- Gartstein, M. A., & Rothbart, M. K. (2003). Studying infant temperament via the revised infant behavior questionnaire. *Infant Behavior & Development, 26*(1), 64-86. doi: 10.1016/S0163-6383(02)00169-8

- Gerdes, A., Hoza, B., Arnold, L., Pelham, W., Swanson, J., Wigal, T., & Jensen, P.
 (2007). Maternal depressive symptomatology and parenting behavior: Exploration of possible mediators. *Journal of Abnormal Child Psychology*, 35(5), 705-714.
 doi: 10.1007/s10802-007-9134-3
- Giardino, J., Gonzalez, A., Steiner, M., & Fleming, A. S. (2008). Effects of motherhood on physiological and subjective responses to infant cries in teenage mothers: A comparison with non-mothers and adult mothers. *Hormones and Behavior*, 53(1), 149-158. doi: 10.1016/j.yhbeh.2007.09.010
- Gimpl, G., & Fahrenholz, F. (2001). The oxytocin receptor system: structure, function, and regulation. *Physiological Reviews*, *81*(2), 629-683.
- Goodyer, I. M., Herbert, J., Altham, P. M. E., Pearson, J., Secher, S. M., & Shiers, H. M. (1996). Adrenal secretion during major depression in 8- to 16-year-olds, I. Altered diurnal rhythms in salivary cortisol and dehydroepiandrosterone (DHEA) at presentation. *Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences, 26*(2), 245-256. doi: 10.1017/s0033291700034644
- Goodyer, I. M., Herbert, J., Tamplin, A., & Altham, P. M. E. (2000). First-episode major depression in adolescents: Affective, cognitive and endocrine characteristics of risk status and predictors of onset. *British Journal of Psychiatry*, *176*(2), 142-149. doi: 10.1192/bjp.176.2.142
- Gordon, I., Zagoory-Sharon, O., Leckman, J. F., & Feldman, R. (2010a). Oxytocin and the development of parenting in humans. *Biological Psychiatry*, 68(4), 377-382. doi: 10.1016/j.biopsych.2010.02.005

- Gordon, I., Zagoory-Sharon, O., Leckman, J. F., & Feldman, R. (2010b). Oxytocin, cortisol, and triadic family interactions. *Physiology & Behavior*. doi: 10.1016/j.physbeh.2010.08.008
- Gottman, J. M., Jacobson, N. S., Rushe, R. H., & Shortt, J. W. (1995). The relationship between heart rate reactivity, emotionally aggressive behavior, and general violence in batterers. *Journal of Family Psychology*, 9(3), 227.
- Granger, D. A., Kivlighan, K. T., Fortunato, C., Harmon, A. G., Hibel, L. C., Schwartz, E. B., & Whembolua, G.-L. (2007). Integration of salivary biomarkers into developmental and behaviorally-oriented research: Problems and solutions for collecting specimens. *Physiology and Behavior, 92*, 583-590. doi: 10.1016/j.physbeh.2007.05.004
- Hanington, L., Ramchandani, P., & Stein, A. (2010). Parental depression and child temperament: Assessing child to parent effects in a longitudinal population study. *Infant Behavior and Development, 33*(1), 88-95. doi:

10.1016/j.infbeh.2009.11.004

- Hayes, A. F. (2012). PROCESS: A versatile computational tool for observed variable mediation, moderation, and conditional process modeling [White paper].
 Retrieved from http://www.afhayes.com/public/process2012.pdf
- Heim, C., Newport, D. J., Mletzko, T., Miller, A. H., & Nemeroff, C. B. (2008). The link between childhood trauma and depression: Insights from HPA axis studies in humans. *Psychoneuroendocrinology*, *33*(6), 693-710. doi: 10.1016/j.psyneuen.2008.03.008

- Heim, C., Young, L. J., Newport, D. J., Mletzko, T., Miller, A. H., & Nemeroff, C. B.
 (2009). Lower CSF oxytocin concentrations in women with a history of childhood abuse. *Molecular Psychiatry*, 14(10), 954-958. doi: 10.1038/mp.2008.112
- Hoge, E. A., Pollack, M. H., Kaufman, R. E., Zak, P. J., & Simon, N. M. (2008).
 Oxytocin levels in social anxiety disorder. *CNS Neuroscience & Therapeutics*, 14(3), 165-170. doi: 10.1111/j.1755-5949.2008.00051.x
- Holt-Lunstad, J., Birmingham, W., & Light, K. C. (2011). The influence of depressive symptomatology and perceived stress on plasma and salivary oxytocin before, during and after a support enhancement intervention. *Psychoneuroendocrinology*, *36*(8), 1249-1256. doi: 10.1016/j.psyneuen.2011.03.007
- Insel, T. R. (2010). The challenge of translation in social neuroscience: A review of oxytocin, vasopressin, and affiliative behavior. *Neuron*, 65(6), 768-779. doi: 10.1016/j.neuron.2010.03.005
- Ishak, W. W., Kahloon, M., & Fakhry, H. (2011). Oxytocin role in enhancing well-being: a literature review. *Journal of affective disorders*, 130(1-2), 1-9. doi: 10.1016/j.jad.2010.06.001
- Katz, L. F., & Gottman, J. M. (1995). Vagal tone protects children from marital conflict. Development and Psychopathology, 7(01), 83-92. doi: 10.1017/S0954579400006350
- Kline, P. (1999). *The handbook of psychological testing* (Second ed.). London: Routledge.
- Landgraf, R., & Neumann, I. D. (2004). Vasopressin and oxytocin release within the brain: a dynamic concept of multiple and variable modes of neuropeptide

```
communication. Frontiers in Neuroendocrinology, 25(3-4), 150-176. doi: 10.1016/j.yfrne.2004.05.001
```

- Landis, J. R., & Koch, G. G. (1977). The measurement of observer agreement for categorical data. *Biometrics*, 159-174.
- Leaper, C., Anderson, K. J., & Sanders, P. (1998). Moderators of gender effects on parents' talk to their children: A meta-analysis. *Developmental Psychology*, 34(1), 3-27. doi: 10.1037/0012-1649.34.1.3
- Leckman, J. F., Goodman, W. K., North, W. G., Chappell, P. B., Price, L. H., Pauls, D.
 L., . . . et al. (1994). Elevated cerebrospinal fluid levels of oxytocin in obsessivecompulsive disorder. Comparison with Tourette's syndrome and healthy controls.
 Archives of General Psychiatry, 51(10), 782-792.
- Light, K. C., Grewen, K. M., & Amico, J. A. (2005). More frequent partner hugs and higher oxytocin levels are linked to lower blood pressure and heart rate in premenopausal women. *Biological Psychology*, 69(1), 5-21. doi: 10.1016/j.biopsycho.2004.11.002
- Light, K. C., Kothandapani, R. V., & Allen, M. T. (1998). Enhanced cardiovascular and catecholamine responses in women with depressive symptoms. *International Journal of Psychophysiology*, 28(2), 157-166. doi: 10.1016/s0167-8760(97)00093-7
- Light, K. C., Smith, T. E., Johns, J. M., Brownley, K. A., Hofheimer, J. A., & Amico, J.
 A. (2000). Oxytocin responsivity in mothers of infants: a preliminary study of relationships with blood pressure during laboratory stress and normal ambulatory activity. *Health Psychology*, 19(6), 560-567.
- Lovejoy, M. C., Graczyk, P. A., O'Hare, E., & Neuman, G. (2000). Maternal depression and parenting behavior: A meta-analytic review. *Clinical Psychology Review*, 20, 561-592. doi: 10.1016/S0272-7358(98)00100-7
- Lytton, H., & Romney, D. M. (1991). Parents' differential socialization of boys and girls:
 A meta-analysis. *Psychological Bulletin*, *109*(2), 267. doi: 10.1037/0033-2909.109.2.267
- Maccoby, E. E., Snow, M. E., & Jacklin, C. N. (1984). Children's dispositions and mother—child interaction at 12 and 18 months: A short-term longitudinal study. *Developmental Psychology*, 20(3), 459-472. doi: 10.1037/0012-1649.20.3.459
- MacDonald, K., & MacDonald, T. M. (2010). The peptide that binds: A systematic review of oxycotin and its prosocial effects in humans. *Harvard Review of Psychiatry*, 18(1), 1-21. doi: 10.3109/10673220903523615
- MacKinnon, D. P., Fairchild, A. J., & Fritz, M. S. (2007). Mediation analysis. *Annual review of psychology*, *58*, 593. doi: 10.1146/annurev.psych.58.110405.085542

Mangold. (2010). INTERACT Quick Start Manual V2.4. Mangold International (Ed.).

- Marazziti, D., Dell'Osso, B., Baroni, S., Mungai, F., Catena, M., Rucci, P., . . . Dell'Osso,
 L. (2006). A relationship between oxytocin and anxiety of romantic attachment. *Clinical Practice and Epidemiology in Mental Health, 2*, 28. doi: 10.1186/1745-0179-2-28
- Meyer-Lindenberg, A., Domes, G., Kirsch, P., & Heinrichs, M. (2011). Oxytocin and vasopressin in the human brain: social neuropeptides for translational medicine. *Nature Reviews Neuroscience*, 12(9), 524-538. doi: 10.1038/nrn3044

- Mills-Koonce, W. R., Gariepy, J.-L., Propper, C., Sutton, K., Calkins, S., Moore, G., & Cox, M. (2007). Infant and parent factors associated with early maternal sensitivity: A caregiver-attachment systems approach. *Infant Behavior and Development*, 30(1), 114-126. doi: 10.1016/j.infbeh.2006.11.010
- Moore, G. A., Hill-Soderlund, A. L., Propper, C. B., Calkins, S. D., Mills-Koonce, W. R., & Cox, M. J. (2009). Mother–infant vagal regulation in the face-to-face still-face paradigm is moderated by maternal sensitivity. *Child Development, 80*(1), 209-223. doi: 10.1111/j.1467-8624.2008.01255.x
- Musser, E. D., Ablow, J. C., & Measelle, J. R. (2012). Predicting maternal sensitivity:
 The roles of postnatal depressive symptoms and parasympathetic dysregulation.
 Infant Mental Health Journal, 33(4), 350-359. doi: 10.1002/imhj.21310
- Neumann, I. D., & Landgraf, R. (2012). Balance of brain oxytocin and vasopressin: implications for anxiety, depression, and social behaviors. *Trends in neurosciences*, 35(11), 649-659. doi: 10.1016/j.tins.2012.08.004
- Parker, K. J., Kenna, H. A., Zeitzer, J. M., Keller, J., Blasey, C. M., Amico, J. A., & Schatzberg, A. F. (2010). Preliminary evidence that plasma oxytocin levels are elevated in major depression. *Psychiatry Research*, 178(2), 359-362. doi: 10.1016/j.psychres.2009.09.017
- Porges, S. W. (1985). Method and apparatus for evaluating rhythmic oscillations in aperiodic physiological response systems. US patent, 4,510,944.
- Porges, S. W. (2001). The polyvagal theory: phylogenetic substrates of a social nervous system. *International Journal of Psychophysiology*, 42(2), 123-146. doi: 10.1016/s0167-8760(01)00162-3

- Porges, S. W. (2007). The polyvagal perspective. *Biological Psychology*, 74(2), 116-143. doi: 10.1016/j.biopsycho.2006.06.009
- Porges, S. W., & Carter, C. S. (2011). Neurobiology and evolution: Mechanisms, mediators, and adaptive consequences of caregiving. In S. L. Brown, R. M.
 Brown & L. A. Penner (Eds.), *Moving beyond self interest: Perspectives from Evolutionary Biology, Neuroscience, and the Social Sciences*: Oxford Press.
- Porges, S. W., Doussard-Roosevelt, J., & Maiti, A. K. (1994). Vagal tone and the physiological regulation of emotion. *Monographs of the Society for Research in Child Development*, 59(2/3), 167-186. doi: 10.1111/j.1540-5834.1994.tb01283.x
- Preacher, K. J., & Kelley, K. (2011). Effect size measures for mediation models: Quantitative strategies for communicating indirect effects. *Psychological Methods*, 16(2), 93. doi: 10.1037/a0022658
- Riem, M., Pieper, S., Out, D., Bakermans-Kranenburg, M., & van IJzendoorn, M. (2010). Oxytocin receptor gene and depressive symptoms associated with physiological reactivity to infant crying. *Social Cognitive and Affective Neuroscience*.
- Rothbart, M. K., & Derryberry, D. (1981). Development of individual differences in temperament. In M. E. Lamb & A. L. Brown (Eds.), *Advances in developmental psychology* (Vol. 1, pp. 37-86). Hillsdale, NJ: Erlbaum.
- Rottenberg, J. (2007). Cardiac vagal control in depression: A critical analysis. *Biological Psychology*, 74(2), 200-211. doi: 10.1016/j.biopsycho.2005.08.010
- Rottenberg, J., Clift, A., Bolden, S., & Salomon, K. (2007). RSA fluctuation in major depressive disorder. *Psychophysiology*, 44(3), 450-458. doi: 10.1111/j.1469-8986.2007.00509.x

- Rottenberg, J., Wilhelm, F. H., Gross, J. J., & Gotlib, I. H. (2003). Vagal rebound during resolution of tearful crying among depressed and nondepressed individuals.
 Psychophysiology, 40(1), 1-6. doi: 10.1111/1469-8986.00001
- Sameroff, A. (1975). Transactional models in early social relations. *Human Development*, *18*(1-2), 65-79. doi: 10.1159/000122384
- Saphire-Bernstein, S., Way, B. M., Kim, H. S., Sherman, D. K., & Taylor, S. E. (2011).
 Oxytocin receptor gene (OXTR) is related to psychological resources.
 Proceedings of the National Academy of Sciences of the United States of America, 108(37), 15118-15122. doi: 10.1073/pnas.1113137108
- Scantamburlo, G., Hansenne, M., Fuchs, S., Pitchot, W., Marechal, P., Pequeux, C., . . .
 Legros, J. J. (2007). Plasma oxytocin levels and anxiety in patients with major depression. *Psychoneuroendocrinology*, *32*(4), 407-410. doi: 10.1016/j.psyneuen.2007.01.009
- Sroufe, L. A., Egeland, B., Carlson, E. A., & Collins, W. A. (2005). The development of the person: The Minnesota study of risk and adaptation from birth to adulthood New York: Guilford.
- Takayanagi, Y., Yoshida, M., Bielsky, I., Ross, H., Kawamata, M., Onaka, T., ... Young, L. (2005). Pervasive social deficits, but normal parturition, in oxytocin receptor-deficient mice. *Proceedings of the National Academy of Sciences of the United States of America*, 102(44), 16096.
- Tamis-LeMonda, C. S., Bornstein, M. H., Baumwell, L., & Damast, A. M. (1996). Responsive parenting in the second year: Specific influences on children's

language and play. *Early Development & Parenting*, *5*(4), 173-183. doi: 10.1002/(SICI)1099-0917(199612)5:4<173::AID-EDP131>3.0.CO;2-V

- Taylor, S. E., Gonzaga, G. C., Klein, L. C., Hu, P., Greendale, G. A., & Seeman, T. E.
 (2006). Relation of oxytocin to psychological stress responses and hypothalamicpituitary-adrenocortical axis activity in older women. *Psychosomatic Medicine*, 68(2), 238-245. doi: 10.1097/01.psy.0000203242.95990.74
- Taylor, S. E., Klein, L. C., Lewis, B. P., Gruenewald, T. L., Gurung, R. A. R., & Updegraff, J. A. (2000). Biobehavioral responses to stress in females: Tend-andbefriend, not fight-or-flight. *Psychological Review*, 107(3), 411-429. doi: 10.1037/0033-295x.107.3.411
- Taylor, S. E., Saphire-Bernstein, S., & Seeman, T. E. (2010). Are plasma oxytocin in women and plasma vasopressin in men biomarkers of distressed pair-bond relationships? *Psychological Science*, *21*(1), 3-7. doi: 10.1177/0956797609356507
- Thayer, J. F., Smith, M., Rossy, L. A., Sollers, J. J., & Friedman, B. H. (1998). Heart period variability and depressive symptoms: gender differences. *Biological Psychiatry*, 44(4), 304-306. doi: <u>http://dx.doi.org/10.1016/S0006-3223(98)00008-</u> <u>0</u>
- Tops, M., van Peer, J. M., Korf, J., Wijers, A. A., & Tucker, D. M. (2007). Anxiety, cortisol, and attachment predict plasma oxytocin. *Psychophysiology*, 44(3), 444-449. doi: 10.1111/j.1469-8986.2007.00510.x

- Turner, R. A., Altemus, M., Enos, T., Cooper, B., & McGuinness, T. (1999). Preliminary research on plasma oxytocin in normal cycling women: investigating emotion and interpersonal distress. *Psychiatry*, 62(2), 97-113.
- Uvnas-Moberg, K. (1996). Neuroendocrinology of the mother--child interaction. *Trends in Endocrinology and Metabolism, 7*(4), 126-131. doi: 10.1016/1043-2760(96)00036-7
- van den Boom, D. C., & Hoeksma, J. B. (1994). The effect of infant irritability on mother-infant interaction: A growth-curve analysis. *Developmental Psychology*, 30(4), 581-590. doi: 10.1037/0012-1649.30.4.581
- Von Elm, E., Altman, D., Egger, M., Pocock, S., Gotzsche, P., & Vandenbroucke, J. (2007). Strengthening the reporting of observational studies in epidemiology (STROBE) statement: guidelines for reporting observational studies. *Lancet, 370*, 1453-1457.
- White-Traut, R., Watanabe, K., Pournajafi-Nazarloo, H., Schwertz, D., Bell, A., & Carter, C. S. (2009). Detection of salivary oxytocin levels in lactating women. *Developmental Psychobiology*, 51(4), 367-373. doi: 10.1002/dev.20376
- Wideman, L., Montgomery, M. M., Levine, B. J., Beynnon, B. D., & Shultz, S. J. (2012).
 Accuracy of calendar-based methods for assigning menstrual cycle phase in women. *Sports Health: A Multidisciplinary Approach*. doi: 10.1177/1941738112469930
- Windle, R. J., Shanks, N., Lightman, S. L., & Ingram, C. D. (1997). Central oxytocin administration reduces stress-induced corticosterone release and anxiety behavior in rats. *Endocrinology*, 138(7), 2829-2834. doi: 10.1210/en.138.7.2829

Yeragani, V. K., Pohl, R., Balon, R., Ramesh, C., Glitz, D., Jung, I., & Sherwood, P. (1991). Heart rate variability in patients with major depression. *Psychiatry Research*, 37(1), 35-46. doi: 10.1016/0165-1781(91)90104-w

Figure 1.

Mediation model



Table 1.

Descriptive Statistics for Mother and Infant Measures

Measure	N	М	SD	Range
Maternal self-report measures				
BDI-II score	70	6.61	7.24	0-43
Number of CTQ maltreatment categories	70	1.2	1.51	0-5
Physiological mediators				
RSA baseline	67	6.23	1.95	3.15 - 14.44
RSA stressor	62	5.95	2.07	3.26 - 13.90
RSA reunion	58	5.63	1.86	3.56 - 14.37
RSA suppression	61	-0.16	1.40	-2.21 - 6.96
RSA recovery	57	-0.08	1.02	-2.71 - 2.86
OT baseline (pg/mL)	70	19.54	9.16	4.74 - 56.15
OT post-stressor (pg/mL)	69	21.76	13.29	5.21 - 94.28
OT change (post-stressor minus	69	2.15	10.17	-13.25 - 63.99
baseline)				
Parenting measures				
% Time in Motherese	66	73.72	19.60	19.98 - 100
% Time in Affectionate Touch	66	21.05	18.97	0-83.09
% Time in Gaze to Face	66	76.37	12.35	22.33 - 91.79
% Time in Positive Affect	66	33.98	17.42	3.61 - 77.42
Active, engaged parenting	69	0.00	1.00	-2.14 - 1.57
Sensitive, responsive parenting	69	0.00	1.00	-4.01 - 1.28
Withdrawal	69	0.00	1.00	-1.20 - 2.86
Infant measures				
IBQ-R Negative Affectivity score	67	4.02	0.41	3.25 - 5.09

Note: BDI-II: Beck Depression Inventory – Second Edition; CTQ: Childhood Trauma Questionnaire; RSA: Respiratory Sinus Arrhythmia; OT: oxytocin; IBQ-R: Infant Behavior Questionnaire – Revised

Mea	sure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.
<u>1</u> .	BDI-II	•	.08	.41**	.08	.03	05	24	16	.10	10	11	05	.35**	.49***	.20
5.	RSA baseline		ı	19	90.	18	.13	.15	02	0~	.11	.24	.16	14	07	.15
ω.	RSA supp.			ı	23	14	.03	0~	30*	.04	.03	.01	07	.14	.27*	27*
4.	RSA recovery				ī	08	.15	24	04	17	02	.01	.01	.13	05	.17
5.	OT baseline ¹					ı	18	.05	07	03	03	05	15	15	.05	.17
6.	OT response ¹						ı	08	.30*	.02	01	02	.28*	05	14	10
7.	% motherese							ı	16	.46***	.41	.51***	11	60***	03	07
8.	% touch								ī	29*	14	10	60 [.]	21	01	08
9.	% gaze									I	.30*	.12	.03	18	08	20
10.	% pos. affect										ı	.35**	.27*	49***	14	09
11.	Active, eng.											ı	.33**	46***	03	.05
12.	Sens. resp.												ı	13	05	13
13.	Withdrawal													ı	.13	09
14.	CTQ # cat.														ı	.12
15.	IBQ-R NA															ı
*	$(.05; *_p < .01; *_p$	> d	.001													

Intercorrelations among depression, physiological mediator, parenting, and moderator variables

¹Partial correlations controlling for assay batch Note: BDI-II, Beck Depression Inventory-II; RSA, respiratory sinus arrhythmia; OT, oxytocin; CTQ, Childhood Trauma Questionnaire, IBQ-R NA, Infant Behavior Questionnaire-Revised Negative Affectivity

Table 2.

Table 3.

Hypothesis 1: Estimates of the mediated effect of baseline respiratory sinus arrhythmia in the association between depression symptoms and parenting behaviors

Parenting behavior	Estimate	SE	LL 95% CI	UL 95% CI
Continuous coded behaviors				
Motherese	.04	.07	02	.26
Affectionate touch	001	.04	09	.09
Gaze at infant's face	001	.04	08	.10
Positive affect	.03	.10	05	.32
Rated behaviors				
Active engaged	.003	.01	002	.02
Sensitive responsive	.002	.003	002	.01
Withdrawn	002	.004	01	.001

Table 4.

Hypothesis 2: Estimates of the mediated effect of respiratory sinus arrhythmia suppression in response to a stressor in the association between depression symptoms

Parenting behavior	Estimate	SE	LL 95% CI	UL 95% CI
Continuous coded behaviors				
Motherese	.18	.16	08	.49
Affectionate touch	24	.19	55	.11
Gaze at infant's face	.02	.09	20	.20
Positive affect	.11	.15	14	.46
Rated behaviors				
Active engaged	.01	.01	01	.02
Sensitive responsive	01	.01	03	.01
Withdrawn	002	.01	01	.01

and parenting behaviors

Table 5.

Hypothesis 3: Estimates of the mediated effect of baseline oxytocin in the association between depression symptoms and parenting behaviors

Parenting behavior	Estimate	SE	LL 95% CI	UL 95% CI
Continuous coded behaviors				
Motherese	.003	.07	10	.19
Affectionate touch	003	.06	16	.09
Gaze at infant's face	001	.05	12	.07
Positive affect	002	.08	10	.24
Rated behaviors				
Active engaged	0002	.002	01	.01
Sensitive responsive	001	.01	01	.01
Withdrawn	001	.01	02	.01

Table 6.

Hypothesis 4: Estimates of the mediated effect of respiratory sinus arrhythmia recovery from a stressor in the association between depression symptoms and parenting behaviors

Parenting behavior	Estimate	SE	LL 95% CI	UL 95% CI
Continuous coded behaviors				
Motherese	03	.09	16	.20
Affectionate touch	004	.04	11	.06
Gaze at infant's face	01	.04	14	.05
Positive affect	001	.04	08	.09
Rated behaviors				
Active engaged	.0002	.003	01	.01
Sensitive responsive	.0002	.001	01	.01
Withdrawn	.001	.003	01	.01

Table 7.

Hypothesis 5: Estimates of the mediated effect of oxytocin change in response to a stressor in the association between depression symptoms and parenting behaviors

Parenting behavior	Estimate	SE	LL 95% CI	UL 95% CI
Continuous coded behaviors				
Motherese	.01	.11	07	.33
Affectionate touch	03	.13	16	.34
Gaze at infant's face	002	.02	04	.05
Positive affect	.002	.05	15	.05
Rated behaviors				
Active engaged	.0001	.004	002	.01
Sensitive responsive	002	.01	02	.01
Withdrawn	.0002	.002	01	.003

Table 8.

Hypothesis 6a: Estimates of moderation by number of CTQ maltreatment categories on the mediated effect of baseline respiratory sinus arrhythmia in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	04	.02	-1.89	.06
b' paths				
Continuous coded behaviors				
Motherese	79	1.07	74	.46
Affectionate touch	1.76	1.15	1.53	.13
Gaze at infant's face	70	.67	-1.05	.30
Positive affect	13	1.14	12	.91
Rated behaviors				
Active engaged	01	.07	10	.92
Sensitive responsive	.04	.07	.55	.58
Withdrawn	.03	.06	.53	.60

Table 9.

Hypothesis 6b: Estimates of moderation by number of CTQ maltreatment categories on the mediated effect of respiratory sinus arrhythmia suppression in response to a stressor in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	.02	.01	1.39	.17
b' paths				
Continuous coded behaviors				
Motherese	.13	1.07	.12	.90
Affectionate touch	1.26	1.04	1.21	.23
Gaze at infant's face	13	.70	19	.85
Positive affect	17	1.17	14	.89
Rated behaviors				
Active engaged	02	.06	29	.77
Sensitive responsive	.01	.07	.14	.89
Withdrawn	08	.06	-1.31	.20

Table 10.

Hypothesis 6c: Estimates of moderation by number of CTQ maltreatment categories on the mediated effect of baseline oxytocin in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	08	.08	-1.04	.30
b' paths				
Continuous coded behaviors				
Motherese	20	.17	-1.23	.22
Affectionate touch	12	.16	77	.44
Gaze at infant's face	.001	.11	.01	.99
Positive affect	.24	.15	1.62	.11
Rated behaviors				
Active engaged	.002	.01	.21	.84
Sensitive responsive	.01	.01	.51	.61
Withdrawn	.01	.01	.65	.52

Table 11.

Hypothesis 6d: Estimates of moderation by number of CTQ maltreatment categories on the mediated effect of respiratory sinus arrhythmia recovery from a stressor in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	.01	.01	.36	.72
b' paths				
Continuous coded behaviors				
Motherese	.40	1.43	.28	.78
Affectionate touch	.23	1.53	.15	.88
Gaze at infant's face	-1.20	.87	-1.38	.18
Positive affect	-1.58	1.52	-1.03	.31
Rated behaviors				
Active engaged	07	.08	80	.43
Sensitive responsive	05	.09	49	.62
Withdrawn	.03	.08	.36	.72

Table 12.

Hypothesis 6e: Estimates of moderation by number of CTQ maltreatment categories on the mediated effect of oxytocin change in response to a stressor in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	16	.11	-1.49	.14
b' paths				
Continuous coded behaviors				
Motherese	04	.21	20	.84
Affectionate touch	14	.19	71	.48
Gaze at infant's face	.03	.14	.26	.80
Positive affect	03	.19	16	.88
Rated behaviors				
Active engaged	01	.01	44	.66
Sensitive responsive	.01	.01	1.13	.26
Withdrawn	01	.01	66	.51

Table 13.

Hypothesis 7a: Estimates of moderation by IBQ-R NA on the mediated effect of baseline respiratory sinus arrhythmia in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	22	.14	-1.53	.13
b' paths				
Continuous coded behaviors				
Motherese	-2.25	2.57	88	.38
Affectionate touch	-3.65	2.73	-1.34	.19
Gaze at infant's face	-3.32	1.45	-2.28	.03
Positive affect	-6.65	3.14	-2.12	.04
Rated behaviors				
Active engaged	.01	.07	.07	.95
Sensitive responsive	.10	.16	.58	.56
Withdrawn	.18	.16	1.11	.27

Table 14.

Hypothesis 7b: Estimates of moderation by IBQ-R NA on the mediated effect of respiratory sinus arrhythmia suppression in response to a stressor in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	05	.07	81	.42
b' paths				
Continuous coded behaviors				
Motherese	22	5.33	04	.97
Affectionate touch	-1.10	8.88	12	.90
Gaze at infant's face	2.87	5.98	.48	.63
Positive affect	3.71	6.92	.54	.59
Rated behaviors				
Active engaged	.16	.69	.23	.82
Sensitive responsive	003	.53	01	1.00
Withdrawn	.02	.32	.07	.94

Table 15.

Hypothesis 7c: Estimates of moderation by IBQ-R NA on the mediated effect of baseline oxytocin in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	.25	.66	.38	.70
b' paths				
Continuous coded behaviors				
Motherese	17	.87	19	.85
Affectionate touch	.45	.93	.48	.63
Gaze at infant's face	.53	.52	1.01	.32
Positive affect	1.15	1.22	.94	.35
Rated behaviors				
Active engaged	.003	.05	.07	.95
Sensitive responsive	01	.06	17	.87
Withdrawn	.01	.04	.12	.90

Table 16.

Hypothesis 7d: Estimates of moderation by IBQ-R NA on the mediated effect of respiratory sinus arrhythmia recovery from a stressor in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	.11	.08	1.44	.16
b' paths				
Continuous coded behaviors				
Motherese	.72	7.03	.10	.92
Affectionate touch	-10.86	6.26	-1.74	.09
Gaze at infant's face	1.29	4.21	.31	.76
Positive affect	4.51	3.88	1.16	.25
Rated behaviors				
Active engaged	.25	.48	.52	.61
Sensitive responsive	03	.42	07	.94
Withdrawn	.18	.31	.57	.57

Table 17.

Hypothesis 7e: Estimates of moderation by IBQ-R NA on the mediated effect of oxytocin change in response to a stressor in the association between depression symptoms and

Parenting behavior	Coefficient	SE	t	р
a' path	-1.24	1.16	-1.07	.29
b' paths				
Continuous coded behaviors				
Motherese	04	.21	20	.84
Affectionate touch	14	.19	71	.48
Gaze at infant's face	.03	.14	.26	.80
Positive affect	03	.19	16	.88
Rated behaviors				
Active engaged	01	.01	44	.66
Sensitive responsive	.01	.01	1.13	.26
Withdrawn	01	.01	66	.51

parenting behaviors

Table 3.

Hypothesis 1: Estimates of the mediated effect of baseline respiratory sinus arrhythmia in the association between depression symptoms and parenting behaviors

Parenting behavior	Estimate	SE	LL 95% CI	UL 95% CI
Continuous coded behaviors				
Motherese	.04	.07	02	.26
Affectionate touch	001	.04	09	.09
Gaze at infant's face	001	.04	08	.10
Positive affect	.03	.10	05	.32
Rated behaviors				
Active engaged	.003	.01	002	.02
Sensitive responsive	.002	.003	002	.01
Withdrawn	002	.004	01	.001

Table 4.

Hypothesis 2: Estimates of the mediated effect of respiratory sinus arrhythmia suppression in response to a stressor in the association between depression symptoms

Parenting behavior	Estimate	SE	LL 95% CI	UL 95% CI
Continuous coded behaviors				
Motherese	.18	.16	08	.49
Affectionate touch	24	.19	55	.11
Gaze at infant's face	.02	.09	20	.20
Positive affect	.11	.15	14	.46
Rated behaviors				
Active engaged	.01	.01	01	.02
Sensitive responsive	01	.01	03	.01
Withdrawn	002	.01	01	.01

and parenting behaviors

Table 5.

Hypothesis 3: Estimates of the mediated effect of baseline oxytocin in the association between depression symptoms and parenting behaviors

Parenting behavior	Estimate	SE	LL 95% CI	UL 95% CI
Continuous coded behaviors				
Motherese	.003	.07	10	.19
Affectionate touch	003	.06	16	.09
Gaze at infant's face	001	.05	12	.07
Positive affect	002	.08	10	.24
Rated behaviors				
Active engaged	0002	.002	01	.01
Sensitive responsive	001	.01	01	.01
Withdrawn	001	.01	02	.01

Table 6.

Hypothesis 4: Estimates of the mediated effect of respiratory sinus arrhythmia recovery from a stressor in the association between depression symptoms and parenting behaviors

Parenting behavior	Estimate	SE	LL 95% CI	UL 95% CI
Continuous coded behaviors				
Motherese	03	.09	16	.20
Affectionate touch	004	.04	11	.06
Gaze at infant's face	01	.04	14	.05
Positive affect	001	.04	08	.09
Rated behaviors				
Active engaged	.0002	.003	01	.01
Sensitive responsive	.0002	.001	01	.01
Withdrawn	.001	.003	01	.01

Table 7.

Hypothesis 5: Estimates of the mediated effect of oxytocin change in response to a stressor in the association between depression symptoms and parenting behaviors

Parenting behavior	Estimate	SE	LL 95% CI	UL 95% CI
Continuous coded behaviors				
Motherese	.01	.11	07	.33
Affectionate touch	03	.13	16	.34
Gaze at infant's face	002	.02	04	.05
Positive affect	.002	.05	15	.05
Rated behaviors				
Active engaged	.0001	.004	002	.01
Sensitive responsive	002	.01	02	.01
Withdrawn	.0002	.002	01	.003

Table 8.

Hypothesis 6a: Estimates of moderation by number of CTQ maltreatment categories on the mediated effect of baseline respiratory sinus arrhythmia in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	04	.02	-1.89	.06
b' paths				
Continuous coded behaviors				
Motherese	79	1.07	74	.46
Affectionate touch	1.76	1.15	1.53	.13
Gaze at infant's face	70	.67	-1.05	.30
Positive affect	13	1.14	12	.91
Rated behaviors				
Active engaged	01	.07	10	.92
Sensitive responsive	.04	.07	.55	.58
Withdrawn	.03	.06	.53	.60

Table 9.

Hypothesis 6b: Estimates of moderation by number of CTQ maltreatment categories on the mediated effect of respiratory sinus arrhythmia suppression in response to a stressor in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	.02	.01	1.39	.17
b' paths				
Continuous coded behaviors				
Motherese	.13	1.07	.12	.90
Affectionate touch	1.26	1.04	1.21	.23
Gaze at infant's face	13	.70	19	.85
Positive affect	17	1.17	14	.89
Rated behaviors				
Active engaged	02	.06	29	.77
Sensitive responsive	.01	.07	.14	.89
Withdrawn	08	.06	-1.31	.20

Table 10.

Hypothesis 6c: Estimates of moderation by number of CTQ maltreatment categories on the mediated effect of baseline oxytocin in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	08	.08	-1.04	.30
b' paths				
Continuous coded behaviors				
Motherese	20	.17	-1.23	.22
Affectionate touch	12	.16	77	.44
Gaze at infant's face	.001	.11	.01	.99
Positive affect	.24	.15	1.62	.11
Rated behaviors				
Active engaged	.002	.01	.21	.84
Sensitive responsive	.01	.01	.51	.61
Withdrawn	.01	.01	.65	.52

Table 11.

Hypothesis 6d: Estimates of moderation by number of CTQ maltreatment categories on the mediated effect of respiratory sinus arrhythmia recovery from a stressor in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	.01	.01	.36	.72
b' paths				
Continuous coded behaviors				
Motherese	.40	1.43	.28	.78
Affectionate touch	.23	1.53	.15	.88
Gaze at infant's face	-1.20	.87	-1.38	.18
Positive affect	-1.58	1.52	-1.03	.31
Rated behaviors				
Active engaged	07	.08	80	.43
Sensitive responsive	05	.09	49	.62
Withdrawn	.03	.08	.36	.72

Table 12.

Hypothesis 6e: Estimates of moderation by number of CTQ maltreatment categories on the mediated effect of oxytocin change in response to a stressor in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	16	.11	-1.49	.14
b' paths				
Continuous coded behaviors				
Motherese	04	.21	20	.84
Affectionate touch	14	.19	71	.48
Gaze at infant's face	.03	.14	.26	.80
Positive affect	03	.19	16	.88
Rated behaviors				
Active engaged	01	.01	44	.66
Sensitive responsive	.01	.01	1.13	.26
Withdrawn	01	.01	66	.51

Table 13.

Hypothesis 7a: Estimates of moderation by IBQ-R NA on the mediated effect of baseline respiratory sinus arrhythmia in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	22	.14	-1.53	.13
b' paths				
Continuous coded behaviors				
Motherese	-2.25	2.57	88	.38
Affectionate touch	-3.65	2.73	-1.34	.19
Gaze at infant's face	-3.32	1.45	-2.28	.03
Positive affect	-6.65	3.14	-2.12	.04
Rated behaviors				
Active engaged	.01	.07	.07	.95
Sensitive responsive	.10	.16	.58	.56
Withdrawn	.18	.16	1.11	.27
Table 14.

Hypothesis 7b: Estimates of moderation by IBQ-R NA on the mediated effect of respiratory sinus arrhythmia suppression in response to a stressor in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	05	.07	81	.42
b' paths				
Continuous coded behaviors				
Motherese	22	5.33	04	.97
Affectionate touch	-1.10	8.88	12	.90
Gaze at infant's face	2.87	5.98	.48	.63
Positive affect	3.71	6.92	.54	.59
Rated behaviors				
Active engaged	.16	.69	.23	.82
Sensitive responsive	003	.53	01	1.00
Withdrawn	.02	.32	.07	.94

Note: Estimate based on bootstrapping with 10,000 samples using the Preacher & Hayes PROCESS macro for SPSS 20.0. This is unadjusted data.

Table 15.

Hypothesis 7c: Estimates of moderation by IBQ-R NA on the mediated effect of baseline oxytocin in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	.25	.66	.38	.70
b' paths				
Continuous coded behaviors				
Motherese	17	.87	19	.85
Affectionate touch	.45	.93	.48	.63
Gaze at infant's face	.53	.52	1.01	.32
Positive affect	1.15	1.22	.94	.35
Rated behaviors				
Active engaged	.003	.05	.07	.95
Sensitive responsive	01	.06	17	.87
Withdrawn	.01	.04	.12	.90

Note: Estimate based on bootstrapping with 10,000 samples using the Preacher & Hayes PROCESS macro for SPSS 20.0. Estimates are adjusted for OT assay batch.

Table 16.

Hypothesis 7d: Estimates of moderation by IBQ-R NA on the mediated effect of respiratory sinus arrhythmia recovery from a stressor in the association between depression symptoms and parenting behaviors

Parenting behavior	Coefficient	SE	t	р
a' path	.11	.08	1.44	.16
b' paths				
Continuous coded behaviors				
Motherese	.72	7.03	.10	.92
Affectionate touch	-10.86	6.26	-1.74	.09
Gaze at infant's face	1.29	4.21	.31	.76
Positive affect	4.51	3.88	1.16	.25
Rated behaviors				
Active engaged	.25	.48	.52	.61
Sensitive responsive	03	.42	07	.94
Withdrawn	.18	.31	.57	.57

Note: Estimate based on bootstrapping with 10,000 samples using the Preacher & Hayes PROCESS macro for SPSS 20.0. This is unadjusted data.

Table 17.

Hypothesis 7e: Estimates of moderation by IBQ-R NA on the mediated effect of oxytocin change in response to a stressor in the association between depression symptoms and

Parenting behavior	Coefficient	SE	t	р
a' path	-1.24	1.16	-1.07	.29
b' paths				
Continuous coded behaviors				
Motherese	04	.21	20	.84
Affectionate touch	14	.19	71	.48
Gaze at infant's face	.03	.14	.26	.80
Positive affect	03	.19	16	.88
Rated behaviors				
Active engaged	01	.01	44	.66
Sensitive responsive	.01	.01	1.13	.26
Withdrawn	01	.01	66	.51

parenting behaviors

Note: Estimate based on bootstrapping with 10,000 samples using the Preacher & Hayes PROCESS macro for SPSS 20.0. Estimates are adjusted for OT assay batch.