

Biological Sensitivity to the Effects of Maternal Postpartum Depressive Symptoms on
Children's Behavior Problems

by

Jennifer Somers

A Thesis Presented in Partial Fulfillment
of the Requirements for the Degree
Master of Arts

Approved April 2017 by the
Graduate Supervisory Committee:

Linda Luecken, Chair
Keith Crnic
Tracy Spinrad

ARIZONA STATE UNIVERSITY

December 2017

ABSTRACT

The theory of biological sensitivity to context (BSC; Boyce & Ellis, 2005) posits that specific biological characteristics, such as vagal tone, may confer risk for physical and mental health outcomes for some children but promote health for others. High levels of resting respiratory sinus arrhythmia (RSA), an index of vagal tone, may confer susceptibility to the effects of the caregiving environment on child development. Consistent with BSC, I expected that, relative to infants with lower RSA, infants with higher RSA would demonstrate fewer behavior problems if their mothers reported fewer postpartum depressive symptoms, but more behavior problems if their mothers reported more postpartum depressive symptoms. I also evaluated whether observed child social engagement with their mothers mediated children's biological sensitivity to the effects of postpartum depressive symptoms on behavior problems in early childhood. I evaluated a mediated moderation model among a sample of 322 low-income Mexican American mother-infant dyads. As expected, the RSA x maternal depressive symptoms interaction, controlling for covariates, was a significant predictor of internalizing, externalizing and total behavior problems, and high vagal tone conferred susceptibility for externalizing behavior problems. Contrary to my hypothesis, children with low RSA may be more susceptible to the effects of maternal postpartum depressive symptoms on children's internalizing and total behavior problems, and child social engagement did not account for these effects. Among infants in economically disadvantaged families, lower RSA and fewer maternal depressive symptoms may promote resilience, and more research is needed to understand behavioral mediators of biological sensitivity.

ACKNOWLEDGMENTS

This research was supported by the National Institute of Mental Health (R01 MH083173-01, PIs: Linda Luecken, Keith Crnic, Nancy Gonzales) and by a National Science Foundation Graduate Research Fellowship to Jennifer Somers.

TABLE OF CONTENTS

	Page
LIST OF TABLES.....	v
LIST OF FIGURES.....	vi
INTRODUCTION.....	1
Current Study.....	9
METHODS.....	13
Participants.....	13
Recruitment.....	14
Planned Missingness.....	15
Procedures.....	15
Measures.....	17
Data Analysis.....	22
RESULTS.....	25
Preliminary Analysis.....	25
Primary Results.....	30
Exploratory Analyses.....	47
DISCUSSION.....	47
Vagal Tone as a Sensitivity Factor to Environmental Context.....	47
Social Engagement.....	50
Strengths and Limitations.....	52
Future Directions.....	54

	Page
Conclusions	56
REFERENCES.....	57

LIST OF TABLES

Table		Page
1.	Descriptive Statistics for the EPDS	19
2.	Descriptive Statistics of Primary Study Variables	25
3.	Zero-Order Correlations Among Primary Study Variables	26
4.	Estimates for One-Factor Confirmatory Models of Social Engagement.....	28
5.	Model Predicting Total Problems, Without Covariates	31
6.	Model Predicting Total Problems, With Covariates	32
7.	Final Model Predicting Total Problems.....	34
8.	Model Predicting Internalizing & Externalizing Problems, Without Covariates	37
9.	Model Predicting Internalizing & Externalizing Problems, With Covariates	39
10.	Final Model Predicting Internalizing & Externalizing Problems	41

LIST OF FIGURES

Figure		Page
1.	Anticipated Findings for Hypothesis 1	11
2.	Anticipated Findings for Hypothesis 2.....	12
3.	Conceptual Model.....	13
4.	Confirmatory Factor Analysis of Social Engagement – Teaching Tasks	29
5.	Confirmatory Factor Analysis of Social Engagement – Free Play.....	30
6.	Maternal Depressive Symptoms x Infant RSA on Total Problems.....	36
7.	Maternal Depressive Symptoms x Infant RSA on Internalizing Problems..	44
8.	Maternal Depressive Symptoms x Infant RSA on Externalizing Problems	45
9.	Postpartum Symptoms x Infant RSA on Maternal Depressive Symptoms..	46

INTRODUCTION

Whereas there is a long tradition of evaluating risk and protective factors for the development of psychopathology, recent theoretical and empirical work indicates that certain individual characteristics may confer risk for physical and mental health outcomes for some people but *promote* health for others. Biological responses to stress are one such characteristic that may be related to either positive or negative health outcomes, depending on an individual's context. The theory of biological sensitivity to context (BSC; Boyce & Ellis, 2005; Ellis & Boyce, 2008; Ellis, Boyce, Belsky, Bakermans-Kranenburg & van IJzendoorn, 2011; Ellis, Essex & Boyce, 2005) posits that the magnitude of biological stress responses accounts for variability in the extent to which individuals are affected by their social environments, for better and for worse.

Autonomic nervous system functioning has received considerable attention within the context sensitivity literature. The myelinated vagus (tenth cranial nerve) acts as a parasympathetically-mediated "brake" on sympathetic influence on the heart's sinus node, which lowers heart rate (Porges, 2007). High vagal tone is thought to reflect one's ability to effectively modulate visceral states in response to both stressful and safe environments, which in turn enables rapid engagement and disengagement with the environment and people in the environment (Beauchaine, 2001; Bornstein & Suess, 2000; Porges, 1996; Porges, 2007). Evidence of the association between high respiratory sinus arrhythmia (RSA), an index of vagal functioning, and emotion reactivity and regulation in infancy is mixed. Several studies have demonstrated positive associations between higher resting RSA and infant's behavioral reactivity, negative emotionality and behavior problems (e.g., Degangi, Dipietro, Greenspan & Porges, 1991; Porges, Doussard-

Roosevelt, Portales & Suess, 1994; Stifter & Fox, 1990), whereas other studies have found the opposite pattern of associations (Huffman et al., 1998; Porter, Porges & Marshall, 1988). Others have suggested resting levels of RSA may be a biological marker of sensitivity to context, which manifests differently in high versus low stress social environments (Ellis & Boyce, 2008). In stressful social environments with few resources, a highly sensitive vagal brake (reflected by greater resting levels of RSA) may lead to psychological maladjustment, whereas in protective social environments that offer numerous resources, it may promote exceptional wellbeing (Ellis et al., 2011).

There is mounting evidence that high resting levels of RSA may confer susceptibility to one's environment, specifically the caregiving environment. Eisenberg and colleagues (2012) demonstrated that baseline RSA and environmental quality (a composite of parental education, family income and marital adjustment) at 18 months interacted to predict developmental trajectories of children's aggression from 18 to 54 months in a community sample. At high levels of environmental quality, there was a significant negative relation between RSA and aggression, such that mothers of toddlers with high baseline RSA rated their children as being less aggressive over time and at 54 months. Whereas evidence of BSC was only found in supportive (high quality) environments, it is possible that the restricted range on the measure of environmental quality employed in Eisenberg and colleagues' (2012) study, in which most of the families were educated and working- or middle-class, precluded detection of susceptibility to low levels of environmental quality.

More consistent with BSC, in a longitudinal study of 73 low-income, predominantly European American mother-child dyads, Conradt, Measelle and Ablow

(2013) demonstrated a significant cross-over interaction effect between children's resting RSA at 5 months and children's attachment security at 17 months on children's problem behaviors at 17 months of age. Whereas there was no difference in problem behaviors among low baseline RSA infants based on their attachment (secure-disorganized), among high baseline RSA infants, problem behaviors were contingent upon attachment organization. Securely attached infants who had high baseline RSA demonstrated levels of problem behaviors that were lower than established norms for low-risk, community samples of children. In contrast, disorganized infants who had high baseline RSA displayed significantly more problem behaviors than their secure counterparts. Thus, even among a high-risk, low-income sample, children who are most susceptible to the effects of the early caregiving environment can reap the benefits of positive parent-child relationships, but may also be most adversely impacted by dysregulated parent-child relationships.

Children with high resting RSA may also be more sensitive to the presence or absence of maternal psychopathology. Among a socioeconomically and ethnically diverse sample overselected for conduct problems, Bandon and colleagues (2008) demonstrated a significant interaction effect of maternal depressive symptoms and children's baseline RSA at four years of age on the development of children's emotion regulation abilities over the following three years. Maternal depressive symptoms when children were four years of age were negatively associated with mother-reports of children's emotion regulation abilities at seven years of age, but only for children with high baseline RSA at age four (Bandon et al., 2008). These effects were further conditioned by an interaction with children's age. Among children with low baseline

RSA, when mothers exhibited high levels of depressive symptoms, emotion regulation abilities remained stable from four to seven years of age, whereas when mothers exhibited low levels of depressive symptoms, emotion regulation abilities increased gradually. Conversely, for children with high baseline RSA, emotion regulation abilities increased the most rapidly when mothers exhibited low levels of depressive symptoms, and decreased the most rapidly when mothers exhibited high levels of depressive symptoms. These findings are consistent with BSC, suggesting that high levels of RSA may act as biological sensitivity factor to the effects of maternal depressive symptoms on children's developing emotion regulation abilities.

Postpartum depression represents a specific form of early-life environmental adversity that confers risk for children. Postpartum depression may affect a mother's ability to provide her infant with support and emotional warmth during one of the most sensitive developmental periods (Nelson et al., 2007; Perry, 2002). A large body of work suggests that on average, depressed mothers display fewer positive emotions and more negative emotions, are less verbal, and disengage more during interactions with their infant children (Ashman & Dawson, 2002; Goodman & Tully, 2006; Lovejoy et al., 2000). In addition, depressed mothers are slower to respond and are less sensitively responsive to their infants than nondepressed mothers (Ashman & Dawson, 2002; Goodman & Gotlib, 1999). For example, relative to nondepressed mothers, depressed mothers are more likely to respond to their infant's regulatory signals by withdrawing, physically moving the infant away, and terminating or rejecting the infant's initiation of contact (Dawson, 1999, cited in Ashman & Dawson, 2002). Whereas some depressed women develop a withdrawn interactional style, others may behave in ways that are

intrusive, such as engaging in overstimulating verbal and physical behavior (Cohn et al., 1990). Women who are not clinically depressed, but demonstrate high levels of depressive symptoms on self-report scales, also exhibit poorer parenting behaviors (Goodman & Tully, 2006; Harnish et al., 1995; Moehler et al., 2006; Weinberg et al., 2001). Overall, research suggests that depressed mothers are typically less competent at fostering an environment that nurtures children's developing emotion regulation abilities (Ashman & Dawson, 2002), especially depressed mothers from disadvantaged socioeconomic backgrounds (Goodman & Tully, 2006).

Whereas there is evidence that maternal depressive symptoms in the first few months postpartum influence infant and toddler behavior problems (Avan et al., 2010; Beck, 1998; Choe, Sameroff & McDonough, 2013; Guyon-Harris et al., 2015; Murray & Cooper, 1997), few researchers have explored whether these effects extend to problems in the early childhood years (3-5 years of age; Walker et al., 2013). This is a critical gap in the literature as early childhood represents an important period in children's socioemotional development. Psychological symptoms in toddlers have been shown to persist over time (Briggs-Gowan et al., 2006; Mesman et al., 2001) and to predict later distress (Mesman & Koot, 2001). Further, in their review of the effects of postpartum depression on child development, Murray and Cooper (1997) note that the early childhood outcomes of children of mothers who had postpartum depression are less consistent than findings obtained in infancy, a conclusion echoed over a decade later by Walker and colleagues (2013).

Some studies have demonstrated associations between maternal postpartum depressive symptoms and behavioral outcomes in early childhood. Jusiene and colleagues (2015)

demonstrated that children with stable, high levels of attention, behavior, and emotion regulation problems from 18 months to 4 years of age were more likely to have had mothers with higher depressive symptoms when they were 3 months of age as compared to children with more typical trajectories of regulatory difficulties (Jusiene, Breidokiene & Pakalniskiene, 2015). Similarly, Carter and colleagues (2001) found self-reported depressive symptoms at 30 days postpartum were associated with children's problem behaviors and competencies at 30 months (Carter et al., 2001). In addition, Field and colleagues (1996) demonstrated dysphoric mothers rated their children as having more internalizing and externalizing problems in preschool (Field et al., 1996). However, in this longitudinal study, because women were excluded if they "crossed-over" from dysphoric to non-dysphoric groups, dysphoria when children were 3 months of age was confounded with dysphoria when children were in preschool.

Other studies have failed to detect a relation between maternal postpartum depression and children's psychological functioning. One prospective study of mostly white, middle-class women found postpartum depression did not predict mother reports of children's internalizing or externalizing behaviors at 4.5 years (Philipps & O'Hara, 1991). Among a nationally representative sample of Canadian women with postpartum depression, Walker and colleagues (2013) found that postpartum depression in the first year of the children's life was associated with children's anxiety, but not with their hyperactivity-inattention, physical aggression-opposition, or with their separation anxiety, after controlling for obstetric, environmental and sociodemographic factors (including current maternal depression) at 2-3 years of age.

These studies raise questions about what contributes to the variability in the consequences of maternal postpartum depressive symptoms on youth development. Guided by a BSC perspective, this study aims to address whether variability in the extent to which postpartum depressive symptoms affect behavior problems in early childhood can be accounted for by biological sensitivity to context. Specifically, one aim of this study is to assess whether children with high resting levels of RSA are more sensitive to the effects of maternal postpartum depressive symptoms on children's behavior problems at 36 months.

In addition to identifying biological sensitivity factors, it is important to evaluate behavioral processes by which these factors impact health outcomes later in life. With the search for sensitivity factors in its infancy, little is known about the mechanisms of these “for better and for worse processes” (Ellis et al., 2011). Ultimately, however, the effects of biologically-based heightened sensitivity to the environment must arise from susceptible individuals' behavioral interactions in their social context (Ellis & Boyce, 2011; Ellis et al., 2011). Highly susceptible individuals are presumed to adapt to their early social environments in ways that maximize survival and fitness, but may have negative long-term health consequences if these environments are aversive.

Research on the unique regulatory strategies children of depressed mothers develop may shed some light on the potential behavioral mechanisms of biological sensitivity to postpartum depressive symptoms. According to Tronick and Gianino's mutual regulation model, when mothers fail to sensitively respond to their infant's signals, infants become withdrawn and minimize use of “approach” strategies in favor of less sophisticated, independent self-regulatory strategies, such as self-soothing and gaze

aversion (Ashman & Dawson, 2002; Gianino & Tronick, 1988; Tronick & Gianino, 1986). Numerous empirical studies demonstrate that infants of depressed mothers display a less positive, more withdrawn interactional style with their mothers as well as with strangers (Ashman & Dawson, 2002), and these findings have been replicated across diverse cultural and socioeconomic groups (Cohn & Campbell, 1992). Whereas the development of attachment bonds is one of the most salient developmental tasks during the second six months of life, research suggests infants of depressed mothers are less likely to be affectionate and socially engaged with their mothers during free play, and are less likely to touch their mothers to gain their attention during a moderately challenging situation during this period (Cohn & Campbell, 1992; Dawson et al., 1999; Feldman et al., 2009).

Highly susceptible infants may demonstrate different levels of engagement with their mothers, depending on their mothers' levels of depressive symptoms during the postpartum period. Even if their mothers are no longer depressed at the end of the first postpartum year, infants may still display impaired behavioral patterns (Cohn & Campbell, 1992). Paradoxically, although disengagement from mothers may be an adaptive response to depressed mothers' inconsistent, unresponsive, or harsh parenting, it may make children miss out on opportunities for developing co-regulation skills and positive social interactions (Goodman & Gotlib, 1999). Social engagement during mother-child play predicts children's developing regulatory abilities (Feldman et al., 2012). Infants and their caregivers are expected to engage in co-regulatory processes; infants' own regulatory capacities are limited and poorly implemented, and therefore infants need external support to effectively regulate their internal states (Gianino &

Tronick, 1988; Tronick & Gianino, 1986). Without parental support and scaffolding in developing social and emotional skills, children's regulatory repertoire is narrowed and children are at a disadvantage in developing the ability to effectively manage stressors on their own (Gianino & Tronick, 1988; Goodman & Gotlib, 1999; Tronick & Gianino, 1986; Tronick & Reck, 2009). Over time, children's inability to cope with stressors on their own leads to dysregulation and increases risk for developing emotional and behavioral problems in early childhood (Contreras et al., 2000; Dawson et al., 2003; Tronick & Reck, 2009). Children who have difficulty regulating their internal state or behaviors may not only be ill-prepared for school entry, but may also be starting on a developmental trajectory to internalizing or externalizing problems (Mesman et al., 2001).

Current Study

The current study assessed whether resting RSA acts as a sensitivity factor for the effects of maternal postpartum depressive symptoms on children's internalizing and externalizing behavior problems and evaluated whether children's social engagement is a mediating behavioral pathway from biological sensitivity to behavior problems. These processes were evaluated among very low-income, Mexican American women and their children who participated in a broader, longitudinal study of children's development (*Las Madres Nuevas*). It is important to address these questions in this population for several reasons. First, nearly a quarter of low-income women meet criteria for a depressive disorder in the first three months postpartum (Yonkers et al., 2001; Hobfoll et al., 1995), and low-income, Hispanic women are at elevated risk for postpartum depressive symptoms (Chaudron et al., 2005; Howell, Mora & Leventhal, 2005; Kuo et al., 2004). In

addition, low-income women's parenting and their children's regulatory behaviors are more likely to be affected by postnatal depressive symptoms (Cohn & Campbell, 1992; Goodman & Tully, 2006). Although many low-SES children show resilience to the effects of early environments, those who receive little parental nurturance and warmth may be at even greater risk for health problems later in life (Miller & Chen, 2013; Perry, 2002). Finally, children raised in low-income, Mexican American families are also at risk for self-regulation and interpersonal skill deficits (Galindo & Fuller, 2010).

The first aim of the study was to examine whether children with high resting RSA at 6 weeks of age are more sensitive to the effects of maternal depressive symptoms during the postpartum period (child age 6 weeks to 6 months) on behavioral problems at 36 months of age. In the present study, children's biological sensitivity (resting RSA) was assessed at 6-weeks of age, at the start of the time period during which maternal postpartum depressive symptoms were measured. Relative to children with lower resting RSA, I predicted children with higher resting RSA will exhibit more problem behaviors if raised by a mother who had elevated postpartum depressive symptoms but fewer problem behaviors if raised by a mother who had minimal postpartum depressive symptoms. Given the association between maternal depression and other risk factors for children's behavior problems (e.g., genetic influence; prenatal programming; exposure to other stressors outside of the social environment), the association between postpartum depressive symptoms and problem behaviors was expected to be positive for all children, although the expected association is smaller in magnitude for children with lower resting RSA.

The second aim of this study was to evaluate whether observed child social engagement with his/her mother acts as a behavioral mechanism by which biological sensitivity impacts development. Higher RSA children whose mothers experienced higher postpartum depressive symptoms are expected to engage less with their mothers, whereas higher RSA children whose mothers experienced fewer postpartum depressive symptoms are expected to exhibit more social engagement. Among lower RSA children, child social engagement levels should not be strongly related to maternal postpartum depressive symptoms (see Figure 1 for anticipated findings).

Figure 1.

Anticipated Findings for Hypothesis 1

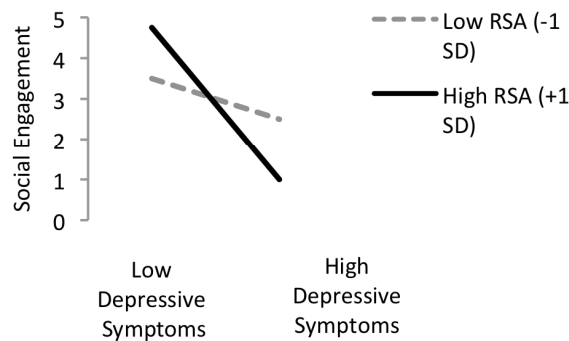


Figure 1. Anticipated Findings for Hypothesis 1

Further, child social engagement with their mothers during a potentially frustrating task at 12 months may predict child problem behaviors at 36 months of age. Thus, the current study's final aim was to evaluate a longitudinal mediated moderation model where 1) resting RSA at 6 weeks of age and maternal depressive symptoms from 6 weeks to 6 months postpartum interact to predict child social engagement at 12 months

and 2) child social engagement at 12 months negatively predicts internalizing and externalizing problem behaviors at 36 months (see Figures 2 and 3).

Figure 2.

Anticipated Findings for Hypothesis 2

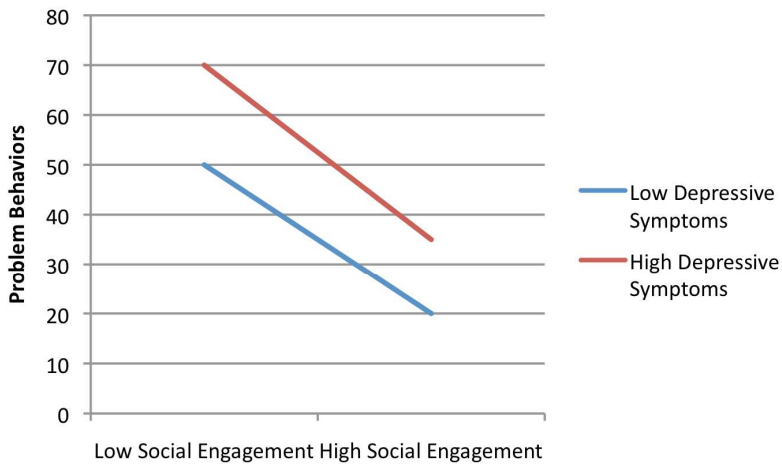
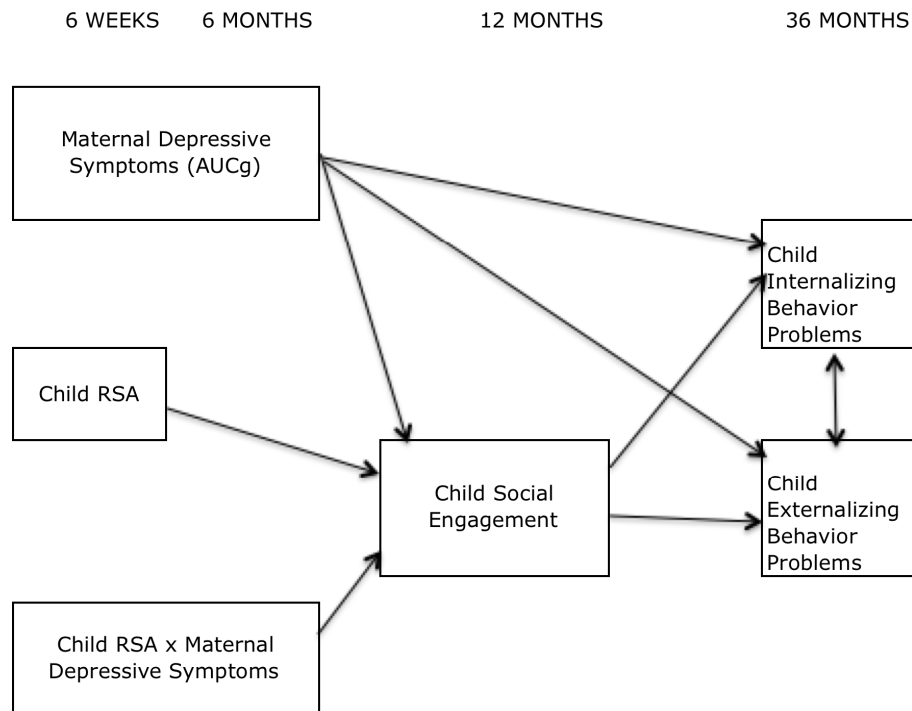


Figure 2. Anticipated Findings for Hypothesis 2

Figure 3.

Conceptual Model



METHODS

Participants

The sample consists of 322 women and their children who participated in a broader examination of very low-income, Mexican American children's development, *Las Madres Nuevas*. Eligibility criteria for the study included: 1) self-identification as Mexican or Mexican American, 2) fluency in English or Spanish, 3) 18 years of age or older, 4) low-income status (defined as family income below \$25,000 or eligibility for Medicaid or Federal Emergency Services coverage for childbirth), and 5) anticipated delivery of a singleton birth based on an ultrasound. The Arizona State University IRB

and the Maricopa Integrated Health System IRB approved all study procedures prior to study inception.

Women were between 18 and 42 years of age when they entered the study; the average age at entry was 27.8 years (SD = 6.5). Most women (86.3%) who participated in the study were born in Mexico. On average, women had moved to the United States 17 years prior to study entry (ranging from less than a year to 35 years ago). Women had an average of 10 years of education. Most (83.8%) were not employed at the time of study entry; 8.7 percent were working part-time and 7.8 percent were working full-time. The modal family income was \$10,001 - \$15,000 for an average household size of 4.34. Approximately forty-six percent of women were living with a partner but not legally married; 29.9 percent were married and living together; 15.3 percent were never married and not living with a partner; 7.5 percent were separated; and 1.9 percent were married but not living together with their partner. On average, women in the sample had two other biological children. Approximately twenty-two percent of the sample was first-time mothers; of the multiparous women, the number of other biological children ranged from one to nine. Twenty-three percent of the sample had one other child, nineteen percent had two other children, eighteen percent had three other children, and seventeen percent had more than three other children. The women who participated gave birth to 147 (45.9%) boys and 173 (53.7%) girls.

Recruitment

During pregnancy, women were recruited from hospital-based prenatal clinics that serve low-income women by a bilingual interviewer from the research team. A bilingual female interviewer from the research team obtained informed consent in the women's

homes between 26-39 weeks gestation. Data for the present study comes from depressive symptom inventories completed with women every three weeks from 6 weeks to 6 months postpartum, and laboratory visits completed when children were 12 months and 36 months of age.

Planned Missingness

To reduce participant burden, a “planned missing” design was employed. Participants were randomly assigned to miss either the 12-, 18- or 24-week postpartum visit, which produces data missing completely at random (MCAR) and does not introduce bias into parameter estimates (Enders, 2010). Data were corrected for planned missingness using Full Information Maximum Likelihood (FIML; Allison, 2003).

Of the 322 women who met inclusion criteria and consented to participate in the study during the initial home visit, 312 (97%) completed the 6-week assessment, 307 (95%) completed the 9-week assessment, 203 (99% of expected completers) completed the 12-week assessment, 302 (94%) completed the 15-week assessment, 209 (96% of expected completers) completed the 18-week assessment, 299 (94%) completed the 21-week assessment, and 209 (93% of expected completers) completed the 24 week assessment.

Procedures

Interviews at 6-weeks, 12-weeks, 18-weeks, and 24-weeks postpartum were conducted in participants’ homes. The 9-week, 15-week and 21-week assessments were conducted over the telephone. Approximately eighty percent of interviews were conducted in Spanish. Given the varying degrees of literacy in the sample, a bilingual interviewer read survey questions aloud to all participants, and participants were given

visual aids with written and graphic depictions of item response formats. Women were compensated \$50 and small gifts for the child (e.g., bibs, rattles) were provided for the 6, 12, 18, and 24-week interviews. Women were compensated \$10 for each telephone assessment they completed.

The 12-month and 36-month visits were conducted in the laboratory. During these visits, women completed questionnaires and several interaction tasks with their children, including a five-minute free play interaction and four four-minute teaching tasks. Mothers were first lead to the filming room, where there was a bin of toys, and were instructed to play with their baby as they would at home. After having their baby clean up the toys, they completed the four teaching tasks. During the 12-month visit teaching tasks, mothers were instructed to have their infants take shapes out of a box, roll a ball back and forth, make a tower with blocks, and use a pop-up toy. These teaching tasks are part of the commonly-used Crowell Procedure (Crowell & Feldman, 1988; Crowell, Feldman & Ginsberg, 1988). An experimenter in another room called the mother on the phone before each task to let her know what to do, and again after four minutes, to prompt the mother to move onto the next task. These teaching tasks were selected to be slightly beyond children's developmental abilities and therefore potentially frustrating for the child. Women were compensated \$100 for their time during each of the laboratory visits. Transportation to the laboratory was provided or women were reimbursed up to \$50 for their travel costs.

Measures

Resting RSA

Respiratory sinus arrhythmia (RSA), the degree of change in heart rate during the respiratory cycle, is a widely-used index of parasympathetically-mediated influences on cardiac output (Beauchaine, 2001). Mean levels of resting RSA gradually increase from birth through early childhood, and research suggests the rank-order of resting RSA is stable in the first 5 years of life in middle-to-upper-SES children (Bornstein & Suess, 2000) as well as in low-income Latino children (Alkon, Boyce, Davis & Eskenazi, 2011). However, given that there is also preliminary evidence to suggest that among some children, changes in resting RSA levels may be affected by early childhood experiences such as harsh parenting and marital conflict (El-Sheikh & Hinnant, 2011; Hinnant, Erath & El-Sheikh, 2015), it is important to assess children's RSA prior to prolonged environmental exposure. RSA was assessed at 6 weeks of age, at the start of the period (from 6 weeks to 6 months postpartum) in which maternal depressive symptoms were assessed.

At 6 weeks of age, infants were seated upright at rest and a research assistant placed electrodes on the infants' left shoulder and right and left waist in a standard lead configuration. Child heart rate data were recorded at 256 Hz with electrocardiography (ECG) equipment from Forest Medical, LLC (Trillium 5000; East Syracuse, NY, USA) during the 7-minute resting period.

QRSTool software 1.2.2 (Allen, Chambers, & Towers, 2007) was used to process the data and automatically obtain R-spikes from the ECG data. Trained undergraduate and graduate coders then used the QRSTool software to manually correct misidentified or

unidentified R-spikes, and obtain R-R interval data. Using CardioBatch software (Brain-Body Center, 2007), a moving polynomial filter was then applied to the R-R interval data to extract heart rate variability in the frequency band of RSA (for infants, 0.3-1.3 Hz). The resting RSA estimates from this analysis were log-transformed, and a mean resting RSA value averaged from 30-second epochs during the first 5 minutes of the resting period was obtained.

Maternal postpartum depressive symptoms

The Edinburgh Postnatal Depression Scale (EPDS; Cox, Holden & Sagovsky, 1987) was administered every three weeks from 6 weeks to 6 months (at 6 weeks, 9 weeks, 12 weeks, 15 weeks, 18 weeks, 21 weeks, and 24 weeks). The EPDS is a 10-item, widely used measure of perinatal depressive symptoms experienced in the past week. Women respond using a scale from 0 to 3, and higher scores correspond to more severe depressive symptoms. The EPDS has been validated in English and Spanish (Garcia-Esteve, Escaso, Ojuel & Navarro, 2001).

An error in the response set rendered 82 of the participants' data unusable at the 6-week, 50 participants' data unusable at the 12-week, 42 participants' data unusable at the 18-week and 43 participants' data unusable at the 24-week time points. For these participants, multiple imputation (Mplus 7; Muthén & Muthén, 2012) was used to impute item-level values for these participants. The imputation variables included parity, survey administration language, the two depression items, and parcels created by averaging the remaining eight EPDS items. Across all waves, total scores on the EPDS ranged from 0 to 26; average scores ranged from 4.55 (*SD*: 4.98) at 6-weeks to 3.81 (*SD*: 4.49) at 24-weeks. Means, standard deviations, skewness and at each wave are presented in Table 1.

Depressive symptoms at each wave were highly positive correlated with each other, with correlations ranging from $r = .46$ (6-week with 21-week) to $r = .69$ (12-week with 15-week). Depressive symptoms from 6 weeks to 6 months were calculated as area under the curve with respect to ground (AUCg), to capture total depressive symptoms during this postpartum period. AUCg offers advantages over other summary measures (e.g., average depressive symptoms or total depressive symptoms) in that it reflects the overall magnitude of depressive symptoms over multiple time points during a specified period (Pruessner et al., 2003).

Table 1.

Descriptive statistics for the EPDS at each wave from 6 weeks to 6 months postpartum

EPDS Wave	Mean	SD	Skewness	Kurtosis
6-week	4.29	4.83	1.24	0.86
9-week	3.61	4.24	1.56	2.51
12-week	3.65	4.22	1.20	0.65
15-week	3.30	3.95	1.41	1.42
18-week	3.95	4.44	1.24	0.85
21-week	3.46	4.25	1.39	1.39
24-week	3.67	4.37	1.54	2.63

Maternal depressive symptoms

The Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977) was administered to assess mothers' depressive symptoms when their children were 36 months of age. The CES-D is a 20-item, widely used measure of depressive symptoms.

Women are asked to report the extent to which they experienced somatic, affective,

cognitive and behavioral depressive symptoms in the past week. Sample items include “I was bothered by things that usually don’t bother me” and “I felt that everything I did was an effort.” Items are rated on a scale from 0-4, with higher scores indicating greater frequency of depressive symptoms. The CES-D has good psychometric properties (Radloff, 1977) and has been validated in Spanish among Mexican Americans (Roberts, Vernon & Rhoades, 1989). Internal consistency at 36 months was good, Cronbach’s alpha = .892.

Child social engagement

Child social engagement during each of the four teaching tasks and free play was assessed using the Coding Interactive Behavior (CIB; Feldman, 1998) rating system. The CIB contains 43 global codes for rating parent-infant interactions and has good psychometric properties, including adequate test-retest reliability, convergent validity with other observational coding schemes, and predictive validity with respect to children’s self-regulation, behavior problems and social competence (Feldman, 2012). The CIB has been validated among dyads from diverse ethnic and socioeconomic backgrounds (Feldman & Masalha, 2007; Dollberg et al., 2013). Children’s social engagement has been shown to increase over time but to demonstrate within-person stability from infancy to childhood (Feldman, 2010). In studies conducted in multiple countries, children’s social engagement with mothers has been negatively related to maternal depressive symptoms in infancy and early childhood (Apter-Levy et al., 2013; Feldman, 2012; Feldman et al., 2009).

Child social engagement is based on ratings of children’s initiation, vocalizations/verbal output, joint attention, expression of positive affect, and negative

emotionality/fussiness (reverse-coded). Each scale is rated from 1 to 5, where higher scores indicate more frequent displays of a behavior. Composite scores can be created from the CIB global codes. Scores on the social engagement composite can therefore range from 5 to 25, with higher scores indicating more frequent displays of social engagement. Alternatively, factor scores on child social engagement can be estimated given an acceptable factor solution; infant social engagement factor scores were treated as a manifest variable in the present analyses.

Trained undergraduate coders coded these scales; 20% of videos were checked against master coders to continually assess reliability. Of these videos, the percent agreement averaged 92.6% across the free play tasks and 91.1% across the teaching tasks, which exceeds the standard of 85% set forth in Feldman's (1998) coding manual. Cronbach's alpha for social engagement was .819 for the teaching tasks; reliability within tasks ranged from .518 to .742.

Child problem behaviors

Mothers reported on their children's behavior problems at 36 months using the Child Behavior Checklist (CBCL/1.5-5; Achenbach & Rescorla, 2000). The CBCL is a 113-item questionnaire that yields information on children's internalizing, externalizing and total behavior problems. Child total behavior problems include internalizing, externalizing, sleep and other problems. Scale scores are created by summing responses to the individual items corresponding to the scales; there was no missing data on any of the individual items. The CBCL is validated in English and Spanish (Achenbach & Rescorla, 2000; Rubio-Stipec, Bird, Canino, & Gould, 1990).

The CBCL internalizing and externalizing scales were highly correlated, $r = .763$, $p < .001$. Thus, two sets of analyses were conducted, one in which internalizing and externalizing symptoms were correlated in the model, and another in which a composite of total problem behaviors was predicted.

Potential covariates

Child gender and date of birth, birth outcomes (birth weight, gestational age, APGAR score), and mother's country and age at prenatal visit were obtained through medical record review. Parity (number of biological children) was obtained from mother's report at the prenatal visit.

Data Analysis

Missing data

Of the 322 cases, 65 (20.2%) participants were missing on the measure of infant RSA. Reasons for missing RSA data included participant attrition (3.7%) and unusable data (16.5%). Because funding was not initially available for an in-person visit at 1-year postpartum, when funding was available, only 266 out of 322 children were eligible for the 1-year visit (i.e., child age between 10.5 and 13.5 months); of eligible families, 206 (77%) completed the 12-month visit. Of the 206 women who completed the 12-month visit, 173 (84.0%) had available data on parent-child interactions during teaching tasks and 157 (76.2%) had available data on parent-child interactions during free play. One hundred and sixty-five (51.2%) participants were missing on the measure of infant social engagement during free play. Reasons for missing data of those who completed the laboratory visit included the task not being coded at the time of data analysis, mother failing to follow instructions (e.g., not completing the task; $n = 2$) or videorecording error

($n = 1$). Two hundred and fifteen women completed the 36-month laboratory visit. One hundred and seven (33.2%) participants were missing on the measures of internalizing, externalizing and total behavior problems.

Participants with missing data on infant RSA did not differ from participants with complete data with respect to maternal age at prenatal visit; maternal country of origin; number of biological children; child birth outcomes (1-minute APGAR, gestational age, birth weight); child sex; maternal depressive symptoms at 36 months; or child internalizing, externalizing or total behavior problems at 36 months (all p 's < .05). Participants with missing data on infant social engagement had children with more biological siblings, $t(318) = 3.347, p = .001$, lower birth weight, $t(319) = 2.303, p = .022$, and were more likely to have to have been born in the United States, $\chi^2(2) = 14.7, p = .001$. Participants with missing data on child internalizing, externalizing and behavior problems were more likely to be younger at the prenatal visit, $t(319) = 2.70, p = .007$. All analyses were conducted with Mplus v. 7.2 (Muthen & Muthen, 1998-2012) which uses all available values and maximum likelihood estimation, which is superior to pairwise or listwise deletion (Enders, 2001).

Outliers on primary study variables

No potential outliers (3 SD from mean) were identified on infant RSA or social engagement. One case was a potential outlier on depressive symptoms from 6 to 24 weeks (AUCg); the participant exhibited high levels of depressive symptoms at 5 of the 7 time points, but was only 3 SD above the mean at two of these time points (21- and 24-weeks). The pattern of results did not significantly change when this case was excluded from the analyses; therefore, the data were retained for analyses. Two potential outliers

were identified on child internalizing problems; one of these was also a potential outlier on child externalizing problems and child total behavior problems at 36 months. The pattern of results did not change when these cases were excluded from the analyses; therefore, they were retained for the analyses presented here.

Preliminary analyses

Preliminary analyses were conducted to identify the latent construct of social engagement. Social engagement was assessed during four teaching tasks and during free play. Separate confirmatory factor models with a unitary latent social engagement construct with loadings on positive affect, negative affect (reverse-coded), vocalizations, gaze and initiation were tested for both contexts (teaching tasks and free play).

Primary analyses

Primary analyses tested mediation models of the interaction effect of maternal postpartum depressive symptoms and infant RSA on children's internalizing and externalizing or total behavior problems by infant social engagement. I tested these mediation models by examining the statistical significance of the indirect paths. The indirect effect from the interaction of maternal postpartum depressive symptoms and infant RSA to infant social engagement to child behavior problems was expected to be significant.

Three sets of analyses were conducted. In the first set (i.e., the base model), only infant social engagement, maternal postpartum depressive symptoms, infant RSA, and their interaction were included in the model. In the second set, covariates identified during preliminary analyses were accounted for in the model. In the final set of analyses, which represents a more stringent test of the hypotheses, maternal depressive symptoms

at 36 months were also accounted for in the model, in addition to identified covariates. Significant interaction effects were probed by testing the significance of the simple slopes of the regression of behavior problems on maternal postpartum depressive symptoms at average, low (-1 SD) and high (+1 SD) levels of infant RSA (Aiken & West, 2001).

RESULTS

Preliminary Analysis

Descriptive statistics

Table 2 presents descriptive statistics for the primary study variables. Table 3 presents zero-order correlations.

Table 2.

Descriptive statistics of primary study variables

	N	Mean	SD	Skewness	Kurtosis	Range
Infant RSA at 6 weeks	257	2.52	0.96	1.09	3.66	0.46, 8.02
Maternal depressive symptoms from 6 weeks to 6 months (AUCg)	322	21.95	20.98	1.16	0.67	0, 100.50
Infant social engagement during free play at 12 months	157	0.00	0.56	-0.07	0.29	-1.69, 1.55
Child internalizing problems at 36 months	215	8.09	6.82	1.54	2.56	0, 36.00
Child externalizing problems at 36 months	215	9.95	7.63	1.00	1.15	0, 43.00
Child total problems at 36 months	215	28.25	21.27	1.13	2.26	1.00,

Table 3.

Zero-order correlations among primary study variables

	1	2	3	4	5	6
1. Infant RSA at 6 weeks	---					
2. Maternal depressive symptoms from 6 weeks to 6 months (AUCg)	.041	---				
3. Infant social engagement during free play at 12 months	-.064	.015	---			
4. Child internalizing problems at 36 months	-.142	.192	-.172	---		
5. Child externalizing problems at 36 months	-.036	.114	-.089	.763	---	
6. Child total problems at 36 months	-.079	.161	-.153	.927	.927	---

Note. Correlation coefficients presented in bold are statistically significant, $p < .05$.

Several potential covariates (maternal country of origin, maternal age, number of other biological children and child sex) were correlated with primary study variables. Maternal country of origin was correlated with infant RSA, $r = -.185$, $p = .003$, and with children's internalizing, $r = -.163$, $p = .017$, externalizing, $r = -.258$, $p < .001$, and total behavior problems, $r = -.231$, $p = .001$. Children whose mothers were born in the United States had higher RSA and more internalizing, externalizing and total behavior problems. Maternal age at prenatal visit was correlated with maternal postpartum depressive symptoms, $r = .121$, $p = .030$, and with children's internalizing, $r = -.153$, $p = .025$, externalizing, $r = -.182$, $p < .008$, and total behavior problems, $r = -.181$, $p = .008$. Child

sex was correlated with children's externalizing problems, $r = -.134, p = .05$. Boys ($M = 11.11, SD = 8.77$) exhibited more externalizing problems than did girls ($M = 9.05, SD = 6.50$). Number of other biological children was positively correlated with maternal age at prenatal visit, $r = .612, p \leq .001$, child birth weight, $r = .203, p \leq .001$, and negatively correlated with child externalizing behavior problems, $r = -.157, p = .022$, and child total behavior problems, $r = -.141, p = .040$. Mothers who were born in Mexico had more biological children, $t(318) = -4.645, p \leq .001$.

Maternal depressive symptoms at 36 months were correlated with infant RSA, $r = -.177, p = .022$, with maternal postpartum depressive symptoms, $r = .349, p \leq .001$, and with children's internalizing, $r = .466, p \leq .001$, externalizing, $r = .436, p \leq .001$, and total behavior problems, $r = .482, p \leq .001$.

Preliminary results

Teaching tasks. For the model of social engagement during the teaching tasks, positive affect, negative affect, vocalizations, gaze and initiation were treated as secondary latent constructs with loadings on the four observed items (e.g., the four observed measures of positive affect during each of the teaching tasks loaded onto the latent measure of positive affect; See Figure 1). The positive and negative affect factors were correlated, and items within task were correlated. This model fit the data better than simpler models of social engagement during the teaching tasks (i.e., models where positive and negative affect were not correlated and where items within task were not correlated). Although this model was the best-fitting model of social engagement during the teaching tasks, $\chi^2(124) = 194.978, p \leq .001$, RMSEA = 0.058 (90% CI: 0.042, 0.073), TLI = 0.871, the secondary factors did not appear to form a higher-order social

engagement factor. As shown in Figure 4 and Table 4, only the gaze and positive affect factors loaded significantly onto the social engagement factor; negative affect, initiation and vocalizations did not load significantly onto the social engagement factor. Given these issues, subsequent analyses examined the construct during free play.

Table 4.

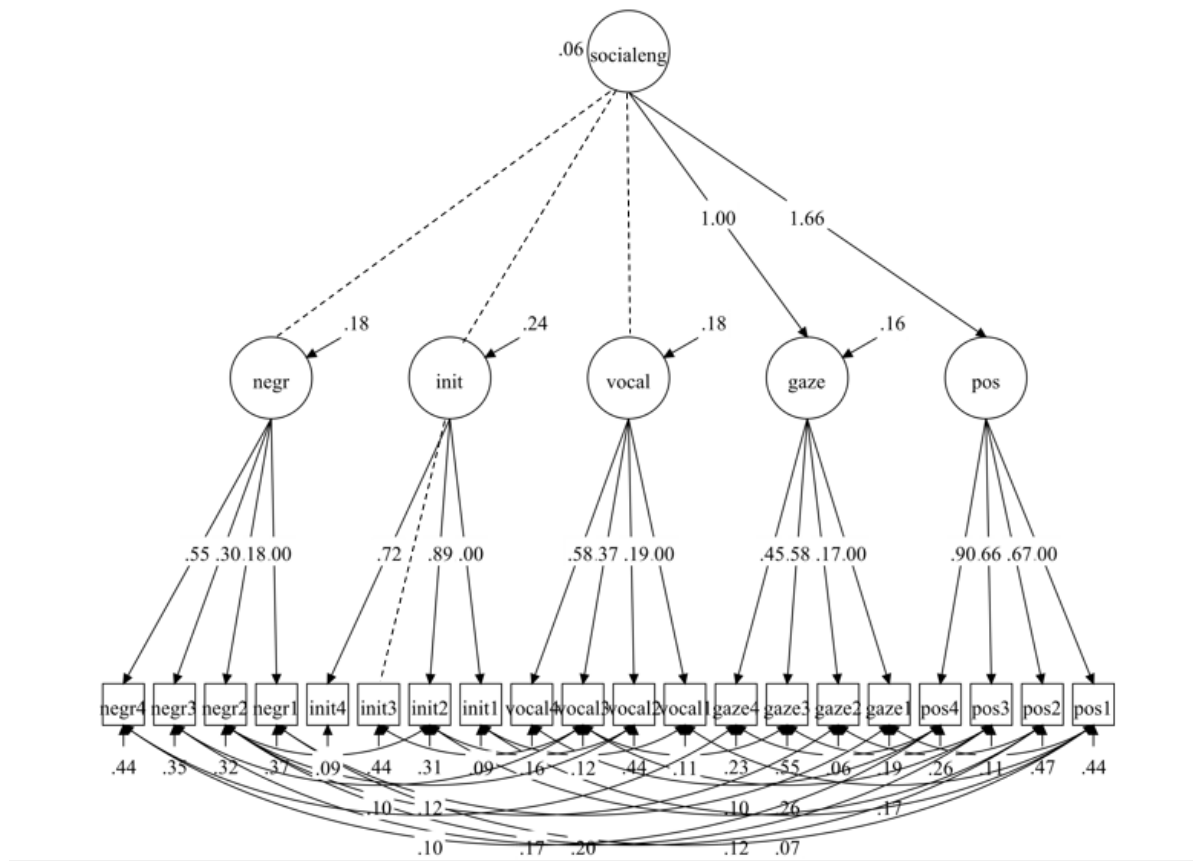
Estimates for one-factor confirmatory model of social engagement during teaching tasks (n=173) and free play (n= 157)

Item/ Construct	Teaching Tasks					Free Play				
	Estimate	SE	p-value	R ²	p-value	Estimate	SE	p-value	R ²	p-value
Positive affect	1.658	0.817	0.042	0.909	0.012	1.000	0.000	999.000	0.753	0.000
Negative affect (reverse-coded)	1.336	0.704	0.058	0.385	0.096	0.441	0.127	0.001	0.223	0.012
Vocalizations	0.960	0.493	0.051	0.245	0.151	0.469	0.146	0.001	0.125	0.049
Initiation	0.449	0.323	0.165	0.050	0.429	0.323	0.111	0.004	0.167	0.013
Gaze	1.000	0.000	9.999	0.284	0.046	---	---	---	---	---

Note. Dashes indicate this construct was not included in the model

Figure 4.

Confirmatory factor analysis of social engagement – teaching tasks

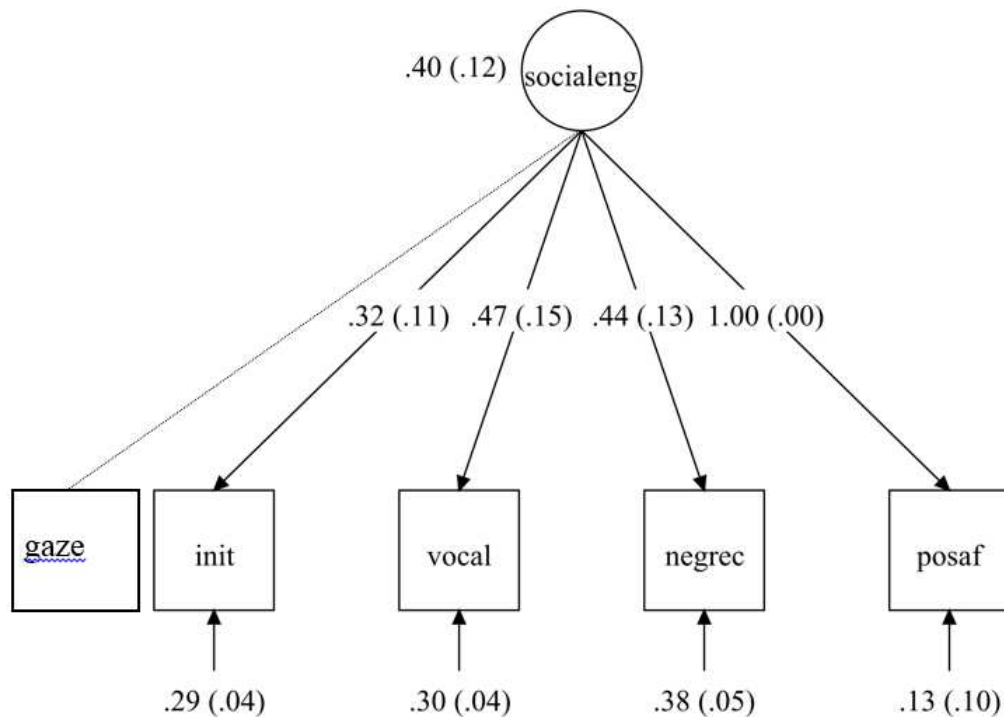


Free play. For the model of social engagement during free play, there was only one observation per item, and therefore, the item-level measures loaded onto the latent construct of social engagement (see Figure 5). With all of the items included, the model was not a good fit for the data, $\chi^2(5) = 12.949$, $p = 0.024$, RMSEA = 0.101 (90% CI: 0.034, 0.170), TLI = 0.780. Gaze did not significantly load onto the social engagement factor, estimate = 0.187, SE = 0.104, $p = 0.07$, although the other items did load onto this

factor. Thus, gaze was removed from the model, resulting in a good fit for the data, $\chi^2(2) = 2.945, p = .23, RMSEA = 0.055$ (90% CI: 0.000, 0.177), TLI = .955. Estimates of loadings are presented in Table 4. Based on the factor loadings and goodness-of-fit indicators, the model of social engagement during free play appeared to fit the data well, and was used for the primary analyses.

Figure 5.

Confirmatory factor analysis for social engagement – free play



Parameter estimates and standard errors are presented on the arrows. Round dotted line indicates item not included in final model.

Primary results

Models predicting total problems. Table 5 presents the results of the model predicting total problems, without covariates included. The interaction term of maternal

postpartum depressive symptoms and infant RSA significantly predicted children’s total behavior problems at 36 months, $B = -0.219$, $SE B = 0.069$, $p = .001$. However, neither infant RSA, $p = .44$, maternal postpartum depressive symptoms, $p = .77$, nor their interaction, $p = .99$, were statistically significant predictors of infant social engagement and the relation of infant social engagement to total behavior problems did not reach statistical significance, $p = .06$. The sum of the indirect effects from the interaction effect of maternal postpartum depressive symptoms and infant RSA was also not statistically significant, $p = .99$.

In the context of a significant interaction, maternal postpartum depressive symptoms were also significantly associated with children’s total behavior problems, $B = 0.195$, $SE B = 0.065$, $p = .003$. The sum of the indirect effects from maternal postpartum depressive symptoms to total behavior problems was not statistically significant, $p = .77$.

Table 5.

Model Predicting Total Problems, Without Covariates

DV	IV	B	SE B	p	R ²
Child total behavior problems					0.105*
	Infant social engagement	-5.541	2.882	0.055	
	Infant RSA	-1.946	1.778	0.274	
	Maternal depressive symptoms	0.195	0.065	0.003	
	RSA x Depressive symptoms	-0.219	0.069	0.001	
Social Engagement					0.006

Infant RSA	-0.044	0.057	0.437
Maternal depressive symptoms	0.001	0.002	0.768
RSA x Depressive symptoms	0.000	0.002	0.990

Note. * indicates statistically significant, $p < 0.05$.

Table 6 presents the results of the model predicting total problems, adjusting for covariates. After adjusting for maternal country of birth, $p = .003$, maternal age, $p = .053$, number of biological children, $p = .74$, and child sex, $p = .46$, the interaction effect of maternal postpartum depressive symptoms and infant RSA on children’s total behavior problems remained statistically significant, $p \leq .001$. Maternal postpartum depressive symptoms also remained a significant predictor of children’s total behaviors, $p = .001$. None of the indirect effects to children’s total behavior problems through social engagement were statistically significant.

Table 6.

Model Predicting Total Problems, Adjusting for Covariates.

DV	IV	B	SE B	p	R^2
Child total behavior problems					0.223**
	Infant social engagement	-5.068	2.739	0.064	
	Infant RSA	-2.103	1.716	0.220	
	Maternal depressive symptoms	0.201	0.063	0.001	

	symptoms			
	RSA x depressive	-0.254	0.066	0.000
	symptoms			
	Country of birth	-15.350	5.232	0.003
	Maternal age	-0.537	0.277	0.053
	Number of biological	0.363	1.102	0.742
	children			
	Child sex	-1.998	2.713	0.462
Social Engagement				0.008
	Infant RSA	-0.051	0.058	0.373
	Maternal depressive	0.000	0.002	0.823
	symptoms			
	RSA x depressive	0.000	0.002	0.992
	symptoms			

Note. ** indicates statistically significant, $p < .001$.

Table 7 presents the results of the model predicting total behavior problems, adjusting for covariates and concurrent maternal depressive symptoms. After adjusting for covariates and concurrent maternal depressive symptoms, $p \leq .001$, the interaction effect of maternal postpartum depressive symptoms and infant RSA on children's total behavior problems remained statistically significant, $p = .009$. Maternal postpartum depressive symptoms were no longer a statistically significant predictor of children's

total behavior problems, $p = .55$. None of the indirect effects to children's total behavior problems through social engagement were statistically significant.

Table 7.

Model Predicting Total Problems, Adjusting for Covariates and Concurrent Maternal Depressive Symptoms

DV	IV	B	SE B	P	R ²
Child total behavior problems					0.335**
		Infant social engagement	-3.079	2.456	0.210
		Infant RSA	-0.029	1.633	0.986
		Maternal depressive symptoms	0.037	0.062	0.549
		RSA x depressive symptoms	-0.166	0.063	0.009
		Country of birth	-14.056	4.709	0.003
		Maternal age	-0.455	0.252	0.071
		Number of biological children	0.196	1.002	0.845
		Child sex	-0.538	2.468	0.827
		Concurrent depressive symptoms	1.119	0.170	0.000
Social Engagement					0.009
	Infant RSA	-0.054	0.058	0.345	

Maternal depressive symptoms	0.001	0.002	0.811
RSA x depressive symptoms	0.000	0.002	0.899

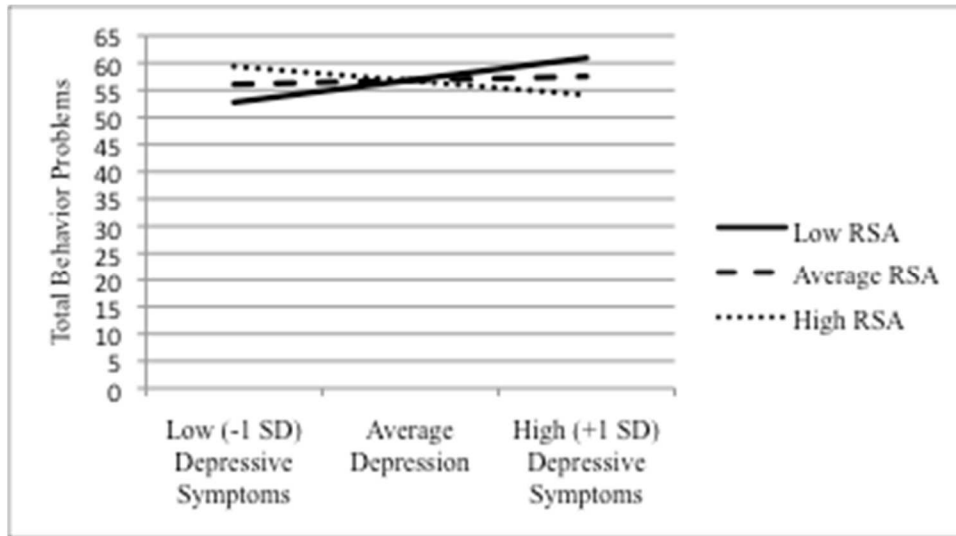
Note. ** indicates statistically significant, $p < .001$.

Post-hoc probing of the interaction effect of maternal postpartum depressive symptoms and infant RSA on children's total behavior problems from the aforementioned model (adjusting for covariates and 36-month maternal depression) revealed that the simple slope of total behavior problems on maternal postpartum depressive symptoms was significant for children with low RSA, $B = 0.194$, $p = 0.044$, but not for children with average RSA, $B = 0.035$, $p = .58$ or children with above average RSA, $B = -0.125$, $p = .11$.¹ As shown in Figure 6, among children with below average RSA, fewer postpartum depressive symptoms were associated with fewer behavior problems, and higher depressive symptoms were associated with more problems. In contrast, children with average and above average RSA showed similar levels of behavior problems at each level of maternal depressive symptoms.

¹The pattern of findings was similar for results of less stringent models; therefore, only the results of the final model are presented here.

Figure 6.

Maternal depressive symptoms x infant RSA on child total problems



Model predicting internalizing and externalizing problems. Table 8 presents the model predicting internalizing and externalizing problems, without covariates included. There were significant interaction effects of maternal postpartum depressive symptoms and infant RSA on children's internalizing behavior problems, $B = -0.082$, $SE B = 0.022$, $p \leq .001$, and on children's externalizing behavior problems, $B = -0.068$, $SE B = 0.025$, $p = .007$. Children's internalizing behavior problems were significantly associated with externalizing behavior problems, $B = 34.691$, $SE B = 4.034$, $p \leq .001$. However, neither infant RSA, $p = .43$, maternal postpartum depressive symptoms, $p = .76$, nor their interaction, $p = .85$, were statistically significant predictors of infant social engagement. Infant social engagement was a statistically significant predictor of children's internalizing behavior problems, $p = .027$, but not children's externalizing behavior problems, $p = .58$. The sum of the indirect effects from the interaction effect of maternal postpartum depressive symptoms and infant RSA to children's internalizing behavior

problems was not statistically significant, $p = .85$. The sum of the indirect effects from infant RSA to children's externalizing behavior problems was also not statistically significant, $p = .64$.

In the context of a significant interaction, maternal postpartum depressive symptoms were also significantly associated with children's internalizing behavior problems, $B = 0.076$, $SE B = 0.020$, $p \leq .001$, as was infant RSA, $p = .029$. Maternal postpartum depressive symptoms were also significantly associated with children's externalizing behavior problems, $B = 0.050$, $SE B = 0.024$, $p = .035$. The sum of the indirect effects from maternal postpartum depressive symptoms to internalizing behavior problems was not statistically significant, $p = .77$. The sum of the indirect effects from maternal postpartum depressive symptoms to children's externalizing behavior problems was also not statistically significant, $p = .79$.

Table 8.

Model Predicting Internalizing & Externalizing Problems, Without Covariates.

DV	IV	B	SE B	P	R ²
Child internalizing behavior problems					0.164*
	Infant social engagement	-1.919	0.868	0.027	
	Infant RSA	-1.234	0.565	0.029	
	Maternal depressive symptoms	0.076	0.020	0.000	
	RSA x depressive symptoms	-0.082	0.022	0.000	

Child externalizing behavior problems				0.054+
	Infant social engagement	-0.645	1.152	0.576
	Infant RSA	-0.319	0.643	0.620
	Maternal depressive symptoms	0.050	0.024	0.035
	RSA x depressive symptoms	-0.068	0.025	0.007
Social Engagement				0.006
	Infant RSA	-0.044	0.056	0.431
	Maternal depressive symptoms	0.001	0.002	0.764
	RSA x depressive symptoms	0.000	0.002	0.851

Note. * indicates statistically significant, $p < .05$. + indicates marginally significant, $p < 0.10$.

Table 9 presents the model predicting internalizing and externalizing problems, adjusting for maternal country of origin, maternal age at prenatal visit, number of biological children and child sex. After adjusting for maternal country of birth, $p = .015$, maternal age, $p = .05$, number of biological children, $p = .52$, and child sex, $p = .79$, infant social engagement remained significantly associated with children's internalizing behavior problems, $p = 0.027$, as did infant RSA, $p = .025$, maternal postpartum depressive symptoms, $p \leq .001$, and the interaction effect of maternal postpartum depressive symptoms and infant RSA, $p \leq .001$. After adjusting for maternal country of

birth, $p = .001$, maternal age, $p = .12$, number of biological children, $p = .98$, and child sex, $p = .23$, maternal postpartum depressive symptoms, $p = .026$, and the interaction effect of maternal postpartum depressive symptoms and infant RSA, $p = .001$, remained statistically significant predictors of children's externalizing behavior problems. None of the indirect effects to children's internalizing or externalizing behavior problems through social engagement were statistically significant.

Table 9.
Model Predicting Internalizing & Externalizing Problems, With Covariates

DV	IV	B	SE B	P	R ²
Child internalizing behavior problems					0.244**
	Infant social engagement	-1.881	0.848	0.027	
	Infant RSA	-1.253	0.558	0.025	
	Maternal depressive symptoms	0.078	0.020	0.000	
	RSA x depressive symptoms	-0.091	0.021	0.000	
	Country of birth	-3.716	1.669	0.026	
	Maternal age	-0.175	0.088	0.047	
	Number of biological children	0.224	0.350	0.522	
	Child sex	-0.167	0.861	0.846	
Child externalizing behavior problems					0.190**

Infant social engagement	-0.569	1.069	0.595	
Infant RSA	-0.395	0.622	0.525	
Maternal depressive symptoms	0.051	0.023	0.026	
RSA x depressive symptoms	-0.081	0.024	0.001	
Country of birth	-6.143	1.892	0.001	
Maternal age	-0.158	0.101	0.230	
Number of biological children	0.009	0.401	0.981	
Child sex	-1.184	0.987	0.230	
Social Engagement				0.008
Infant RSA	-0.052	0.057	0.365	
Maternal depressive symptoms	0.001	0.002	0.806	
RSA x depressive symptoms	0.001	0.002	0.785	

Note. ** indicates statistically significant, $p \leq .001$.

Table 10 presents the model predicting internalizing and externalizing problems, adjusting for covariates and concurrent maternal depressive symptoms. After adjusting for maternal country of birth, $p = .030$, maternal age, $p = .07$, number of biological children, $p = .60$, child sex, $p = .77$, and concurrent maternal depressive symptoms, $p \leq .001$, the interaction effect of maternal postpartum depressive symptoms and infant RSA remained a significant predictor of children's internalizing behavior problems, $p = .002$. After adjusting for maternal country of birth, $p = .001$, maternal age, $p = .16$, number of biological children, $p = .90$, child sex, $p = .46$, and concurrent maternal depressive symptoms, $p \leq .001$, the interaction effect of infant RSA and maternal postpartum depressive symptoms remained a significant predictor of children's externalizing behavior problems, $p = .027$. None of the indirect effects to children's internalizing or externalizing behavior problems through social engagement were statistically significant.

Table 10.

Final Model Predicting Internalizing & Externalizing Problems

DV	IV	B	SE B	P	R ²
Child internalizing behavior problems					0.322**
	Infant social engagement	-1.377	0.781	0.078	
	Infant RSA	-0.670	0.555	0.228	
	Maternal depressive symptoms	0.033	0.020	0.101	
	RSA x depressive	-0.067	0.021	0.002	

	symptoms			
	Country of birth	-3.348	1.539	0.030
	Maternal age	-0.151	0.082	0.067
	Number of biological	0.173	0.326	0.596
	children			
	Child sex	0.233	0.803	0.771
	Concurrent maternal	0.309	0.055	0.000
	depressive symptoms			
Child externalizing				0.300**
behavior problems				
	Infant social engagement	0.111	0.979	0.909
	Infant RSA	0.293	0.593	0.621
	Maternal depressive	-0.006	0.023	0.795
	symptoms			
	RSA x depressive	-0.051	0.023	0.027
	symptoms			
	Country of birth	-5.708	1.726	0.001
	Maternal age	-0.131	0.093	0.159
	Number of biological	-0.047	0.369	0.899
	children			
	Child sex	-0.678	0.907	0.455
	Concurrent maternal	0.388	0.062	0.000
	depressive symptoms			
Social Engagement				0.009
	Infant RSA	-0.055	0.057	0.341

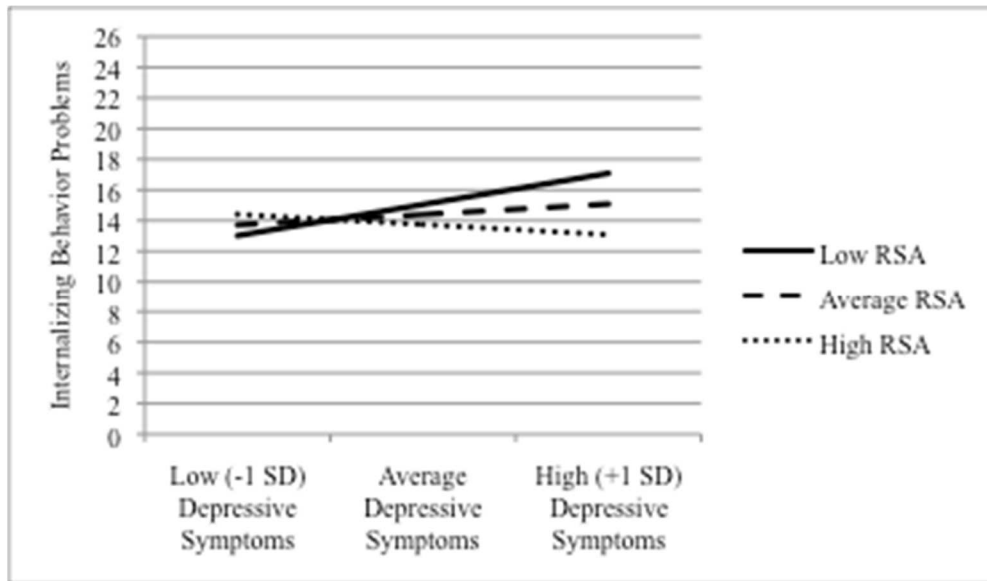
Maternal depressive symptoms	0.001	0.002	0.808
RSA x depressive symptoms	0.001	0.002	0.761

Note. ** indicates statistically significant, $p < .001$.

Post-hoc probing of the interaction effect of maternal postpartum depressive symptoms and infant RSA on children's internalizing behavior problems from the aforementioned model (adjusting for covariates and 36-month maternal depression) revealed that the simple slope of maternal depressive symptoms on child internalizing problems was significant for children with low levels of RSA, $B = 0.097$, $p = .003$, but not for children with average, $B = 0.033$, $p = .12$, or high levels of RSA, $B = -0.032$, $p = .22$. As shown in Figure 7, among children with below average RSA, lower levels of postpartum depressive symptoms were associated with fewer internalizing behavior problems, but higher levels of postpartum depressive symptoms were associated with more behavior problems. In contrast, children with average and above average RSA showed similar levels of internalizing behavior problems at each level of maternal postpartum depressive symptoms.

Figure 7.

Maternal depressive symptoms x infant RSA on child internalizing behavior problems

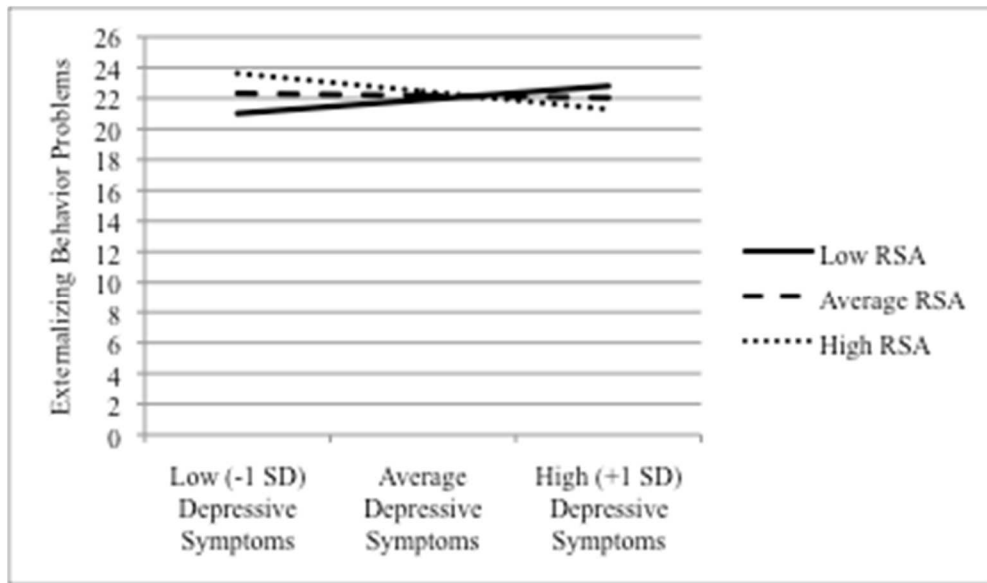


Post-hoc probing of the interaction effect of maternal postpartum depressive symptoms and infant RSA on children's externalizing behavior problems also revealed that the simple slope of maternal depressive symptoms on externalizing problems was marginally significant for children with high levels of RSA, $B = -0.056$, $p = 0.051$, but not for children with average, $B = -.007$, $p = .78$, or children with below average levels of RSA, $B = 0.043$, $p = .23$. As shown in Figure 8, among children with above average RSA, lower levels of postpartum depressive symptoms were associated with more externalizing behavior problems, but higher levels of postpartum depressive symptoms were associated with fewer externalizing behavior problems. In contrast, children with

average and below average RSA showed similar levels of externalizing behavior problems at each level of maternal postpartum depressive symptoms.²

Figure 8.

Maternal depressive symptoms x infant RSA on child externalizing behavior problems



Exploratory analyses

Post-hoc paired samples t-tests suggested that the teaching tasks were stressful, on average, for infants. Positive affect decreased significantly from free play to the first teaching task, $p \leq .001$, and to the last teaching task, $p \leq .001$, and negative irritability/fussiness increased significantly from free play to the first teaching task, $p = .025$, and to the last teaching task, $p = .047$.

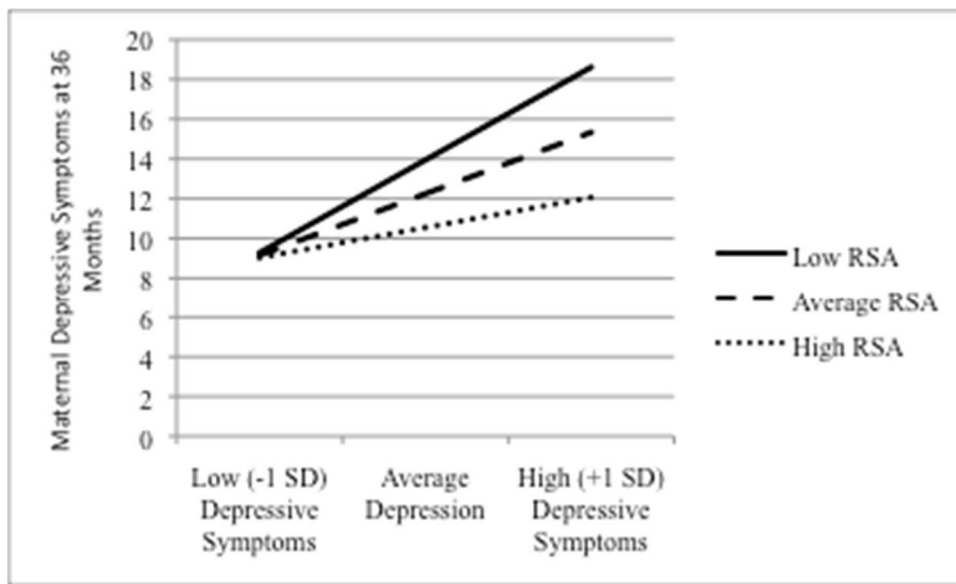
In the models predicting total behavior problems, internalizing and externalizing behavior problems, adjusting for covariates and concurrent maternal depressive symptoms, maternal depressive symptoms at 36 months were significantly predicted by

²The pattern of findings was similar for results of less stringent models; therefore, only the results of the final model are presented here.

the interaction between maternal postpartum depressive symptoms and infant RSA,. Post-hoc probing of the interaction effect revealed that the simple slopes of maternal postpartum depressive symptoms on maternal depressive symptoms at 36 months were positive and significant for children with higher RSA, $B = 0.072$, $SE B = 0.032$, $p = 0.023$, average levels of RSA, $B = 0.147$, $SE B = 0.024$, $p < .001$; and lower RSA, $B = .222$, $SE B = 0.036$, $p \leq .001$; See Figure 9.

Figure 9.

Postpartum depressive symptoms x infant RSA on maternal depressive symptoms



A series of analyses were also conducted to test whether children’s gaze during free play or the square of children’s gaze mediated the interaction effect of infant RSA and maternal postpartum depressive symptoms on children’s behavior problems at 36 months. Gaze and gaze-squared were not associated with any primary study variables, all p ’s $< .05$. Gaze was also not significantly associated with the interaction of infant RSA and maternal postpartum depressive symptoms, $p = .64$, children’s total behavior problems, p

= .78, internalizing behavior problems, $p = .74$, or with children's externalizing behavior problems, $p = .79$.

DISCUSSION

I evaluated biological sensitivity (Boyce & Ellis, 2005) to the effects of mothers' postpartum depressive symptoms on their children's behavior problems, as well as a behavioral pathway by which sensitivity may unfold, among very low-income Mexican American families. I hypothesized that children with higher vagal tone (as indexed by resting RSA) at 6 weeks of age would be more sensitive to the effects of maternal postpartum depressive symptoms on behavior problems at 36 months of age. However, contrary to expectations, the associations between maternal postpartum depressive symptoms and children's internalizing and total behavior problems were only statistically significant for children with *lower* RSA. Additionally, although the association between maternal postpartum depressive symptoms and children's externalizing behavior problems was statistically significant for children with higher RSA, children with higher RSA exhibited fewer externalizing behavior problems when exposed to more maternal postpartum depressive symptoms and more externalizing behavior problems when exposed to fewer maternal postpartum depressive symptoms. Finally, children's observed social engagement with their mothers during free play did not mediate the interaction effect on children's behavior problems. These results held even after controlling for covariates and concurrent maternal depressive symptoms.

Vagal Tone as a Sensitivity Factor to Environmental Context

Children with relatively lower RSA demonstrated more internalizing and total behavior problems when exposed to higher levels of maternal postpartum depressive

symptoms but fewer internalizing and total behavior problems when exposed to lower levels of maternal postpartum depressive symptoms; in contrast, children with average and higher levels of RSA demonstrated similar levels of internalizing and total behavior problems regardless of maternal postpartum depressive symptoms. Although the empirical literature has supported the role of higher vagal tone as a sensitivity factor, there is ongoing debate as to whether lower vagal tone could confer susceptibility to environmental influences. In a recent review, Obradovic (2012) argued that low parasympathetic nervous system (PNS) reactivity might better capture susceptibility than higher PNS activity based on evidence that children with elevated PNS activity are less affected by their environmental context. Studies have demonstrated stronger associations between a range of environmental contexts and children's behavior problems for children with lower, rather than higher, resting RSA (El-Sheikh, Harger & Whitson, 2001; El-Sheikh, 2005; Hastings & De, 2008; McLaughlin et al., 2015).

The mixed state of the literature may be reconciled by considering differences in the environments under investigation and the range of environmental quality experienced by a sample in a given study (Del Giudice, 2017). The research that has demonstrated differences in sensitivity to environmental contexts among children with lower RSA has focused on mild to moderate environmental stressors in lower-risk samples. For example, children with lower resting RSA exhibited more internalizing and externalizing behavior problems in response to verbal marital conflict, but they did not differ from their higher RSA counterparts in the extent to which their adjustment was affected by physical marital conflict (El-Sheikh, Harger & Whitson, 2001). Lower resting RSA may confer sensitivity to milder environmental stressors, characterized by a lack of warmth and support, but not

to stressors characterized by danger or potential harm. Bolstering this interpretation is the fact that higher resting RSA appears to confer sensitivity to caregiving environments associated with trauma and danger, such as disorganized attachment, and in higher-risk samples (e.g., Conradt et al., 2013; Blandon et al., 2008). The women in the present sample endorsed relatively few depressive symptoms, which likely represent a mild-to-moderate stressor for their children.

As the search continues for *which* level of RSA confers susceptibility, an overlooked possibility is that vagal tone differentially predicts the impact of environmental adversity on internalizing versus externalizing problems. Whereas high vagal tone is associated with infants' increased affective expressivity and reactivity and active engagement, lower vagal tone is associated with lower activity levels (Fox, 1989; Stifter, Fox & Porges, 1989), which may have different implications for sensitivity to the effects of varying caregiving contexts. Prior research has suggested the importance of considering whether depressed caregivers show intrusive or withdrawn behaviors (Tronick & Reck, 2009), and differentiating between depressed-intrusive and depressed-withdrawn parenting may inform our understanding of children's sensitivity. Children with low RSA and a withdrawn mother, who may provide fewer opportunities for rewarding social interactions, may be at risk for future internalizing behavior problems. On the other hand, children with high RSA may be at risk of over-stimulation by more active caregivers, increasing their risk for externalizing behavior problems in the context of other forms of adversity (e.g., socioeconomic hardship). Higher RSA children may show fewer externalizing behavior problems if raised by a more intrusive caregiver who exerts more control over their activities and limits their opportunities to be disruptive,

hyperactive, or destructive. One must also consider whether externalizing behaviors at three years of age (e.g., high activity level, poor impulse control, notable noncompliance) are problematic in-and-of-themselves, or whether they are better viewed as a risk factor for problems later in childhood or adolescence.

Social Engagement

Despite its use in prior research, I could not confirm the proposed factor structure of social engagement during the teaching tasks; failure to do so may be attributed to several sources of variability in the tasks. Post-hoc paired samples t-tests suggested that the teaching tasks were stressful, on average, for infants (i.e., positive affect decreased and negative irritability/fussiness increased from free play to the teaching tasks), but there was variability in the extent of affective changes. Similarly, numerous factors (e.g., maternal depression, education level, home environment, teaching style, infant developmental abilities) may have contributed to variability in how stressful the tasks were for mothers, which may have affected their behavior during the tasks. These differences are ecologically valid representations of the variations in the caregiving environments in which children are expected to engage. Evaluating children's social engagement in a manner that also directly captures differences in caregiver responsiveness and reciprocal engagement may be a more predictive measure than focusing exclusively on infants' social engagement behaviors during a teaching task paradigm.

Given these limitations, I decided to evaluate infants' social engagement during free play, consistent with most of the relevant empirical literature (e.g., Feldman, 2010; Feldman & Masalha, 2007, 2010; Feldman et al., 2009). Assessing social engagement during free play, in which there are no task demands, may offer advantages over

assessing social engagement during the teaching tasks, which are mother-directed by design. Free play may provide a better opportunity to see how the child initiates interactions with their caregivers (Asok et al., 2013), which is important because mothers' contingent responsiveness to their children's non-distress cues (e.g., during play) has been related to children's physiological and behavioral regulatory capacities (Feldman, 2012). However, prior theory and research suggests social engagement during a stressor may reflect children's developing capacity for regulating emotional experiences (e.g., Dollberg et al., 2010; Feldman, 2010; Tronick & Gianino, 1986) and may therefore be predictive of behavior problems; in contrast, social engagement during free play may be a better predictor of positive outcomes (e.g., social competence or prosocial behaviors).

In this study, infant RSA, maternal depressive symptoms, and their interaction did not predict social engagement during free play. It is possible that children's social engagement may be more strongly associated with maternal depression in clinical populations (Cohn & Campbell, 1992) and susceptibility to low social engagement may be difficult to detect in nonclinical samples, or subclinical levels of depressive symptoms may not be associated with social engagement. For example, in contrast to Feldman and colleagues' (2009) study, which used an extreme case design and found decreased social engagement among children of depressed mothers, I focused on depressive symptoms rather than clinical diagnoses of postpartum depression. It is also possible social engagement during free play may become more notably differentiated later in development. At 12 months of age, children may still be adapting their social engagement behaviors with their caregivers to suit environmental conditions. Children's developing

motor and physical competencies could allow them to more flexibly adjust their social engagement behaviors, such as gaze and vocalizations, later in life.

Social engagement during free play did predict children's internalizing, but not externalizing or total, behavior problems. Consistent with expectations, less social engagement was associated with more internalizing behavior problems. However this association did not hold after controlling for concurrent maternal depressive symptoms, which may be due to the influence of continued exposure to depressive symptoms or common method variance. Social engagement may be a precursor to internalizing symptoms, or the association between social engagement and internalizing symptoms may reflect a heterotypic continuity of underlying withdrawn-depressed behavior, which may also share a common genetic source with maternal depressive symptoms.

Despite the lack of strong association in infancy, social engagement at a later stage may be a stronger predictor of behavioral outcomes. While Feldman (2010) found that adolescents with fewer internalizing and externalizing behavior problems had higher levels of social engagement from 3 months to 13 years, differences in mean levels of social engagement did not emerge until they were 2 years of age, with the divergence in social engagement among well-adapted and less well-adapted children increasing from infancy through adolescence. These intriguing results suggest there may be a sleeper effect, where lower social engagement earlier in life sets children on a trajectory of low social engagement, which predicts behavior problems later in development (e.g., starting in toddlerhood).

Strengths and Limitations

My results add to a growing literature demonstrating that the variability in the extent to which caregiving environments impact children's behavioral adjustment may be explained by biological sensitivity, indexed by RSA. These results build on prior research in several ways. First, there is limited research on sensitivity to caregiving environments in the broader context of socioeconomic adversity (e.g., Conradt et al., 2013), and we assessed sensitivity to a mild-to-moderate stressor among an underrepresented sample of very low-income, Mexican American families. Second, whereas most studies have focused on sensitivity to externalizing behavior problems (Stoltz et al., 2017; see El-Sheikh, 2005 for an exception), or total behavior problems (e.g., Conradt et al., 2013), this study evaluated sensitivity to internalizing, externalizing and total behavior problems among younger children. Third, most prior research has assessed the caregiving environment at a single time point; the multiple measures of maternal postpartum depressive symptoms in this study strengthen the interpretation of maternal depressive symptoms as an environmental exposure. Fourth, in contrast to studies that have employed cross-sectional designs that simultaneously assess children's environment and outcomes (e.g., Conradt et al., 2013), this investigation's longitudinal design addresses questions of causality by offering repeated measures of the environment prior to assessing children's behavior problems. Further, my results held even after adjusting for concurrent maternal depressive symptoms, the most stringent evaluation of causality. Finally, while prior research has focused on identifying sensitivity factors, the present study is the first to my knowledge to evaluate a behavioral mechanism by which children's sensitivity to the influence of an early caregiving environment unfolds.

Despite its strengths, there were several limitations of the present investigation. Although multiple sources of data (biological, observational and questionnaire) were modeled, mothers reported on both their depressive symptoms and children's behavior problems, which could have affected the main effects of depressive symptoms on children's behavior problems by overinflating the associations between measures from the same rater, but not the interaction effect between mother-reported depressive symptoms and a biological measure of infant sensitivity. Women endorsed relatively low levels of depressive symptoms; it is noteworthy that I detected sensitivity to a narrow range of environmental exposure, but future research is needed to assess whether the findings would be consistent among clinical populations. Additionally, I evaluated my hypotheses among a sample of very low-income, Mexican American families; it is unclear whether the results would generalize to families from different ethnic or socioeconomic backgrounds. Finally, biological sensitivity may be affected by other factors that were not addressed in the present study (e.g., genetic, child sex, or cultural factors).

Future Directions

An important next step for future research is to evaluate the longer-term implications of biological sensitivity for mental health. Future research should address whether low vagal tone confers susceptibility to the effects of maternal depressive symptoms as children enter school and approach puberty. It is possible that these associations could change across development, especially during periods where stress response systems are recalibrated (Del Giudice, Ellis & Shirtcliff, 2011). In addition, internalizing and externalizing behavior problems display inverse growth patterns across

development, with externalizing behavior problems reaching their peak around two years of age and internalizing behavior problems continuing to increase (Gillion & Shaw, 2004). Yet, even though internalizing and externalizing behavior problems can be reliably differentiated during the preschool years, few studies have evaluated the developmental origins of internalizing behavior problems (Mesman et al., 2001). Longitudinal research on susceptibility may help account for the multifinal pathways from maternal postpartum depressive symptoms to children's resilience or internalizing or externalizing behavior problems.

Another avenue for future research on biological sensitivity is to evaluate wider ranges of environments and outcomes. Biological sensitivity differs from traditional stress-diathesis models in that it suggests variability in response to environmental context in a *for better or for worse* manner. Providing evidence in support of biological sensitivity therefore requires both evaluating the range of environments to which children may be exposed (e.g., from the most to the least supportive) as well as a range of outcomes. One way to capture a wider range of environments is to include finer-grained classification of the caregiving environment (e.g., considering maternal stress levels and the availability of social support in addition to depressive symptoms). Similarly, although the literature has primarily focused on negative child outcomes, future research should also consider developmental competencies that may be heightened in susceptible children, such as prosocial behaviors.

Finally, there are several ways in which other investigations could build on the present study's novel contribution of evaluating a behavioral mediator of sensitivity. Repeated assessments of children's social engagement would help address if and when

this mediational process becomes apparent, as well as the possible role of change in social engagement. Biological sensitivity may affect change in children's social engagement behaviors (Hopp et al., 2013), but not their mean level of social engagement. Future models should evaluate the influence of children's level of social engagement, and the amount of change in social engagement, on their subsequent behavior problems.

Conclusions

The present study provided evidence of low-income, Mexican American children's biological sensitivity to the effects of maternal postpartum depressive symptoms on internalizing, externalizing and total behavior problems. Low vagal tone conferred susceptibility to the effects of maternal postpartum depressive symptoms on internalizing and total behavior problems, and high vagal tone conferred susceptibility for externalizing behavior problems. In the context of the existing literature, these results suggest sensitivity may exist at both high and low levels of vagal tone. My results also point to the utility of separately considering children's internalizing and externalizing symptoms as children's susceptibility to these problems may differ. Biological sensitivity, indexed by vagal tone, may shed light onto which children are at greatest risk for and which children are most resilient to developing behavior problems in the context of maternal postpartum depressive symptoms.

REFERENCES

- Asok, A., Bernard, K., Roth, T.L., Rosen, J.B. & Dozier, M. (2013). Parental responsiveness moderates the association between early-life stress and reduced telomere length. *Development and Psychopathology*, 25(3), 577-585.
- Achenbach, T.M. & Rescorla, L.A. (2000). Manual for the ASEBA preschool forms & profiles. Burlington, VT: University of Vermont, Research Center for Children, Youth & Families.
- Alkon, A., Boyce, W.T., Davis, N.V. & Eskenazi, B. (2011). Developmental changes in autonomic nervous system resting and reactivity measures in Latino children from 6 to 60 months of age. *Journal of Developmental and Behavioral Pediatrics*, 32, 668-677.
- Allen, J. J. B., Chambers, A. S., & Towers, D. N. (2007). The many metrics of cardiac chronotropy: A pragmatic primer and a brief comparison of metrics. *Biological Psychology*, 74(2), 243-262.
doi:<http://dx.doi.org/10.1016/j.biopsycho.2006.08.005>
- Allison, P.D. (2003). Missing data techniques for structural equation models. *Journal of Abnormal Psychology*, 112, 545-557.
- Apter-Levy, Y., Feldman, F., Vakart, A., Ebstein, R.P. & Feldman, R. (2013). Impact of maternal depression across the first 6 years of life on the child's mental health, social engagement, and empathy: The moderating role of oxytocin. *American Journal of Psychiatry*, 170, 1161-1168.
- Avan, B., Richter, L.M., Ramchandani, P.G., Norris, S.A. & Stein, A. (2010). Maternal postnatal depression and children's growth and behaviour during the early years of life: Exploring the interaction between physical and mental health. *Archives of Disease in Childhood*. doi:10.1136/adc.2009.164848
- Ashman, S.B. & Dawson, G. (2002). Maternal depression, infant psychobiological development, and risk for depression. In Sherryl H. Goodman & Ian H. Gotlib (Eds.), *Children of depressed parents: Mechanisms of risk and implications for treatment* (pp. 37-58). Washington, D.C.: American Psychological Association.
- 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, 183-214.
- Beck, C.T. (1998). The effects of postpartum depression on child development: A meta analysis. *Archives of Psychiatric Nursing*, 12(1), 12-20.

- Blandon, A.Y., Calkins, S.D., Keane, S.P. & O'Brien, M. (2008). Individual differences in trajectories of emotion regulation processes: The effects of maternal depressive symptomatology and children's physiological regulation. *Developmental Psychology, 44*(4), 1110-1123.
- Bornstein, M.H. & Suess, P.E. (2000). Child and mother cardiac vagal tone: Continuity, stability, and concordance across the first 5 years. *Developmental Psychology, 36*(1), 54-65.
- Boyce, T. & Ellis, B.J. (2005). Biological sensitivity to context: I. An evolutionary developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology, 17*, 271-301.
- Briggs-Gowan, M., Carter, A. S., Bosson-Heenan, J., Guyer, A. E., & Horwitz, S. M. (2006). Are infant-toddler social-emotional and behavioral problems transient? *Journal of the American Academy of Child & Adolescent Psychiatry, 45*(7), 849-858. doi:<http://dx.doi.org/10.1097/01.chi.0000220849.48650.59>
- CardioBatch software. Brain-Body Center, University of Illinois at Chicago. 2007.
- Carter, A.S., Garrity-Rokous, F.E., Chazan-Cohen, R., Little, C. & Briggs-Gowan, M.J. (2001). Maternal depression and comorbidity: Predicting early parenting, attachment security, and toddler social-emotional problems and competencies. *Journal of the American Academy of Child and Adolescent Psychiatry, 40*(1), 18-26.
- Chaudron, L.H., Kitzman, H.J., Peifer, K.L., Morrow, S., Perez, L.M. & Newman, M.C. (2005). Prevalence of maternal depressive symptoms in low-income Hispanic women. *Journal of Clinical Psychiatry, 66*(4), 418-423.
- Choe, D.E., Sameroff, A.J. & McDonough, S.C. (2013). Infant functional regulatory problems and gender moderate bidirectional effects between externalizing behavior and maternal depressive symptoms. *Infant Behavior and Development, 36*(3), 307-318. doi:10.1016/j.infbeh.2013.02.004.
- Cohn, J.F. & Campbell, S.B. (1992). Influence of maternal depression on infant affect regulation. In D. Cicchetti & S.L. Toth (Eds.), *Developmental perspectives on depression. Rochester symposium on developmental psychopathology* (pp. 103-130). Rochester, NY, US: University of Rochester Press.
- Cohn, J.F., Campbell, S.B., Matias, R. & Hopkins, J. (1990). Face-to-face interactions of postpartum depressed and nondepressed mother-infant pairs at 2 months. *Developmental Psychology, 26*(1), 15-23.
- Conradt, E., Measelle, J. & Ablow, J.C. (2013). Poverty, problem behavior, and promise:

- Differential susceptibility among infants reared in poverty. *Psychological Science*, 24(3), 235-242. doi:10.1177/0956797612457381.
- Contreras, J.M., Kerns, K.A., Weimer, B.L., Gentzler, A.L. & Tomich, P.L. (2000). Emotion regulation as a mediator of associations between mother-child attachment and peer relationships in middle childhood. *Journal of Family Psychology*, 14(1), 111-124.
- Cox, J.L., Holden, J.M., & Sagovsky, R. (1987) Detection of postnatal depression. *British Journal of Psychiatry*, 150, 786.
- Crowell, J.A. & Feldman, S.S. (1988). Mothers' internal models of relationships and children's behavioral and developmental status: A study of mother-child interaction. *Child Development*, 59, 1273-1285.
- Crowell, J., A., Feldman, S.S. & Ginsberg, N. (1998). Assessment of mother-child interaction in preschoolers with behavior problems. *Journal of the American Academy of Child and Adolescent Psychiatry*, 27(3), 303-311.
- Dawson, G. (1999). *The Effects of Maternal Depression on Children's Emotional and Psychobiological Development*. Paper presented at the National Institute of Mental Health Conference on Parenting, October, Bethesda, MD.
- Dawson, G., Frey, K., Panagiotides, H., Yamada, E., Hessel, D. & Osterling, J. (1999). Infants of depressed mothers exhibit atypical frontal electrical brain activity during interactions with mother and with a familiar, nondepressed adult. *Child Development*, 70(5), 1058-1066.
- Dawson, G., Ashman, S.B., Panagiotides, H., Hessel, D., Self, J., Yamada, E. & Embry, L. (2003). *Child Development*, 74(4), 1158-1175.
- Degangi, G. A., DiPietro, J.A., Greenspan, S.I. & Porges, S.W. (1991). Psychophysiological characteristics of the regulatory disordered infant. *Infant Behavior & Development*, 14(1), 37-50.
- Del Giudice, M. (2017). Statistical tests of differential susceptibility: Performance, limitations, and improvements. *Development and Psychopathology*.
- Del Giudice, M., Ellis, B.J. & Shirtcliff, E.A. (2011). The adaptive calibration model Of stress responsivity. *Neuroscience and Biobehavioral Reviews*, 35(7), 1562-1592.

- Dollberg, D., Feldman, R. Keren, M. & Guedeney, A. (2006). Sustained withdrawal behavior in clinic-referred and nonreferred infants. *Infant Mental Health Journal*, 27(3), 292-309. doi: 10.1002/imhj.20093
- Eisenberg, N., Sulik, M.J., Spinrad, T.L., Edwards, A., Eggum, N.D., Liew, J., Sallquist, J., Popp, T.K., Smith, C.L. & Hart, D. (2012). Differential susceptibility and the early development of aggression: Interactive effects of respiratory sinus arrhythmia and environmental quality. *Developmental Psychology*, 48(3), 755-768.
- El-Sheikh, M. (2005). Does poor vagal tone exacerbate child maladjustment in the context of parental problem drinking? A longitudinal examination. *Journal of Abnormal Psychology*, 114(4), 735-741.
- El-Sheikh, M., Harger, J. & Whitson, S.M. (2001). Exposure to interparental conflict and children's adjustment and physical health: The moderating role of vagal tone. *Child Development*, 72(6), 1617-1636.
- El-Sheikh, M. & Hinnant, J.B. (2011). Marital conflict, respiratory sinus arrhythmia, and allostatic load: Interrelations and associations with the development of children's externalizing behavior. *Development and Psychopathology*, 23, 815-829. doi:10.1017/S0954579411000320
- Ellis, B. J. & Boyce, W.T. (2008). Biological sensitivity to context. *Current Directions in Psychological Science*, 17(3), 183-187.
- Ellis, B.J. & Boyce, W. T. (2011). Differential susceptibility to the environment: Toward an understanding of sensitivity to developmental experiences and context. *Development and Psychopathology*, 23, 1-5. doi:10.1017/S095457941000060X
- Ellis, B.J., Boyce, W.T., Belsky, J., Bakermans-Kranenburg, M.J. & van IJzendoorn, M.H. (2011). Differential susceptibility to the environment: An evolutionary-neurodevelopmental theory. *Development and Psychopathology*, 23, 7-28. doi:10.1017/S0954579410000611
- Ellis, B.J., Essex, M.J., & Boyce, W.T. (2005). Biological sensitivity to context: II. Empirical explorations of an evolutionary-developmental theory. *Development and Psychopathology*, 17, 303-328.
- Feldman, R. (1998). Coding interactive behavior manual. Unpublished manual, Israel: Bar-Ilan University.
- Feldman, R. (2010). The relational basis of adolescent adjustment: Trajectories of

- mother-child interactive behaviors from infancy to adolescence shape adolescents' adaptation. *Attachment & Human Development*, 12(1-2), 173-192.
- Feldman, R. (2012). Parenting behavior as the environment where children grow. In L. Mayes, & M. Lewis (Eds.), *The Cambridge handbook of environment in human development*. New York, NY: Cambridge University Press.
- Feldman, R. & Masalha, S. (2010). Parent-child and triadic antecedents of children's social competence: Cultural specificity, shared process. *Developmental Psychology*, 46(2), 455-467.
- Feldman, R. & Masalha, S. (2007). The role of culture in moderating the links between early ecological risk and young children's adaptation. *Development and Psychopathology*, 19, 1-21. doi: 10.1017/S0954579407070010
- Feldman, R., Granat, A., Pariente, C., Kanety, H., Kuint, J., & Gilboa-Schechtman, E. (2009). Maternal depression and anxiety across the postpartum year and infant social engagement, fear regulation, and stress reactivity. *Journal of the American Academy of Child and Adolescent Psychiatry*, 48(9), 919-927.
- Field, T., Lang, C., Martinez, A., Yando, R., Pickens, J., & Bendell, D. (1996). Preschool follow-up of infants of dysphoric mothers. *Journal of Clinical Child Psychology*, 25(3), 272-279.
- Fox, N.A. (1989). Psychophysiological correlates of emotional reactivity during the first year of life. *Developmental Psychology*, 3, 364-372.
- Galindo, C. & Fuller, B. (2010). The social competence of Latino kindergartners and growth in mathematical understanding. *Developmental Psychology*, 46(3), 579-592.
- Garcia-Esteve, L., Ascaso, C., Ojuel, J., & Navarro, P. (2003). Validation of the Edinburgh Postnatal Depression Scale (EPDS) in Spanish mothers. *Journal of Affective Disorders*, 75, 71-76.
- Gianino, A. & Tronick, E.Z. (1988). The mutual regulation model: The infant's self and interactive regulation and coping and defensive capacities. In Tiffany M. Field, Philip M. McCabe, & Neil Schneiderman (Eds.), *Stress and coping across development* (pp. 47-68). Hillsdale, N.J: Lawrence Erlbaum Associates, Publishers.
- Gillion, M. & Shaw, D.S. (2004). Codevelopment of externalizing and internalizing problems in early childhood. *Development and Psychopathology*, 16, 313-333.
- Goodman, S.H. & Gotlib, I.H. (1999). Risk for psychopathology in the children of

- depressed mothers: A developmental model for understanding mechanisms of transmission. *Psychological Review*, 106(3), 458-490.
- Goodman, S.H. & Tully, E. (2006). Depression in women who are mothers: An integrative model of risk for the development of psychopathology in their sons and daughters. In Corey L.M. Keyes & Sherryl H. Goodman (Eds.), *Women and Depression: A handbook for the social, behavioral, and biomedical sciences*. New York: Cambridge University Press.
- Guyon-Harris, K., Huth-Bocks, A., Lauterbach, D. & Janisse, H. (2015). Trajectories of maternal depressive symptoms across the birth of a child: Associations with toddler emotional development. *Arch Womens Ment Health*. doi:10.1007/s00737-015-0546-8
- Harnish, J.D., Dodge, K.A., Valente, E. & Conduct Problems Prevention Research Group. (1995). Mother-child interaction quality as a partial mediator of the roles of maternal depressive symptomatology and socioeconomic status in the development of child behavior problems. *Child Development*, 66, 739-753.
- Hastings, P. D. & De, I. (2008). Parasympathetic regulation and parental socialization of emotion: Biopsychosocial processes of adjustment in preschoolers. *Social Development*, 17, 211–238. doi:10.1111/j.1467-9507.2007.00422.x
- Hinnant, J.B., Erath, S.A. & El-Sheikh, M. (2015). Harsh parenting, parasympathetic activity, and development of delinquency and substance use. *Journal of Abnormal Psychology*, 124(1), 137-151. doi: 10.1037/abn0000026.
- Hobfoll, S.E., Ritter, C., Lavin, J., Hulsizer, M.R. & Cameron, R.P. (1995). Depression prevalence and incidence among inner-city pregnant and postpartum women. *Journal of Consulting and Clinical Psychology*, 63(3), 445-453.
- Hopp, H., Shallcross, A.J., Ford, B.Q., Troy, A.S., Wilhelm, F.H. & Mauss, I.B. (2013). High cardiac vagal control protects against future depressive symptoms under conditions of high social support. *Biological Psychology*, 93, 143-149.
- Howell, E.A., Mora, P.A., Horowitz, C.R. & Leventhal, H. (2005). Racial and ethnic differences in factors associated with early postpartum depressive symptoms. *Obstetrics and Gynecology*, 105(6), 1442-50.
- Huffman, L. C., Bryan, Y.E., del Carmen, R., Pedersen, F.A., Doussard-Roosevelt, J.A., & Porges, S.W. (1998). Infant temperament and cardiac vagal tone: Assessments at twelve weeks of age. *Child Development*, 69(3), 624-635.

- Jusiene, R., Breidokiene, R. & Pakalniskiene, V. (2015). Developmental trajectories of mother reported regulatory problems from toddlerhood to preschool age. *Infant Behavior & Development, 40*, 84-94.
- Kuo, W.H., Wilson T.E., Holman S., Fuentes-Afflick, E., O'Sullivan, M.J., Minkoff, H. (2004). Depressive symptoms in the immediate postpartum period among Hispanic women in three U.S. cities. *Journal of Immigrant Health, 6*, 145–153.
- 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*(5), 561-592.
- McLaughlin, K.A., Rith-Najarian, L., Dirks, M.A. & Sheridan, M.A. (2015). Low vagal tone magnifies the association between psychosocial stress exposure and internalizing psychopathology in adolescents. *Journal of Clinical Child & Adolescent Psychology, 44*(2), 314-328.
- Mesman, J., Bongers, I.L. & Koot, H.M. (2001). Preschool developmental pathways to preadolescent internalizing and externalizing problems. *Journal of Child Psychology and Psychiatry, 42*(5), 679-89.
- Mesman, J. & Koot, H.M. (2001). Early preschool predictors of preadolescent internalizing and externalizing DSM-IV diagnoses. *Journal of the American Academy of Child and Adolescent Psychiatry, 40*(9), 1029-36.
- Miller, G.E. & Chen, E. (2013). The biological residue of childhood poverty. *Child Development Perspectives, 7*(2), 67-73. doi:10.1111/cdep.12021.
- Moehler, E., Brunner, R., Wiebel, A., Reck, C. & Resch, F. (2006). Maternal depressive symptoms in the postnatal period are associated with long-term impairment of mother-child bonding. *Archives of Women's Mental Health, 9*, 273-278.
- Murray, L. & Cooper, P.J. (1997). Postpartum depression and child development. *Psychological Medicine, 27*, 253-260.
- Muthén, L.K. and Muthén, B.O. (1998-2012). Mplus User's Guide. Seventh Edition. Los Angeles, CA: Muthén & Muthén
- Nelson, C.A., Zeanah, C.H., Fox, N.A., Marshall, P.J., Smyke, A.T. & Guthrie, D. (2007). Cognitive recovery in socially deprived young children: The Bucharest early intervention project. *Science, 318*, 1937-1940.

- Obradovic, J. (2012). How can the study of physiological reactivity contribute to our understanding of adversity and resilience processes in development? *Development and Psychopathology*, 24, 371-387.
- Perry, B.D. (2002). Childhood experience and the expression of genetic potential: What childhood neglect tells us about nature and nurture. *Brain and Mind*, 3, 79-100.
- Philipps, L.H.C. & O'Hara, M.W. (1991). Prospective study of postpartum depression: 4½-Year follow-up of women and children. *Journal of Abnormal Psychology*, 100(2), 151-155.
- Porges, S. W. (1995). Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A polyvagal theory. *Psychophysiology*, 32, 301– 318.
- Porges, S.W. (2007). The polyvagal perspective. *Biological Psychology*, 74(2), 116-143.
- Porges, S. W., Doussard-Roosevelt, J..A., Portales, A.L. & Suess, P.E. (1994). Cardiac vagal tone: Stability and relation to difficultness in infants and 3-year-olds. *Developmental Psychobiology*, 27(5), 289-300.
- Porter, F. L., Porges, S.W. & Marshall, R.E. (1988). Newborn pain cries and vagal tone: Parallel changes in response to circumcision. *Child Development*, 59(2), 495-505.
- Radloff, L.S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, 1(3), 385-401. doi: 10.1177/014662167700100306
- Roberts, R.E., Vernon, S.W. & Rhoades, H.M. (1989). Effects of language and ethnic status on reliability and validity of the Center for Epidemiologic Studies-Depression Scale with psychiatric patients. *The Journal of Nervous and Mental Disease*, 177(10), 581-592.
- Rubio-Stipec, M., Bird, H., Canino, G. & Gould, M. (1990). The internal consistency and concurrent validity of a Spanish translation of the Child Behavior Checklist. *J Abnorm Child Psychol*, 18(4), 393-406.
- Stifter, C. A. & Fox (1990). Infant reactivity: Physiological correlates of newborn and 5 month temperament. *Developmental Psychology*, 26(4), 582-588.
- Stifter, C. A., Fox, N. A., & Porges, S. W. (1989). Facial expressivity and vagal tone in 5- and 10-month-old infants. *Infant Behavior and Development*, 12, 127 – 137.

- Stoltz, S., Beijers, R., Smeekens, S. & Dekovic, M. (2017). Diathesis stress or differential susceptibility? Testing longitudinal associations between parenting, temperament, and children's problem behavior. *Social Development*, 00, 1-14.
- Tronick, E.Z. & Gianino, A.F. (1986). The transmission of maternal disturbance to the infant. In E.Z. Tronick & T. Field (Eds.), *Maternal Depression and Infant Disturbance. New Directions for child development, no 34*. San Francisco: Jossey-Bass.
- Tronick, E. & Reck, C. (2009). Infants of depressed mothers. *Harvard Review of Psychiatry*, 17, 147-156.
- Walker, M.J., Davis, C., Al-Sahab, B. & Tamim, H. (2013). Reported maternal postpartum depression and risk of childhood psychopathology. *Maternal and Child Health Journal*, 17, 907-917. doi:10.1007/s10995-012-1071-2
- Weinberg, M.K., Tronick, E.Z., Beeghly, M., Olson, K.L., Kernan, H. & Riley, J.M. (2001). Subsyndromal depressive symptoms and major depression in postpartum women. *American Journal of Orthopsychiatry*, 71(1), 87-97.
- Yonkers, K.A., Ramin, S.M., Rush, A.J., Navarrete, C.A., Carmody, T., March, D., Heartwell, S.F. & Leveno, K.J. (2001). Onset and persistence of postpartum depression in an inner-city maternal health clinic system. *American Journal of Psychiatry*, 158, 1856-1863.