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Research Paper

Infant negativity moderates trajectories of maternal emotion across pregnancy and the peripartum period

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ABSTRACT

Background: Although the effects of maternal behavior on the development of child emotion characteristics is relatively well-established, effects of infant characteristics on maternal emotion development are less well known. This gap in knowledge persists despite repeated calls for including child-to-mother effects in studies of emotion. We tested the theory-based postulate that infant temperamental negativity moderates longitudinal trajectories of mothers' perinatal symptoms of anxiety and depression.

Method: Participants were 92 pregnant community women who enrolled in a longitudinal study of maternal mental health; symptoms of anxiety and depression were assessed during the second and third trimesters of pregnancy and again at infant age 4 months. A multimethod assessment of infants' temperament-based negative reactivity was conducted at infant age 4 months.

Results: Maternal symptoms of anxiety showed smaller postnatal declines when levels of infant negativity were high. Mother rated and observed infant negative reactivity was related to smaller postnatal declines in maternal anxiety, while infant negative reactivity, at the level of neuroendocrine function, was largely unrelated to longitudinal changes in maternal anxiety symptoms. Infant negativity was related to early levels, but largely unrelated to trajectories of maternal symptoms of depression.

Limitations: Limitations of this work include a relatively small and low-risk sample size, the inability to isolate environmental effects, and a nonexperimental design that precludes causal inference.

Conclusions: Findings suggest that levels of infant negativity are associated with differences in the degree of change in maternal anxiety symptoms across the perinatal period.

1. Infant negativity and moderates trajectories of maternal emotion across pregnancy and the peripartum period

Although maternal propensities for positive and negative emotion predict offspring risk for emotional problems as early as infancy (Crockenberg and Leerkes, 2004; Kagan and Snidman, 1999; Madigan et al., 2018), there remains a dearth of scientific work testing infant characteristics as predictors of trajectories of risk for mental illness in mothers. Such predictive value may be particularly beneficial for highly prevalent postpartum syndromes, like depressive or anxiety conditions, which impact millions of women each year (Andersson et al., 2006; Giardinelli et al., 2012; Reck et al., 2008) and have established patterns of normative development across the peripartum period (Blair et al., 2011; Heron et al., 2004; Ross and McLean, 2006). Delineating such an effect would extend existing models of emotional development in the mother-infant dyad and offer a foundation for longitudinal process models of dyadic emotional development. To address this gap in the literature and begin to build just such a foundation for future research, we investigated infant negative emotion as a moderator of longitudinal trajectories of maternal symptoms of anxiety and depression across the

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peripartum period.

2. Normative symptoms of depression and anxiety in mothers

Roughly 9–19% of postpartum women suffer from major depressive or dysthymic conditions, compared to 6–10% of women in the general population (O'Hara and McCabe, 2013). Peripartum depressive symptoms follow a normative nonlinear trend, onsetting during pregnancy and gradually declining after childbirth (Heron et al., 2004). Symptom onset in pregnancy, rather than postnatally, suggests that a mother's experience of her infant as highly negative is less likely to impact symptom onset than symptom trajectory, as a mother cannot experience her infant as negative until after they are born. Thus, we hypothesized that infant negativity would predict individual differences in the decline of peripartum symptoms, with mothers of highly negative infants showing smaller declines (i.e., greater persistence) in symptoms of depression during the peripartum period.

Anxiety symptoms are also common in peripartum and postpartum mothers, with a prevalence of roughly 11% (Dennis et al., 2017) or higher when considering subclinical symptoms (Kingston et al., 2018). Symptoms of anxiety following childbirth most commonly manifest as elevated worry as women transition to a new role (Ross and McLean, 2006). Prenatal symptoms may similarly manifest or may appear as worries specifically related to fetal health and childbirth (Blackmore et al., 2016). Conventional and pregnancy-related symptoms have distinct trajectories. Both conditions onset before childbirth (Ross and McLean, 2006). However, pregnancy-specific concerns decline across pregnancy (Ekelin et al., 2009; Tsartsara & Johnson, 2006) while conventional symptoms follow a trajectory similar to depressive symptoms, increasing and then declining postpartum (Blair et al., 2011; Ross and MacLean, 2006). Again, normative postpartum declines in conventional symptoms set an expectation for nonlinear trajectories and a likelihood that infant characteristics impact the symptom course rather than onset.

3. Infant negative affect across levels of assessment

The propensity to experience and express negative emotion, or infant negativity, is a biologically-rooted temperament characteristic present in the first months of life (Goldsmith et al., 1982). In infancy, negativity may take the form of heightened motor activity, crying, and/or changes in body posture or facial expressions in response to emotional challenge (Goldsmith and Rothbart, 1999). Negativity observed during fear-eliciting paradigms as early as infant age 4 months is moderately stable through age three, with greater stability for infants with more extreme temperament classifications (Pfeifer et al., 2002). Because temperament is rooted in biological function, reactivity has also been operationalized as increased neuroendocrine output (specifically in the hypothalamic-pituitary-adrenocortical [HPA] axis) in response to an emotional stressor (Gunnar, 1992). Neuroendocrine output can be noninvasively measured as the concentration of cortisol, a major HPA product, in infant saliva. Increases in salivary cortisol, though not specific to the infant stress response, correlate with other indices of temperament-based negative reactivity (Buss et al., 2003). Given conceptual convergence, the ideal for studies of temperament is a multimethod approach that assesses reactivity at both behavioral and biological levels. Though single composites are not always justified across measures (Goldsmith et al., 2012; Zentner and Bates, 2008), assessment at multiple levels offers unique insight regarding conclusions that can be drawn from the work. Namely, findings that converge across measurement levels underscore the relevance of a single, broad-level construct (Campbell and Fiske, 1959) whereas divergent findings highlight nuances in complex developmental processes (Goldsmith et al., 2012).

Anxious mothers are likely to have infants who are high in temperament-based negative reactivity as assessed via parent report (Vaughn et al., 1987), observation (Gartstein et al., 2010) and/or psychophysiological function (Brennan et al., 2008), though single studies rarely include multiple levels of measurement. More negative child emotion characteristics predict more negative and less sensitive and responsive maternal behaviors (Gouze et al., 2017; Ravindran et al., 2019), though it is less clear whether negativity in infants might predict parental emotions, such as symptoms of depression or anxiety. Such associations may be particularly salient during sensitive periods such as the pre- to postpartum transition (Brennan et al., 2008). Cross-sectional work suggests that infant negativity positively predicts maternal depressive (Murray et al., 1996) and anxiety symptoms (Britton, 2011). Longitudinal designs similarly show that temperamental negativity in infants' the first years of life predict subsequent levels of maternal anxiety (Brooker et al., 2015; Buss et al., 2021) and depression (Cioffi et al., 2021; Gross et al., 1994) and indirectly predict maternal mental health over time (Kuckertz et al., 2018). While this work is important, an absence of prospective longitudinal designs allowing a test of child-based effects on trajectories of maternal mental health leave a dearth of information on emotional development in mothers of infants. Moreover, an absence of such work across the perinatal period maintains the possibility that differences were present prior to the infant's birth, consistent with mother-to-child rather than child-to mother effects.

4. Current study

We conducted a prospective longitudinal multimethod investigation to test whether infant negativity moderated trajectories of maternal depression and anxiety symptoms. We conducted this investigation across the peripartum period for three reasons. First, there is both established variability and normative patterns of maternal symptoms across this time, allowing for the replication of known trajectories and test of our hypothesis of moderation. Second, maternal symptoms of anxiety and depression are highly prevalent and influential across the peripartum period, with consequences for both maternal and child outcomes. Finally, assessing prenatal through postnatal change controls for pre-existing associations between infant and mother characteristics, as infants were not yet born at the initial assessment. We hypothesized that symptoms of depression and anxiety would generally follow the nonlinear pattern of increases followed by decreases over time. Moreover, we hypothesized that infant negativity would moderate nonlinear change such that postnatal declines in symptoms of anxiety and depression would be reduced when infants were high in negative reactivity. We tested our hypothesis using a longitudinal, multi-method design to allow for an examination of convergence or divergence across indices of negative reactivity.

5. Method

5.1. Participants

Approval for study procedures was obtained from the Human Subjects Committee of the Institutional Review Board at Montana State University. Our minimum target sample size was $N = 80$, based on an *a priori* power analysis, so we attempted to enroll 100 total participants to account for an overall attrition rate of roughly 20%. Eight mothers indicated interest in the study but never participated, resulting in a sample of 92 pregnant women. Most mothers were recruited through informational brochures distributed to women at their regular checkups through local physicians (26%), e-mail announcements through the local university (25%), and word-of-mouth referrals (27%). Mothers were also recruited through flyers at local businesses (13%), referrals from the local Women, Infants, and Children office (7%), and announcements in local clubs (2%). Both primigravida and multigravida mothers were allowed to enroll.

Participation included two prenatal laboratory visits: one each during the second ($M = 21.15$ weeks; SD=3.79) and third trimester ($M = 35.92$ weeks; SD=1.47), and a postnatal visit at infant age 4 months (M

= 4.27, *SD*=0.62). All infants were born between 34 and 44 weeks of age (*M* = 39.64, *SD*=1.75) The target gestational age for each visit was selected based on study goals and funding: because we wanted to cover both pregnancy and postnatal periods, assessments occurred in the second trimester (~20 weeks), third trimester (~36 weeks, selected based on records of gestational age for live, singleton births), and as early in the postnatal period as infants could be reliably tested using our behavioral paradigms (~4 months). Including three data points is consistent with gold standards in the field and allowed us to capture nonlinear change in maternal symptoms.

Rolling recruitment resulted in 81 mothers in the second trimester, 86 mothers in the third trimester visit (12 new participants, 1 skipped because infant was born early, 6 withdrew from study), and 75 mothers postnatally (8 additional withdrawals, 3 participants whose infants were not yet 4 months old when the study was ended because the PI switched institutions).

Most mothers (89%) were White (Asian = 8%, American Indian = 1%, African-American = 1%, Mixed Race = 1%) and Non-Hispanic (96%). The mode level of education was a college degree (36%). Gross annual income ranged from less than $15,000 to more than $91,000; most families reported earning $51,000–$60,000 (17%) or more than $91,000 (25%) per year. On average, mothers were in their early thirties at enrollment (*M* = 30.49, *SD*=4.22).

Pregnancy complications were reported by mothers at the postnatal visit. Using a questionnaire adapted from previous work (Marceau et al., 2013), mothers reported the presence or absence of 28 conditions (e.g., flu, lead exposure, diabetes, etc.), experiences (e.g., induction, Caesarian section, etc.), and medications taken at any time during their pregnancy. Numbers of pregnancy and labor and delivery complications were low (*M* = 3.35, *SD*=2.22). The most commonly-reported prenatal complication was a fever or chills (*n* = 7) and the most common delivery-related risk factor was labor induction (*n* = 21). Mothers reported using very few medications during pregnancy (*M* = 1.02, *SD*=1.13), most frequently reporting treatments for heartburn or nausea. Given the low-risk nature of the sample, pregnancy and delivery complications were not considered further.

5.2. Procedure

5.2.1. Prenatal visits

Procedures were identical for the prenatal visits. Two weeks prior to the laboratory visit, mothers were mailed a packet of questionnaires to complete, including assessments of anxiety and depressive symptoms. A full outline of study procedures and measures are available through the Open Science Framework.

5.2.2. Postnatal visit

Questionnaires for the postnatal visit were identical to the prenatal assessments, but additionally assessed pregnancy and childbirth complications and infant temperament. Infants completed a series of 5 emotion-eliciting episodes (fear, anger, positivity). Infant baseline cortisol was collected upon arrival and at the end of the laboratory visit.

5.2.3. Measures

Our assessment of maternal symptoms focused on conventional symptoms of maternal anxiety and depression given our interest in investigating a trajectory that included equivalent postpartum symptoms.

5.2.4. Maternal symptoms of depression

Maternal depressive symptoms were assessed using the Edinburgh Postnatal Depression Scale (EPDS; Cox et al., 1987). The EPDS is a 10-item measure that asked mothers to rate, on a 4-point scale (0=*absence of the symptoms*, 3=*most frequent experience of the symptoms*), the degree to which they experienced a variety of symptoms over the last 7 days (e.g., feeling sad or miserable). Internal consistency was acceptable at each visit (second trimester α=0.82, third trimester α=0.88, postnatal visit α=0.82). The EPDS has demonstrated validity across the peripartum period (Ji et al., 2011). Mothers reported experiencing nearly the full range of symptoms across the three visits. No mothers reported experiencing zero symptoms. Fifteen mothers (16%) reported levels of depressive symptoms above the traditional threshold for clinical significance (>12; Cox et al., 1987).

5.2.5. Maternal symptoms of anxiety

Maternal anxiety symptoms were assessed using the Penn State Worry Questionnaire (PSWQ; (Meyer et al., 1990), a 16-item measure that asked mothers to describe, using a 5-point scale (1=*not at all typical*, 5=*very typical*), the degree to which items were characteristic of them (e.g., worrying all the time). The PSWQ showed acceptable internal consistency (second trimester α=0.91, third trimester α=0.93, postnatal α=0.93) and has demonstrated validity across the peripartum period (Wenzel et al., 2005). Mothers reported experiencing nearly the full range of symptoms across the three visits. No mothers reported experiencing zero symptoms. Twenty-four mothers (26.09%) reported levels of anxiety symptoms comparable to those reported by individuals with a clinically diagnosed anxiety disorder (>53; Brown et al., 1992).

5.2.6. Maternal-rated infant negativity

Mothers reported on infant negativity via the Infant Behavior Questionnaire – Revised version (IBQ-R; Gartstein and Rothbart, 2003), a 191-item measure that asked mothers to describe, using a 7-point scale (1=*never*, 7=*always*), the extent to which various behaviors were typical of their child over the past 1–2 weeks. Given the hypotheses of the current study, we focused on the Negative Affectivity scale, comprising 66 items tapping sadness (e.g., crying when left alone), distress to limitations (e.g., frustration at being unable to change circumstances), fear (e.g., distress to novelty), and falling reactivity (e.g., ease of sleeping). Individual items were averaged to create a mean composite. Internal consistency was acceptable (α=0.83).

5.2.7. Observed negativity with mother

Infant negativity was also assessed at age 4 months using the Masks paradigm from the Prelocomotor version of the Laboratory Temperament Assessment Battery (Lab-TAB; Goldsmith and Rothbart, 1999), which allows for the observation of fear in a nonsocial context. Infants were buckled into a car set affixed to the top of a table and surrounded on three sides by grey wooden walls mounted on the tabletop. The front wall, directly facing the infant, included a 12″ by 12″ opening in the center and a 3″ by 6″ opening to the immediate right. The larger opening, covered by a black curtain, was used for stimulus presentation. The smaller opening was used for videotaping from a camera mounted on a tripod behind the structure. The grey enclosure walls and openings were constructed per Lab-TAB manual instructions. Mothers were seated beside their infant, but out of view, approximately 1 m away. The experimenter, seated behind the large window of the enclosure, drew the infant's attention toward the curtain by knocking on the booth wall. When the infant's attention was focused on the curtain, the experimenter displayed a mask for 10 s. The experimenter then removed the mask from the infant's view, letting the curtain down for 5 s before displaying the next mask. This sequence was repeated to display a total of 4 masks in increasing order of putative threat: an evil queen, an old man, a glow-in the dark vampire, and a gas mask.

Because mothers are asked to remain uninvolved, the traditional version of Masks does not necessarily offer information about infants' fear reactivity in contexts where mothers are available. To get a more valid assessment of infants' levels of fear reactivity during interactions that include mothers, we repeated the episode with a validated modification (Crockenberg and Leerkes, 2004; 2006). The procedure was repeated in exactly the same way except that mothers were told that they could talk to and interact with their infants as they normally would. The two administrations always occurred in the same order to maintain

the novelty of the masks during assessments of temperament. Because the goals of the current study center around putative effects of infant negativity on mothers, we focused on the mother-involved episode, as it most directly assesses how infant negativity manifests during interactions with mothers. That is, if there is any suspicion that infant behavior may differ when infants are with mother versus when infants are not with their mother, it stands to reason that patterns of infant behavior during infant-mother interactions would more directly influence maternal behaviors than infant behaviors in mother's absence.

Consistent with Lab-TAB instructions (Goldsmith and Rothbart, 1999), trained behavioral coders recorded the intensity of facial fear, bodily fear, facial sadness, escape behaviors, and the intensity of vocal distress across eight 5-s coding epochs (2 per mask). Facial fear and sadness were rated on a 4-point scale (0=no codable fear/sadness across three regions of the face [i.e., brows, eyes, mouth], 3=visible fear/-sadness in all three regions or an intense expression in two areas). Vocal distress was rated on a 5-point scale (0=complete absence, 5=full-intensity cry/scream). Bodily fear was rated on a 4-point scale (0=no signs of bodily fear, 3=maximum intensity). Latency to the first observation of negative affect was recorded, in seconds, from the beginning of the episode. Latencies were then reversed to reflect speed such that higher scores reflected earlier expressions. All episodes were double coded by two independent scorers who had been trained by a master coder. Each coder was required to achieve a minimum reliability (κ=0.70) with a master coder before coding independently. Ratings across coders were highly correlated (facial fear $r = 0.76$; facial sadness $r = 0.88$; vocal distress $r = 0.95$; bodily fear $r = 0.58$; latency to fear $r = 0.76$; latency to sadness $r = 0.79$) and so were averaged across coder. Fear and sadness composites were then created using regression-based factor scores from a principal components analysis (PCA) containing all coded behaviors. The PCA (orthogonal rotation) returned a two-factor solution accounting for 69.05% of the variance in the original variables (mean communality=0.69) Thus, each infant received a single score reflecting overall intensity of fear displays (facial fear, bodily fear, speed to fear) and one score that reflected overall intensity of sadness displays (facial sadness, vocal distress, speed to sadness). Higher scores reflected greater intensity of expression during the mother-involved masks episode. Correlations among variable used in the composites, which further justify the creation of two separate composites, are provided in Supplementary Table 1. All infants showed either fear or sadness during the episode.

5.2.8. Cortisol reactivity

To assess neuroendocrine reactivity to a negative emotion elicitation, we collected two saliva samples from infants. The first was collected upon arrival at the laboratory; the second was collected at the end of the visit, approximately 30 min after the completion of a series of positive and negative affect-eliciting tasks (mobile [Kagan & Snidman, 1991], arm restraint, peek-a-boo, and puppet show [Goldsmith & Rothbart, 1991]), which have previously been shown to be stressful for infants (Buss, 2011; Buss et al., 2013). Saliva samples were collected by the primary experimenter using an infant swab (Salimetrics, LLC part 5501.20). Experimenters were instructed to hold the swab under the infant's tongue for a minimum of 90 s. If it was not possible to place the swab under the tongue, experimenters were instructed to place the swab in the corner of the mouth. All samples were stored at −80 °C until they were thawed and centrifuged. Samples were assayed in duplicate using a highly sensitive enzyme immunoassay (Salimetrics, Suffolk, UK) with a detection range from 0.012μg/dL-3μg/dL. Samples were included in analyses only if the coefficient of variation was less than 20% (pre-visit $M = 7.18$, $SD = 0.15$, post-visit $M = 6.92$, $SD = 4.82$).

Raw cortisol values were log10 transformed to reduce negative skew. A reactivity score was created from the transformed values by subtracting pre-visit cortisol levels from post-visit cortisol levels; higher values reflect greater neuroendocrine reactivity to the emotion-eliciting episodes (Gunnar, 1992). Cortisol reactivity scores were normally distributed and were unrelated to sample times (mean $r = -0.13$), morning wake time ($r = 0.06$), time since waking (mean $r = -0.14$), or hours of sleep the previous night ($r = -0.13$). Cortisol reactivity was also unrelated to maternal reports of infants having napped on the day of the visit ($t(54) = 0.90$, $p = 0.37$), being on antibiotics ($t(54) = 0.26$, $p = 0.79$) or other medications ($t(51) = -0.02$, $p = 0.99$), or having had an "out of the ordinary, potentially stressful" event recently occur ($t(53) = 0.36$, $p = 0.72$). Because the inclusion of non-significant covariates introduces more noise than predictive value into the statistical model, and given noted potential problems with interpreting partialed variables (Vize et al., 2018), these nonsignificant covariates were not considered further.

5.2.9. Missing data

Symptoms of depression and anxiety were missing from 15 participants during the second trimester visit (12 not yet enrolled, 2 did not return questionnaires, 1 unknown), 12 participants during the third trimester visit (6 withdrew from study, 1 did not return questionnaires, 1 skipped this assessment, 4 unknown) and 26 participants at the postnatal visit (14 withdrew from study, 3 infants were too young for the postnatal visit when the study ended due to the PI switching institutions, 9 unknown). IBQ data were missing from 1 additional infant with incomplete questionnaires.

An analysis of patterns of missing data suggested that data were missing at random pattern (Little's MCAR $\chi 2(96) = 111.41$, $p = 0.14$). Missing data were handled using a full-information maximum likelihood (FIML) procedure that allows one to take advantage of the full sample from which at least some data are available. FIML is appropriate with MAR data (Enders, 2010). The final analytic sample comprised 92 mother-infant dyads.

5.2.10. Plan for analysis

Data and syntax supporting the results presented here are available at https://osf.io/wyjd7/?view_only=618e5037640247738f3f7297089d6bba. Analyses were conducted in Mplus 7 Version 1.4 (Muthén and Muthén, 1998) using latent growth curve models (Hox and Stoel, 2014). Structural equation models were created that identified intercepts, linear slopes, and quadratic slopes as latent variables with loadings for each of the assessments. Intercepts for the manifest variables (i.e., loadings for the manifest variables onto the latent intercept) were fixed to zero to make the latent intercept interpretable as the first assessment occasion. Unequal spacing between assessments was accounted for in the loadings of the manifest variables on the latent linear and quadratic slope factors. Errors of manifest variables were constrained to zero. In each model, latent growth curve parameters (i.e., intercept, linear slope, quadratic slope) were regressed onto manifest variables assessing infant negative reactivity.

Recognizing overlap between symptoms of anxiety and depression, we regressed concurrent symptoms of depression out of anxiety symptoms in mothers (and concurrent symptoms of anxiety out of measures of depression). This step was intended to assure that any similarities in patterns of results were not simply a product of comorbid symptoms.

We tested each indicator of infant negativity (parent-reported negativity, observed negativity, and HPA reactivity) in separate models given our interest in understanding the degree of convergence for patterns of results across multilevel indicators of temperamental reactivity. Achieved power, derived from tests based on work by Satorra and Bentler (1985), differed by measure, with statistical powers ranging from 69.1% in the model that included parent-rated negativity to 87.7% in the model that included observed infant negativity.

Based on the literature, we expected that maternal symptoms would already be elevated by the second trimester and that this effect would be visible as a significant (nonzero) intercept term. We also expected increases in symptoms of anxiety and depression during pregnancy followed by a decline, a pattern that would be visible as significant positive linear and negative quadratic effects. Significance was expected for both

linear and quadratic terms given that we did not expect symmetry in the rise and fall of symptoms over time.

We expected patterns of change in maternal symptoms to be moderated by infant negativity such that the size of the quadratic effects of maternal depression and anxiety would be reduced when infant negativity was high. This would be visible as a significant prediction of linear and quadratic effects by infant negativity and the expectation that the quadratic slope at high levels of infant negativity would be smaller than the quadratic slope at low levels of infant negativity. The quadratic effect is key in our hypothesis because it contains the postnatal changes anticipated for the sample (leveling off/slow declines).

6. Results

6.1. Preliminary analyses

Descriptive statistics (Table 1) suggested substantial individual differences in maternal symptoms of both depression and anxiety. Correlations (Table 2) suggested overlap between symptoms of anxiety and depression, with the greatest comorbidity during the postnatal period and moderate stability over time. Mother-reported and observed negativity in infants were moderately correlated, as is typical for infant temperament characteristics. Maternal ratings of infant negativity were largely unassociated with maternal symptoms and cortisol reactivity in infants. Greater cortisol reactivity across the laboratory visit was linked to less observed negativity. Thus, individual measures of infant negativity were overlapping, but nonredundant, and patterns of correlations (ranging from $r = 0.00 – 0.36$) supported the maintenance of separate measures rather than a single composite.

Sex of infant was unrelated to any of the study variables ($|t|$s $\langle 1.57$, $ps\rangle$ 0.10). Given no *a priori* hypotheses for different effects for male vs. female infants, sex was not considered further. A series of one-way ANOVAs showed that household income was unrelated to all study variables ($Fs \langle = 2.01$, $ps \rangle$ 0.05) except maternal depressive symptoms (second trimester [$F(9, 71) = 2.24$, $p = 0.03$], third trimester [$F(9, 72] = 3.16$, $p < 0.01$), postnatal [$F = 1.82$, $p = 0.09$]). Therefore, household income was added, as a covariate, for analyses examining maternal depressive symptoms.

6.2. Unconditional models

We ran an unconditional model containing only the latent growth curve parameters for each type of maternal symptoms that represented average sample-level change in symptom over time. Models including the quadratic term fit the data as well or better than intercept-only models (Table 3).

For the unconditional model of maternal trajectories of depression (Fig. 1), there was a significant intercept (β=1.58, $p<0.01$, 95%CI [1.25, 1.92]) suggesting elevated depressive symptoms by the second trimester. Linear (β=−0.17, $p = 0.14$, 95%CI [−0.40, 0.06]) and quadratic (β=0.14, $p = 0.24$, 95%CI [−0.10, 0.37]) slopes were nonsignificant.

For the unconditional model of maternal trajectories of anxiety (Fig. 1), there was a significant intercept (β=3.68, $p<0.01$, 95%CI [3.08, 4.29]) suggesting elevated anxiety symptoms by the second trimester. Linear (β=0.05, $p = 0.66$, 95%CI [−0.18, 0.28]) and quadratic (β=−0.03, $p = 0.83$, 95%CI [−0.26, 0.21]) slopes were nonsignificant.

Table 1
Descriptive statistics for variables.

	N	Min	Max	*M*	*SD*	*t*
Second trimester anxiety symptoms	78	19	68	40.63	11.01	−0.84
Third trimester anxiety symptoms	81	22	69	40.70	12.04	−0.22
Postpartum anxiety symptoms	67	22	74	40.90	12.24	−0.29
Second trimester depressive symptoms	78	1	16	6.42	4.10	−0.59
Third trimester depressive symptoms	82	0	25	5.46	4.74	0.29
Postpartum depressive symptoms	67	0	21	5.30	4.26	−0.98
Mother-rated infant negativity	66	1.89	4.31	2.94	0.60	0.49
Observed fear	62	−2.62	2.00	0.00*	1.00*	−1.56
Observed sadness	62	−1.66	2.92	0.00*	1.00*	−0.99
Cortisol reactivity	56	−1.36	0.35	−0.07	0.30	0.79

Note: Table reflects observed values prior to imputation. *t* values are from an independent samples *t*-test examining differences based on sex of infant. All *t* values were nonsignificant at $p<0.05$. *standardized values.

6.3. Maternal symptoms of depression

We then tested infant negativity as a predictor of changes in symptoms of depression over time. Model fit indices are shown in Table 3, though their interpretation is cautioned given that final models were fully saturated. For all three models, residual variances for growth curve parameters were significant, suggesting that infant negativity did not fully account for changes in maternal symptoms of depression ($|\beta|s$ = 0.31–0.99, $ps<0.01$).

6.4. Mother-rated infant negativity

Mother-reported infant negativity was not related to intercept values (β=−0.01, $p = 0.99$, 95%CI [−0.25, 0.38]), or linear (β=0.12, $p = 0.89$, 95%CI [−0.28, 0.31]) or quadratic (β=0.00, $p = 1.00$, 95%CI [−0.37, 0.26]) changes in maternal symptoms.

6.5. Observed negativity with mother

Observed sadness did not predict intercept values (β=−0.05, p = 0.68, 95%CI [−0.32, 0.21]), or linear (β=0.29, $p = 0.06$, 95%CI [−0.01, 0.59]) changes but was associated with quadratic changes in maternal depressive symptoms (β=−0.31, $p = 0.05$, 95%CI [−0.61, −0.00]). These effects demonstrate statistically significant *changes* from the original values, or that greater sadness predicted more quadratic changes in maternal symptoms over time (Δβ=−0.49–0.31 =−0.80).

Observed fear was associated with intercept values of maternal symptoms of depression (β=−0.33, $p = 0.01$, 95%CI [−0.58, −0.08]), but not linear (β=0.00, $p = 0.98$, 95%CI [−0.32, 0.33]) or quadratic (β=0.07, $p = 0.17$, 95%CI [−0.25, 0.40]) change.

6.6. Infant neuroendocrine reactivity

Neuroendocrine reactivity did not predict intercepts (β=−0.15, p = 0.33, 95%CI [−0.44, 0.15]), or linear (β=0.23, $p = 0.16$, 95%CI [−0.09, 0.55]) or quadratic (β=−0.26, $p = 0.17$, 95%CI [−0.59, 0.07]) changes in maternal symptoms of depression.

6.7. Maternal symptoms of anxiety

Residual variances for growth curve parameters predicting maternal anxiety symptoms were significant, suggesting that infant negativity did not fully account for changes in maternal symptoms ($|\beta|s = 0.90 – 0.99$, $ps<0.01$).

6.8. Mother-rated infant negativity

Mother-rated infant negativity was unrelated to intercepts (Fig. 2) but was marginally related to linear and quadratic changes in maternal anxiety. Again, these effects demonstrate statistically significant *changes* from the original values, or that greater infant negativity predicts smaller linear increases (Δβ=0.97–0.23=0.74) and less negative

Table 2
Bivariate Correlations among primary variables.

	1	2	3	4	5	6	7	8	9
1. Second trimester anxiety symptoms									
2. Third trimester anxiety symptoms	0.74**								
3. Postpartum anxiety symptoms	0.73**	0.69**							
4. Second trimester depressive symptoms	0.45**	0.45**	0.53**						
5. Third trimester depressive symptoms	0.27*	0.55**	0.39**	0.52**					
6. Postpartum depressive symptoms	0.44**	0.36**	0.72**	0.35**	0.32*				
7. Mother-rated infant negativity	0.06	−0.01	0.19	0.10	0.13	0.23+			
8. Observed fear	−0.03	−0.02	0.02	−0.18	−0.02	0.07	0.03		
9. Observed sadness	0.10	−0.04	0.16	−0.03	0.09	0.09	0.36**	0.00	
10. Cortisol reactivity	0.24+	0.22	0.05	−0.07	0.18	−0.09	−0.31*	−0.25+	−0.12

Note: $^{**}p < 0.01$, $^{*}p < 0.05$, $+p < 0.10$.

Table 3
Model Fit Indices.

	AIC	BIC	RMSEA	CFI	TLI	Chi Square
Anxiety Symptoms: Unconditional Model						
Intercept Only	1667.65	1677.78	0.13	0.92	0.95	12.84*
+Linear Term	1755.27	1770.48	0.58	0.08	0.08	96.48
+Quadratic Term	1664.82	1687.61	0.00	1.00	1.00	0.00*
Depressive Symptoms: Unconditional Model						
Intercept Only	1284.98	1302.71	0.00	1.00	1.05	1.06
+Linear Term	1287.92	1310.71	0.00	1.00	1.00	0.00*
+Quadratic Term	1287.92	1310.71	0.00	1.00	1.00	0.00*
Anxiety Symptoms: Test Model						
Mother-rated negativity	3420.43	3498.94	0.17	0.83	0.71	49.66
Observed negativity	3664.37	3765.67	0.17	0.82	0.67	52.39
Cortisol reactivity	3323.34	3401.85	0.18	0.82	0.69	52.39
Depressive Symptoms: Test Model						
Mother-rated negativity	3395.72	3491.95	0.09	0.96	0.89	10.94
Observed negativity	3638.53	3760.10	0.09	0.97	0.89	10.56
Cortisol reactivity	3295.10	3391.34	0.08	0.97	0.91	10.15

$^{*}p<0.05$.

quadratic change ($\Delta\beta = -1.18+0.24=-0.94$). This pattern suggests that heightened infant negativity is associated with stably elevated or slight increases in maternal anxiety symptoms followed by less steep declines over time.

6.9. Observed negativity with mother

Intercepts of maternal anxiety were not significantly predicted by observed infant fear or sadness (Fig. 3). Observed fear was unrelated to linear or quadratic change in maternal anxiety symptoms over time. However, observed sadness predicted linear and quadratic change in maternal anxiety symptoms. Again, these associations reflect changes from original values; they demonstrate that greater infant sadness predicts smaller linear increases ($\Delta\beta=-0.08–0.27=-0.35$) and less negative quadratic change ($\Delta\beta=-0.04+0.29 = 0.25$) over time.

6.10. Infant neuroendocrine reactivity

Infant neuroendocrine reactivity was associated with intercept values but not with linear or quadratic change in maternal symptoms (Fig. 4). Greater maternal prenatal anxiety was associated with greater neuroendocrine reactivity in infants at 4 months postpartum.

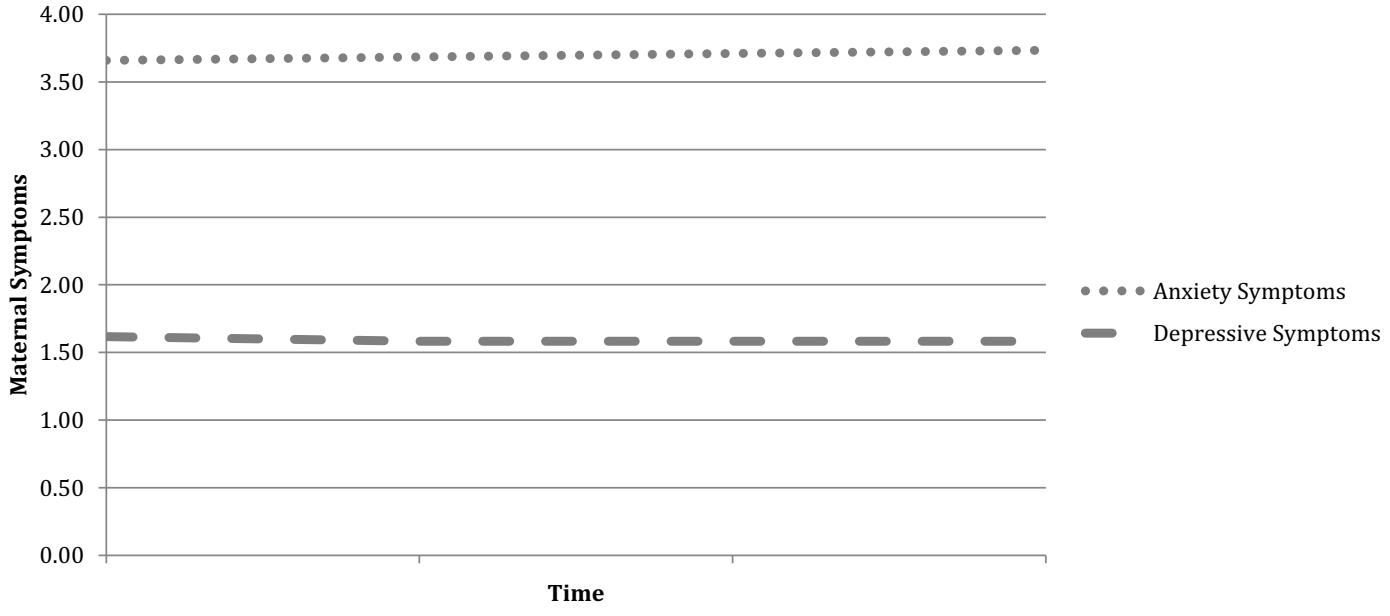

Fig. 1. Trajectories of maternal anxiety and depression from the unconditional model. Trajectories are plotted on standardized scales to allow for simultaneous plots; scores are standardized within scale.

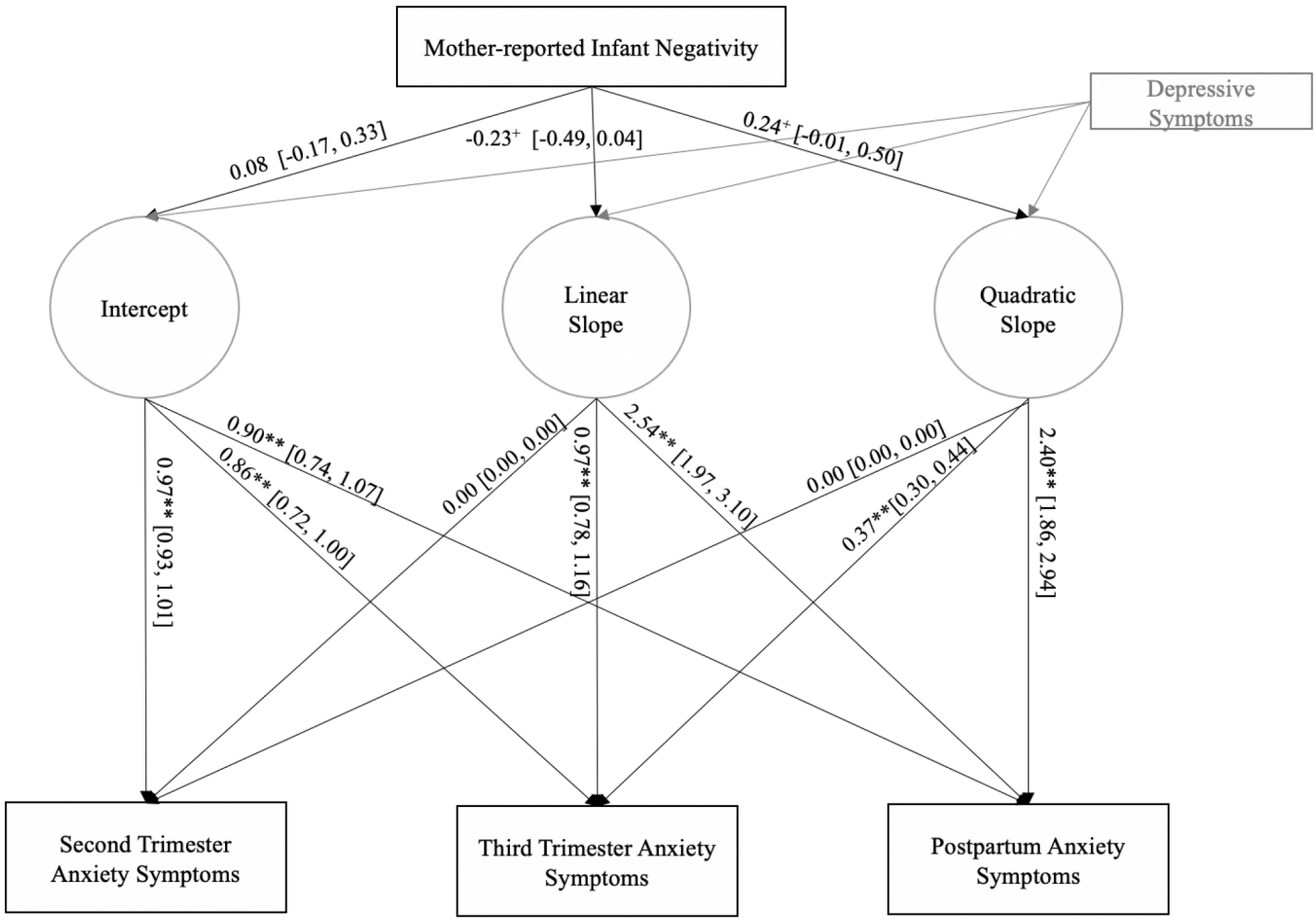

Fig. 2. Latent growth model predicting trajectories of change in maternal anxiety symptoms from mother-rated infant negativity. $^{**}p < 0.01$, $+p < 0.10$. Boxes reflect manifest variables; circles reflect latent variables. Model controlled for maternal symptoms of depression at each assessment. All loadings are shown on a standardized scale. Intercept was marginally correlated with linear ($r = -0.23$, $p = 0.06$) but not quadratic ($r = 0.08$, $p = 0.57$) slope. Linear and quadratic slopes were also correlated ($r = -0.96$, $p < 0.01$).

7. Discussion

We found small but consistent effects of infant negativity on nonlinear trajectories of maternal anxiety symptoms. The negative quadratic "shape of change" in maternal anxiety symptoms is consistent with prior work showing nonsignificant linear change (Skouteris et al., 2009) in maternal anxiety symptoms, which generally increase during the prenatal period and then decline postpartum (Grimm et al., 2011). Critically, quadratic change was dampened when observed infant negativity was high, an effect consistent with expectations that high infant negativity would impede postnatal declines in anxiety symptoms.

With one exception, associations between infant negativity and trajectories of maternal symptoms replicated across maternal-reported and observational measures. Given noted differences between maternal and self-report measures (Goldsmith et al., 2012; Zentner and Bates, 2008), this makes for a strong conceptual replication of our primary hypothesis. In contrast, although similar for maternal-reported and observed infant negativity, findings did not replicate for our psychophysiological measure. It is perhaps reasonable that effects hypothesized to be rooted in behavioral exchanges may weaken as focus shifts further from behavior to more basic physiological processes. Moreover, it is not uncommon for very young infants to show inconsistent cortisol reactivity to emotion stressors (Gunnar et al., 2009), suggesting that this effect may be more likely in slightly older children. Our pattern of results may also highlight a need for infant behavior to manifest at a level perceptible for mothers (i.e., reflected in maternal report) or observers (i.e., reflected in observational coding). That is, our results suggest that mothers need to encounter infant behaviors to such an extent that they are observable or reportable, before they are related to trajectories of maternal symptoms.

Unexpectedly, effects of observed negativity were limited to infant sadness. It is possible that this pattern reflects a critical role for infant sadness in mother-child dynamics. By toddlerhood, children express sadness more frequently and at greater levels of intensity than other emotions when looking to their mothers during emotional challenge (Buss and Kiel, 2004), perhaps because sadness expressions are most likely to elicit maternal intervention. Although previous work has largely investigated mother-directed expressions of sadness relative to expressions of anger, there is some suggestion that children generalize the contingent relation between expressions of sadness and maternal responding to other affective domains (Malatesta and Haviland, 1982), like our fear context.

Maternal depressive symptoms were also slightly elevated but stable over time. Infant negativity was associated with maternal symptoms in the second trimester, an effect that may reflect shared propensities for emotional blunting, but was not associated with maternal symptoms over time. Findings are perhaps most consistent with the notion that increased maternal depression during pregnancy influences "set points" for reactivity at the biological level that predispose infants to be more emotionally reactive (Goldsmith et al., 1997; Shirtcliff and Ruttle, 2010). As depressed mothers are less responsive to infant emotion

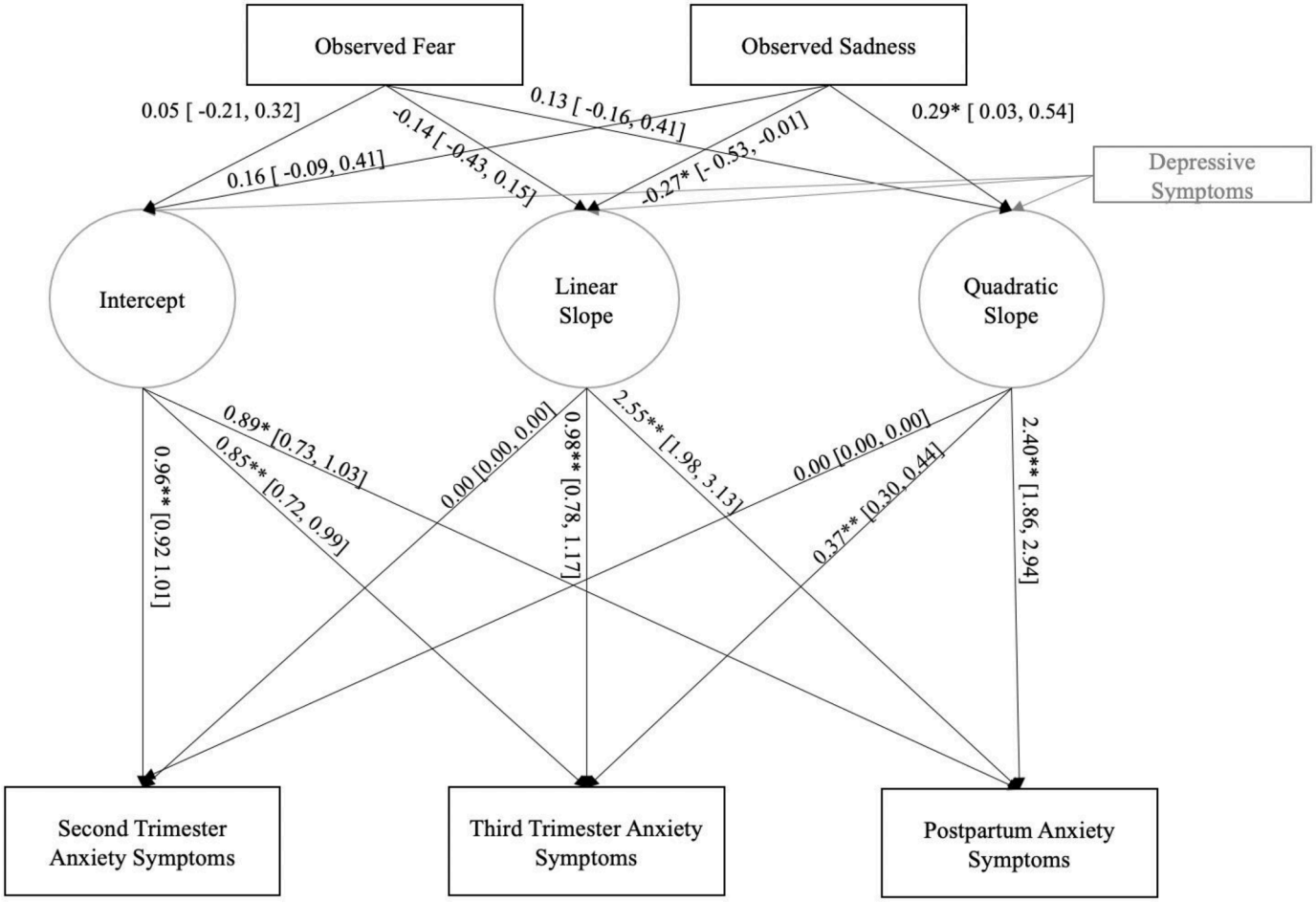

Fig. 3. Latent growth model predicting trajectories of change in maternal anxiety symptoms from observed infant negative affect. $^{**}p < 0.01$, $+p < 0.10$. Boxes reflect manifest variables; circles reflect latent variables. Model controlled for maternal symptoms of depression at each assessment. All loadings are shown on a standardized scale. Intercept was marginally correlated with linear ($r = -0.21$, $p = 0.08$) but not quadratic ($r = 0.06$, $p = 0.67$) slope. Linear and quadratic slopes were also correlated ($r = -0.96$, $p < 0.01$).

behaviors (Campbell et al., 2004), the effects of infant behaviors could be less robust for depressed than for anxious mothers.

Although we did not have *a priori* hypotheses regarding the intercept terms, which are necessary components of the growth model, we recognized that they may be significantly associated with infant negativity. Such a possibility may seem counterintuitive given that a mother in her second trimester of pregnancy has not yet interacted with her infant. However, there exists a theoretical framework for interpreting effects of infant negativity on *prenatal* measures of maternal symptoms. Behavioral genetics work has shown predictive effects of birth mother characteristics on adoptive parent behaviors *despite the two never interacting* (Klahr et al., 2017) and recognizes that the association represents a shared variance that is common to the two variables even if it is unmeasured directly. In adoption samples, this is interpreted as connections mediated through the adopted child, namely parent characteristics inherited from the birth parent but experienced by the adoptive parent that are not directly measured by another variable in the study (Hajal et al., 2015). In non-adoptive samples such as ours, this likely reflects other unmeasured sources of shared risk for whatever morbidity is under study and, in our biologically related parents and children, almost certainly includes shared genetic variation.

This work is nonexperimental and precludes causal inferences. In addition, replication will be needed in larger samples that differ in genetic-relatedness and level of risk for psychological syndromes. Also, given the design to test a broad effect of infant negativity on maternal symptoms, we cannot comment on specific mechanisms by which infant negativity may influence maternal outcomes. This will be an important avenue for future work. Finally, regressing concurrent symptoms from our anxiety and depression variables may increase the difficulty of interpreting effects relative to the general population, for whom comorbidity is typical.

Despite limitations, this work responds to repeated calls for empirical tests of child-to-parent effects in early development. This work extends previous findings linking child emotion characteristics to maternal behaviors (Buss et al., 2021; Kiel et al., 2021) to demonstrate associations between infant negative emotion and maternal mental health. In addition, our findings expand on previous work linking infant negative emotion to trait level parent emotion characteristics (Cioffi et al., 2021) and is, to our knowledge, the first study to prospectively demonstrate child-based effects on the trajectories of maternal symptoms. Critically, this pattern is demonstrated during the perinatal period, when earlier levels of symptoms are measured and controlled. Although replication is needed, these findings are an important step in moving away from theoretical representations of parents and children as active and passive agents, respectively, in developmental change. Rather, these results offer an important foundation for extending particularly models of co-occurring emotional development in both mothers and young children.

Contributors

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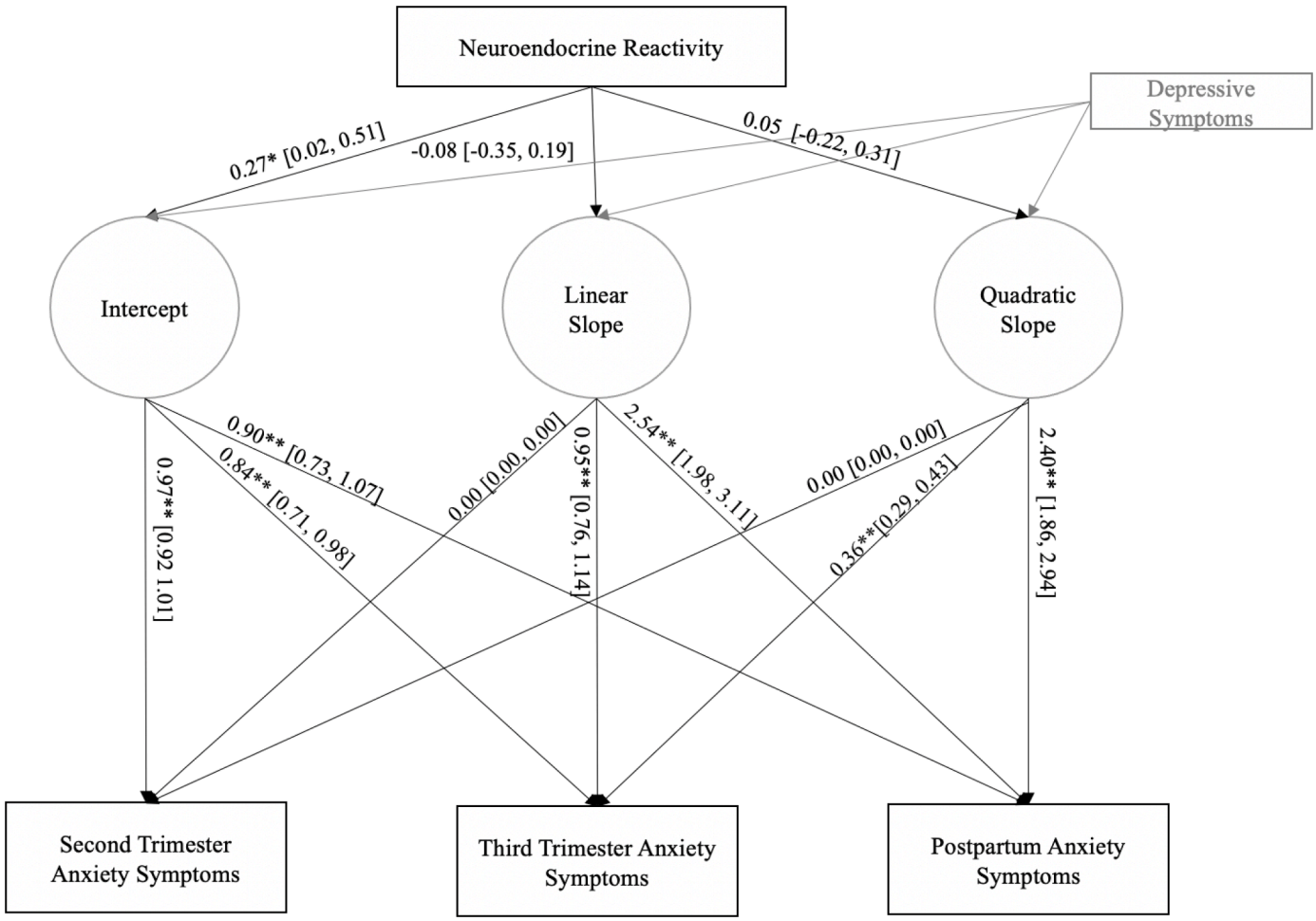

Fig. 4. Latent growth model predicting trajectories of change in maternal anxiety symptoms from infant neuroendocrine reactivity. $^{**}p < 0.01$, $^{*}p < 0.05$. Boxes reflect manifest variables; circles reflect latent variables. Loadings for second trimester anxiety symptoms (shown in gray) were fixed. Intercept was correlated with linear ($r = -0.24$, $p = 0.05$) but not quadratic ($r = 0.10$, $p = 0.47$) slope. Linear and quadratic slopes were correlated ($r = -0.96$, $p < 0.01$).

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jadr.2023.100481.

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