



Published in final edited form as:

Biol Psychol. 2023 September ; 182: 108625. doi:10.1016/j.biopsycho.2023.108625.

Examining Transactional Associations between Maternal Internalizing Symptoms, Infant Negative Emotionality, and Infant Respiratory Sinus Arrhythmia

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Abstract

The current study examined transactional associations between maternal internalizing symptoms, infant negative emotionality, and infant resting respiratory sinus arrhythmia (RSA). We used data from the Longitudinal Attention and Temperament Study (N=217) to examine the associations between maternal internalizing symptoms, infant negative emotionality, and infant resting RSA from 4-months to 18-months using a random-intercepts cross-lagged panel model. We found that mothers with higher average internalizing symptoms have infants with higher levels of resting RSA. However, there were no stable, between-individual differences in infant negative emotionality across time. Additionally, we found significant negative within-dyad cross-lagged associations from maternal internalizing symptoms to subsequent measures of infant negative emotionality, as well as a significant negative cross-lagged association from maternal internalizing symptoms to child resting RSA after 12-months of age. Lastly, we find evidence for infant-directed effects of negative emotionality and resting RSA to maternal internalizing symptoms. Results highlight the complex, bidirectional associations in maternal-infant dyads during the first two years of life, and the importance of considering the co-development of infant reactivity and regulatory processes in the context of maternal internalizing symptoms.

Keywords

negative emotionality; respiratory sinus arrhythmia; temperament; infancy; maternal internalizing symptoms

Infancy is characterized by rapid development in both biological and behavioral domains. During the first two years of life, changes in infants' emotional reactivity and regulation mark important and meaningful developmental transitions and lead to the development of

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individual differences associated with later socioemotional outcomes, including risk for psychopathology (Bornstein et al., 2014; Klein et al., 2012). During infancy, caregivers play a large role in providing the environmental context and social experiences that may shape socioemotional development. It is important, then, to consider how parental factors, such as maternal distress, may play a role in the development of individual differences in reactivity and regulation.

One method of assessing maternal distress is through maternal internalizing symptoms, which include both depression and anxiety symptoms. Maternal internalizing symptoms are often associated with child socioemotional outcomes, and numerous studies have demonstrated intergenerational associations between maternal and child internalizing symptoms (Eley et al., 2015; Goodman, 2020; McAdams et al., 2015). Despite evidence for links between maternal and child internalizing symptoms, the mechanisms are still complex and open to study. One possibility is that maternal internalizing symptoms may impact the development of children's emotional reactivity and regulation, which in turn lead to increased risk for children's development of internalizing behaviors (Goodman & Gotlib, 1999). Specifically, the interaction between parasympathetic nervous system (PNS) activity and early childhood temperament may be one pathway for the development of associations between maternal and child internalizing symptoms. For example, maternal internalizing symptoms are associated with greater autonomic arousal in their infants, which is associated with higher levels of temperamental fear in toddlerhood (de Vente et al., 2020). Drawing from biopsychosocial models of development (e.g., Gottlieb, 2007; Sameroff, 2010) and prior research, the child's daily context is likely impacting the development of both emotional reactivity and regulation. However, less is known about how within-individual changes in maternal internalizing symptoms, PNS activity, and negative emotionality development may be associated with one another, and how negative emotionality and PNS activity may co-develop across infancy.

Additionally, there are bidirectional associations between the infant and parents that occur across this developmental period, which may have implications for both infant development as well as parental mental health and behaviors. Infant and child negative emotionality (i.e., fear, sadness, and anger) elicits negative affect in mothers (Murray, 1979; Soltis, 2004), increases in maternal stress (Pesonen et al., 2008), and increases in anxiety symptoms (Behrendt et al., 2020). Taken together, there may be bidirectional associations within dyads between maternal internalizing symptoms and infant negative emotionality, and developmental changes in infant physiology may modulate these relations over time. The present study leverages a longitudinal design with repeated measures of maternal internalizing symptoms, infant negative emotionality, and infant PNS activity to examine these inter-relations across infancy as a potential mechanism by which internalizing symptoms develop and are maintained in families.

Associations between the Development of Infant Resting Respiratory Sinus Arrhythmia and Maternal Internalizing Symptoms

Maturation in physiological systems underlying reactivity and regulation may act as a marker of risk for the development of internalizing symptoms. The stress response system has three main biological functions: to coordinate physiological and behavioral responses to threats and opportunities, to encode and filter information from the environment, and to translate that information into broad-band individual differences in behavior and physiology (Del Giudice et al., 2011). The Adaptive Calibration Model (ACM) of stress responsivity provides a framework for considering the ways in which individual differences in stress regulation may arise due developmental changes in response to early experiences (Del Giudice et al., 2011). The ACM posits that individual differences in stress regulation develop as the organism modifies its functioning—and in turn, its developmental trajectory—to match the conditions of the social and physical environment. This would suggest that the activation of the child's stress response system during the first few years of life plays a key role in recalibration of physiological systems underlying stress regulation, as early activation signals important information about the environment in which development is occurring. For example, early activation may lead to signaling that there are environmental dangers and threats that the body needs to cope with, that may lead to up- or down-regulation of the stress-response system (Del Giudice et al., 2011). This highlights the importance of considering the early environment in the development of physiological systems.

One branch of the autonomic nervous system (ANS) is the parasympathetic nervous system (PNS), which plays an important role in how the body copes with stress. The PNS responds very quickly to environmental cues as a first-response coping system by allowing individuals to mobilize resources and engage with objects and/or other individuals to achieve homeostasis (Porges, 2007). PNS activity is commonly assessed through respiratory sinus arrhythmia (RSA), which captures variability in the inter-beat interval of the heart and the impact of the vagus nerve (Porges, 2007). Resting RSA is thought to reflect the general capacity for behavioral and self regulation (including emotion regulation), with higher levels of resting RSA reflecting capacity for *better* emotion regulation in the face of contextual stressors. While task-based measures of RSA capture regulatory processes during specific contexts, resting RSA is putatively less context-specific and can capture individual differences in emotion regulation more broadly, and, perhaps ironically, remove 'noise' created by individual differences in emotional and behavioral responses to task-based triggers. Resting RSA has been associated with risk for internalizing symptom development as a transdiagnostic marker of emotion regulation associated with various forms of psychopathology, with lower levels of resting RSA associated with higher levels of internalizing symptoms (Beauchaine, 2015).

Recent work provides evidence that the ANS matures throughout the fetal period and in infancy (Mulkey & du Plessis, 2019; Porges & Furman, 2011), and may be susceptible to environmental influences during early development. There is some emerging evidence that postnatal maternal factors may shape the development of infant RSA. Factors such as maternal psychopathology (history of psychiatric disorder) and marital conflict that are

associated with maternal distress are also associated with higher infant heart rate and lower infant resting RSA (Dierckx et al., 2009; Moore, 2010; Propper & Holochwost, 2013). This body of work provides evidence that postnatal maternal distress, such as maternal internalizing symptoms, are associated with infant ANS activity. It is important to note that most prior research examining associations between maternal internalizing symptoms and infant RSA does not use repeated measures of maternal internalizing symptoms or infant RSA, which limits our understanding of timing of associations between maternal internalizing symptoms and infant RSA.

Thus, one gap that remains in the literature and aligns with the goals of this study is how postnatal maternal distress may be associated with developmental change in RSA across infancy. Overall, researchers have found that there is rank-order stability in resting RSA across infancy and toddlerhood, with mean increases across time (Bornstein & Suess, 2000; Wagner et al., 2021). However, Wagner and colleagues's systematic review (2021) concluded that while there is moderate stability in resting RSA across the first three years, there are considerable individual differences in initial levels of resting RSA and variability in trajectories that follow. Additionally, one limitation in previous work is the lack of examination of between-individual versus within-individual changes in resting RSA during development. While there is some evidence for rank-order stability (Bornstein & Suess, 2000), prior analyses examining growth trajectories of RSA reflect a combination of between-individual and within-individual change. Taken together, this suggests that while there may be trait-like components to resting RSA, resting RSA may also be open to environmental influences during this infancy period that may contribute to within-individual changes across time.

Associations between the Development of Negative Emotionality and Maternal Internalizing Symptoms

Temperament traits are biologically-based individual differences that include the domains of activity, reactivity, emotionality, and sociability (Shiner et al., 2012). One dimension of temperamental reactivity is negative emotionality, which typically includes the expression of fear, sadness, and anger. Individual differences in emotional reactivity (i.e., the intensity and duration of negative emotionality in response to contextual demands) may be modulated by emotion regulation processes, and are distinct but related constructs (Davidson, 1998; Sheppes et al., 2015). Anger, sadness, and fear individually, as well as negative emotionality more broadly, have been associated with the development of children's internalizing symptoms (e.g., Buss et al., 2018; Klein et al., 2012; Crawford et al., 2011; Liu et al., 2018; Eisenberg et al., 2009).

While higher levels of negative emotionality are associated with greater risk for developing internalizing behaviors, not all children high in negative emotionality go on to develop high levels of internalizing symptoms (Buss & McDoniel, 2016; Klein et al., 2012). One possible reason for this inconsistency is that in addition to normative (mean-level) developmental changes in negative emotionality, patterns of change in negative emotionality may also affect risk for developing internalizing symptoms. Longitudinal work on temperamental traits has

mainly focused on rank order stability across early childhood, and demonstrates that there is stability across infancy through childhood across multiple temperamental traits (Durbin et al., 2007; Roberts & DelVecchio, 2000). Negative emotionality increases across infancy and toddlerhood, with increases in parent-reported negative emotionality from 9-months to 27-months (Cioffi et al., 2021a; Lipscomb et al., 2011). Studies examining individual domains of parent-reported negative emotionality also find increases across the first year of life in fear (Braungart-Rieker et al., 2010; Gartstein et al., 2010, 2018) and anger (Braungart-Rieker et al., 2010; Dollar & Calkins, 2019). However, studies examining change in rank-order stability of temperament has shown that there is relatively less stability in temperamental traits during infancy (Lemery et al., 1999).

Individual patterns of continuity and discontinuity in negative emotionality across development could be indicative of risk status for internalizing symptom development (Aksan & Lemery, 2000; Goldsmith & Lemery, 2000). Goldsmith and Lemery (2000) suggest that there may be more stability in negative emotionality across time for children who have higher levels of negative emotionality compared to other children their age, which could be indicative of higher risk of developing internalizing disorders. Consistent with this perspective, past research suggests that temperament profiles characterized by more reactivity show more stability across time (Beekman et al., 2015). Additionally, empirical studies in children have consistently demonstrated that stable, high levels of fear are associated with greater anxiety and depressive symptoms at a later assessment (Brooker et al., 2013; Van Hulle et al., 2017). Thus, it is important to consider between-individual differences within normative changes in negative emotionality during infancy, and as well as what factors may contribute to the development of negative emotionality.

Developmental changes in temperament may stem from maturation in systems underlying regulation. These systems have a protracted developmental trajectory, and maturation of these systems (such as the stress response system) may lead to changes in the expression of temperament (Rothbart & Bates, 2006; Shiner et al., 2012). In particular, self-regulation increases across development, and this increase in regulation may begin to influence domains of temperamental reactivity, leading to increases in stability (Shiner et al., 2012). Children's development of negative emotionality may be more sensitive to social and contextual influences earlier in development as biological mechanisms underlying reactivity and regulation are maturing.

Parents are one source of social influence on the development of reactivity. For example, there is some evidence that parental internalizing symptoms are associated with changes in negative emotionality, including fear and anger (e.g., Potapova et al., 2014; Gartstein et al., 2010). Gartstein and colleagues (2010) demonstrated that fear increased in a linear trend over the first year of life, and that infants with mothers who reported more depressive symptoms exhibited greater increases in fear across the first year and had higher levels of toddler anxiety. Both mothers' and fathers' internalizing symptoms have been demonstrated to influence infants' negative emotionality during the first year of life, although parental internalizing symptoms are often assessed at a single time point. It is less clear how stability (or lack thereof) in parental internalizing symptoms across the infancy period may be associated with developmental changes in infant negative emotionality.

Taken together, it appears there may be both between-individual differences in negative emotionality and within-individual processes that may have important implications for the development of internalizing psychopathology risk. Additionally, examining developmental changes in negative emotionality in the context of infants' social environment is particularly important as parents play a large role in infant development. Given that individual differences in negative emotionality may play a role in the intergenerational transmission of internalizing symptoms, it is important to consider these between-infant differences and within-infant changes in negative emotionality in the context of maternal internalizing symptoms.

Infant Directed Effects on Maternal Internalizing Symptoms

Lastly, it is important to consider the role that the infant may have on parents. Mothers' own internalizing symptoms change across time, especially during the early years of parenthood (Ashman et al., 2008; Gump et al., 2009; Skipstein et al., 2012; van der Waerden et al., 2015). Transactional models of development (e.g., Sameroff, 2010) highlight the importance of considering bidirectional influences. Prior work finds evidence for transactional processes within families that maintain anxiety and depressive symptoms of both parents and children across time (e.g., Ahmadzadeh et al., 2019). While there are some studies that have measured maternal internalizing symptoms at multiple time points, most of the work examining associations between maternal internalizing symptoms and either infant negative emotionality or infant resting RSA have only examined maternal internalizing symptoms at a single time point. As a result, less is known about how changes in maternal internalizing symptoms may be associated with the development of infants' reactivity and regulation.

Additionally, children may play a role in regulating their parent's affective states and may elicit both greater parent negative affect and stress that, in turn, lead to changes in maternal internalizing symptoms. Infants with more difficult temperaments may exacerbate depressed mothers' moods (Whiffen & Gotlib, 1989). More specifically, infant and child negative emotionality elicits stronger negative affect in mothers (Murray, 1979; Soltis, 2004), increases in maternal stress (Pesonen et al., 2008), parents' depressive symptoms (Cioffi et al., 2021b), and anxiety symptoms (Behrendt et al., 2020). Mothers who have internalizing symptoms may struggle to regulate their emotional responses when faced with infant distress, which could negatively impact both early dyadic interactions as well as increase subsequent maternal internalizing symptoms.

Less work has been conducted on how children's physiological regulation may be associated with changes in maternal internalizing symptoms, making this a critical gap this study will address. One study has demonstrated that trajectories of stable, higher levels of maternal depressive symptoms across time are associated with greater child RSA during a stressor (Gump et al., 2009). Although the work conducted by Gump and colleagues (2009) does not directly test child-directed effects on maternal internalizing symptoms, it suggests that stability/instability in parent internalizing symptoms over time may be associated with children's physiological regulation. However, there is a lack of evidence for transactional influences with repeated measures of both RSA and maternal internalizing symptoms. In addition, it is unknown if child-directed effects are driven by behavioral responses to

stress that are coupled with physiological responses, or if there may be specific biological processes that are influencing the parent.

The Present Study

Understanding the inter-related associations between maternal internalizing symptoms, infant negative emotionality, and infant PNS activity is important because it may help researchers better understand how internalizing symptoms develop and are maintained in families through (1) the development of child negative emotionality and physiological regulation as precursors to child internalizing symptoms, as well as (2) infant-directed effects on maternal postnatal distress. Prior studies have often assessed infant negative emotionality, resting RSA, and maternal internalizing symptoms at a single time point, limiting our understanding of how these constructs may co-develop across time. The current study aims to examine whether the associations between infant markers of risk (i.e., negative emotionality and resting RSA) and maternal internalizing symptoms suggest between-dyad trait-like levels across time, or if there may be time-varying, within-dyad processes. Specifically, the present study examined associations between maternal internalizing symptoms, infant negative emotionality, and infant PNS activity using a random-intercepts cross-lagged panel model (RI-CLPM; Hamaker et al., 2015; Mulder & Hamaker, 2021).

The RI-CLPM is an analytic strategy that allows for a more nuanced understanding of bidirectional associations within dyads across multiple measures, and addresses limitations in other analytic strategies that conflate between- and within-person associations (Hamaker et al., 2015). Using an RI-CLPM, we can examine both within- and between-dyad associations for maternal internalizing symptoms and negative emotionality, as well as maternal internalizing symptoms and resting RSA. Additionally, we will be able to examine within-person and between-person (trait level) associations of negative emotionality and resting RSA.

We hypothesized that we would find associations between trait-levels of maternal internalizing symptoms and negative emotionality, such that higher levels of maternal internalizing symptoms are associated with higher levels of negative emotionality. We also hypothesized that there would be negative associations between trait levels of maternal internalizing symptoms and resting RSA, as well as negative associations between trait levels of negative emotionality and resting RSA. Additionally, we predicted cross-lagged associations between maternal internalizing symptoms and negative emotionality, as well as maternal internalizing symptoms and resting RSA. Lastly, we hypothesized that the only direct child-to-parent effects we would see in the cross-lagged associations would be from negative emotionality to maternal internalizing symptoms, as prior studies suggest that child-directed effects on maternal internalizing symptoms may be behaviorally mediated, as they are the risk factors most directly visible to parents. Thus, we expected to find that RSA may be indirectly associated with maternal internalizing symptoms via children's negative emotionality.

Method

Participants.

357 infants (50.7% female, 49.2% male) and their parents were recruited from areas surrounding State College, PA, Harrisburg, PA, and Newark, NJ (Perez-Edgar et al., 2021). 298 infants were recruited when infants were 4-months of age ($M_{age} = 4.80$ months; $SD_{age} = 0.80$), with an additional 46 participants enrolled at 8-months ($M_{age} = 8.83$ months, $SD_{age} = 0.73$) and 13 participants enrolled at 12 months ($M_{age} = 12.73$, $SD_{age} = 1.12$). Caregivers identified 16% of infants as African American/Black, 3% as Asian/Pacific Islander, 22% as Latinx, 50% as white, and 8% as mixed race. Across all participants, 14% families reported a household income of \$15,000 or less, 6% reported \$16,000–20,000, 6% reported \$21,000–30,000, 5% reported \$31,000–40,000, 6% reported \$41,000–50,000, 8% reported \$51,000–60,000, and 39% reported an income above \$60,000. 17% caregivers declined to provide their annual household income.

For mother's education, 3% completed grade school only, 5% had some high school, 10% graduated from high school, 16% had some college or trade/technical degree, 20% were college graduates, 16% had graduate training, and 19% had a graduate degree. 11% did not provide their education level. For fathers, 3% completed grade school only, 4% had some high school, 14% graduated from high school, 17% had some college or trade/technical degree, 20% were college graduates, 12% had graduate training and 16% had a graduate degree. 15% did not provide this information.

For the present study, a subset of 217 infants (51.6% female) were included, as they provided at least one time point of usable data for our variables of interest, and mothers were the caregiver that completed the questionnaires.

Procedures and Measures.

As part of the larger study, data were collected when infants were 4-, 12-, 18-, and 24-months. Due to the COVID-19 pandemic prohibiting in lab data collection and the amount of missing physiology data at 24-months (79% of full sample), only data at 4-, 12-, and 18-months were used in the present study.

Questionnaires.—Most parents completed questionnaires via Qualtrics before each laboratory visit at 4-, 8-, 12-, and 18-months. For parents who were unable to complete the questionnaires ahead of time, they were instead completed using a laptop during a laboratory visit. Versions of each questionnaire were available in both English and Spanish and were administered based on the caregiver's first language.

Maternal internalizing symptoms.—Maternal internalizing symptoms were assessed using the Beck Depression Inventory (BDI) and Beck Anxiety Inventory (BAI), which were collected at 4-, 8-, 12-, and 18-months. We created a mean score of the BDI and BAI scores at each time point as a composite of maternal internalizing symptoms given the high positive correlations between both measures across timepoints ($r_s = .50$ to $.71$).

The BDI is a 21-item self-report questionnaire for evaluating the severity of depression in healthy and psychiatric populations. Each item consists of a group of related statements and respondents self-report how they have been feeling for the past week on a four-point scale (0 - *symptoms absent*, 1 - *mildly*, 2 - *moderately*, 3 - *severe symptoms*). The BDI is scored by adding the ratings for all 21 items, with a score range from 0 to 63. The BDI has good to excellent scale reliability (4M $\alpha = .89$; 8M $\alpha = .86$; 12M $\alpha = .90$; 18M $\alpha = .91$).

The BAI is a 21-item self-report questionnaire for evaluating the severity of anxiety, designed to distinguish cognitive and somatic symptoms of anxiety from symptoms of depression. Parents rated individual symptoms of anxiety (e.g., fear of losing control) in the past month using a four-point Likert scale (0 - *not at all*, 1 - *mildly*, 2 - *moderately*, 3 - *severely*). The BAI is also scored by adding the ratings for all 21 items, for a score range from 0 to 63. For both measures, higher scores indicate higher levels of symptom severity. The BAI has good to excellent scale reliability (4M $\alpha = .91$; 8M $\alpha = .88$; 12M $\alpha = .93$; 18M $\alpha = .93$).

Infant Temperament.—Dimensions of infant temperament were assessed using mothers' ratings on the Infant Behavior Questionnaire-Revised (IBQ; Putnam et al., 2014) and the Toddler Behavior Assessment Questionnaire (TBAQ; Goldsmith, 1996). Prior studies have demonstrated measurement equivalence between the subscales of the IBQ and the TBAQ (e.g., Goldsmith, 1996). For the present study, a composite of negative emotionality consisting of subscales assessing fear, sadness, and anger (distress to limitations) was used.

The IBQ-R is a 191-item survey designed to assess general patterns of behavior associated with temperament in infancy (3–12 months). Parents rated how often they observed a behavior in the past week at the 4-, 8-, and 12-month time points. Each item describes an infant's behavior (e.g., *During feeding, how often did the baby lie or sit quietly?*) using a 7-point scale (1 = never, 7 = always). Parents are also given a “not applicable” response option for use when the infant has not been observed in the situation described. Each item loads onto one of 14 subscales: Activity Level, Distress to Limitations, Fear, Duration of Orienting, Smile/Laughter, High-intensity Pleasure, Low-intensity Pleasure, Soothability, Falling Reactivity, Cuddliness, Perceptual Sensitivity, Sadness, Approach, and Vocal Reactivity. The Distress to Limitations consists of 16 items and has acceptable scale reliability (4M $\alpha = .77$; 8M $\alpha = .84$; 12M $\alpha = .80$). The Fear subscale consisted of 16 items and had excellent scale reliability (4M $\alpha = .90$, 8M $\alpha = .92$, 12M $\alpha = .91$). The Sadness scale consists of 14 items and has acceptable scale reliability (4M $\alpha = .82$, 8M $\alpha = .83$, 12M $\alpha = .78$). The IBQ-R has demonstrated good internal consistency, reliability, and validity, including correlations with laboratory observations (Gartstein & Marmion, 2008; Goldsmith & Campos, 1990; Parade & Leerkes, 2008).

The TBAQ is a 120-item survey designed to assess general patterns of behavior associated with temperament in young children (2–3 years). It was collected at the 12-, and 18-month time points. Parents rated how often their toddler displayed a specific behavior in the past month using a 7-point Likert scale (1 = never, 7 = always). Each item loads onto one of 11 subscales: Activity Level, Anger, Appropriate Attention Allocation, Inhibitory Control, Interest, Object Fear, Perceptual Sensitivity, Pleasure, Sadness, Social Fear, Soothability.

Items from each subscale are averaged to obtain scale scores. Goldsmith (1996) reported high levels of convergence with various subscales of the IBQ. There was acceptable scale reliability at 18-months for the anger subscale ($\alpha = .77$), the sadness subscale ($\alpha = .64$), the object fear subscale ($\alpha = .82$), and the social fear subscale ($\alpha = .73$).

Respiratory Sinus Arrhythmia (RSA).—Infants and their parents participated in a lab visit when infants were 8-, 12-, and 18-months of age. Electrocardiograph (ECG) data from the infant was continuously recorded during a resting baseline. Gelled sensors were placed by the experimenter on the child's right collarbone, and lower left and right rib prior to resting baseline. Resting RSA was 4 minutes in duration, and the infant was sitting on a parent's lap and given non-stimulating toys to keep them occupied. Parents were instructed to avoid interacting with their infants and to keep as neutral as possible. Ambulatory data collection equipment (PDA) was attached to the back of the parent's chair. At the 18-month visit, the infant wore the PDA inside a small backpack to allow for locomotor activity, although infants were seated on their mothers' laps during the resting task.

ECG was sampled at a rate of 500ms using Mindware MW1000A PDA devices and BioLab system (Mindware Technologies Ltd., Westerville, OH). Data were analyzed offline using the Mindware editing program Mindware HRV, Versions 3.1.4 and 3.1.5, which identified interbeat intervals, and detected physiologically improbable intervals using a validated algorithm. Trained personnel visually inspected ECG data for R-peak and artifact identification. Artifacts in the data from infant crying were excluded from RSA calculations. RSA was calculated in 30-second epochs using the 0.240–1.040 Hz power band, consistent with prior work establishing this range as appropriate for infants and toddlers (Bar-Haim et al., 2000). For participants with less than 4 minutes of data, a minimum of 10 seconds in an epoch at the end of the episode were required to be included in the calculation in order to reduce data loss. Epoch length did not significantly predict RSA after accounting for nesting by participant within our data. On average, participants had 7.91 epochs across all ages. The mean RSA value of all epochs across the resting task was used for analyses. 96.0% of participants' data were usable at 8-months, 94.7% of participants' data were usable at 12-months, and 99.2% of participants' data were usable at 18-months.

Analytic Plan.

Missing Data.—Of note, the full sample ($N=357$) contained data that was not missing not at random, as participants missing RSA data differed significantly on negative emotionality, with infants who did not provide any RSA data at subsequent timepoints having significantly higher negative emotionality scores at 4M than infants who provided at least 1 time point of usable data. Thus, the decision was made to use a subsample of infants who provided at least 1 time point of usable data for our variables of interest. While the subsample of data ($N=217$) was not missing completely at random (MCAR) based on Little's MCAR test (Little, 1988), we tested if missing data differed based on our variables of interest as well as demographic data (e.g., if mothers with and without internalizing symptom scores at 4-months significantly differed based on infant negative emotionality scores). There were no significant differences between data that was and was not missing within the subsample, thus data was established to be missing at random. Thus, full information

maximum likelihood (FIML) was used to account for missing data. FIML estimator in multiple regression models with missing data have shown to produce less biased parameter estimates, especially compared to listwise deletion, pairwise deletion and mean imputation (Enders, 2001).

Random-Intercept Cross-Lagged Panel Model.—A random-intercept cross-lagged panel model (Hamaker et al., 2015) is an extension of the traditional cross-lagged panel model that helps account for stable, trait-like differences between units. A model including random intercepts for maternal internalizing symptoms, infant negative emotionality, and infant RSA, autoregressive paths for maternal internalizing symptoms, infant negative emotionality and infant RSA, and cross-lagged paths between maternal internalizing symptoms, infant negative emotionality and infant RSA was fit to the data using the *lavaan* package (Rosseel, 2012) in the R Environment for Statistical Computing. Random intercepts are latent variables with the repeated measures as its indicators, and all factor loadings are fixed to 1. Auxiliary variables in the model included biological sex of the child, and prenatal medication use. While we do not hypothesize sex (as assigned at birth) differences in our analyses as prior studies on gender differences in temperament only found a very small difference between boys and girls in fear, and no differences in anger (Else-Quest et al., 2006). However, there are findings that suggest parents' responses to children's negative emotionality may differ based on child sex (e.g., Park et al., 1997). Thus, child sex was entered as an auxiliary variable. Significant prenatal medication use (including selective serotonin reuptake inhibitor use, anxiolytics, and psychotics) was included as an auxiliary variable as there is some evidence for associations between prenatal medications and infant neurobiology (e.g., Gemmel et al., 2018; Gentile, 2010). The *lavaan* package uses FIML to account for unbiased estimates of data when data is missing at random. Model fit was evaluated using χ^2 , the comparative fit index (CFI), standardized root mean square residual (SRMR), and the root mean square error of approximation (RMSEA). Good model fit is indicated by $p < .05$, CFI $\geq .95$, TLI $\geq .95$, SRMR $\leq .05$, RMSEA $\leq .05$ (Hu & Bentler, 1999).

Results

Descriptive Statistics

Table 3.1 contains the descriptive statistics and correlations among all key variables. Infant negative emotionality was significantly correlated across all timepoints ($r_s = .46$ to $.70$). Maternal internalizing symptoms were significantly correlated across all timepoints ($r_s = .48$ to $.72$). RSA was also significantly correlated across timepoints ($r_s = .39$ to $.54$).

Random-Intercept Cross-Lagged Panel Model.

The RI-CLPM fit the data well (Figure 1), $\chi^2 = 20.40$, $p = .01$, CFI = 0.99, TLI = 0.97, RMSEA = .03, SRMR = .03.

Intercept factors in a RI-CLPM account for trait-like levels, and our model showed that there was significant variance in maternal internalizing symptoms (10.77, $p < .01$) and infant RSA (0.473, $p < .01$), indicating stable, between-persons differences across time. The intercepts

for maternal internalizing symptoms and infant RSA were also significantly associated ($B=1.50, p < .01$). There was no significant variance in negative emotionality in this sample, suggesting that there are no stable between-infant differences, and that each infant fluctuates around the same individual level means across time. The non-significant variance for infant negative emotionality was subsequently fixed to 0 given that including a non-significant intercept in the model is redundant (Mulder & Hamaker, 2021). Thus, negative emotionality scores reflect both trait-like levels as well as within-person fluctuations as there was no meaningful random intercept.

Concurrent Covariances—Concurrent covariances were included in the model, but not included in Figure 1. At 4-months, there was a significant covariance between maternal internalizing symptoms and child negative emotionality, $B=-0.64, p=.04$. At 8-months, there was a significant covariance between maternal internalizing symptoms and RSA, $B=-1.06, p < .01$. At 12-months, there was a significant covariance between maternal internalizing symptoms and child RSA, $B=-1.08, p=.02$. Finally, at 18-months, there was a significant covariance between maternal internalizing symptoms and child negative emotionality, $B=-1.25, p=.03$.

Autoregressive Paths—Autoregressive paths for maternal internalizing symptoms were significant from 4-months to 8-months ($B=0.34, p < .01$), from 8-months to 12-months ($B=0.52, p < .01$), and from 12-months to 18-months ($B=0.26, p=.03$). For child negative emotionality, there was a significant autoregressive path from 4-months to 8-months ($B=0.80, p < .01$), from 8-months to 12-months ($B=0.74, p < .01$), and from 12-months to 18-months ($B=0.73, p < .01$). There were no significant autoregressive paths for infant RSA.

Cross-Lagged Paths—For interpretation of cross-lagged paths after accounting for the random intercepts, levels of maternal internalizing symptoms and infant RSA are relative to an individual's mean across time. Across all time points, higher maternal internalizing symptoms relative to the individual's mean across time were associated with infant negative emotionality at the subsequent time point. Higher maternal internalizing symptoms at 4-months was associated with lower negative emotionality at 8-months ($B=-0.03, p < .01$). Higher maternal internalizing symptoms at 8-months was associated with lower negative emotionality at 12-months, $B=-0.06, p < .01$. Similarly, higher maternal internalizing symptoms at 12-months was also associated with lower negative emotionality at 18-months, $B=-0.06, p < .01$. Additionally, 8-month maternal internalizing symptoms were negatively associated with 12-month infant resting RSA ($B=-0.09, p < .01$). Similarly, 12-month maternal internalizing symptoms were negatively associated with 18-month infant resting RSA ($B=-0.09, p < .01$).

Negative emotionality at 12-months was associated with resting RSA at 18-months ($B=0.76, p=.03$). There were no cross-lagged paths from RSA to infant negative emotionality.

Lastly, the results supported the hypothesis that there would be infant-directed effects on maternal internalizing symptoms. Negative emotionality was negatively associated with maternal internalizing symptoms at the subsequent time point prior to 18-months: 4-months to 8-months ($B=-1.24, p=.04$), from 8-months to 12-months ($B=-2.37, p=.02$). Additionally,

infant RSA at 12-months was associated with maternal internalizing symptoms at 18-months ($B=-2.07, p=.01$) but not at any earlier timepoints.

Discussion

The present study aimed to better understand the inter-relations between maternal internalizing symptoms, infant negative emotionality, and infant PNS activity across the first two years of life as a possible mechanism by which intergenerational transmission of internalizing symptoms may occur. Specifically, our model was able to examine transactional associations that furthered our understanding of (1) the co-development of child negative emotionality and physiological regulation in the context of maternal internalizing symptoms, and (2) infant-directed effects on maternal postnatal internalizing symptoms. In particular, by accounting for between-person levels, we are able to examine how within-person changes relative to one's mean in one construct may lead to within-person changes in another, and vice versa. While associations between observed variables may be informative for understanding how constructs are generally associated with one another, parsing out the between- and within-dyad associations can provide targets for intervention based on individual differences in risk as well as optimal points during development based on within-dyad fluctuations.

While we were not able to directly test the association between infant negative emotionality, infant resting RSA, and child internalizing symptoms, the present study provides important preliminary evidence that maternal internalizing symptoms may impact the development of child risk for developing internalizing symptoms and furthers our understanding of how internalizing symptoms may develop and be maintained within families during infancy.

Individual differences in maternal internalizing symptoms and resting RSA

We found stable, between-persons differences in both maternal internalizing symptoms during the postnatal period from 4-months to 18-months. This is consistent with prior person-centered work examining maternal internalizing symptom trajectories across infancy to childhood. For example, Ashman and colleagues (2008) show that there are three trajectories of maternal internalizing symptoms from infancy to childhood: stable, low levels of internalizing symptoms, stable/persistent high internalizing symptoms, and decreasing internalizing symptoms. Similarly, we found evidence for stable, between-persons differences in infant resting RSA, consistent with findings from prior work that demonstrates that there are individual differences in levels of resting RSA during infancy (Wagner et al., 2021). It is important to note there was no evidence for a "trait" component of resting RSA during the first two years of life in more diverse samples (e.g., low-income Mexican American infants) (Jewell et al., 2018). This work suggests that continuity in levels of resting RSA may not be universal, and the broader environment (beyond the parent-child dyad) may also play a role.

Contrary to our hypotheses, we did not find a significant intercept for infant negative emotionality, which suggests that there were no stable, between-infant differences across time, and that each infant fluctuates around the same individual level mean. One possibility is that temperamental traits such as negative emotionality are not stable during infancy,

and thus we do not find stable, between-infant differences from 4- to 18-months. This is consistent with work by Lemery and colleagues (1999) that show that different dimensions of temperament, including distress-anger and fear, are less stable from 3-months to 18-months. It may be that individual differences are not emerging during this early developmental period as there is a heavy reliance on caregivers for regulation. Future research should examine these associations into early childhood and school entry as a possible transitional period where individual differences in negative emotionality may emerge.

An alternative explanation for the lack of stable, between-person differences in infant negative emotionality is rooted in our specific analytic sample, as infants who provided usable resting RSA data had significantly lower levels of negative emotionality relative to the full sample. Our RSA data were not missing at random, as infants without any usable RSA data at any time point had significantly higher scores for 4-month negative emotionality than infants who provided usable RSA data for at least one time point. It is possible that stability in negative emotionality is only present for infants who are at the higher end of the distribution compared with their same-aged peers, and thus at higher risk for internalizing symptom development (Goldsmith & Lemery, 2000). Brooker and colleagues (2013) found four different trajectories of stranger fear from 6-months to 36-months, but only 11.8% of infants remained high and stable across time while the rest of the infants exhibited change across time. In particular, the infants in the high, stable group were associated with later behavioral inhibition. Behavioral inhibition, in turn, is a temperament trait associated with sensitivity to social novelty in early childhood, and the later emergence of internalizing difficulties (Perez-Edgar & Fox, 2005). Indeed, the specific association between behavioral inhibition and social anxiety emerges for children with high, and stable, levels of temperamental risk. It is possible that infants in this study who may be at the higher end of the distribution may not have provided usable physiological data, and thus we did not find significant variance in the random intercept of negative emotionality.

Associations between Maternal Internalizing Symptoms and Infant Negative Emotionality

Results from the present study also provided evidence for within-dyad associations between maternal internalizing symptoms and infant negative emotionality and infant resting RSA. Higher levels of maternal internalizing symptoms (relative to the mean) were associated with lower levels of negative emotionality at the next time point from 4-months to 12-months. This is contrary to our hypotheses, as we expected to find that higher levels of maternal internalizing symptoms would be associated with higher levels of infant negative emotionality given prior research reporting positive associations between maternal internalizing symptoms and higher levels of negative emotionality (e.g., Gartstein et al., 2010).

One possible explanation is that our sample is relatively low-risk with respect to overall low levels of maternal internalizing symptoms. Prior research has often recruited mothers who meet clinical criteria for an anxiety or depression diagnosis. In our sample, our measure of maternal internalizing symptoms may not reflect psychopathology, and instead reflect normative levels of maternal distress. Additionally, there is some emerging evidence that a

midrange of anxiety symptoms may be associated with better infant outcomes as a function of higher maternal sensitivity to infant cues (e.g., Lemus et al., 2022). Our sample was not recruited for elevated levels of maternal internalizing symptoms, and thus our findings of negative associations between maternal internalizing symptoms and subsequent infant negative emotionality may reflect this idea that some maternal internalizing symptoms may be optimal for infant socioemotional development, as mild to moderate levels of maternal internalizing symptoms may be mediated by positive parenting behaviors.

It is also possible that when we examine maternal postpartum distress as a combination of maternal depressive and anxiety symptoms, we are not accounting for differential effects of maternal depressive and anxiety symptoms on infant socioemotional development that may be mediated by different parenting behaviors. Of note, there were low rates of maternal depressive symptoms in our sample, and so our findings may be explained by relatively higher levels of anxiety. For example, there is research that demonstrates that clinically anxious mothers do not show deficits in sensitivity; rather, higher levels of maternal anxiety symptoms were associated with increased attunement to 12-month-old infants' emotional arousal (Kaitz et al., 2010; Smith et al., 2021). Recent work also demonstrates that maternal sensitivity is associated with lower toddler negative emotionality, but that these associations were moderated by maternal emotion dysregulation (Bailes & Leerkes, 2023). Future research should aim to examine if there are dimensions of parenting behaviors, such as parental sensitivity, that may help potentially explain the negative associations between within-person change in maternal internalizing symptoms on infant negative emotionality.

While we did not find significant variance in the intercept factor for negative emotionality, we found continuity in negative emotionality across time. All autoregressive paths for negative emotionality were significantly associated, representing within-person carry-over effects. That is, an infant higher on negative emotionality at one time point is likely to experience higher negative emotionality at the next occasion as well. This is consistent with work that demonstrates increases in negative emotionality across infancy and toddlerhood (Aktar & Pérez-Edgar, 2020; Brooker et al., 2013; Gartstein et al., 2010).

Associations between Maternal Internalizing Symptoms and Infant Resting RSA

We also found a significant, positive association between the trait-like levels of maternal internalizing symptoms and infant RSA. Mothers who have higher overall maternal internalizing symptoms across time have infants with higher levels of resting RSA. Higher levels of resting RSA are often thought to reflect a greater capacity for physiological regulation, and are linked with better socioemotional outcomes (Beauchaine, 2001; Porges, 2007). Infants with mothers higher in internalizing symptoms may be exposed to more situations that require self-regulation than infants with mothers with lower internalizing symptoms, thus developing higher levels of resting RSA in order to meet contextual demands. However, another interpretation is that higher levels of resting RSA also reflect greater sensitivity to the environment. For instance, studies have shown that resting RSA often moderates associations between environmental factors and child socioemotional outcomes (e.g., Bandon et al., 2008). Thus, it is possible that infants with higher trait levels

of RSA are more sensitive to their environments, which in turn supports the emergence of positive associations between maternal traits and infant outcomes.

Additionally, there were within-dyad associations between maternal internalizing symptoms and subsequent levels of infant resting RSA even after accounting for between-dyad associations. Within each mother-infant dyad, mothers exhibiting higher levels of internalizing symptoms relative to their mean had infants with subsequent lower levels of resting RSA relative to their mean from 8- to 18-months. Importantly, the between-person associations were opposite of the within-person's associations between maternal internalizing symptoms and resting RSA. While higher levels of overall maternal internalizing symptoms are associated with higher levels of infant resting RSA, there are negative cross-lagged associations indicating that maternal internalizing symptoms at the prior time point are associated with later lower resting RSA relative to trait levels. Mothers who exhibit higher levels of internalizing symptoms relative to their mean may have children whose resting RSA are becoming lower relative to their mean, indicating the development of poorer physiological regulation.

Co-Development of Infant Negative Emotionality and Infant Resting RSA in the context of Maternal Internalizing Symptoms

After accounting cross-lagged paths from maternal internalizing, we did not find reciprocal associations between infant negative emotionality and infant resting RSA from 4- to 12-months. Our results suggest that negative emotionality and RSA are not associated with one another during the first year of life. At 12-months, higher levels of negative emotionality were positively associated with subsequent 18-month RSA. Prior work has demonstrated negative associations between resting RSA and emotional reactivity during infancy, such that higher levels of resting RSA were associated with shorter latency to negative affect during an affective task (Calkins, 1997). However, these associations were found during concurrent measures of negative emotionality and resting RSA during early childhood (Calkins, 1997).

Turning to literature on behavioral measures of reactivity and regulation during infancy, studies have found that 5-month reactivity was negatively associated with 10-month regulation (Braungart-Rieker & Stifter, 1996). Our findings of positive associations between negative emotionality and resting RSA are opposite to findings from prior work. It is important to note that the co-development of negative emotionality and resting RSA is examined in the present study within the context of the mother-infant dyad. The direction of effects may be, in part, due to accounting for associations with maternal internalizing symptoms, as well as accounting for trait-like levels of resting RSA. The discrepancy in findings highlight the importance of considering how infants are developing within a social context, as parental characteristics may have influences on infants' emotional reactivity and regulation. After accounting for the effects of maternal internalizing symptoms on 18-month RSA, higher levels of negative emotionality are associated with higher than expected levels of RSA after accounting for an infant's mean resting RSA across time. Additionally, given that we did not find between-individual differences in negative emotionality and could not examine associations with RSA on a between-individuals level, it may be that we

see a positive association emerge in the cross-lagged associations instead. Similarly, one way to interpret the positive cross-lagged association may be that resting RSA reflects greater sensitivity to the environment such that higher levels of negative emotionality (as a marker of greater reactivity to contextual demands) is positively associated with greater physiological sensitivity to context.

Additionally, maternal internalizing symptoms have been associated with infant negative emotionality via the development of the ANS (de Vente et al., 2020). Prior work on developmental changes in temperament also suggests that maturational changes in biological systems in the individual underlies within-person change in temperament (e.g., Gartstein et al., 2010). However, our findings did not support this hypothesis, as we did not find cross-lagged associations from resting RSA to negative emotionality. It may be that there are different biological processes underlying negative emotionality that resting RSA does not influence at this point in development, or relations are found at more extreme levels of negative emotionality. Future work should examine other biological indices that may be associated with the development of negative emotionality or other behavioral indices of reactivity.

Infant-Directed Effects on Maternal Internalizing Symptoms

Consistent with our hypotheses and extant literature, we also found evidence for infant-directed effects on maternal internalizing symptoms. Both infant negative emotionality and infant resting RSA may elicit changes in maternal internalizing symptoms, although cross-lagged associations between infant resting RSA and subsequent maternal internalizing symptoms only emerge after the first year of life.

We found negative associations between infant negative emotionality and subsequent change in maternal internalizing symptoms during the first year of life, such that higher negative emotionality was associated with lower maternal internalizing symptoms relative to the mean. This is contrary to our hypotheses as we expected to find that higher levels of negative emotionality would be associated with higher levels of maternal internalizing symptoms at the next time point. Infants expressing higher levels of negative affect and negative emotionality often elicit stronger maternal negative affect and increased internalizing symptoms (Whiffen & Gotlib, 1989; Murray, 1979; Behrendt et al., 2020). However, it is important to note that prior studies did not account for trait levels of maternal internalizing symptoms, and did not examine repeated measures of infant or maternal constructs. Effects that were not evident in prior studies, as well as bivariate correlations within the same dataset, highlight the importance of considering both individual differences and temporal precedence to understand how maternal internalizing symptoms and child negative emotionality may co-develop.

One interpretation of this finding is that not all negative emotionality may be indicative of risk for the development of socioemotional problems. It is likely that negative emotionality plays an adaptive role during infancy when infants are reliant on caregivers for regulation. From a functionalist perspective on emotion, fear, sadness, and anger have adaptive functions: for example, anger is associated with restoration of progress towards a goal and effecting a change in the behavior of a social other, while fear is associated with

avoidance of pain, and alerting others to avoid the situation or help (Campos et al., 1983; Bornstein et al., 2014). Negative emotionality may be adaptive and elicit parenting behaviors such as maternal sensitivity as it alerts parents to the needs of the infant. Prior research has demonstrated that child distress is also associated with children seeking out comfort from parents (Buss et al., 2004). It may be that the adaptive nature of negative emotionality during this developmental period may be associated with fluctuations in maternal internalizing symptoms and lower parental distress. It is important to note that our measure of infant negative emotionality captures broad dimensions of negative emotionality in multiple contexts, and future research should consider examining negative emotionality in different contexts as a potential risk factor that may be positively associated with maternal internalizing symptoms. For example, work by Buss and colleagues (2021) demonstrate that behavioral fear in low-threat contexts during toddlerhood was associated with both maternal overprotection, as well as maternal general anxiety symptoms. Researchers may find different associations between negative emotionality and maternal internalizing symptoms when considering infant negative emotionality in different contexts.

Next, the finding that higher levels of resting RSA at 12-months relative to an infant's average RSA was associated with lower maternal internalizing symptoms relative to trait levels of maternal internalizing symptoms suggests that better regulatory capability is associated with subsequent decrease in mothers' internalizing symptoms. We found a direct effect from resting RSA to maternal internalizing symptoms from 12-months to 18-months, and did not find evidence that this association may be behaviorally mediated by negative emotionality. Infants higher in resting RSA relative to their own mean may be exhibiting more regulatory behaviors contributing to the decrease in maternal distress at the subsequent time point. As negative emotionality is capturing dimensions of behavioral reactivity, there may be behavioral regulation that may be mediating associations between resting RSA and maternal internalizing symptoms. Taken together, there is evidence for joint contributions of reactivity and regulation that may play a role in changes in maternal internalizing symptoms across infancy.

Limitations and Future Directions

It is important to interpret findings from the current study with limitations in mind. One consideration is that mothers rated both their internalizing symptoms as well as infant temperament. There have been some mixed findings on how maternal internalizing disorders or symptoms may be associated with their perceptions of infant behavior. For example, there is some evidence that mothers higher in depressive symptoms were more likely to report more problems than their child's self-report (van der Toorn et al., 2010). However, Olino and colleagues (2021) found little psychometric evidence for maternal psychopathology biasing reports of child psychopathology symptoms. In the current study, our measure of infant temperament may be a better reflection of mothers' interpretations of infant negative emotionality. Future work should aim to replicate these findings using a different caregiver's report of temperament, or external observer scores of negative emotionality to ensure that maternal symptoms are not biasing infant negative emotionality scores.

Another methodological consideration of the physiology data used in the current study is that we did not collect movement and respiration data. While movement artifacts were visually inspected by trained personnel and accounted for in data processing, respiration rate was not accounted for. Thus, measures of RSA that do not account for respiration rate may reflect a combination of both cardiac vagal control and other changes in respiration independent of the vagus nerve (Houtveen et al., 2002; Allen et al., 2007; Ritz et al., 2012). However, there are also studies that establish that RSA uncorrected for respiration is accurately reflecting PNS activity during tasks where respiration is not expected to greatly vary (Houtveen et al., 2002; Allen et al., 2007). Given that we did not expect respiration to greatly vary during our resting task, our physiology data may still primarily reflect vagal activity even without accounting for respiration rate.

Additionally, we do not have measures of prenatal maternal internalizing symptoms and distress and therefore we are unable to account for any associations between prenatal maternal internalizing symptoms, infant negative emotionality, and infant RSA. This is important as there is research suggesting that prenatal maternal distress can play a role in fetal programming, especially with stress response systems (Del Giudice, 2012). Additionally, empirical studies have found associations between prenatal maternal internalizing symptoms and infant RSA (Propper & Holochwost, 2013), as well as associations between prenatal maternal distress and behavioral fear (Nolvi et al., 2019). However, one study that accounted for both prenatal and postnatal developmental periods only found associations between maternal emotion dysregulation and infant RSA during the postnatal period, but did not find evidence for associations between prenatal maternal emotion dysregulation and postnatal infant RSA (Gao et al., 2022). These studies suggest that the associations between maternal internalizing symptoms, infant negative emotionality, and infant RSA may be largely due to genetic and environmental processes as opposed to prenatal programming—however, future studies should aim to test these associations directly to disentangle prenatal programming from environmental processes.

Paternal mental health during infancy is also associated with infant socioemotional development. For example, there is a study showing that paternal anxiety symptoms are associated with infant temperament (Potapova et al., 2014). Additionally, infant negative emotionality is also associated with fathers' depressive symptoms (Cioffi et al., 2021b). More broadly, paternal anxiety is prospectively associated with child anxiety, but maternal anxiety is also associated with subsequent paternal anxiety (Ahmadzadeh et al., 2019), highlighting the importance of considering the family unit as a whole.

An additional consideration is the range of negative emotionality in infants and maternal internalizing symptoms in this sample. As aforementioned, infants that did not provide any RSA data were significantly higher in negative emotionality at 4-months than the infants that provided usable RSA data. Therefore, we cannot assume that these associations hold when there are infants who exhibit higher negative emotionality. Using imputation methods may help account for missing data, but one major limitation of doing so would be assuming that the associations between variables are linear even at the higher end of the distribution for negative emotionality. Similarly, our sample was not recruited for elevated, clinical levels of postpartum maternal internalizing symptoms. It is less clear if these associations would be

the same in samples that include a wider range of maternal internalizing symptoms. Future research should aim to examine if these associations remain the same when samples include both infants with higher levels of negative emotionality and mothers with higher levels of internalizing symptoms.

Conclusions

The present study extends the current literature by examining both between- and within-dyad associations between maternal internalizing symptoms, infant negative emotionality and infant resting RSA during the first two years of life. Specifically, the findings from the present study provide evidence for both mother-to-infant as well as infant-to-mother associations over time. The bidirectional associations from the present study suggest that while maternal internalizing symptoms are associated with the development of risk for child internalizing behaviors, infant-directed effects may also serve to maintain the presence of internalizing behaviors and symptoms within families. A better understanding of these transactional processes provides some evidence for processes that may underlie intergenerational associations between parent and child internalizing behaviors, and may have implications for targets for prevention and intervention for both mothers and their children during infancy and toddlerhood. The use of longitudinal methods can help elucidate the nuances in development that occur on multiple levels of analysis, and further our understanding of the importance of parental internalizing symptoms in the co-development of child reactivity and regulation.

Acknowledgments

Data for this study is supported by grants from National Institutes of Health awarded to Pérez-Edgar, Buss, and LoBue (R01MH109692). Drs. Buss and Pérez-Edgar's Psychology Professorships are supported by the Social Science Research Institute of The Pennsylvania State University, and endowments through the Tracy Winfree and Ted H. McCourtney Professorship in Children, Work, and Families (Buss) and the McCourtney Professorship of Child Studies (Pérez-Edgar). The authors declare they have no conflicts of interest. We would like to thank the families involved with the study, as well as the staff and larger research team for their contributions to the study.

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Highlights:

- Maternal factors may play a role in infant reactivity and regulation development
- Infants may also influence their mothers' internalizing symptoms

Findings show complex, bidirectional associations between mothers and infants

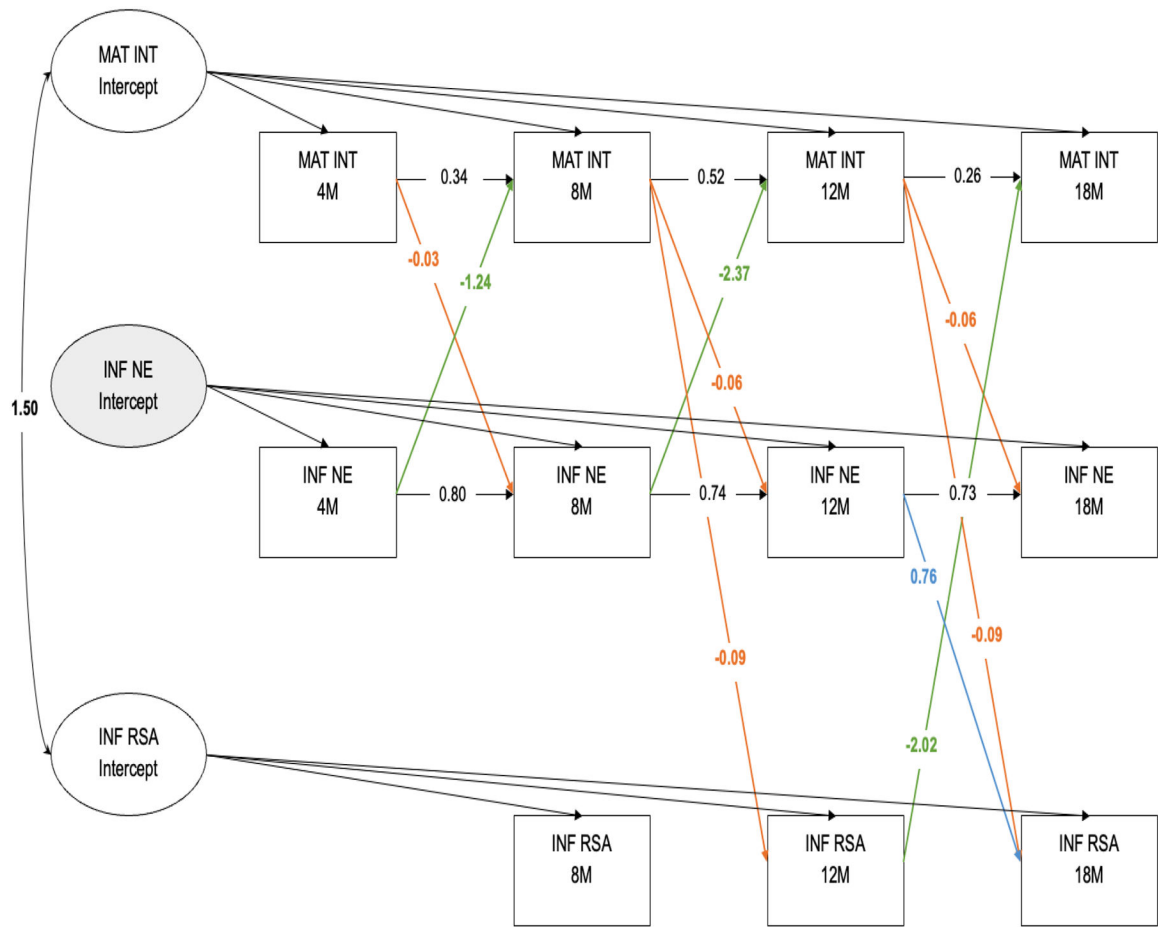


Figure 1. Random-Intercepts Cross-Lagged Panel Model of Maternal Internalizing Symptoms, Infant Negative Emotionality and Infant Resting RSA from 4-months to 18-months. Significant cross-lagged paths ($p < .05$) are indicated by estimates and solid lines. MAT INT = Maternal Internalizing Symptoms composite. INF NE = Infant Negative Emotionality. INF RSA = Infant Resting RSA

Table 1.

Means, standard deviations, and correlations with confidence intervals.

Variable	<i>M</i> (<i>SD</i>)	1	2	3	4	5	6	7	8	9	10
1. INF NE 4	2.94 (0.68)										
2. INF NE 8	3.23 (0.73)	.70** [.61,.77]									
3. INF NE 12	3.45 (0.71)	.49** [.36,.60]	.62** [.51,.71]								
4. INF NE 18	0.64 (0.64)	.53** [.39,.64]	.47** [.33,.58]	.46** [.33,.58]							
5. MAT INT 4	6.01 (5.63)	.09 [-.06,.23]	.11 [-.05,.26]	.11 [-.06,.27]	.12 [-.06,.28]						
6. MAT INT 8	4.86 (4.98)	.13 [-.03,.28]	.22** [.08,.36]	.16 [-.00,.32]	.12 [-.05,.28]	.72** [.63,.79]					
7. MAT INT 12	6.11 (7.06)	.07 [-.11,.24]	.22* [.05,.38]	.23** [.07,.38]	-.03 [-.20,.14]	.52** [.38,.64]	.64** [.52,.73]				
8. MAT INT 18	5.69 (6.56)	.04 [-.13,.21]	.15 [-.02,.31]	.17 [-.00,.33]	-.01 [-.17,.15]	.48** [.33,.61]	.60** [.47,.70]	.55** [.41,.66]			
9. RSA 8	3.54 (0.97)	-.02 [-.17,.14]	.05 [-.09,.20]	.08 [-.08,.23]	.01 [-.16,.17]	.02 [-.13,.18]	.11 [-.04,.25]	.10 [-.08,.27]	.15 [-.02,.31]		
10. RSA 12	3.78 (0.91)	.11 [-.07,.28]	.20* [.03,.36]	.11 [-.06,.27]	.16 [-.01,.33]	-.08 [-.26,.10]	.12 [-.06,.30]	.01 [-.17,.19]	-.05 [-.23,.13]	.54** [.41,.65]	
11. RSA 18	3.93 (0.98)	.08 [-.12,.28]	.12 [-.09,.31]	.07 [-.13,.26]	.14 [-.05,.33]	-.14 [-.33,.07]	-.03 [-.23,.18]	-.05 [-.26,.16]	.07 [-.13,.27]	.39** [.22,.55]	.51** [.35,.65]

Note. *M* and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation.

* indicates $p < .05$.

** indicates $p < .01$.