The relation between maternal depression and the infant stress response system: The role of positive parenting

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Abstract

Objective To examine the effect of the parenting style of the mother on the relation between maternal depression and the infant's stress response system. **Method** The sample consisted of 55 mothers and their infants. Maternal depressive symptoms were assessed during pregnancy and when the infant was six months old, by means of the Beck Depression Inventory-II. Maternal parenting style was observed from mother-infant interaction during the Still Face Paradigm, when the infant was six months old. When the infant was 12 months old, a stress-paradigm was conducted during which both physiological and behavioral measures of infant stress reactivity were obtained. **Results** Maternal postnatal depressive symptoms were found to be negatively related to maternal sensitivity. In addition, maternal prenatal as well as postnatal depressive symptoms were found to predict infant baseline heart rate, infant stress reactivity and infant heart rate recovery, but only in interaction with certain characteristics of the mother's parenting style. **Conclusions** The present study underlines that the way a mother interacts with her child is very important for the development of the child's neurobiological system and that her parenting style can either buffer or enhance the effect of maternal psychopathology. Teaching mothers at risk how to interact positively and adequately with their child and how to respond to its needs, could possibly decrease the deleterious effects of maternal depression on child development.

Keywords: maternal depression, maternal psychopathology, maternal sensitivity, intrusiveness, infant stress reactivity, Still Face Paradigm, stress paradigm, HPA-axis, heart rate

Introduction

Many studies have shown that maternal depression during the postpartum period influences the ability of a mother to care for her child (e.g. Burt & Stein, 2002; Seretti, 2006). In addition, maternal depression during the postnatal as well as the prenatal period has been found to be associated with poor child outcomes (Field et al., 2004; Field, 2011). More specifically, longitudinal research has shown that the offspring of depressed mothers tend to show poor outcomes across childhood and adolescence, such as impaired cognitive, neuropsychological, social, and emotional skills (e.g. Beck, 1998; Cichetti & Toth, 1998; Goodman & Gotlib, 1999; Murray, Woolgar, Cooper, & Hipwell, 2001; Weinberg & Tronick, 1998), as well as higher rates of medical problems and early mortality (Weissman et al., 2006). Previous studies suggest that deficits in emotion regulation and affective synchrony in children of depressed mothers are already present from infancy onwards (Tronick & Reck, 2009). Moreover, studies have shown that maternal depression is associated with physiological dysregulation of the child, especially reflected in impairments of the stress response system (e.g. Kaufman & Ryan, 1999). Dysregulation of the HPA-axis, a very important stress system of the body, has been suggested to be one of the mechanisms explaining intergenerational transmission of risk for negative outcomes such as psychopathology, from depressed parents to their children (Dougherty, Tolep, Smith, & Rose, 2013; Goodman & Gotlib, 1999). In general, deviating (especially heightened) HPA-axis activity has been associated with processes linking stress to illness (e.g. Holsboer, 2000). Because continuity has been found of depression, from mother to child and again from child to adolescent and adult (e.g. Fombonne, Wostear, Cooper, Harrington, & Rutter, 2001; Kaitz, Maytal, Devor, Bergman, & Mankuta, 2010), studying the effect of maternal depression on child development at an early stage is crucial in order to develop effective prevention and intervention programs for children at risk. Examining behavioral as well as neuroendocrine dysfunctions in children of mothers with depressive symptoms contributes to our insight in trajectories and mechanisms underlying the intergenerational transmission of risk for negative outcomes later in life.

Maternal depression and parenting

Parenting styles have been found to have an early and powerful influence on the child's social, emotional and cognitive development. With respect to depression, a meta-analysis of Gaynes et al. (2005) found that based on the symptoms, between 8.5% and 11% of the women would be diagnosed with a clinical prenatal depression, while 6.5% to over 12% would be diagnosed in the postpartum period. Moreover, a subgroup was detected with a depression during the postpartum period with symptom onset in the prenatal period, causing their children to be exposed to depression both in the pre- and postnatal period (Stowe, Hostetter, & Newport, 2005). In addition, up to 50% of women in low socioeconomic status (SES) households have been found to report depressive symptoms, often without being diagnosed or treated (Leadbeater & Linares, 1992). In light of explaining how maternal depression affects infant development, depressed mothers have been found to provide a less caring and stimulating environment for their children (Goodman & Gotlib, 1999). More specifically, depressed mothers have been found to be less sensitive in interaction with their children and to show less affective expression (Cicchetti & Toth, 1998; Feldman, 2007). Maternal sensitivity, or the way in which a mother perceives the signals of her child and responds to them promptly and adequately (Ainsworth, Bell, & Stayton, 1971; 1974) is highly important for infants in order to learn how to regulate their emotions and feelings of stress, and also for social behavior, causing reduced sensitivity of depressed mothers to be an additional risk factor for emotional dysregulation of their children (Feldman et al., 2009). Depressed mothers have been found to show less interaction with their children, to show less warmth and affection, and to show more negative and hostile parenting (Feldman et al., 2009; Lovejoy, Graczyk, O'Hare, & Neuman, 2000). Compared to for example anxious mothers, who have been found to show more intrusive behavior in interaction with their children, depressed mothers have been found to be unavailable to their children while showing a withdrawn and flattened affect (Feldman et al., 2009). In the study of Feldman and colleagues (2009), both anxious and depressed mothers showed lower sensitivity than controls, with depressed mothers showing the lowest sensitivity, while anxious mothers showed high

intrusiveness compared to depressed mothers and controls. Intrusiveness, as well as unavailability, can both be considered insensitive maternal styles that might negatively influence the social-emotional development ofchildren.

Development of the stress response system

In the second half of the infant's first year of life, a developmental transition with respect to emotion regulation takes place, in which for example intentional action emerges, including the upcoming ability to control stressful situations (e.g. Calkins & Fox, 2002; Tronick, 1989). During this period, the infant's emotion regulation skills increase, including turning away from a stressor, manipulating objects and exploring unfamiliar situations, all strategies to actively regulate distress, increasingly replacing more infantile strategies in which the caregiver is alerted through for example fussing or crying (Feldman et al., 2009). With respect to maternal depression, the literature has suggested that the mother's impaired affect regulation disturbs her provision of a regulatory framework to her child, in turn withholding the child to shift from the infantile strategies to more active forms of coping and regulation (Gianino & Tronick, 1986).

Mechanisms of fear-regulation

Individuals respond differently to stressful situations, such as unfamiliar situations or other situations that can be considered frightening. Fearful individuals tend to inhibit their behavior and responses, avoid stressful situations and risks en tend to get physiologically aroused in stressful or unfamiliar situations (Hirshfield, Biederman, Brody, Faraone, & Rosenbaum, 1997; Hirshfield-Becker et al., 2003). Studies have shown that fearfulness at a young age is associated with internalizing symptoms in adolescence and adulthood, such as anxiety and depression, through the mechanism of behavioral inhibition (Biederman et al., 1993; Schwartz, Snidman, & Kagan, 1999). In contrast, fearless individuals tend to disinhibit their behavior, by showing approach to stressful and unfamiliar situations (Hirshfield-Becker et al., 2003). Fearless individuals have been found to more often show externalizing symptoms (Colder, Mott, & Berman, 2002), for example by being more engaged in

antisocial behavior such as aggression (Raine, 1993). In short, both extremes of mechanisms of fear are associated with the development of psychopathology and other adversities later in life.

The development of temperament

It is important to study the development of children's temperament already at a very young age, as temperament is an underlying factor that fosters the social-emotional functioning of children later in life (Blandon, Calkins, Keane, & O'Brien, 2010). Although not yet quite clearly, stability of temperament from infancy onwards has been proven in some studies. For example, Casalin, Luyten, Vliegen, & Meurs (2012) found stability of different dimensions of temperament from infancy into toddlerhood, while Komsi and colleagues (2006) even found stability of temperament into middle childhood. However, physiological indicators of temperament have been found to be less stable in infancy. For example, in a study of Baker and colleagues (2012), it was found that the behavioral measures of temperament (i.e. fear) remained stable from the first until the third year of the children, while the physiological measures were not stable over time, as both heart rate and skin conductance decreased from the first to the third year (Baker et al., 2012). Moreover, other studies claim that the development of fear in general is only stable from 14 months (Kochanska, 2001) or even from 24 months onwards (Lemery, Goldsmith, Klinnert, & Mrazek, 1999). Taken together, more insight is needed regarding the stability of temperament and its components.

Assessing the stress response system in infants

When studying temperamental concepts in infants, such as emotion regulation or stress reactivity, it is important to include physiological measures as well as behavioral measures, as they are sensitive to changes in psychological state and less sensitive to human interpretation (Campos, 1976). Both measures are needed to retain an as complete and reliable representation as possible (Lewis & Ramsay, 1999). In many studies measures of heart rate (HR) and skin conductance level (SCL) have already been used in infants (e.g. Baker et al., 2012; Fracasso, Porges, Lamb, & Rosenberg, 1994; Hernes et al., 2002) and have been found to be reliable, sensitive and valid physiological

indicators of temperament (e.g. Campos, 1976; Nigg, 2006). However, stress-induced psychophysiological assessments have not often been performed with infants (i.e., Laboratory Temperament Assessment Battery (Lab-TAB); Goldsmith & Rothbart, 1999), although they have been used with older children more often (e.g. Calkins & Dedmon, 2000; Kagan, Reznnick & Snidman, 1987).

In order to increase the understanding of risk pathways and their development, physiology has been included in the field of developmental psychopathology (Cicchetti & Gunnar, 2008). Physiological dysregulation is often detected in children who show internalizing as well as externalizing problem behavior (Baker et al., 2012). For example, high resting HR, low HR variability and high cortisol levels in children have been found to predict more severe fear and inhibition (Kagan, 1997) and more behavioral inhibition in the long run (Kagan, Reznick, Clarke, Snidman, & Garcia-Coll, 1984), while low resting HR, low baseline SCL and low cortisol levels have been associated with externalizing problem behavior (Scarpa, Raine, Venables, & Mednick, 1997; Snoek, van Goozen, Matthys, Buitelaar, & van Engeland, 2004; Van Goozen, Matthys, Cohen-Kettenis, Buitelaar, & van Engeland, 2000). However, less is known about physiological reactivity in response to a stressor in infants, although it has been suggested that physiological reactivity at this very young age also identifies individuals who are at risk for developing externalizing or internalizing problems (Baker et al., 2012).

Maternal depression and the stress response system of the child: Behavior and physiology

Studies have shown that maternal depression influences the stress reactivity and emotion regulation of the infant, particularly of negative emotions such as fear and novelty (Murray & Cooper, 1997). For example, in the study of Feldman et al. (2009), where a fear-paradigm with frightening masks was used with 9-month-olds, infants of mothers with a current major depressive disorder (MDD) cried more often and were more fussy compared to children of anxious mothers and controls. In addition, infants from depressed mothers showed less regulatory behavior during the

paradigm than controls (Feldman et al., 2009). Additional studies examining behavioral stress reactivity in infants of depressed mothers by means of a stress-paradigm are lacking. However, studies have shown that maternal depression influences the child's physiological response to stress. Many studies suggest that early experience such as parental stress or psychopathology, prenatally as well as postnatally, may affect neurobiological functioning such as the stress response system, both immediately as well as in the long term (e.g. Francis, Diorio, Liu, & Meaney, 1999; Talge, Neal, & Glover, 2007). For example, animal studies have shown that animal rearing conditions such as maternal deprivation or reduced skin contact between mother and her young influence the functioning of the HPA-axis (Champagne, 2008; Levine, 2005). With respect to humans, deviating diurnal cortisol rhythms, as well as high baseline cortisol levels and increased cortisol reactivity have been found in children of depressed mothers (Brennan et al., 2008; Field & Diego, 2008; Goodyer, Park, & Herbert, 2001). Moreover, those deviating patterns have been found to remain present at least up to adolescence, indicating that maternal depression can lead to long-term disturbances of the stress response system. Feldman and colleagues (2009) found a relation between current maternal depression and cortisol reactivity of 9-month old infants, with infants of depressed mothers showing heightened stress reactivity compared to controls. In addition, the cortisol peak of children of depressed mothers remained high after the paradigm, indicating that the stress system of those infants took more time to recover compared to controls and children of anxious mothers. In addition, children of mothers who have been depressed prenatally have also been found to show greater right frontal EEG activity (Jones, Field, Fox, Luncy, & Davalos, 1997) and lower vagal tone, a marker of activity of the parasympathetic nervous system (Field, Pickens, Fox, Nawrocki, & Gonzalez, 1995). Where a higher vagal tone is typically indicative of more adaptive functioning, a lower vagal tone has been found to correlate with other physiological adversities such as high cortisol levels, but also with behavioral adversities such as less interest and less positive expressiveness of the infants (e.g. Field et al., 1995). In contrast, in a study of Fernald, Burke and Gunnar (2008), a hyporesponse to a stressor was found in toddlers of depressed mothers with a low SES, in form of a blunted cortisol

response, which could possibly be explained by downregulation of the HPA-axis as a consequence of chronic elevations in cortisol earlier in the children's life due to exposure to chronic economic and social stress (Fries, Hesse, Hellhammer, & Hellhammer, 2005; Gunnar & Vazquez, 2001).

Taken together, the stress response system (i.e. HPA-axis) of children of depressed mothers seems to function differently, in a way that those children experience more stress and are less able to regulate those feelings of stress in a healthy way (e.g. Field et al., 1995). The literature suggests that offspring of depressed mothers shows dysregulation of the stress response system reflected in the HPA-axis, which may contribute to the emergence of psychopathology later in life. In general, findings of prenatal influence of parental psychopathology support the prenatal programming hypothesis (Barker, 1995), suggesting that the in utero environment, such as the mother's HPA-axis activity, influences the development of the fetal brain, leading to possibly maladaptive behavioral and neuroendocrinological pathways during the child's development (Wadhwa, 2005; O'Connor, Heron, Golding, & Glover, 2003; Welberg & Seckl, 2001). In addition, postpartum maternal depression can also contribute to early adversities in the child's life, such as negative mother-child interactions and exposure to stressors in general. Prenatal influences as well as early experience can both be considered biological mechanisms involved in the transmission of intergenerational risk of psychopathology. However, research involving young infants of depressed mothers is sparse, with respect to behavioral as well as physiological measures.

Maternal depression and child stress reactivity: The influence of parenting

Research has shown that characteristics of the environment of the child might moderate the relation between parental adversity and the child's neuroendocrine functioning (Luecken & Appelhans, 2006). However, due to possible confounding effects, it is not yet clear whether familial vulnerability, exposure to a mother with internalizing problems and her parenting, or a combination of those factors lead to neuroendocrine abnormalities in children. In line with the diathesis-stress model in which individuals are thought to be differentially susceptible to their environmental

experiences (Zuckerman, 1999), interactions between a familial vulnerability and maternal style possibly predict different patterns of stress responses in children, both behaviorally and physiologically. For example, in a study of Kaplan, Evans and Monk (2008) neither a prenatal diagnosis of maternal depression nor maternal sensitivity alone predicted the infant baseline cortisol level at 4 months, but their interaction was significant. More specifically, higher cortisol levels were found in infants of women with a prenatal diagnosis, but only when mothers showed less sensitive parenting (Kaplan, Evans, & Monk, 2008). In addition, because maternal depression has been found to strongly influence parenting, it might be plausible that parenting would explain part of the effect of maternal depression on child characteristics. Because research examining the influences of both pre- and postnatal maternal depression on the infant stress response system is sparse and because the exact role of parenting on this relationship is not yet clear, the present study aims to examine the effect of the parenting style of the mother on the relation between maternal depression and the infant's stress response system.

The current study

The current study focuses on maternal depressive symptoms, both prenatally and postnatally, and their possible influence on the infant's stress response system. The stress reactivity of the child, both behaviorally and physiologically, is studied when the child is 12 months old, by means of a stress-paradigm. Based on the majority of the literature, it is hypothesized that infants of mothers who report more depressive symptoms show enhanced stress reactivity during the paradigm, both behaviorally and physiologically. Moreover, infants of mothers with more depressive symptoms are expected to show a higher baseline amount of stress and to recover less quickly after the paradigm. In addition, the current study aims to examine the role of the maternal style with respect to the hypothesized relationship between maternal depressive symptoms and infant stress reactivity, where maternal style will be observed during mother-child interaction when the child is 6 months old. It is hypothesized, that mothers who report more depressive symptoms, will interact

more negatively with their child, for example by showing more negative affect or responding less sensitively to their child. If the previously described relations are found, it is hypothesized that the maternal style towards the child mediates the effect of maternal depressive symptoms on infant stress reactivity, in a way that higher maternal depressive symptoms predict higher reactivity of the child, partly or fully explained by a more negative maternal style. Alternatively, the maternal style could also moderate the effect, in a way that the interaction between maternal depressive symptoms and maternal style would alter the infant's stress-response. In that case, it is hypothesized that a positive maternal style, i.e. sensitivity, would buffer the negative effect of maternal depressive symptoms on the infant's stress response system and that a negative maternal style, i.e. intrusiveness, would augment this negative effect. Finally, prenatal and postnatal depressive symptoms are examined independently, but no specific hypotheses are stated with respect to the timing of the symptoms.

Methods

Background Information "A Good Start"

The mothers and infants included in the present study participate in a large longitudinal study conducted in The Netherlands, called "A Good Start", in which mothers are followed from pregnancy onwards until the infant is 2,5 years old. At 27 weeks of pregnancy mothers are interviewed and have to fill out questionnaires on many domains. Subsequently, infants are assessed at 6 and 20 months of age during a home visit, and at 12 and 30 months of age during a laboratory visit. Based on an intake-interview during pregnancy, mothers are either assigned to the control group, or to the high-risk group where they again are randomly assigned to an intervention-group which receives individual coaching until the infants are 2,5 years old, or to the group without intervention (only care-as-usual). In order to be assigned to the high-risk group, mothers have to be deemed "lacking in self-sufficiency", which is operationalized as presenting with minimally one of the following risk factors: Unemployment, financial problems, housing problems, restricted or

inadequate social support, single motherhood, psychopathology (e.g. depression- and anxiety complaints, borderline symptoms, or aggression), or drug (ab)use (e.g. smoking, alcohol or drugs). Mothers are recruited from all parts of the Netherlands by advertisements and recruitment at several health care facilities and pregnancy- and parenthood-fairs. Mothers are included in the study when they are between 17 and 25 years of age during pregnancy, when they are pregnant of their first infant and when they are sufficiently fluent in the Dutch language. Mothers are excluded in case of severe drug addiction, severe psychiatric problems or an IQ < 70 (estimated during the first appointment), and when severe medical complications of either mother or infantwould render study participation too difficult or impossible. “A Good Start” was approved bythe ethics board of the Faculty of Social Sciences as well as by the medical ethical board of Leiden University Medical Center.

Participants

The sample used for the present study consists of 55 mothers living in the Netherlands and their first-born infant. To be included in the present study, data of the dyads had to be present on all variables of interest from the first, second and third appointments. During the first appointment, 89 mothers completed the depression questionnaire, and 85 at the second appointment. However, for one participant no maternal behavior was coded during the second appointment and due to technical problems at the third appointment, the majority of the physiological data of 29 participants was lost. Overall, complete data was present for 55 dyads. Demographic characteristics were not significantly different for those who were included in the study and those who were excluded (e.g., maternal and infant age, maternal education). Background characteristics of the present sample are depicted in Table 1. Mean age of the mothers during the first appointment was 22.9 years. Mean age of the infants (27 boys, 28 girls) was 5.9 months during the second appointment and 12.4 months during the third appointment. Participating dyads were mostly Caucasian (81.8%) and the highest level of education that was achieved was secondary school for a majority of the mothers (45.5%) (Table 1).

Table 1. *Demographics*

	N	*%*	*Mean*	*SD*
Infant age II (months)			5.9	0.4
Infant age III (months)			12.4	0.7
Infant gender				
Male	27			
Female	28			
Maternal age I (years)			22.9	2.5
Maternal ethnicity				
Caucasian	45	81,8		
Antillean	4	6,7		
Indonesian	3	5,5		
Other	3	5,5		
Maternal education				
Primary school	8	14,5		
Secondary school	25	45,5		
Community college	15	27,3		
Higher education (bachelor)	6	10,9		
University of science (master)	1	1,8		

Note: I = First appointment, II = second appointment, III = third appointment.

Procedure

The first home visit with the mother took place around the 27th week of pregnancy, in which an interview was conducted and several questionnaires, including one measuring depressive symptoms, had to be filled out. Subsequently, when the infant was six months of age, mother and infant were visited at home by two researchers. During the home visit, several tasks were conducted with mother and infant. A free play session of mother and infant took place in which the mother could freely interact with her infant for three minutes. In addition, the Still Face Paradigm (SFP) was conducted. At the end of the visit the mother filled out some questionnaires, again including a questionnaire measuring depressive symptoms.

Six months later, during the first laboratory visit when the infant was 12 months of age, again several tasks were conducted with the infant. The session was concluded with a stress paradigm, which is used in the present study. At the end of the laboratory session, the mother filled out some

questionnaires. During the second and third visit, physiological responses of the infant were recorded during the appointment.

Measures

Beck Depression Inventory-II. The mother filled out the Beck Depression Inventory-II (BDI-II) during pregnancy (prenatal maternal depressive symptoms) and when the infant was six months old (postnatal maternal depressive symptoms). The 21-item self-report questionnaire is widely used and designed to measure the severity of current depressive symptoms (Beck, 1978). Items are rated on a scale of 0 to 3, resulting in a total score ranging from 0 to 63. Scores of above 9 are considered to indicate elevated depressive symptoms and a risk for Major Depressive Disorder (Kendall, Hollon, Beck, Hammen, & Ingram, 1987). However, the BDI-II does not provide a psychiatric diagnosis. The reliability and validity of the BDI-II are considered to be satisfactory and a high internal consistency of .92 has been found (Osman, Barrios, Gutierrez, Williams, & Bailey, 2008).

Mother-infant interaction

Still Face Paradigm. The Still Face Paradigm (SFP) was conducted with mother and infant when the infant was six months and consisted of three consecutive two-minute episodes (Tronick, Als, Adamson, Wise, & Brazelton, 1978). The infant was placed into a car seat on a table, in which it was only able to look forward, facing the mother sitting on a chair in front of him. First, the 'play-episode' was conducted, in which the mother could interact with her infant without toys, followed by the 'still face-episode', in which the mother was asked to remain immobile and freeze her face, finished by a 'reunion-episode' in which the mother could re-engage with her infant. When infants became highly distressed, mothers were allowed to abort the still face episode and move on to the reunion episode (Tronick et al., 1978). After the SFP a recovery phase of about 5 minutes took place in whichthe mother was allowed to pick up the infant and calm it down if necessary.

For the present study, the Still Face Paradigm (SFP) is only used to code maternal behavior during the 'play episode' and the 'reunion episode', in which mother was allowed to interact with the

infant the way she wanted. In general, all behavioral reactions on the SFP were coded by two trained researchers, who carefully watched the fragments on video. Both the play episode and the reunion episode were scored for the full two minutes. The mother received a global score for each episode. In order to determine interrater reliability, 20 videos (36%) were double coded and intraclass-correlation coefficients were computed (*M* = .81, range .46 to 1.0). Maternal behavior in the SFP was coded in the categories sensitivity, intrusiveness, positive affect, interest/involvement, internalizing/helpless behavior and hostile/angry behavior during the two minutes of the play and reunion episode. However, because the scores of interest/involvement, positive affect, internalizing/helpless behavior and hostile/angry behavior lacked variability, those behaviors were not included in the analyses and are therefore not discussed.

It was decided to include maternal behavior during the SFP in the present study, because mother-infant interaction during this paradigm takes place in a structured setting and includes an interaction under relatively relaxed circumstances (play episode) as well as an interaction after a stressful situation (reunion episode). Because the mother is challenged in two different ways, a broad concept of the several maternal behaviors is targeted.

Free play. The interactive behavior of the mother with the infant at 6 months was also measured during a free play session in which the mother was allowed to interact with her infant the way she wanted, with different kinds of toys available to play with. The interaction was coded by two trained researchers. Interactive behaviors assessed both during the two episodes of the SFP and during Free Play included:

Successful positive engagement. Successful positive engagement was defined as the extent to which a mother succeeded in positively engaging her infant in interaction or play. It was coded in what way the mother invited the infant to engage and in what way the infant responded to the mothers attempts. In other words, the mother had to initiate positive interaction with the infant that succeeded. Examples of maternal initiatives are initiating eye-contact, smiling, vocalizing or showing

toys, examples of infant reactions are joint attention, eye-contact, laughter or other vocalizations towards its mother or joint play. The more often the infant responded to the initiatives of its mother, the higher score she received on a scale of 0 to 3.

Maternal sensitivity. Maternal sensitivity included acknowledging the infant's communications and adequate responding to it, with the mother constantly behaving infant-centered in interaction. It was coded whether a mother was able to read (e.g. by verbalizing) and follow her infants cues (e.g. responding to the infant reaching for toys or reaching to be picked up). In times of distress, appropriate maternal behavior would be the soothing of the infant. The more the mother showed appropriate responses, the higher score she received on a scale of 0 to 3.

Intrusiveness. Intrusiveness included the way the mother interacted roughly with her infant and whether she interfered with the infants needs, interests or actual behavior, which was also coded on a scale from 0 to 3. The infant did not need to respond negatively in response to its mother's actions in order for the mother to be considered intrusive. Examples of intrusive behavior include a shrill tone of voice in the infant's face, roughly touching the infant or restricting movements.

Stress paradigm

The stress paradigm used in the present study is based on the protocol of the Lab-TAB (Goldsmith & Rothbart, 1996), although a remote-controlled robot was used instead of the mechanical toy dog. In addition, in contrast to the paradigm of Goldsmith and Rothbart (1996), the mother was asked to leave the room. During the stress paradigm, the mother and the familiar researcher left the room followed by a stranger entering the room with a remote-controlled robot that walked back and forth towards the infant a few times, who was sitting strapped into a low chair. The unfamiliar experimenter made the robot approach the infant, stop approximately 15 cm from the infant for 15 seconds while making movements and sounds, walk back again and stop for 5 seconds. Corresponding to the Lab-TAB protocol, this procedure was repeated three times. Next to

behavioral measures, physiological measures of the infant were recorded during the paradigm. After the stress paradigm, the mother was allowed to re-enter the room and comfort the infant if necessary during the recovery phase. Physiological data were recorded during the stress paradigm as well as the recovery phase.

In order to perform the behavioral coding of the paradigm, the Lab-TAB's guidelines were followed using video recordings of the paradigm (Goldsmith & Rothbart, 1996). During the task, frontal shots of the infant's body and face were made in order to facilitate coding at micro level. All three trials of the robot's approach and movement in front of the infant were separated into three episodes (walking towards the infant, moving in front of the infant part one and moving in front of the infant part two). In the present study, behavior that was coded when the robot was walking backwards was not included. This resulted in 9 episodes in total that were scored separately per behavioral variable. The behavioral parameters that were coded and used for the present study all target stress reactivity of the infant and are based on those found in the Lab-TAB (Goldsmith & Rothbart, 1996), namely intensity of facial fear (0–3), intensity of facial sadness (0-3), intensity of distress vocalizations (0–5), intensity of bodily fear (0–3) and the presence of a startle response (0-1).

Physiological measures

In order to measure the electrocardiogram (ECG), the impedance cardiogram (ICG) and the skin conductance level (SCL) of the infant, the Vrije Universiteit-Ambulatory Monitoring System (VU-AMS) was used. Seven electrodes were used to measure the ECG and ICG and were attached to the chest and the back of the infant, while two electrodes were connected to its foot in order to measure the SCL. The three ECG electrodes were placed slightly below the right collar bone to the right of the sternum, on the right side between the lowest two ribs and on the left side slightly below the nipple (Van Dijk et al., 2013). Subsequently, all electrodes were attached to the VU-AMS device, which was attached to the back of the infant during the appointment. Two baselines were included in order to record physiological measures in a resting state, the first one before the tasks started and the second

one prior to the stress paradigm. During both baselines the infant got to watch a movie or was sung to, in order to keep the infant as calm as possible. In addition, physiological data were recorded during the stress paradigm and during the recovery phase following immediately after the paradigm. The accuracy of the time registration during the appointment was confirmed using a control marker. Afterwards, data were analyzed using the VU-DAMS software program in order to extract the physiological indices. In order to obtain the HR, the R-peak derived from the ECG was used as an indication (Figure 1). If R-peaks could not be detected by the program for part of the recordings, they were inserted in the ECG by using the mean. Subsequently, the average HR in beats per minute was calculated every 30 seconds for each participant, resulting in an average of Baseline 1, Baseline 2, six intervals of the stress paradigm and ten intervals of the recovery phase. Eventually, the mean HR across the six intervals during the stress paradigm was used as an indicator of HR during stress and the mean HR across the ten intervals of the recovery as an indicator of heart rate recovery.

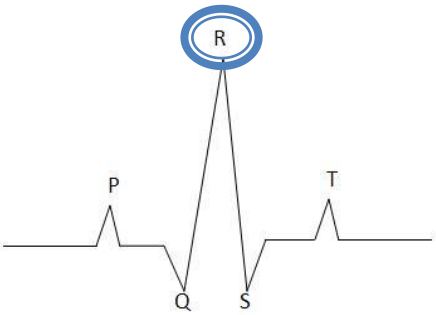

Figure 1. R-peak in ECG-complex.

Data analysis

Due to technical problems, some of the physiological data could not be used for half of the participants, i.e. the pre-ejection period (PEP) to be derived from the ICG, and the SCL. Because too many participants would be lost and the power of the study would decrease substantially, it was decided to only include heart rate as a physiological reactivity measure in the present study, thereby only using the ECG measure.

All statistical analyses were performed using the Statistical Package for the Social Sciences (IBM SPSS; Version 21.0). Prior to the main analyses, data were inspected thoroughly with respect to normality, missing values and outliers. A score was considered an outlier when it deviated at least three standard deviations from the mean. Outliers were imputed to the most extreme score within three standard deviations from the mean. With respect to all variables, correlations were inspected. Composite scores were only created if variables correlated significantly. In addition, all variables were inspected in relation to the following background variables: Infant gender, infant and maternal age, maternal education and maternal ethnicity. In case of a significant correlation, the background variable was controlled for in future analyses.

With respect to heart rate, repeated measures analysis was performed in order to detect a change in heart rate from baseline, to stress paradigm, to recovery. Degrees of freedom were corrected if necessary. To examine the role of maternal depression on infant HR, repeated measures analysis was performed with maternal depressive symptoms as covariate and HR during baseline, stress paradigm and recovery as within-subjects variable. Contrasts were inspected in order to examine possible differences.

Regarding all variables, data were further analyzed if a substantial (not necessarily significant) correlation was present. Regression analyses were used to predict behavioral and physiological stress reactivity. With respect to maternal depressive symptoms, in all analyses symptoms measured at the other time point were controlled for, in order to examine the unique effect of either prenatal or postnatal depressive symptoms. With respect to HR as an outcome measure in regression analyses, HR measured during the stress paradigm was controlled for HR at baseline, while HR measured during recovery phase was controlled for HR during the stress paradigm.

Mediation would only be examined in case requirements as stated by Baron and Kenny (1986) were met. Moderation analyses were performed to examine possible effects between maternal depressive symptoms and maternal style towards the infant in predicting infant behavioral

and physiological stress reactivity, using linear multiple regression analyses. With respect to interaction effects, in order to prevent multicollinearity, independent variables were centered first by subtracting their mean and then multiplied for their interaction. Main effects and background characteristics were entered in the first model and also in the second, but then with the interaction term added to the model as well. Results were considered significant in case of a *p*-value of <.05.

Results

Preliminary analyses

Depression*.* On the BDI-II measured during pregnancy at the first appointment, the mean total score of depressive symptoms (*N* = 55, range 0 to 33) was 9.91, *SD* = 6.73 and during the second appointment, when the infant was six months, the mean total score (*N* = 55, range 0 to 44) was 8.91, *SD* = 7.84. However, distributions of the total scores of the mothers on the BDI-II at both time points were highly skewed and were therefore log-transformed for further analyses. The correlation between (log-transformed) prenatal and postnatal depressive symptoms was .38, $p < .01$. Prenatal depressive symptoms did not significantly correlate with prenatal anxiety symptoms, measured by the State-Trait Anxiety Inventory (STAI; Spielberger, Gorssuch, Lushene, Vagg, & Jacobs, 1983), $r = .07$, $p = .61$. The correlation between depressive and anxiety symptoms measured when the infant was six months was also non-significant, $r = -.02$, $p = .89$. Background characteristics (maternal age, maternal ethnicity and maternal education) did not significantly correlate with prenatal or postnatal depressive symptoms, although an almost significant negative correlation was found between postnatal depressive symptoms and maternal age, $r = -.23$, $p = .10$.

Maternal style*.* Correlations of maternal behavior during the two episodes of the Still Face Procedure and the Free Play session are depicted in Table 2. Because correlations of the several behaviors during the Free Play and the Still Face Paradigm were considered too low/inconsistent and the correlations between maternal behaviors during the play and reunion episodes of the SFP are all

significant, it was decided to create a composite score of maternal behavior during the play and reunion episodes of the SFP and not to include the measures of the free play session in further analyses. In addition, because not all maternal behaviors during the SFP consistently correlate with each other, it was decided not to create composite scores to target maternal style but independently examine the three maternal behaviors. With respect to background characteristics, maternal sensitivity positively correlated with maternal age (r = .28, p < .05) and maternal education (r = .37, p < .01), while maternal intrusiveness negatively correlated with maternal age (r = -.31, p < .05).

Table 2. *Correlations maternal behavior during Free Play and two SFP episodes (N = 55).*

	Free Play			SFP Play			SFP Reunion		
	PE	SE	IN	PE	SE	IN	PE	SE	IN
Free play									
Positive engagement (PE)	1	.48**	-.18	-.02	.16	-.20	-.04	.15	-.16
Sensitivity (SE)		1	-.57**	.13	.24	-.02	-.02	.22	-.21
Intrusiveness (IN)			1	-.08	-.08	.15	-.08	-.06	.01
SFP Play									
Positive Engagement (PE)				1	.43**	-.05	.38**	.21	.05
Sensitivity (SE)					1	-.44**	.20	.60**	-.22
Intrusiveness (IN)						1	-.10	-.62**	.61**
SFP Reunion									
Positive Engagement (PE)							1	.25	.01
Sensitivity (SE)								1	-.49**
Intrusiveness (IN)									1

Note: * is significant at p < .05, ** at p < .01.

Physiology. In order to target the physiological response of the infants, heart rate was recorded during the laboratory session when the infant was 12 months. Before the stress paradigm took place, the baseline of the infant's HR was recorded twice during the appointment. Because the second baseline in general is considered to be more sensitive to movement (the infant is strapped into a chair and also already has completed several tasks) and indeed was higher on average (M = 126.8, SD = 9.0), the first baseline (M = 124.2, SD = 9.6) is used as an indicator of HR during rest .

With respect to the stress paradigm, the second baseline will be used in analyses because it took place right before the paradigm. Repeated measures analysis was performed in order to detect a change in HR due to the stress paradigm. Because the assumption of sphericity was violated (Mauchly's test, $X^2(2) = 53.91$, $p < .001$), degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\varepsilon = .61$). The results revealed that HR significantly changed from second baseline to stress paradigm, $F(1, 54) = 51.52$, $p < .001$, $\eta^2 = .49$ and again from stress paradigm to recovery, $F(1, 54) = 20.39$, $p < .001$., $\eta^2 = .27$, indicating that the stress paradigm in general elicited a physiological stress response in the infants. Heart rate means are depicted in Table 3.

Table 3. *Descriptives Heart Rate (N = 55)*

	Baseline 1	Baseline 2	Stress paradigm	Recovery
Mean HR *(SD)*	124.2 *(9.6)*	126.8 *(9.0)*	148.2 *(21.6)*	136.2 *(9.7)*

Note: Heart rate is reported in beats per minute.

Infant behavioral reactivity. All children received a mean score on each behavioral variable (facial fear, facial sadness, distress vocalizations, bodily fear and startle response) across the 9 episodes of the stress paradigm. The behavior of one child could not be coded, because the camera view was blocked. Because the distribution of facial fear was skewed, it was log-transformed. Correlations of the several reactivity measures, including heart rate, are depicted in Table 4. Because facial sadness and distress vocalizations correlated almost perfectly and therefore measure the same concept, only facial sadness is included in further analyses. As expected, facial fear correlated negatively with facial sadness ($r = -.45$, $p < .01$), as those behaviors are mutually exclusive in the coding system. Facial sadness, distress vocalizations and bodily fear all correlated positively and significantly with HR (Table 4). Background characteristics did not correlate significantly with any behavioral measure.

Table 4. *Correlations infant stress reactivity (N = 54)*

	Facial fear	Facial sadness	Distress vocalizations	Bodily fear	Startle	Mean heart rate
Facial fear (log)	1	-.47**	-.46**	.06	.33*	-.16
Facial sadness		1	.95**	.40**	-.08	.75**
Distress vocalizations			1	.50**	-.06	.78**
Bodily fear				1	.35*	.44**
Startle					1	.07
Mean heart rate						1

Note: * is significant at $p < .05$, ** at $p < .01$.

Main analyses

Maternal depressive symptoms predicting infant stress reactivity. Maternal prenatal depression, whether or not postnatal symptoms were partialled out, did not significantly correlate with any infant behavioral reactivity measureand was therefore not further examined in regression analyses (Table 5). In addition, controlling for baseline HR, regression analysis revealed a non-significant effect of maternal prenatal depressive symptoms on infant HR during the stress paradigm, $B = -.10$, $t = -.021$, $p = .983$. When repeated measures analyses were performed, maternal prenatal depressive symptoms also did not predict a different pattern of HR from baseline, to stress paradigm, to recovery.

With respect to maternal postnatal depressive symptoms assessed when the infant was six months, whether or not prenatal symptoms were partialled out, again no significant correlations with infant behavioral measures were found (Table 5). In addition, controlling for baseline HR, regression analysis revealed a non-significant effect of maternal postnatal depressive symptoms on infant HR during the stress paradigm, $B = -.85$, $t = -.214$, $p = .832$. When repeated measures analyses were performed, maternal postnatal depressive symptoms did not predict a different pattern of HR from baseline, to stress paradigm, to recovery either.

Table 5. *Correlations maternal depressive symptoms and infant stress reactivity.*

	Maternal prenatal depression	Maternal postnatal depression
Maternal prenatal depression	1.00	.38**
Maternal postnatal depression	.38**	1.00
Infant facial fear	-.13	-.12
Infant bodily fear	.05	-.04
Infant facial sadness	.07	.01
Infant startle response	-.01	-.01
Infant mean HR baseline 1	-.07	-.08
Infant mean HR stress paradigm (corrected for Baseline 2)	-.02	-.03
Infant mean HR recovery (corrected for stress paradigm)	-.11	-.01

Note: * is significant at $p < .05$, ** at $p < .01$.

Maternal style predicting infant reactivity. No significant correlations were found between maternal characteristics in interaction with her infant and variables with respect to infant reactivity (Table 6). However, a trend was found of maternal intrusiveness predicting the infant's heart rate during the stress paradigm, $B = -4.362$, $t = -1.957$, $p = .056$, indicating that infants of intrusive mothers showed less HR reactivity during the paradigm.

Table 6. *Correlations maternal behaviors and infant stress reactivity.*

	Positive engagement	Sensitivity	Intrusiveness
Infant facial fear	-.01	.16	.04
Infant bodily fear	-.05	-.06	.06
Infant facial sadness	-.01	-.15	-.02
Infant startle response	.12	.03	-.02
Infant mean HR baseline 1	.14	.19	.15
Infant mean HR stress paradigm (corrected for Baseline 2)	-.04	.01	-.25
Infant mean HR recovery (corrected for stress paradigm)	.16	.19	.07

Note: * is significant at $p < .05$, ** at $p < .01$.

Maternal depressive symptoms predicting maternal style. Prenatal depressive symptoms did not correlate significantly with any of the mother variables (Table 7). However, maternal depressive symptoms at six months did significantly and negatively correlate with maternal sensitivity (r = -.36, p < .01). Regression analysis revealed that, controlled for maternal age and maternal education, maternal depressive symptoms at six months significantly negatively predicted maternal sensitivity, B = -.55, t = -2.437, p = .018, R^2 = .086.

Table 7. *Correlations maternal depressive symptoms and maternal style.*

	Maternal prenatal depression	Maternal postnatal depression
Maternal prenatal depression	1.00	.38**
Maternal postnatal depression	.38**	1.00
Successful positive engagement	.06	.01
Sensitivity	-.09	-.36**
Intrusiveness	.18	.19

Note: * is significant at p < .05, ** at p < .01.

The joint effect of maternal depression and maternal style on infant reactivity. Because no significant effects of depression on infant reactivity (neither behaviorally nor physiologically) were found, the requirement of a significant relation between the independent and dependent variable in order to test mediation was not met (Baron & Kenny, 1986). Therefore, mediation analyses could not be performed. However, in order to examine whether the effect of depression on infant reactivity is influenced by maternal behavior towards the child, moderation analyses were performed.
With respect to maternal prenatal depressive symptoms, four significant interactions were found on infant heart rate. First, controlled for postnatal symptoms and maternal age and education, a significant interaction was observed between prenatal depressive symptoms and maternal sensitivity on infant HR during Baseline 1, B = -6.02, t = -3.384, p < .01, R^2 = .18 (Appendix, Table 8). When more depressive symptoms were present during pregnancy, infants of low sensitive mothers showed a higher baseline HR (Figure 2). Second, controlled for postnatal symptoms and maternal age, a significant interaction was observed between prenatal depressive symptoms and maternal

intrusiveness on infant heart rate during Baseline 1, $B = 6.06$, $t = 4.359$, $p < .01$, $R^2 = .27$ (Appendix, Table 9). When more depressive symptoms were present during pregnancy, infants of highly intrusive mothers showed a higher baseline HR (Figure 2). In addition, when the interaction was added to the model, the effect of maternal intrusiveness on infant HR became significant, $B = 2.41$, $t = 2.47$, $p = .017$, $R^2 = .09$, in a way that infants of more intrusive mothers had a higher baseline HR (Appendix, Table 9).

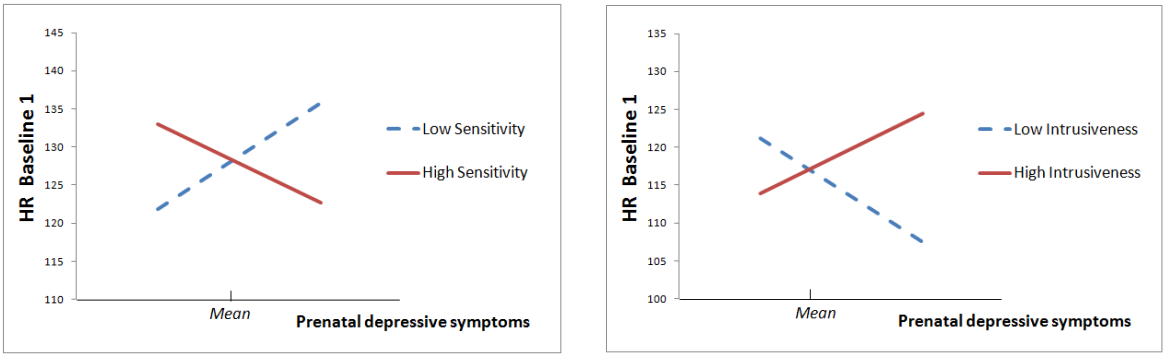

Figure 2. Interactions prenatal depressive symptoms and sensitivity/intrusiveness on HR during Baseline 1.

Third, controlled for postnatal symptoms, HR during paradigm, maternal age and education, a significant interaction was observed between prenatal depressive symptoms and sensitivity on HR during the recovery phase, $B = -5.75$, $t = -3.464$, $p < .01$, $R^2 = .16$ (Appendix, Table 10). When more depressive symptoms were present during pregnancy, infants of highly intrusive mothers showed a higher HR during recovery (Figure 3). Fourth, controlled for postnatal symptoms, HR during paradigm and maternal age, a significant interaction was observed between prenatal depressive symptoms and intrusiveness on HR during the recovery phase, $B = 5.07$, $t = 3.726$, $p < .01$, $R^2 = .18$ (Appendix, Table 11). When more depressive symptoms were present during pregnancy, infants of low sensitive mothers showed a higher HR during recovery (Figure 3).

In addition, again with respect to prenatal depressive symptoms, two significant interactions were found on behavioral reactivity during the stress paradigm. First, controlled for postnatal symptoms and maternal age and education, a significant interaction was observed between prenatal depressive symptoms and sensitivity on infant facial sadness during the paradigm, $B = .44$, $t = 2.063$,

$p < .05$, $R^2 = .08$ (Appendix, Table 12). When more depressive symptoms were present during pregnancy, infants of highly sensitive mothers showed more facial sadness during the stress paradigm (Figure 4). Second, controlled for postnatal symptoms and maternal age, a significant interaction was observed between prenatal depressive symptoms and intrusiveness on infant bodily fear during the paradigm, $B = -.28$, $t = -3.09$, $p < .01$, $R^2 = .16$ (Appendix, Table 13). When more depressive symptoms were present during pregnancy, infants of highly intrusive mothers showed more bodily fear during the stress paradigm (Figure 4).

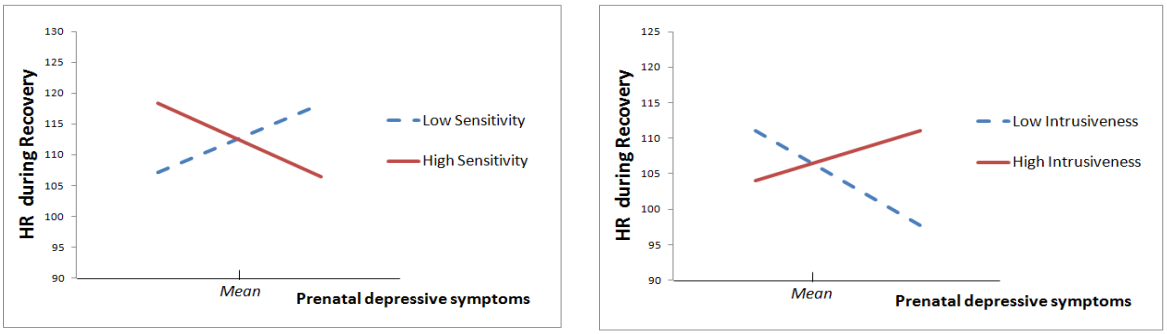

Figure 3. Interactions prenatal depressive symptoms and sensitivity/intrusiveness on HR during Recovery.

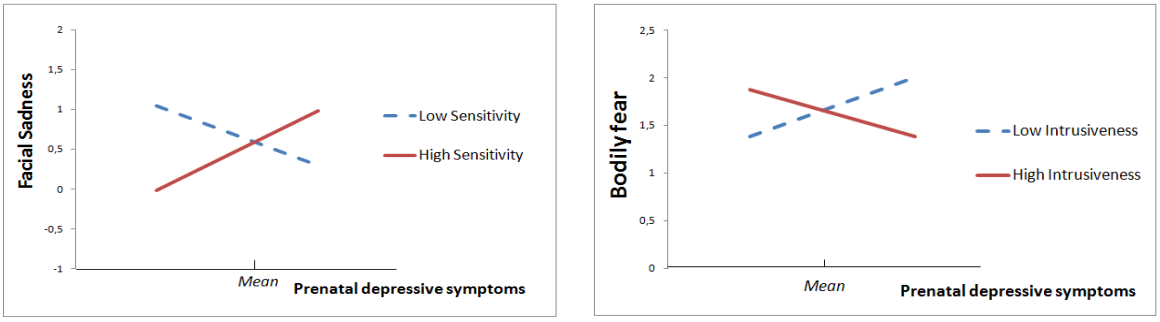

Figure 4. Interactions prenatal depressive symptoms and sensitivity/intrusiveness on behavioral reactivity during the paradigm.

With respect to maternal postnatal depressive symptoms measured when the infant was six months, two significant interactions were found on infant heart rate. First, a significant interaction was found of postnatal depressive symptoms and intrusiveness, on infant HR during Baseline 1, $B = 4.47$, $t = 3.608$, $p < .01$, $R^2 = .20$ (Appendix, Table 14). When more depressive symptoms were present when the infant was six months, infants of high intrusive mothers showed a higher baseline HR

(Figure 5). In addition, when adding the interaction term, a trend was found of maternal intrusiveness on infant baseline HR, $B = 1.86$, $t = 1.86$, $p = .069$, $R^2 = .05$, in a way that infants of more intrusive mothers tended to have a higher baseline HR.

Second, a significant interaction was found of postnatal depressive symptoms and intrusiveness, on infant HR during the recovery phase, $B = 4.07$, $t = 3.435$, $p < .01$, $R^2 = .16$ (Appendix, Table 15). When more depressive symptoms were present when the infant was six months, infants of high intrusive mothers showed a higher HR during recovery (Figure 5).

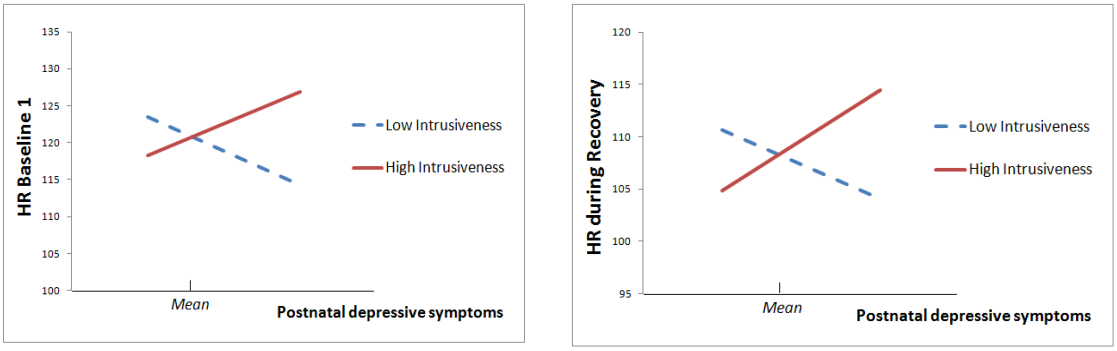

Figure 5. Interactions postnatal depressive symptoms and intrusiveness on Baseline HR and HR during Recovery.

Finally, a significant interaction was found of postnatal depressive symptoms and intrusiveness, on infant bodily fear during the stress paradigm, $B = -.22$, $t = -2.888$, $p < .01$, $R^2 = .15$ (Appendix, Table 16). When more depressive symptoms were present when the infant was six months, infants of highly intrusive mothers showed less bodily fear (Figure 6).

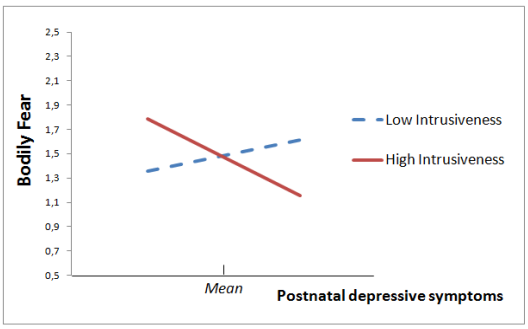

Figure 6. Interaction postnatal depressive symptoms and intrusiveness on Bodily Fear during the paradigm.

Discussion

Maternal depression has been associated with poor parenting skills and lower levels of mother-child interaction quality, with negative child outcomes as a consequence (e.g. Cohn & Tronick, 1983; Cox, Puckering, Pound, & Mills, 1987; Hops et al., 1987; Zahn-Waxler, McKnew, Cummings, Davenport, & Radke-Yarrow, 1984). The body's stress response system, i.e. HPA-axis, is considered an important link between environmental influences and child behavior and therefore very fruitful to study in relation to maternal depression. In addition, in order to decrease adverse outcomes or even prevent them, it is important to detect adverse effects of maternal depression on child development and their precursors as early as possible. The relation between maternal depression and stress reactivity in infants has not been studied often and also the influence of maternal behavior on this relationship in such young children is not yet quite clear. Therefore, the present study aimed to examine the effect of maternal depressive symptoms on infant stress reactivity and whether the way a mother interacts with her infant would influence this effect.

Predicting infant stress reactivity from maternal depressive symptoms

However, no direct relationships were found between maternal depressive symptoms, neither prenatal nor postnatal, and infant stress reactivity. A few possible explanations can be given for not finding this relationship. First, the stress paradigm used in the present study was found to elicit great stress responses in most infants participating in the study. At such a young age, an evolutionary very adaptive response would be to start crying in response to a threatening situation, a reaction that was observed in a large part of the infants. The role of the HPA-axis is to convert the perception of danger into a bodily state of arousal, attention and a fight/flight response (Cozolino, 2006). In extremely stressful situations such as the stress paradigm used in the present study, a quick response of arousal can be considered an adaptive response. According to Bowlby (1982) and many others studying attachment theory, a forceful protest during the early absence of a caregiver is a good initial strategy to alert the caregiver and thereby promote survival, as humans are born in a

developmentally immature and highly dependent state (Bowlby, 1982). This particular stress paradigm, in which the mother leaves the room as well, might be extremely stressful for most infants, and heightened stress reactivity might be a very adaptive response for all infants. In addition, infants of depressed mothers might be more used to her unavailability, therefore becoming even less upset when being left alone by their mother, compared to children of non-depressed mothers. For example, in the present study, infants of highly intrusive mothers tended to physiologically show less distress during the paradigm, which might be explained by them being more used to stressful experiences, compared to infants from a relatively secure environment. Because this particular stress paradigm has not been used before with such young infants, its relatively extreme content might explain why direct relationships between maternal depression and child stress reactivity found in previous studies were not present.

Another explanation would be that fear, as a specific component of temperament, is not stabile yet in these young infants. More specifically, studies that have investigated the stability of fear from infancy onwards failed to find consistency. For example, Kochanska (2001) investigated fear from 9 months on, finding stability only from 14 months onwards. In another study of Lemery and colleagues, fear was investigated from 3 months onwards and stability was only found from 24 months onwards (Lemery et al., 1999). It might be that, although stability of temperament has been found in some studies from infancy onwards (e.g. Casalin et al., 2012; Komsi et al., 2006), the development of fear stabilizes at older age, why the two types of responses to fear (i.e. inhibition and disinhibition) possibly weren't yet developed in the 12-month-olds in the present study. In addition, fear expresses itself differently in young infants compared to older children, because they do not have other tools yet to cope with stressors but infantile strategies as crying. Other regulatory skills, e.g. turning away from the stressor, might not have been developed in most of these infants yet, as those skills are found to start developing towards the end of the first year (Feldman et al., 2009). Moreover, infant crying is a difficult emotional expression to elucidate, as crying can be considered expression of feelings of fear and despair on the one hand, and an adaptive response to

both regulate fear and to alert caregivers on the other hand, but it is impossible to distinguish among those motives. When children grow older, emotions such as fear, sadness and despair differentiate more and more, making it easier to label a specific response. Taken together, within the same sample, differences should be reexamined when the children are older in order to investigate whether or not the hypothesized differences would develop at older age.

The relation between maternal depressive symptoms and maternal style

In contrast, with respect to the relation between depressive symptoms and maternal behavior, the hypothesized relation between maternal depressive symptoms during the child's life and maternal sensitivity was found, in a way that mothers with more depressed symptoms were found to be less sensitive in interaction with their children. According to the literature, mothers with more depressive symptoms are more involved with themselves and their own perspective, causing them to perceive and respond to signals of their infant less adequately (e.g. Feldman et al., 1999). The quality of maternal caregiving is thought to be the key theoretical precursor of attachment security and is found to influence neurological systems in the child that regulate responses to stress. Not finding a relation between maternal sensitivity and infant reactivity, again might be explained by the extreme character of the paradigm used in the present study, causing heightened reactivity to be an adaptive response. In general, low maternal sensitivity has been found to be associated with insecure attachment and thereby with several adverse child outcomes, i.e. less self-regulation (Thompson & Meyer, 2007), less sociability and more negative affect (Hane & Fox, 2006), more conflict with siblings, peers and partners (e.g. Sroufe, 2005) and a more negative self-concept (Doyle, Markiewicz, Brendgen, Lieberman, & Voss, 2000). The present study provides additional evidence that mothers with depressive symptoms are at risk to interact less sensitively with their children, thereby putting their children at risk for adversities later in life.

The joint effect of maternal depressive symptoms and maternal style on behavioral reactivity

Although no significant predictors of infant stress reactivity were found, significant interactions were found with respect to three behavioral variables during the stress paradigm. First, if more prenatal depressive symptoms were present, high maternal sensitivity lead to more facial sadness during the paradigm. Considering heightened reactivity an adaptive response in this specific paradigm, it seems that infants of more sensitive mothers have more adaptive coping skills and start crying during extreme stress, compared to mothers of low sensitive mothers. However, this was only the case when mothers reported a higher amount of depressive symptoms during pregnancy. For infants of mothers with little depressive symptoms, enhanced sensitivity was related to less facial sadness. Although hard to explain, it might be that infants of mothers with little prenatal symptoms who are also highly sensitive, do have better regulatory skills, also maybe because of other characteristics not targeted in the present study. Maybe sensitivity in mothers with more depressive symptoms, evokes a strategy for these children to regulate their distress (i.e. crying), which is less needed for infants of mothers with less symptoms. However, this explanation is rather speculative and moreover, the interaction was only found for prenatal depressive symptoms and not for postnatal, which is also difficult to explain. However, for prenatal as well as postnatal depressive symptoms, an interaction with intrusiveness was present on infant bodily fear during the paradigm, in a way that when more depressive symptoms were present, infants of highly intrusive mothers showed less bodily fear. It seems that, when more depressive symptoms are present, positive parenting leads to enhanced infant stress reactivity. Again, it seems more adaptive to show enhanced reactivity and it seems that infants of mothers with negative parenting skills who are also depressed, are less frightened during the paradigm thereby showing a less adaptive response. Moreover, it might be the case that children of mothers with depressive symptoms are even more used to stressful situations when mothers are also intrusive or little sensitive and therefore show less reactivity during extreme stress.

The joint effect of maternal depressive symptoms and maternal style on physiology during non-distress

In addition, infants of mothers with more depressive symptoms showed enhanced physiological arousal without the presence of a stressor, as interactions were found with respect to baseline HR and HR during the recovery phase after the paradigm. First, infants of mothers with enhanced prenatal symptoms who were also less sensitive towards their child showed a relatively higher baseline HR, while infants of mothers with enhanced prenatal symptoms who were more sensitive showed a relatively lower baseline HR. However, for mothers with little prenatal depressive symptoms it was the other way around, in a way that infants of more sensitive mothers had a higher baseline than infants of less sensitive mothers. An explanation for this could be, although rather speculative, that infants of mothers with little depressive symptoms who were also sensitive towards their children, were more explorative and more active when they watched the video during the baseline, causing their HR to be higher. For example, Kalb and Raymond (2003) found that children of depressed mothers are less interested in their surroundings and engage less in explorative behavior, such as play. Second, similar interactions were found of both maternal prenatal and postnatal depressive symptoms and maternal intrusiveness on the infant's baseline HR, in a way that infants of mothers with enhanced depressive symptoms who were also more intrusive showed a higher baseline HR. Enhanced baseline stress, i.e. higher baseline cortisol, has been found in infants of depressed mothers in earlier studies, although maternal behavior was not included in those studies and depression alone caused the effect (Brennan et al., 2008; Field & Diego, 2008). A higher resting HR in children is a marker of physiological dysregulation and has been found to predict more behavioral inhibition and child internalizing problems in the long term (Kagan et al., 1984). However, HR in such young infants has not been found to be stable yet (e.g. Richards & Cameron, 1989), so it is recommended to reexamine their resting HR when they are older.

The same interactions as described above were found predicting HR during the recovery phase. Infants of mothers with more prenatal depressive symptoms who were little sensitive as well,

had a higher HR during the recovery phase, while infants of mothers with either more prenatal or postnatal depressive symptoms who were also highly intrusive, also showed a higher HR during recovery. This indicates that children of mothers with depressive symptoms who show less positive parenting as well, recover more slowly after extreme stress. Feldman and colleagues (2009) found a similar result, as the cortisol response of children of depressed mothers remained elevated after the stress paradigm compared to children of non-depressed mothers. In the most ideal scenario, after perception of and response to threat, an individual returns to a homeostatic balance (i.e. allostasis) again as soon as possible (Cozolino, 2006), a scenario that was less well achieved by infants of mothers with enhanced depressive symptoms with a more negative parenting style. Where an immediate response to a stressor can be considered functional and adaptive, a prolonged stress response can lead to several adverse health consequences. The finding that the stress response system of infants of certain mothers with depressive symptoms needs more time to recover, is in line with the literature stating that infants of depressed mothers show physiological dysregulation (e.g. Kaufman & Ryan, 1999). The finding that infants of mothers with little depressive symptoms who were also highly sensitive or little intrusive also show a higher HR during the recovery phase, again might be because those children sooner got active again and reengaged into play, although this remains speculation.

In sum, the way maternal depressive symptoms influence the infant's stress response system seems to depend on her maternal style when she interacts with her child, in a way that a combination of depressive symptoms (either prenatal or postnatal) and negative parenting leads to less adaptive outcomes, i.e. to less arousal during extreme stress and more arousal during non-distress. These interactions are in line with findings of Kaplan et al. (2008), who also found that prenatal depressive symptoms only predicted higher baseline cortisol in infants in combination with low maternal sensitivity. These interactions underline the differential susceptibility of infants to parenting characteristics and thereby are in line with the diathesis-stress model (Belsky & Pluess, 2009; Zuckerman, 1999).

Furthermore, finding these results with respect to prenatal depressive symptoms as well, provides evidence for the influence of the in utero environment on the child's stress response system, thereby supporting the prenatal programming hypothesis (Barker, 1995). Mothers with enhanced prenatal depressive symptoms might influence the development of the fetal brain of their child, which may alter the functioning of their stress response system. The altered functioning of the stress response system in infants of mothers with enhanced depressive symptoms puts those infants at risk, as physiological dysregulation is often detected in children who show internalizing as well as externalizing problem behavior (Baker et al., 2012). Prenatal depression as well as early experience with a depressed mother during the postnatal period can both be considered biological mechanisms involved in the transmission of intergenerational risk of psychopathology from mother to child. The results of the present study underline the importance of positive parenting towards a child, most particularly when symptoms of maternal psychopathology are present, in order to provide the child with a stress response system that functions in a healthy way, which forms the basis for a lifelong optimal development.

Strengths and limitations

The present study contributes to our insight with respect to the influence of a mother's parenting qualities in relation to depression and the infant's neuroendocrine system. The project "A Good Start" is a prospective, longitudinal study from pregnancy onwards, providing possibilities to longitudinally examine maternal behavior, child behavior, temperament and stress response system, and their development and mutual relations. Research examining the HPA functioning of young infants is limited en future studies should continue studying the stress response system in young children. The present study used a multimethod approach in which physiological as well as behavioral parameters were assessed, which is recommended for future studies as well, in order to retain multiple facets of the child's response to stressors.

However, the present study had some limitations as well. First, maternal depression, mother-child interaction and child stress reactivity were all assessed at different time points, enhancing the possibility for other factors to have influenced the child's stress response system in the meantime. Maternal depressive symptoms, as well as the way she interacted with her infant could have changed in six months, which could not be examined. A follow-up study should examine those variables at the same time point, if possible. Second, because depressive symptoms were indicated by means of a self-report questionnaire that does not provide a diagnosis, the present study cannot provide solid evidence with respect to maternal depression, but can only make statements about mothers with depressive symptoms or a certain vulnerability for depression. However, many mothers remain undiagnosed while they certainly are at risk, causing the present study to be highly relevant anyway. Another limitation involves the recovery phase after the stress paradigm. Because the recovery phase was not standardized and also not coded behaviorally, HR could not be controlled for the activity level of the child during the five minutes of recovery. Many children reengaged into play for example, which makes it difficult to determine whether HR was due to elevated stress after the paradigm, or due to movement because of play activities. In addition, recording baseline HR in such young children has also been a challenge, because it depended on the mood and temperament of the infant whether it could sit still for a while. Another limitation of the present study is that eventually only heart rate could be used as a physiological indicator, while other physiological measures were assessed as well and possibly could have strengthened the present results. Finally, it needs to be pointed out that no causal relations could be examined in the present study and that the findings rather include associations.

Future directions and implications

Dysregulation of the HPA-axis has been suggested to be one of the mechanisms explaining intergenerational transmission of risk for negative outcomes such as psychopathology, from depressed parents to their children (Dougherty et al., 2013; Goodman & Gotlib, 1999). The present

findings underline that especially the combination of maternal depression and negative parenting leads to dysregulation of the stress response system, thereby putting these children at risk to develop psychopathology themselves when they mature. In contrast, infants of mothers with enhanced depressive symptoms with positive parenting characteristics, i.e. maternal sensitivity, showed indications of a more healthy stress response system. Therefore, future studies should continue to include parenting characteristics when examining the effect of maternal psychopathology on child development, because they are of high importance and may explain or alter the effect. Moreover, studying maternal style in relation to child outcomes gives direction to prevention and intervention programs and might be easier to coach than curing the depression itself. In addition, future studies should continue studying infant physiological reactivity, because still little is known about infant physiological responses to a stressful situation and individuals at risk for developing internalizing or externalizing problem behavior might be identified that way (Baker et al., 2012).

Second, although fear has been studied in infants (Kagan, 1982; Kochanska, Coy, Tjebkes, & Husarek, 1998; Schwartz et al., 1999), less research has focused on the development of fear from infancy onwards and stability of this component of child temperament is not yet clear. A follow-up of the present study using the same sample could provide insight in the development of fear in children and its stability. Moreover, physiological markers such as heart rate have not been found to be stable in infants yet either (Baker et al., 2012), so a follow-up study when the children are older would be fruitful. In addition, when a larger sample is used a psychiatric diagnosis of depression could be examined next to the amount of depressive symptoms reported by the mother.

Third, little is known about the relation between fear and emotions that emerge when children are older, such as guilt and empathy, which are also known to be of importance with respect to the development of internalizing and externalizing psychopathology (Stuewig, Tangney, Heigel, Harty, & McCloskey, 2010). It would be interesting to examine this development and possible neuroendocrine correlates, in relation to maternal depression and parenting. Follow-up studies

within the project “A Good Start” could possibly gain insight into the further emotional and behavioral development of the infants that were included in the present study. In addition, more research is needed using the present stress paradigm in young children and its correlates, in order to find out whether the paradigm differentiates enough among children. The research might be compared to research with a milder stress paradigm, in order to find out whether the present paradigm might be too extreme for these young infants to elicit different responses. Finally, it would be fruitful to replicate the present study with a higher amount of participants.

Taken together, the present study underlines that the way a mother interacts with her child is very important for the development of the child’s neurobiological system and that her parenting style can either buffer or enhance the effect of maternal psychopathology. In line with earlier studies, positive maternal behavior (i.e. sensitivity) could form a buffer to the negative effects of maternal depression (Harnish, Dodge, & Valente, 1995; National Institute of Child Health and Human Development Early Child Care Research Network,1999), while maternal intrusiveness might strengthen those effects, both findings providing implications for future prevention and intervention programs. Teaching mothers at risk, for example in low SES communities, how to interact positively and adequately with their child and how to respond to its needs, could possibly decrease the deleterious effects of maternal depression on child development. Initiatives such as the project “A Good Start”, which provide coaching in parenting for young mothers at risk from pregnancy onwards, might be of great importance with respect to prevention of adversities during the development of children, and follow-up studies should gain insight into the longitudinal effects of such coaching programs.

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Appendix

Table 8. *Multiple linear regression predicting HR during Baseline 1.*

Model		*B*	*SE*	β	*t*	*p*	*(part r)*^{2}
1	(Constant)	129.83	13.13		9.89	<.001**	
	Prenatal depressive symptoms	-.78	2.16	-.06	-.36	.719	<.01
	Postnatal depressive symptoms	-.02	1.93	-.01	-.01	.993	<.01
	Maternal sensitivity	-.33	.67	-.09	-.49	.624	.03
	Maternal age	.54	1.85	.05	.29	.771	<.01
	Maternal education	1.54	1.30	.19	1.19	.241	<.01
2	(Constant)	128.33	11.92		10.76	<.001**	
	Prenatal depressive symptoms	.92	2.02	.06	.45	.652	<.01
	Postnatal depressive symptoms	-.74	1.76	-.06	-.42	.677	<.01
	Maternal sensitivity	-.33	.61	-.09	-.53	.596	<.01
	Maternal age	.86	1.68	.08	.51	.609	<.01
	Maternal education	-.49	1.33	-.06	-.37	.715	<.01
	Prenatal depressive symptoms x Maternal sensitivity	-6.02	1.78	-.50	-3.38	.001**	.18

Model 1: R^2 = .044
Model 2: R^2 = .228
* p < .05, ** p < .01

Table 9. *Multiple linear regression predicting HR during Baseline 1.*

Model		*B*	*SE*	β	*t*	*p*	*(part r)*^{2}
1	(Constant)	121.06	13.07		9.27	<.001**	
	Prenatal depressive symptoms	-1.03	2.15	-.07	-.48	.633	<.01
	Postnatal depressive symptoms	-.90	1.81	-.08	-.50	.621	<.01
	Maternal age	.14	.57	.04	.24	.811	<.01
	Maternal intrusiveness	1.38	1.11	.19	1.25	.217	.03
2	(Constant)	116.74	11.25		10.38	<.001**	
	Prenatal depressive symptoms	-.77	1.84	-.05	-.42	.678	<.01
	Postnatal depressive symptoms	-2.17	1.56	-.19	-1.38	.175	.03
	Maternal age	.28	.49	.07	.58	.562	<.01
	Maternal intrusiveness	2.41	.98	.33	2.47	.017*	.09*
	Prenatal depressive symptoms x Maternal sensitivity	6.06	1.39	.54	4.36	<.001**	.27**

Model 1: R^2 = .038
Model 2: R^2 = .307
* p < .05, ** p < .01

Table 10. *Multiple linear regression predicting HR during Recovery.*

Model		*B*	*SE*	β	*t*	*p*	*(part r)*^{2}
1	(Constant)	118.18	14.02		8.43	<.001**	
	Prenatal depressive symptoms	-1.85	1.99	-.13	-.93	.357	.01
	Postnatal depressive symptoms	1.10	1.78	.09	.62	.539	<.01
	Maternal sensitivity	1.83	1.20	.22	1.52	.135	.04
	Maternal age	-.27	.62	-.07	-.43	.669	<.01
	Maternal education	-.95	1.70	-.09	-.56	.579	<.01
	HR during fear paradigm	.18	.06	.41	3.18	.003**	.16**
2	(Constant)	112.53	12.75		8.83	<.001**	
	Prenatal depressive symptoms	-.24	1.85	-.02	-.13	.897	<.01
	Postnatal depressive symptoms	.41	1.62	.04	.25	.801	<.01
	Maternal sensitivity	-.13	1.22	-.02	-.11	.914	<.01
	Maternal age	-.30	.56	-.08	-.54	.594	<.01
	Maternal education	-.64	1.54	-.06	-.42	.678	<.01
	HR during fear paradigm	.22	.05	.49	4.11	<.001**	.22**
	Prenatal depressive symptoms x Maternal sensitivity	-5.75	1.66	-.47	-3.46	.001**	.16**

Model 1: R^2 = .217
Model 2: R^2 = .376
* p < .05, ** p < .01

Table 11. *Multiple linear regression predicting HR during Recovery.*

Model		*B*	*SE*	β	*t*	*p*	*(part r)*^{2}
1	(Constant)	112.4.6	14.66		7.67	<.001**	
	Prenatal depressive symptoms	-1.76	2.02	-.12	-.87	.387	.01
	Postnatal depressive symptoms	.11	1.70	.01	.06	.949	<.01
	Maternal intrusivenesss	.62	1.06	.08	.59	.561	<.01
	Maternal age	-.22	.53	-.06	-.41	.685	<.01
	HR during fear paradigm	.19	.06	.43	3.24	.002**	.17**
2	(Constant)	105.94	13.16		8.05	<.001**	
	Prenatal depressive symptoms	-1.57	1.79	-.11	-.87	.387	.01
	Postnatal depressive symptoms	-.96	1.54	-.08	-.63	.534	<.01
	Maternal intrusiveness	1.57	.98	.21	1.60	.116	.03
	Maternal age	-.11	.48	-.03	-.23	.823	<.01
	HR during fear paradigm	.22	.05	.48	4.02	<.001**	.21**
	Prenatal depressive symptoms x Maternal sensitivity	5.07	1.36	.45	3.73	.001**	.18**

Model 1: R^2 = .19
Model 2: R^2 = .37
* p < .05, ** p < .01

Table 12. *Multiple linear regression predicting Facial Sadness during stress paradigm.*

Model		*B*	*SE*	β	*t*	*p*	*(part r)*^{2}
1	(Constant)	.46	1.45		.32	.750	
	Prenatal depressive symptoms	.19	.24	.12	.80	.427	.01
	Postnatal depressive symptoms	-.14	.21	-.10	-.64	.526	<.01
	Maternal sensitivity	-.24	.14	-.26	-1.66	.104	.05
	Maternal age	.01	.08	.02	.12	.909	<.01
	Maternal education	.27	.21	.23	1.29	.205	.03
2	(Constant)	.58	1.40		.41	.681	
	Prenatal depressive symptoms	.06	.24	.04	.26	.796	<.01
	Postnatal depressive symptoms	-.09	.21	-.07	-.44	.661	<.01
	Maternal sensitivity	-.09	.16	-.10	-.60	.551	<.01
	Maternal age	.01	.07	.01	.08	.937	<.01
	Maternal education	.26	.20	.22	1.27	.209	.03
	Prenatal depressive symptoms x Maternal sensitivity	.44	.21	.32	2.06	.045*	.08*

Model 1: R^2 = .084
Model 2: R^2 = .160
* p < .05, ** p < .01

Table 13. *Multiple linear regression predicting Bodily Fear during stress paradigm.*

Model		*B*	*SE*	β	*t*	*p*	*(part r)*^{2}
1	(Constant)	1.46	.77		1.90	.063	
	Prenatal depressive symptoms	.05	.13	.07	.43	.671	<.01
	Postnatal depressive symptoms	-.05	.11	-.08	-.50	.618	<.01
	Maternal intrusiveness	.02	.07	.05	.33	.740	<.01
	Maternal age	-.01	.03	-.03	-.22	.830	<.01
2	(Constant)	1.66	.71		2.34	.023*	
	Prenatal depressive symptoms	.03	.12	.04	.29	.777	<.01
	Postnatal depressive symptoms	-.01	.10	-.01	-.02	.985	<.01
	Maternal intrusiveness	-.03	.06	-.07	-.50	.622	<.01
	Maternal age	-.02	.03	-.07	-.47	.637	<.01
	Prenatal depressive symptoms x Maternal intrusiveness	-.28	.09	-.43	-3.09	.003**	.16**

Model 1: R^2 = .010
Model 2: R^2 = .174
* p < .05, ** p < .01

Table 14. *Multiple linear regression predicting Baseline HR.*

Model	*B*	*SE*	β	*t*	*p*	*(part r)*^{2}
1 (Constant)	121.06	13.07		9.27	<.001**	
Postnatal depressive symptoms	-.90	1.81	-.08	-.50	.621	<.01
Prenatal depressive symptoms	-1.03	2.15	-.07	-.48	.633	<.01
Maternal intrusiveness	1.38	1.11	.19	1.25	.217	.03
Maternal age	.14	.57	.04	.24	.811	<.01
2 (Constant)	120.71	11.73		10.29	<.001**	
Postnatal depressive symptoms	-.18	1.64	-.02	-.11	.914	<.01
Prenatal depressive symptoms	-2.59	1.97	-.18	-1.31	.196	.03
Maternal intrusiveness	1.86	1.00	.25	1.86	.069	.05
Maternal age	.11	.51	.03	.22	.825	<.01
Postnatal depressive symptoms x Maternal intrusiveness	4.47	1.24	.47	3.61	.001**	.20**

Model 1: R^2 = .038
Model 2: R^2 = .240
* p < .05, ** p < .01

Table 15. *Multiple linear regression predicting HR during Recovery.*

Model	*B*	*SE*	β	*t*	*p*	*(part r)*^{2}
1 (Constant)	112.46	14.66		7.67	<.001**	
Postnatal depressive symptoms	.11	1.70	.01	.06	.949	<.01
Prenatal depressive symptoms	-1.76	2.02	-.12	-.87	.387	.01
HR during fear paradigm	.19	.06	.43	3.24	.002**	.17**
Maternal intrusiveness	.62	1.06	.08	.59	.561	<.01
Maternal age	-.22	.53	-.06	-.41	.685	<.01
2 (Constant)	108.51	13.32		8.15	<.001**	
Postnatal depressive symptoms	.76	1.55	.06	.49	.626	<.01
Prenatal depressive symptoms	-3.21	1.87	-.22	-1.72	.093	<.01
HR during fear paradigm	.22	.06	.49	4.03	<.001**	.22**
Maternal intrusiveness	1.17	.98	.16	1.19	.238	<.01
Maternal age	-.26	.48	-.07	-.53	.599	<.01
Postnatal depressive symptoms x Maternal sensitivity	4.07	1.18	.42	3.44	.001**	.16**

Model 1: R^2 = .185
Model 2: R^2 = .346
* p < .05, ** p < .01

Table 16. *Multiple linear regression predicting Bodily Fear during stress paradigm.*

Model		*B*	*SE*	β	*t*	*p*	*(part r)*^{2}
1	(Constant)	1.46	.77		1.90	.063	
	Postnatal depressive symptoms	-.05	.11	-.08	-.50	.618	<.01
	Prenatal depressive symptoms	.05	.13	.07	.43	.671	<.01
	Maternal intrusiveness	.02	.07	.05	.33	.740	<.01
	Maternal age	-.01	.03	-.03	-.22	.830	<.01
2	(Constant)	1.48	.71		2.07	.044*	
	Postnatal depressive symptoms	-.10	.10	-.14	-.95	.346	.01
	Prenatal depressive symptoms	.12	.12	.15	1.03	.308	.01
	Maternal intrusiveness	-.01	.06	-.02	-.12	.909	<.01
	Maternal age	-.01	.03	-.03	-.21	.833	<.01
	Postnatal depressive symptoms x Maternal intrusiveness	-.22	.08	-.40	-2.89	.006**	.15**

Model 1: R^2 = .010
Model 2: R^2 = .157
* p < .05, ** p < .01