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Maternal Contingent Responses to Distress Facilitate Infant Soothing but Not in Mothers With Depression or Infants High in Negative Affect

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Depression in mothers is consistently associated with reduced caregiving sensitivity and greater infant negative affect expression. The current article examined the real-time behavioral mechanisms underlying these associations using Granger causality time series analyses in a sample of mothers ($N = 194$; 86.60% White) at elevated risk for depression and their 3-month-old infants (46.40% female) living in a major metropolitan area in the United States. Overall, mothers contingently responded to infant distress, and mothers' responses to infant distress increased the likelihood of infant soothing in real time. However, there was no evidence for maternal contingent responding or facilitation of infant soothing in subsamples of mothers who were currently experiencing elevated depression symptoms or in mothers of highly negative infants. These findings suggest real-time behavioral mechanisms by which risks for maladaptive self-regulation may develop.

Public Significance Statement
Overall, mothers contingently respond to infant distress, and their responses to distress facilitate infant soothing. However, in groups of mothers with depression symptoms and groups of infants who show high levels of distress, we do not find evidence for such regulation behaviors. Our results suggest that both mothers and infants play key roles in infants' developing self-regulation.

Keywords: mother–infant interaction dynamics, infant distress, negative emotionality, maternal depression, Granger causality

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A fundamental tenet of attachment theory is that caregivers' responses to infant distress are the foundation of infant's developing social–emotional functioning (Bowlby, 1988). Broadly, a history of caregivers' sensitive, that is, consistent, contingent, and appropriate responses to infant distress are thought to facilitate infants' soothing following distress. Over repeated instances of distress and soothing, infants are thought to develop the expectation that caregivers will provide relief, as well as strategies for modulating their own distress, that is, self-regulation (Kopp, 1989). These theoretical tenets receive strong empirical support; maternal sensitive responding to infants' distress is prospectively linked to children's more adaptive emotion regulation, positive social–emotional functioning, and fewer maladaptive outcomes (Leerkes, 2011; Leerkes et al., 2009; McElwain & Booth-LaForce, 2006).

Both maternal depression and infant negative affect (NA) expression have been associated with maternal insensitivity to infant distress (Field, 2010; Leerkes, 2010; Putnam et al., 2002). Maternal depression symptoms can interfere with their ability to provide appropriate support for the regulation of their infants' distress (Field, 1994, 2010). Among a host of genetic, neurobiological, and environmental factors, such caregiving differences are considered a key mechanism by which risks for which depression are

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transmitted intergenerationally from mothers to children (Goodman, 2020; Goodman & Gotlib, 1999). Yet effect sizes for associations between depression symptom severity and sensitive parenting are small (Crockenberg & Smith, 1982; Perry et al., 2018; van den Bloom & Hoeksma, 1994), suggesting a potential role of moderators, such as infant negativity.

Mothers of infants who express relatively high amounts of NA have lower caregiving self-efficacy (Cutrona & Troutman, 1986) and higher frustration, distress, and concurrent depression symptoms (Fujiwara et al., 2011; Miller et al., 1993; Petzoldt, 2018). This has led some authors to propose a "vicious circle" where the psychological burdens of caring for a highly negative infant may lead these mothers to become less sensitive to their infants over time, ultimately leading to further increases in both infant and maternal distress (Kurth et al., 2011; Papoušek & von Hofacker, 1998). Such dynamics may be exacerbated in the context of mothers predisposed to depression. For example, some work in this area shows that depression in mothers predicted their later *lower* sensitivity only in the context of infant negativity being high (Newland et al., 2016).

Multiple real-time behavioral pathways could account for established associations between maternal sensitivity and infant NA expression in women who are and are not depressed. First, mothers who are depressed or who have infants high in NA expression may be less likely to respond contingently to infant distress relative to control mothers, which may extend the duration of infant distress before soothing. Alternatively, these mothers may contingently respond to infant distress, but their responses to infant distress may not facilitate infant soothing. We know of no studies that have examined these real-time processes directly. However, knowledge about these behavioral pathways has distinct theoretical implications. Consistent with attachment theory, higher rates of mothers' responding to infant distress may lead to more occasions on which infants experience being regulated by their mothers, implying more opportunities for the infant to gain regulatory skills (Perry et al., 2018). However, if maternal responses to infant distress do not facilitate soothing, these interactions may not contribute to infants' developing self-regulation.

In the current study, we examine mothers' real-time responses to individual instances of infant NA to gain insight into the mechanisms by which such regulatory behaviors are associated with maternal depression and infant NA expression. To examine how such distress and regulation dynamics may contribute to the intergenerational transmission of psychopathology, we examined these questions in a sample of women at elevated risk for postpartum depression and their 3-month-old infants, an age at which infants can sustain face-to-face engagement with their caregivers (Beebe & Steele, 2013) but infant regulation strategies are relatively immature (Calkins & Leerkes, 2004) meaning that caregiver regulation efforts are an important contributor to infant soothing. Despite the importance of these unanswered questions and the potential of this approach, we found only two published studies that reported having examined the real-time dynamics of mother–infant distress and regulation behaviors (Crockenberg & Leerkes, 2004; Jahromi et al., 2004). These studies provide evidence that maternal behaviors contingent on infant distress are associated with subsequent decreases in the duration or intensity of infant crying and fussing. Both studies examined mother–infant interactions in "stressor tasks," or situations of heightened infant distress initiated by researchers. In the current study, we build on these articles by studying mother–infant distress and regulation dynamics in response to unprompted instances of infant distress occurring during a free play task. Unprompted infant distress may differ from distress expressed in researcher-designed stressor tasks—for example, briefer or less intense, or unfolding over longer timescales—potentially prompting different responses from mothers. Additionally, given that maternal sensitivity to distress during free play tasks is predictive of infants' later outcomes (Leerkes et al., 2009), free play is a relevant task in which to examine mother–infant distress and regulation dynamics.

In another related study, Beebe et al. (2008) used time series analyses to examine the real-time mother and infant affect dynamics in a sample of women suffering from depression. Relative to controls, mothers with high depressive symptoms displayed more contingent affect coordination during naturalistic free play with their 4-month-old infants, meaning that they more frequently shifted their affect to match their infants' positive and NA states (Beebe et al., 2008). Depressed mothers and their infants also showed lower self- and partner-contingency for both gaze and touch behaviors (Beebe et al., 2008). Relative to mothers with no depression, depressed mothers appeared to be "over vigilant" to their infants' expressions, matching these states to such an extent that they sacrifice the structure and predictability of their own actions; see also Jaffe et al. (2001). However, Beebe et al.'s (2008) analyses do not distinguish the type of infant affect shift that elicited maternal responses (e.g., negative, neutral, positive). Thus, the extent to which mothers contingently shifted their affect in response to the onset of infant *distress*, relative to the onset of other states of infant affect (e.g., shifts to neutral or positive affect) is unknown. This is critical, given that maternal responses to infant distress, specifically, are thought to form a foundation for developing infant self-regulation (Leerkes et al., 2009).

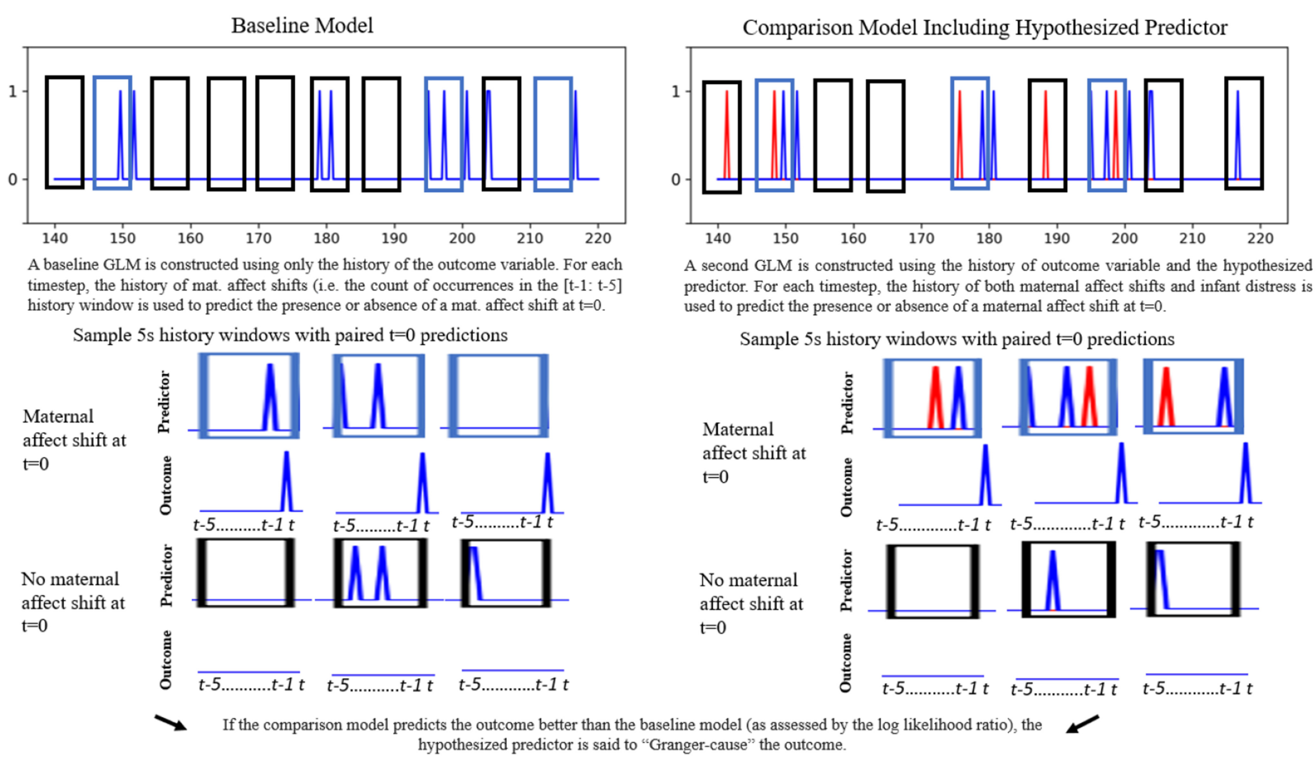
Thus, a key innovation of our analytic approach is that we disaggregate our infant affect data such that we can directly examine mothers' responses to infant distress in particular and the effects of mothers' responses on infant soothing in particular. Specifically, to examine the extent to which mothers' contingently respond to infant distress, we transform continuous infant affect annotations into a time series datastream that represents the timing of only the onset of infant distress. To examine the extent to which maternal contingent responses predict infant soothing, that is, a return to neutral or positive affect from NA, we transform infant affect annotations into a time series datastream that indicates only the timing of the onset of infant soothing following distress. Disaggregating infant affect data in this way allows us to use time series analyses to query the predictors and consequences of infant distress and infant soothing specifically, as per our hypotheses detailed below.

We used two strategies to examine real-time contingencies between mother and infant affect shifts following individual instances of infant distress. First, we used *event-related* analyses to determine the rate at which mothers contingently shifted their affect following the onset of infant distress. Drawing on past literature, we defined a contingent response as occurring within 3 s of infant distress onset (Beebe et al., 2010; Millar, 1972; Ramey & Ourth, 1971; Van Egeren et al., 2001). Additionally, we used *Granger causality* time series analyses to examine predictive sequences of mother and infant affect shifts following infant distress onset and preceding infant soothing. Like other time series analyses (Beebe et al., 2008, 2020; Cohn & Tronick, 1987; Kaye & Fogel, 1980), Granger causality is used to examine temporal relationships between theoretically linked time series. However, the Granger causality framework is used to explicitly test the real-time "causal" influence of hypothesized predictor variables on outcome variables (see Figure 1 for a conceptual model). In particular, a hypothesized predictor variable *Y* is said to Granger-cause the outcome variable *X* if the past values of *Y* improve the prediction of the future values of *X* relative to the past values of *X* alone. For example, to establish whether shifts in mothers' affect "Granger-causes" changes in infant affect, Granger causality examines whether the past values of the mothers' affect improve the prediction of future values of infant affect over and above the extent to which infants' history of affect predicts their own future affect. Granger causality is typically modeled using multivariate autoregressive (MVAR) models when dealing with time series composed of continuous data (Granger, 1969). However, Kim et al. (2011) proposed a framework that enables Granger causality to be applied to binary "spike" data, such as a time series indicating the onset of infant distress. In the case of binary data, likelihood ratio tests are used to compare the goodness-of-fit of the two competing generalized linear models (GLMs): one which includes only the history of the outcome variable *X* as a linear predictor of *X* at the current timepoint, and one which includes both the history of *X* and the history of the hypothesized predictor variable *Y* as linear predictors of the current value of the outcome variable *X* (Kim et al., 2011). The strength of the Granger causal relationship can be determined by the Granger-cause value, which is the averaged influence of *Y* on *X*. The sign of the Granger-cause value indicates whether predictor *Y* increases or decreases the likelihood of outcome *X* occurring (Kim et al., 2011).

In effect, Granger causality takes as its premise that an individual's past behavior is often a very good predictor of their future behavior. To establish "Granger causality," any additional factor must provide some added benefit to predicting future behavior of a variable, relative to the individual's own history. Thus, importantly, where event-related contingency analyses can identify the raw rate of maternal affect shifts contingent on infant distress onset, only analyses that control for the history of past mother and infant behaviors, including Granger causality, can specify whether such affect shifts are occurring more often than would be expected by chance alone. We note that other time series analyses can control for history or autoregressive influence of one time series on another (e.g., Beebe et al., 2008), but have not been used to evaluate the extent of influence of one time series on another in mother–infant interactions as we propose to use Granger causality to do. Finally, our approach is technically similar to other techniques used for modeling dynamic changes between high-density repeated variables over time such as dynamic structural equation models or multilevel models with lagged variables (Asparouhov et al., 2018; Beebe et al., 2016; Lowe et al., 2016; Somers et al., 2022). We selected to use Granger causality because it has recently been extended for use with binary time series (Kim et al., 2011) and additionally for its

Figure 1

Conceptual Overview of Granger Causality Analysis to Determine Whether Infant Distress Onset "Granger-Causes" Maternal Affect Shifts



Note. In our example, the outcome variable is maternal affect shifts (blue spikes), and the hypothesized predictor is the onset of infant distress (red spikes). History of a variable here refers to the count of occurrences of the variable in the 5 s history window $[t-1 : t-5]$ before current timestep $t = 0$. Note that history windows are shown only for selected sample timesteps for visual clarity; in the formal analysis, each timestep in the time series is considered with its paired history window. Additionally, we show only 5 s history windows here; however, in formal analysis, multiple pairs of models with varying sizes of history windows are typically compared. GLM = generalized linear model. See the online article for the color version of this figure.

explicit focus on testing the influence of one time series on another. For additional details on Granger causality and its existing and potential applications within the developmental science community, we point interested readers to several recent publications, including a recently published "code paper" including a general tutorial and code for implementing Granger causality analyses (Hoch et al., 2021; Xu et al., 2017, 2020).

Our specific hypotheses are as follows:

Hypothesis 1 (H1): Mothers contingently respond to infant distress. Building on the reviewed studies, we hypothesized that, for the sample as a whole, mothers will contingently respond to their infants' distress by shifting their affect within 3–5 s of infant distress onset. Specifically, within the Granger causality framework, we examined the extent to which mothers' affect shifts are predicted by the onset of infant distress over and above (i.e., controlling for) mother's tendency to change affect, that is, the history of mothers' affect shifts.

Hypothesis 2 (H2): Mothers' responses to infant distress, that is, contingent affect shifts, will increase likelihood of infant soothing. Next, we hypothesized that overall, at the sample level, mothers' affect shifts following the onset of infant distress would increase the likelihood of infant soothing, that is, a shift in infants' affect from negative to either positive or neutral affect. Within the Granger causality framework, we examined the extent to which mothers' affect changes during infant distress events increase the likelihood of infant soothing over and above the infant's own tendency to cycle between distress and soothing, that is, the history of both infant distress and infant soothing.

Next, we also considered how these two mother–infant distress-regulation dynamics may be moderated by individual differences in infant NA expression (H3) and by mothers' current depression symptom severity status (H4). Namely, we hypothesized the following:

Hypothesis 3 (H3): Mothers of infants high in NA expression will be less likely to contingently respond to infant distress (H3.1), and their responses will be less likely to increase the likelihood of infant soothing (H3.2), relative to mothers of infants low in NA expression. To examine the influence of infant NA expression during the interaction on mothers' responses and their impacts on infant soothing, we split our infants into high and low NA expression groups, as detailed in the methods below, and compared group performance on the Granger causality analyses described for H1 and H2.

Hypothesis 4 (H4): Mothers' currently experiencing clinically significant levels of depression will be less likely to contingently respond to infant distress (H4.1), and their responses will be less likely to increase the rate of infant soothing (H4.2), relative to mothers who were not experiencing clinically significant levels of depression at the time of the study. As in H3, we compared Granger causality results from the analyses described in H1 and H2 two between mothers who were above clinical threshold on our depression measure to those who were not.

Method

Participants

Participants were 202 mothers and their 3-month-old infants drawn from a larger longitudinal study, The Impact of Maternal Depression, Anxiety, and Stress on Infant Vulnerabilities to the Development of Psychopathology (Goodman, primary investigator [PI]), which was one of three projects within National Institute of Mental Health, 1 P50 MH077928-01A1, Perinatal Stress and Gene Influences: Pathways to Infant Vulnerability, a Translational Research Center in Behavioral Science (TRCBS) at Emory University School of Medicine, Zachary Stowe, PI of the Center. We recruited pregnant women through referrals from local obstetrical and mental health practitioners. Referred participants came from their doctors, other clinics, the community, and other research studies. Women were enrolled if they were <16 weeks pregnant and between 18 and 45 years of age. The recruitment strategy was to enroll women with a history of depressive episodes to increase the likelihood of elevated postpartum depression relative to the general population. Thus, the primary entry criterion was all women meeting DSM-IV (i.e., Diagnostic and Statistical Manual of Mental Disorders, 4th Edition) criteria for a previous major depressive episode. Women were excluded for meeting DSM-IV criteria for bipolar disorder, schizophrenia or an active eating disorder, having psychotic symptoms, active suicidality, active homicidality, an active substance use disorder within 6 months before the last menstrual period or positive urine drug screen, illness related to infant outcomes (autoimmune disorders, asthma, and epilepsy), anemia, or abnormal thyroid stimulating hormone. Of the 202 families who participated in the session with their 3-month-old infants, $N = 194$ (96%; 46.40% female) had all of the required data to be included in these analyses, including a minimum of 150 s of affect data coded during free play to ensure adequate data for analyses. Mothers were, on average, 33.81 years old ($SD = 4.50$) at the time of delivery, 86.60% identified as White, 10.30% as Black, and 3.00% reported other racial backgrounds, 46.40% were primiparous, 86.60% were married, they were primarily of middle to upper-middle socioeconomic status ($M = 49.40$), as measured by the Hollingshead scale, and mean educational attainment was 16.52 years ($SD = 2.07$). Mothers who were not primiparous (53.61% of the sample) had an average of 2.48 ($SD = .89$) total children (including the target child), ranging from 2 to 8 children.

Procedure

To confirm that women met diagnostic criteria for at least one major depressive episode, participants completed the Structured Clinical Interview for DSM-IV at their first visit during pregnancy. Mothers and infants visited the lab when infants were 3 months of age. The current study focuses on data collected during the free play task during this visit. Mothers and infants were video-recorded during a 5-min face-to-face play session (Calkins & Leerkes, 2004). Mothers sat in a chair facing their infants and were within an arm's reach from the infant, while the infant sat in an infant seat on a table. Mothers were provided with age-appropriate toys for their infants and were asked to play with their infants as they typically do. All women participated in an informed consent procedure, and all procedures were approved by the Emory University Institutional Review Board (protocol number: IRB00004249). Data were collected between 2007 and 2012 approximately.

Mothers' Depression Status

To assess depression status, mothers completed the Beck Depression Inventory II (BDI-II) at their 3-month study visit. The BDI-II is a reliable and valid 21-item self-report measure of depressive symptoms (possible range 0–63), including during the postpartum time period (Lovejoy et al., 2000), with an internal reliability of approximately .9 (Wang & Gorenstein, 2013). Higher scores indicate more severe depression symptoms, with scores of 0–13 considered minimal depression symptoms, 14–19 considered mild, 20–28 considered moderate, and 29–63 considered severe. Eleven of 194 participants did not complete the depression assessment and were thus not included in the analyses involving maternal depression status. We used a cutoff score of 14 or higher to identify mothers experiencing mild, moderate, or severe depression symptoms (Beck et al., 1996).

Affect Coding

Mothers' and infants' observed moment-to-moment affect was coded from video recordings of the 5-min play session using the software Mangold Interact, coding all changes visible at 24 frames per second (FPS; Mangold, 2010). If participants went out of the camera frame and the affect could not be identified using sound cues, the frames were coded as *uncodeable*.

Maternal Affect

Maternal affect was coded using a 7-point scale modified from Dawson and colleagues (Lovejoy et al., 2000) with three levels of NA (−3: *marked distress, including sadness, disgust, fear, or anger*; −2: *moderate distress*; −1: *tension or worry*), one level of neutral or flat affect, and three levels of positive affect (+1: *positive interest*; +2: *smile or excitement*; and +3: *laughter or giggling*). Interrater reliability was assessed for a randomly selected 20% of videos. Time-unit Kappa reflecting the extent of agreement for positive, neutral, or flat, and negative mothers' affect within a 2-s tolerance was $K = .76$.

Infant Affect

Infants' affect was coded on a 3-point scale (+1: *positive*; 0: *neutral*; and −1: *negative*). Positive affect or "approach" included happy or joyful behaviors such as smiling, giggling, laughing, and cooing. NA or "withdrawal" included sadness and distress behaviors, such as grimacing, furrowed brow, frown, fuss, whimper, protest, precry, cry, back arching, and gaze aversion. Neutral or "flat" affect was coded for instances where infant affect was not clearly positive or negative. Interrater reliability was assessed for a randomly selected subset of 24% videos; time-unit Kappa reflecting the extent of agreement for positive, neutral, and negative infant affect within a 2-s tolerance was $K = .76$.

High Versus Low Infant NA Expression. To examine dynamics between high and low NA expression infants (H3), we compared interactions between infants who expressed the highest and lowest amounts of NA in our sample using an adjusted three-way split. The remaining infants with mid-levels of NA are not included in the main analyses. However, in supplemental analyses, we conducted all analyses for infants with mid-levels of NA expression, finding that their results were equivalent to those of our low NA expression groups (Result S2 in the online supplemental materials).

Thresholds were determined using an even three-way split (33.33%), which we then adjusted using state space grid visualizations (see Hollenstein, 2007) to identify similarities between clusters of infants who were highly and minimally negative. This resulted in three clusters as follows: infants who spent at least 46% time in NA ($n = 42$), infants with <9% time in NA ($n = 87$), and infants with between 9% and 46% time in NA ($n = 65$). State space grid visualizations are available in Result S1 in the online supplemental materials.

Event-Based Contingency Analyses

To contextualize our Granger causality analyses, we calculated several event-based measures of mother and infant affect shifts, including measures of maternal contingent responses to infant distress. Drawing from past literature, we defined a maternal contingent response to infant distress as occurring within 3 s of infant distress onset (Beebe et al., 2010; Millar, 1972; Ramey & Ourth, 1971; Van Egeren et al., 2001). We calculated the rate of contingent responding for each mother by dividing the number of contingent responses by the number of observed instances of infant distress, treating all mothers' affect changes equally, regardless of the directionality of maternal affect change (e.g., from +2 to +3 or +1 to −2). For additional context, we calculated rates of maternal affect shifting overall as well as during periods of infant distress and nondistress.

Granger Causality Contingency Analyses

We conducted all Granger causality time series analyses using publicly available code available from a recent publication by Xu et al. (2020). Using the specified history window size, this script creates two GLM models, a baseline model including only the history of the outcome variable as a predictor and a second GLM which includes the two predictors, the hypothesized predictor along with the history of the outcome variable (see also Figure 1). Inputs for both models include a set of windows signifying time periods of history indicating the presence and absence of predictors paired with their corresponding outcome variable prediction at the current time step. These history windows are constructed to predict the outcome variable at every smallest possible timestep, that is, in our case, every .33 s as our data are sampled at 3 Hz. Next, the model without the hypothesized predictor is compared to including it using the log-likelihood ratio to determine which model is a better fit to predict the outcome variable. If the log-likelihood ratio is <1, then the hypothesized predictor is said to "Granger-cause" the outcome and a causal influence from the hypothesized predictor to the outcome is established. All available code is written in the progamming language MATLAB and is available on GitHub (see link in Xu et al., 2020). The only parameter to set is the history window size, that is, the duration of time before the outcome of interest that is hypothesized to potentially influence the outcome. For example, for a history window of 0–5 s, 0 denotes the time step $t = 0$ at which we predict the outcome variable, and the history window 0–5 s denotes the duration 5 s before timestep $t = 0$. We conducted the Granger causality analyses for five different history window sizes of 1–5 s, with each successive window including all past values, that is, windows of 0–1, 0–2, 0–3, 0–4, and 0–5 s. Rather than examining the significance of responses at individual lags, this allowed us to compare and identify the windows of contingency at which responses were most predictive of behavioral shifts, paralleling the existing literature on contingency.

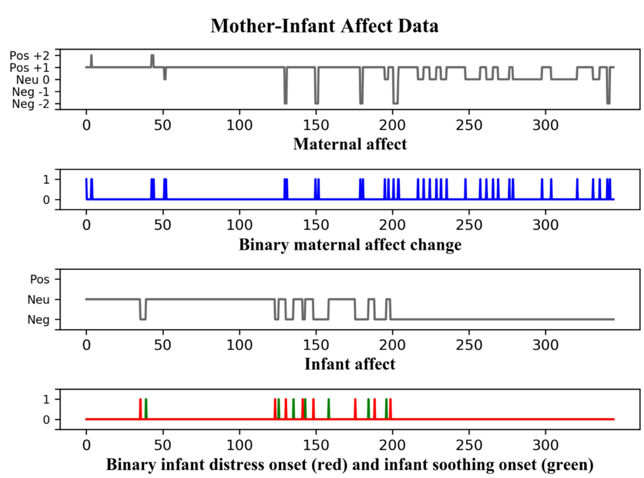
Data Preparation for Granger Causality Analyses

Before running Granger causality analyses, mother and infant affect annotations were transformed into time series format. Whenever an observer coded the infant's or the mother's affect as *uncodeable* (but not both *uncodeable* at the same time), we removed the matching mother and infant frames from the time series, stitching together the adjoining sections. Next, mother and infant affect time series were converted into three binary time series datastreams, as detailed below. Figure 2 illustrates both the raw time series and the derived binary time series for a single dyad. Finally, both the maternal and infant affect time series were downsampled from 24 FPS to three FPS (i.e., one sample taken at every .33 s from the original time series) to reduce the time required to run Granger causality while retaining high-resolution affect change data. This is a common strategy when working with high-density data (Xu et al., 2017).

Onset of Infant Distress and Soothing Time Series. We transformed infant affect data into two complementary binary time series, one corresponding to the onset of infant distress, and one corresponding to the offset of infant distress, which we refer to as the onset of infant soothing. Specifically, the infant distress onset time series is a binary time series that has a value of 1 at those moments in the session where infants shift from positive or neutral affect to NA and zeros otherwise. The infant soothing onset time series is a binary time series with a value of 1 in all instances where infants shift from NA into a state of positive or neutral affect and zeros otherwise. Note that this transformation removes all shifts in infant affect between neutral and positive states (0 to +1, +1–0), as our hypotheses are focused on the processes that follow distress and precede soothing, rather than on distinctions between infant positive and neutral affect.

Figure 2

Sample Visualization of One Dyad's Maternal Affect and Infant Affect Time Series and Their Disaggregation Into Three Binary Time Series Created for Granger Causality Analyses



Note. Maternal affect change is shown in blue; infant distress onset is shown in Red; and infant soothing onset (i.e., offset of infant distress) is shown in green. The *x*-axis indicates time in seconds. Note that the final instance of infant distress beginning approximately just before 200 s continues until the end of the interaction, as indicated by the continuation of the negative affect event in the infant affect datastream and the corresponding data in the binary time series, where a distress onset event (last red spike) is not followed by a soothing event (i.e., no green spike). Pos = positive; Neu = neutral; Neg = negative. See the online article for the color version of this figure.

Maternal Affect Change Time Series. Next, we transformed our mother affect data into a binary time series indicating the timing of *changes* in maternal affect. Specifically, mother affect change time series has a value of 1 at those moments in the session where mothers shift their affect and zeros otherwise. Note that all changes in affect are indicated with the same positive (+1) value, that is, frames in which a mother changes from +2 to a +3, and frames in which mothers shift from +1 to 0 or +1 to −1 all receive the same positive (+1) value in the maternal affect change time series. This removes information about the valence of mother's affect, allowing us to examine whether mothers' contingently respond to infant distress by shifting their affect (whether positively or negatively). It also allows us to examine the extent to which shifts in mothers' affect (whether positive or negative) facilitate infant soothing following infant distress, as we did not aim to test hypotheses about whether mothers' positive versus negative reactions (including, e.g., "woe") to infant distress would affect the likelihood of soothing.

Granger Causality Analysis Plan

Following the affect data transformations, we concatenated together each dyad's datastreams for each category of time series (infant distress onset, infant distress offset, and maternal affect change) as input data for our main hypotheses that overall, mothers would contingently respond to infant distress (H1) and that mothers' responses to infant distress would facilitate infant soothing (H2). To test these hypotheses in subsamples of mothers of infants high in NA (H3) and mothers with current depression symptoms (H4), we created analogous datastreams for the following pairs of dyads: dyads with infants identified as being high versus low in NA expression, and dyads with mothers who were above versus below the depression cutoff at their visit. Concatenation of participant data for Granger causality analyses is a common strategy for obtaining group- and subgroup-level results (Xu et al., 2017).

To test for the presence of contingent maternal responses following infant distress, we used these concatenated time series directly. Specifically, we used Granger causality to test whether infant distress onset (our hypothesized predictor) predicted a change in maternal affect shifts (our outcome variable) controlling for the history of maternal affect shifts (our baseline predictor). Testing whether maternal responses to infant distress facilitated infant soothing involved three predictor variables. Specifically, we tested whether maternal affect shifts during infant distress (our hypothesized predictor) predicted a change in infant soothing onset (our outcome variable) while controlling for the history of infant distress and the history of infant soothing (our two baseline predictors). This study was not preregistered. Data and study materials from this study are not publicly available.

Results

Descriptive Statistics

Table 1 details descriptive statistics of our interactions for the sample as a whole and for infants high versus low in NA

Table 1
Descriptive Summaries of Infant Distress and Maternal Affect Shifts

Sample	Dyad count	Total count of distress episodes	Distress episode duration (s)	Rate of distress episodes	Rate of maternal contingent response	Rate of maternal affect shifts	Rate of maternal affect shifts during distress	Rate of maternal affect shifts during nondistress
Whole sample	194 (100%)	932	5.02 (30.9)	0.92 (0.91)	.33 (.30)	6.23 (3.89)	7.64 (7.84)	5.94 (4.01)
High NA infants	42 (21.65%)	273	11.7 (50.8)†,***	1.20 (0.93)†,***	.3 (.27)	6.32 (5.08)	6.67 (5.43)	5.74 (5.43)
Low NA infants	87 (44.85%)	193	3.08 (3.04)†,***	0.51 (0.38)†,***	.33 (.35)	5.91 (3.35)	9.30 (10.9)	5.72 (3.31)
Dep mothers	38 (19.59%)	219	5.50 (32.3)	1.00 (1.04)	.27 (.29)	6.01 (4.50)	6.16 (5.83)	5.12 (5.15)
Non-dep mothers	145 (74.74%)	681	4.91 (30.4)	0.94 (0.89)	.33 (.30)	6.25 (3.82)	8.12 (7.96)	5.99 (3.76)

Note. Given skewed distributions, duration and rate values provided indicate median (standard deviation). All simple rate variables are calculated to reflect summary measures per minute. Rate of maternal contingent response is calculated as a proportion of the infant distress episodes per dyad. Pairs or groups within a column indicated by matching dagger symbols differ significantly from each other as indicated by one-way analysis of variance. High/low NA infants = infant negative affect clusters, as determined by three-way cutoff; Dep/Non-dep mothers = mothers' depression status as indicated by Beck Depression Inventory II cutoff; s = seconds.
*** $p < .001$.

expression, and for infants whose mothers did and did not have elevated depression symptoms. Overall, infants had a median of four distress episodes per session ($SD = 4.39$), with each episode lasting a median of 5.02 s ($SD = 30.9$) and with a median of 19.9 s ($SD = 32.4$) between episodes of distress. There were large differences in the overall volume of NA expressed by the infants, with infants expressing from 0% to 100% NA in the session. Infants classified as high NA spent a mean of 68.9% time in NA ($SD =$ 18.4%, range 46%–100%), infants classified as low NA spent a mean of 3.06% time in NA ($SD = 2.75\%$, range 0%–9%). As expected, infants classified as high versus low NA differed in the proportion of expressed NA as assessed by one-way analysis of variance (ANOVA), $F(1, 192) = 925.43$, $p < .001$, with post hoc tests indicating that infant mean proportion of NA differed between each pair of groups, all $p < .001$. The rate and duration of NA episodes were greater for infants classified as high (vs. low) in NA expression (see Table 1). Mothers' mean BSD-II depression score was 8.80 ($SD = 7.93$, range = 0–44). Based on the established cutoff of 14 or higher, 20.77% exceeded that cutoff, indicating at least mild symptoms ($M = 21.21$, $SD = 6.87$); the remainder of the sample experienced low levels of depression symptoms ($M = 5.55$, $SD = 4.00$).

Event-Based Analyses

Event-based analyses indicated that mothers shifted affect on average 7.2 times/min, or once every 8.33 s, with no significant differences in affect shifting between depressed versus nondepressed mothers, $F(1, 181) = .34$, $p = .560$, or mothers of infants with high versus low NA expression, $F(1, 192) = 5.47$, $p = .020$. Table 1 provides rates of maternal affective shifts and contingent responding overall and for each pair of subgroups. For the sample as a whole, mothers contingently shifted affect following a median of 33% ($SD = .30$) of infants' distress episodes within 3 s of distress onset. The rate of maternal contingent responses to infant distress did not differ between depressed versus nondepressed mothers, $F(1, 173) = 3.55$, $p = .061$, or mothers of infants with high versus low NA expression, $F(1, 173) = .01$, $p = .914$. That is, event-based analyses indicated that depressed mothers were as likely to shift their affect in the 3 s following infant distress onset as the nondepressed mothers, and mothers of high NA infants were as likely to shift their affect in the 3 s following infant distress onset as mothers of low NA infants. Finally, we note that depressed mothers and mothers of high NA infants both shifted their affect at similar rates during infant distress, depressed mothers: $F(1, 165) = 2.00$, $p = .160$; high NA mothers: $F(1, 173) = .69$, $p = .407$, and nondistress periods, depressed mothers: $F(1, 177) = .02$, $p = .888$; high NA mothers: $F(1, 188) = 1.49$, $p = .223$. Note that event-related analyses do not control for the history of mothers' affect shifting nor can they directly speak to whether observed contingencies were statistically influenced by—or responsive to—infant distress versus random co-occurrence, which we will test using Granger causality analyses detailed below.

Granger Causality Analyses

We report all Granger causality results in Table 2. All models control for the history of the outcome variable, and analyses testing whether maternal affect shifts facilitated infant soothing control for the history of both infant distress and infant soothing, as detailed in the methods. Note that for successive windows to be significant, the larger window must add significant and unique explanatory power for predicting the outcome variable relative to the smaller window. For example, when both 2 (0–2) and 3 s (0–3) history windows are significant, this means that information about the presence or absence of the predictor (e.g., infant distress onset) in the 3 s history window (i.e., the 3 s before $t = 0$) significantly increased ability to predict the presence or absence of the outcome variable (e.g., a maternal affect shift) occurring at $t = 0$, relative to information about the presence or absence of the predictor in a 2 s history window.

Whole Sample Analyses

When analyzing all participants' data, we observe Granger evidence that mothers contingently respond to infant distress ($N =$ 194; H1). That is, the onset of infant distress significantly increases the likelihood of the mother shifting affect in the subsequent 2–5 s, relative to occasions when infants do not become distressed. We also observe Granger evidence that mother's responses to infant distress facilitate infant soothing, when considering all participants' data ($N = 194$; H2). That is, the presence of a maternal affect shift during

Table 2
Granger Causality Results

Sample	Distress episodes count	Maternal contingent response to infant distress			Maternal responses facilitate infant soothing		
		Window (s)	Granger value	p	Window (s)	Granger value	p
Whole sample ($n = 194$)	932	_0–1_	_0.34_	_.409_	0–1	1.70	.066
		_0–2*_	_6.05_	_.002_	_0–2**_	_6.06_	_.002_
		_0–3**_	_9.10_	_<.001_	_0–3**_	_7.53_	_.002_
		_0–4**_	_10.73_	_<.001_	0–4	3.77	.110
		_0–5**_	_11.37_	_<.001_	0–5	−4.36	.121
High NA infants ($n = 42$)	273	0–1	0.01	.902	0–1	0.17	.561
		0–2	0.17	.842	0–2	−0.54	.585
		0–3	0.47	.818	0–3	−1.08	.539
		0–4	0.54	.898	0–4	−1.39	.596
		0–5	0.74	.915	0–5	−2.78	.351
Low NA infants ($n = 87$)	193	0–1	0.22	.511	0–1	0.01	.296
		_0–2*_	_3.96_	_.020_	_0–2**_	_6.49_	_.002_
		_0–3*_	_5.71_	_.010_	_0–3*_	_5.61_	_.011_
		_0–4*_	_7.01_	_.007_	_0–4**_	_7.87_	_.003_
		_0–5*_	_8.41_	_.005_	_0–5**_	_7.87_	_.008_
Depressed mothers ($n = 38$)	219	0–1	0.37	.389	0–1	−0.08	.689
		0–2	1.80	.166	0–2	0.60	.551
		0–3	2.42	.184	0–3	−1.81	.306
		0–4	3.18	.174	0–4	−4.13	.083
		0–5	3.46	.227	0–5	−5.20	.065
Nondepressed mothers ($n = 145$)	681	0–1	0.02	.857	0–1	1.61	.072
		0–2	3.01	.050	_0–2**_	_5.87_	_.003_
		_0–3*_	_4.78_	_.022_	_0–3**_	_6.70_	_.004_
		_0–4**_	_7.25_	_.006_	0–4	4.59	.057
		_0–5*_	_7.58_	_.010_	0–5	4.65	.098

Note. Italics depict positive significance, that is, where the presence of the predictor variable increases the likelihood of the outcome variable. We observed no negative significant results (e.g., where the predictor decreases the likelihood of the outcome variable). We observed no significant Granger causality results for dyads with high NA infants or depressed mothers. NA = negative affect; s = seconds.
* $p < .05$. ** $p < .01$.

an infant distress episode increases the likelihood of infant soothing in the next 2–3 s, relative to occasions when mothers did not shift their affect during infant distress.

Analyses Comparing Infants High Versus Low in NA

In dyads with infants high in NA, we did not observe Granger evidence that mothers contingently respond to infant distress (H3.1)—or that infant soothing is facilitated by maternal affect shifting ($n = 42$; H3.2). That is, in dyads with high levels of infant NA expression, the onset of infant distress did not significantly increase the likelihood of a maternal affect shift in the next 5 s, relative to occasions when infants did become distressed. Additionally, for high infant NA dyads, the presence of a maternal affect shift during infant distress did not increase the likelihood of infant soothing in the following 5 s, relative to occasions when mothers did not shift their affect during infant distress. By contrast, in dyads with infants low in NA expression, we observed Granger evidence that mothers contingently responded to infant distress, and infants were more likely to soothe following maternal affect shifts ($n = 87$), replicating our findings for the whole sample. That is, for dyads of infants low in NA expression, the onset of infant distress increased the likelihood of mothers shifting affect in the next 2–5 s, relative to occasions when infants did not become distressed. Further, in those same dyads, the presence of a maternal affect shift during infant distress increased the likelihood of infant soothing in the subsequent 2–5 s, relative to occasions in which mothers did not shift their affect during infant distress. This suggests that interactions between infants low in NA and their mothers drove the effects we observed for the whole sample.

Analyses Comparing Mothers With Versus Without Elevated Depression Symptoms

In dyads with mothers' currently experiencing elevated depression symptoms, we do not observe Granger evidence that mothers contingently respond to infant distress (H4.1)—or that infant soothing is facilitated by maternal affect shifting (H4.2). That is, the onset of infant distress did not increase or decrease the likelihood of a maternal affect shift in the next 5 s. Additionally, the occurrence of maternal affect shifting during infant distress did not change the likelihood of infant soothing in the following 5 s. By contrast, in dyads with mothers without elevated depression symptoms, we observed significant Granger evidence for maternal contingent responding and maternal facilitation of soothing, paralleling the results we observed in the whole sample. That is, for mothers without elevated depression symptoms the onset of infant distress increased the likelihood of a maternal affect shift in the next 3–5 s. Additionally, in the interactions of dyads with mothers who were not depressed, maternal affect shifts occurring during infant distress significantly increased the likelihood of infant soothing in the next 2–3 s.

Post Hoc Analysis

Our finding that dyads with depressed mothers and highly negative infants showed a similar pattern of null results raised the possibility that these two samples may be highly overlapping, that is, mothers of infants high in NA expressions may have also been more likely to experience elevated levels of depression symptoms. As such, we conducted a post hoc analysis to assess this possibility. Using a chi-squared test of independence showed that there was no significant association between the likelihood of infants being high, relative to low, in NA expression and mothers' having elevated depression or not, $\chi^2(1, n = 120) = .94, p = .333$.

Discussion

To gain insight into the behavioral pathways theorized to underlie infants' development of self-regulation in a high-risk sample of mothers with a history of depression, we examined real-time sequences of mother's responses to infant distress and infant's subsequent likelihood of soothing. In a sample of 194 dyads, we found significant Granger evidence that mothers contingently responded to infants' distress with an affect switch. We also found evidence that mothers affect shifts during infant distress led to an increase in infant soothing. However, although that finding was true for the sample as a whole, subsample analyses revealed that the finding was specific to dyads of infants who were low in observed NA expression and dyads of mothers who were low in current depression symptom levels. That is, we did not find Granger evidence for maternal contingent responding or that maternal responses facilitated infant soothing in subsamples of our data comprising dyads of infants high in NA expression or dyads of mothers who were currently experiencing clinically significant levels of depression symptoms. We detail the implications of our findings below.

Infant NA Expression

We observed very different patterns of interactions between dyads with infants who were characterized as high versus low in NA expression during the interaction. In dyads with infants low in NA expression—who comprised nearly half of our sample and were in distress an average of just 3% of their play session—results matched the patterns of the whole sample—that is, we observed evidence both for maternal contingent responding and facilitation of infant soothing following maternal response to infant distress. However, dyads with infants high in NA—who comprised just over 20% of the sample and expressed NA for nearly 70% of their play sessions, on average—showed neither of these patterns. There are several considerations when interpreting these results. First, we note that nonsignificant findings from Granger causality may result from an insufficient amount of data. However, for each comparison, high NA infants had similar or more counts of target events, that is, distress, soothing, and affect-shifting events relative to our low NA infants (see Table 1). As target events represent the number of positive samples for testing the result, this suggests that if a similar-sized effect was present in the high NA infant sample, there should have been enough data to detect it. Thus, we cautiously interpret our nonsignificant results as meaningfully not significant.

We hypothesized that high levels of infant NA expression could be the result of an infant interacting with a caregiver who makes minimal efforts to soothe infant distress (Crockenberg & Leerkes, 2004). Considering all our results however, we do not believe we have evidence for this claim. Our event-based analyses indicated that mothers of highly negative infants shift affect within 3 s of infant distress at the same rate as mothers of less negative infants. This is consistent with past work showing that mothers of more negative infants show high rates of responding to distress relative to mothers of less negative infants (van den Bloom & Hoeksma, 1994). Critically though, event-based analyses do not control for the mothers' history of affect shifts, and they do not assess whether the presence or absence of infant distress changes the likelihood of maternal affecting shifting. For example, mothers may respond within 3 s of infant distress by virtue of simply having a high rate of affect shifts. When we control for mothers' history of affect shifts, and we use Granger causality to explicitly test whether infant distress onset increases the likelihood of contingent affect shifting, we no longer find evidence that mothers of highly negative infants respond contingently to their distress. Thus, our Granger causality results suggest that for mothers of high NA infants, maternal affect shifts following infant distress onset are not statistically responsive to infant distress onset. That is, while maternal affect shifts did occur in the 3 s following infant distress, this appears to be due to chance occurrence, as including information about infant distress onset did not predict any change in the timing of mothers' affect shifts. Infants with relatively higher expression of NA during face-to-face interactions also appeared to benefit *less* from maternal responses to distress than infants who display less NA. That is, mothers' affect shifts during infant distress did not appear to facilitate soothing in high NA infants, despite our evidence that they did for the group overall.

Infants who were in the group designated as high in NA expression had significantly more and longer episodes of distress than infants low in NA expression. Thus, one possible explanation for our results is that mothers' may have different patterns of affect shifting during periods of infant distress relative to nondistress. For example, if mothers of both high NA and low NA infants had a high rate of affect shifts during infant distress and a relatively lower rate of affect shifts during infant nondistress, then the longer durations of infant distress episodes could lead to higher autocorrelations in responses of mothers of high NA infants relative to mothers of low NA infants, potentially making mothers' responses appear as though they were not statistically responsive to infant distress. However, our event-based analyses indicate that mothers of high NA infants shifted their affect at similar rates as mothers of low NA infants during distress and nondistress periods. This suggests that it is the timing of mothers' responses to infant distress, not the duration of infant distress per se that is driving our results.

Maternal Depression Symptoms

Next, we considered the hypothesis that mothers with current elevated depression symptom levels may be less responsive to infants' distress, and that their infants may be more challenging to soothe. Our results showed that dyads with mothers experiencing elevated depression symptoms followed many of the same patterns as dyads with infants high in NA: that is, mothers with current elevated depression symptom levels did not show contingent responding to infant distress, and when their affect changes during infant distress were not associated with increases in the likelihood of infant soothing. Given that we found significant Granger causality results in our dyads with low NA infants, who had fewer episodes of distress and soothing than did the dyads with mothers with elevated symptoms of depression, we interpret these nonsignificant Granger causality results as meaningful. That is dyads with mothers with elevated symptoms of depression should have had sufficient target events to detect equivalent effects observed in other models.

Additionally, our post hoc analyses showed that there was no significant association between infant NA expression and maternal depression, meaning results associated with infant high NA expression and maternal depression status are independent of one another. Event-based analyses indicated that currently depressed mothers had the same rate of contingent responding and affect shifting as mothers who were not currently depressed. This suggests that it was not mothers' rate of affect shifts per se that differed between the groups but rather the timing in mothers' responses: that is, depressed mothers appeared to be less statistically responsive to infant distress than nondepressed mothers. Beebe et al.'s (2008) found that depressed mothers' facial affect shifts were more responsive to infants' vocal affect than nondepressed mothers, that is, depressed mothers were hypervigilant to changes in infants' affect. Given that Beebe's results assess mothers' responses to any changes in infants' vocal affect, one possibility is that depressed mothers' responsiveness to onset of infant's positive or neutral affect drove their effect. To test this, in supplemental analyses, we examined mothers' responsiveness to all changes in infant affect. However, we did not find Granger evidence that depressed mothers contingently shifted affect following infants affect shifts regardless of the type of affect (i.e., considering infant affect shifts to positive, neutral, or NA; see Result S3 in the online supplemental materials). Another possibility is that the observed patterns of maternal contingent responses to distress are highly modality specific. Our affect coding scheme examined relations between mother and infant affect considering a combination of both facial behavior and vocalizations, whereas Beebe et al., examined relations between mothers' facial affect in response to infants' vocal affect. Such differences in the annotation of mother and infant affect may have led to different patterns of results. For example, Beebe et al. (2008) also found that depressed mothers' gaze and touch were less contingent upon infants' gaze and touch relative to nondepressed mothers, more in line with the results we present here.

Implications

Overall, our results suggest that depressed mothers and mothers of highly negative infants were not statistically responsive to infant distress. These findings are consistent with data that mothers of highly negative infants, and those who are more depressed, are less sensitive in their interactions with infants relative to controls (Papoušek & von Hofacker, 1998). Additional research should consider the relevance of responses to distress that are statistically responsive (over and above being contingent) for the development of self-regulation. One possibility is that infants are sensitive to the statistics of mothers' patterns of behavior, discriminating occasions on which mothers' history of affect shifts changes in response to their distress. Another possibility is that responses that are statistically responsive to infant distress qualitatively differ from contingent but not statistically responsive responses in some dimensions that we did not capture. In addition to these contributions of mothers, infants also contribute to dyadic distress and regulation dynamics. Infants who were high in NA expression and infants of currently depressed mothers were less responsive to mothers' affect shifts. That is, when mothers shifted affect during infant distress, infants were no more likely to soothe than if mothers had not shifted affect. These same responses facilitated soothing in a group of infants with lower levels of NA expression and in infants whose mothers were not depressed. Thus, our results indicate that infants vary in how responsive they are to their mother's efforts to soothe them. This is consistent with temperament research, which indicates that more negative infants are often more difficult to soothe (Crockenberg & Smith, 1982). However, our work is the first to use real-time sequences of data to determine infants' likelihood of soothing in the presence or absence of similar responses by mothers. Although much research focuses on mothers' role in soothing, this finding highlights the role of individual differences in infant NA expression and infant soothability as independent dimensions of dyadic distress dynamics.

Our results also suggest the need to clarify coding schemes. Global measures can obscure or misrepresent the precise real-time dynamics that we observe. Sensitivity rating schemes often incorporate the effectiveness of caregiver soothing as a characteristic of the caregiver. For example, Ainsworth's scales state that a sensitive mother "knows what kind and degree of soothing he requires to comfort him [when he is distressed]" (Ainsworth, 1969). Our analyses of real-time sequences of mother–infant affect indicate that this is problematic, in that it may overly place the responsibility of soothing on the mothers.

Limitations

Although Granger causality is a powerful time series analysis technique, it is necessary to be cautious in the interpretation of Granger causality results. First, as with any statistical analysis, insignificant results can be an indication of inadequate sample size. This is especially relevant given that in each of our analyses, the "smaller" subsample showed more insignificant results. Given that our low NA infants showed significant results with the lowest overall counts of distress, we should have had the power to detect similar-sized effects in our other subsamples. However, designs with larger samples or with longer periods of interaction may reveal additional significant findings. Second, Granger causality analyses control for the history of the outcome of interest. As such, systematic patterns in the history of the outcome of interest—for example, the duration or frequency of infant distress—can act as "hidden" factors that can obscure relationships between predictor and outcome variables, in particular if they also systematically affect the predictor variable, that is, mothers' responses to infant distress, as detailed above. We directly tested the possibility that the duration or frequency of infant NA drove our results, determining that they did not. However, additional factors which we have not considered may also be affecting both our outcome and predictor variables. Thus, without experimental manipulations of mothers' or infants' behavior (e.g., Goldstein et al., 2003) it is not possible to know whether the relations we observed are "truly" causal.

Our free play paradigm also likely constrained the types of NA we observed in our sample, as well as mothers' responses to distress. For example, the fact that mothers and infants were facing one another in chairs likely prevented mothers from picking up and holding infants, known to be the most common response to infant crying (Bornstein et al., 2017). More ecologically valid research examining repeated distress and regulation cycles in everyday settings over hours or days would help to substantiate our results. Additionally, longitudinal data are necessary to examine the processes that might lead to the real-time differences we saw in our sample. For example, it will be essential for future research to test whether depressed mothers and mothers of highly NA infants become less responsive to infant distress over time because they do not receive the ostensibly reinforcing signal of infant soothing when they do respond during episodes of infant distress. Mobile sensing tools to capture and automatically detect infant crying over extended periods of time, for example, hours, days, or weeks can facilitate such future work (de Barbaro, 2019; Micheletti et al., 2022).

Finally, more testing is necessary to assess the extent to which our findings generalize beyond our sample. Our study sample comprised women with a history of depression as they interacted with their 3-month-old infants. Moreover, the women were generally well resourced in terms of education and income. Future efforts could examine these results with women without a history of depression, with older infants who display different patterns of affect expression and soothing (van den Bloom & Hoeksma, 1994), and among families with fewer sociodemographic resources.

Conclusions

Carefully characterizing real-time sequences of mother and infant behaviors provides a "social microscope" (van den Bloom & Hoeksma, 1994) by which we can gain insight into key developmental processes. By applying this microscope to repeated sequences of mother–infant distress and regulation, we aimed to precisely examine the behavioral mechanisms that shape infants' developing self-regulation in a sample at elevated risk for depression (Kopp, 1989). Our work highlights both mother and infant contributions to processes of infant distress and regulation. Although overall, we found evidence that mothers' responses to infant distress facilitated infant soothing, our results suggest possible mechanisms for more maladaptive self-regulation trajectories in highly negative infants and infants of depressed mothers.

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