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Maternal heart rate variability at 3-months postpartum is associated with maternal mental health and infant neurophysiology

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Previous research has demonstrated a critical link between maternal mental health and infant development. However, there is limited understanding of the role of autonomic regulation in postpartum maternal mental health and infant outcomes. In the current study, we tested 76 mother-infant dyads from diverse socioeconomic backgrounds when infants were 3-months of age. We recorded simultaneous ECG from dyads while baseline EEG was collected from the infant; ECG heart rate variability (HRV) and EEG theta-beta ratio and alpha asymmetry were calculated. Dyadic physiological synchrony was also analyzed to better understand the role of autonomic co-regulation. Results demonstrated that lower maternal HRV was associated with higher self-reported maternal depression and anxiety. Additionally, mothers with lower HRV had infants with lower HRV. Maternal HRV was also associated with higher infant theta-beta ratios, but not alpha asymmetry. Exploratory analyses suggested that for mother-infant dyads with greater physiological synchrony, higher maternal HRV predicted increased infant theta-beta ratio via infant HRV. These findings support a model in which maternal mental health may influence infant neurophysiology via alterations in autonomic stress regulation and dyadic physiological co-regulation.

The prevalence of postpartum mental health concerns among new mothers is on a disconcerting rise¹. This is especially concerning as maternal mental health in the perinatal period is known to shape the infant-caregiver relationship and consequent child development²⁻⁴. However, previous research in this domain have primarily used self-reported measures of psychological distress. Self-report measures are prone to bias, resulting in both over and underreporting of symptoms and offer limited mechanistic insight. While self-report measures of maternal mental health are considered the gold-standard in research, there are notable drawbacks of this approach. Namely, widely used self-reported perinatal mental health measures demonstrate poor cross-cultural validity⁵, which raises concerns about the accuracy of these measures for diverse sociocultural populations. Thus, research evaluating objective measures that could offer more accurate assessments of depression and anxiety among new mothers is warranted. To better understand the biological mechanisms underlying individual differences in maternal mental health, physiological indices of maternal emotion regulation can be evaluated. Specifically, by examining the maternal stress physiology system, we can gain insights into how maternal psychological distress can influence infant outcomes. Research in this domain is imperative for advancing our understanding of the interplay between maternal well-being and child development.

Heart rate variability (HRV), a measure of the intervals between heartbeats, is a candidate marker of physiological stress regulation that could help elucidate the relation between maternal mental health and infant outcomes. Heart rate variability (HRV) is commonly used to characterize the fast-acting autonomic nervous system (ANS). The ANS comprises two branches: the sympathetic (SNS) and parasympathetic (PNS) branches, representing the “fight-flight-freeze” and “rest and restore” stress systems, respectively. Although the precise physiological mechanisms contributing to heart rate variability (HRV) are still being explored, literature suggests that HRV reflects the balance between sympathetic and parasympathetic nervous system activity and can be used to index physiological stress regulation, more broadly⁶⁻⁸.

Past research proposes that HRV approximates the capacity for adaptive regulation of autonomic response to perceived threats and exert top-down cognitive control across the lifespan^{9,10}. Specifically, higher HRV is

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associated with greater adaptive autonomic regulation and lower HRV is associated with more maladaptive responses to stress. For example, individuals with lower HRV are shown to be less effective at regulating incoming negative emotional stimuli relative to individuals with higher HRV¹¹. Critically, HRV is not a fixed construct but demonstrates individual-level fluctuations, both improvements and disruptions¹². Neurobiologically, it has been proposed that HRV is linked with the functional activation of neuroanatomical structures responsible for the detection of threat and safety^{12,13}. Thus, individuals who demonstrate consistent maladaptive threat awareness may also show lower HRV, indicative of heightened reactivity and inadequate recovery of the stress response. Continually elevated states of perceived stress can result in wear and tear on both physical and psychological well-being, impacting overall health and mood^{14,15}.

Maternal heart rate variability (mHRV) has predominantly been evaluated during the prenatal period^{16,17}. Studies in this domain have underscored the importance of mHRV in characterizing cardiorespiratory features of maternal health, with findings suggesting its predictive value for conditions like preeclampsia and other prenatal complications^{18–20}. Notably, lower prenatal mHRV scores have been associated with increased prevalence of anxiety disorders during late pregnancy¹⁷. Despite these insights, a significant gap exists in the literature regarding the replication of these findings in the postnatal period. Existing studies measuring mHRV within stress-induced contexts, such as the Still-Face paradigm, have revealed associations between mHRV responsiveness and symptoms of depression and anxiety^{21–23}. However, there is a lack of research evaluating mHRV at baseline, without any experimental emotion induction. Such studies are necessary to characterize ANS function independent of external stressors, given the documented links between baseline mHRV and prenatal mental health outcomes. The postpartum period is marked by significant physiological and emotional fluctuations as mothers adapt to childbirth and child-rearing^{25–27}. Thus, the postpartum period is a critical phase for characterizing biological markers of effective stress regulation systems during this critical phase.

From a psychobiological perspective, assessing caregivers' physiological stress regulation is crucial as caregivers play an important role in shaping their child's own stress regulation system development^{25,28–30}. Infants are born with immature stress response systems, and thus rely on caregivers for stress regulation²⁴. This co-regulation can occur through behavioral (e.g., soothing) or covert physiological means (e.g., heart-rate synchrony)^{31,32}. When caregivers struggle to manage their own physiological stress, it can lead to atypical patterns of autonomic stress regulation in their infants^{33–36}. This is because the developing infant ANS and brain are highly receptive to cues from caregivers^{26,33,37,38}. Indeed, research has shown that mHRV changes can regulate infants' HRV in real-time during maternal breathing exercises³⁹. Consequently, the varied stress cues provided by caregivers may induce changes in the infant's physiology, including variability (HRV and neural activity)^{29,40}.

Two candidate neural markers of infant cognitive and emotion regulation measured using EEG are theta-beta ratio and frontal alpha asymmetry, respectively. Theta-beta ratio is a relative EEG power metric that indicates the abundance of lower frequency power relative to high frequency power. It is believed to reflect the maturation of cortical-subcortical networks that support emerging attentional control processes^{41,42}. Indeed, research has found that higher theta-beta ratios (indicating more relative theta power) at 10 months is predictive of differences in cognitive control over one year later⁴³. Additionally, relative EEG power metrics are sensitive to variations in environmental input. Studies have shown that infants of higher stressed mothers exhibit higher relative theta power compared to higher frequency power^{44,45}. Relatedly, infant frontal alpha asymmetry has been predictive of later emotion regulation abilities in childhood^{46–48} and has been suggested as a potential mechanism explaining intergenerational transmission of depression^{49–51}.

The current study leverages unique triadic data collected through concurrent recordings of maternal and infant ECG and infant EEG (N = 76) at 3-months of age. Our study objectives were threefold. Firstly, we examined the associations between mHRV and self-reported experiences of depression, anxiety, and stress. Secondly, we investigated how individual differences in average mHRV levels correlated with infant stress physiology and neural function. Lastly, in an exploratory analysis we examined dyadic physiological synchrony between infant and maternal HRV as a moderator and mediator of associations between mHRV and infant neurophysiology. To incorporate and consolidate diversity in HRV calculations, we employed factor analysis to derive a composite mHRV factor score, encompassing frequency, and non-linear quantifications of heart rate activity. We chose a factor analysis approach primarily due to our theoretical assumption that the latent factor of autonomic regulation underlies observed HRV variables. Similar approaches have been adopted in studies linking HRV to cognition and mood in healthy adults⁵². This factor score incorporated high-frequency (HF) band power, low-frequency (LF) to HF power ratio, and a non-linear HRV measure, namely DFA Alpha 1, to assess the complexity and irregularity of heart rate dynamics. We hypothesized that lower mHRV would be associated with higher maternal reports of depression, anxiety, and perceived stress. We also hypothesized that higher mHRV would be concurrently associated with higher infant HRV, lower infant theta-beta ratios, and lower infant frontal alpha asymmetry.

Results

Heart rate variability (HRV) factor score

Using confirmatory factor analysis (CFA), we computed a single latent factor score for heart rate variability (HRV) using high-frequency (HF) band power, low-frequency to HF power (LFHF) ratios, and DFA Alpha 1. The decision to estimate a single factor score was justified by the significant correlations observed among HRV variables across different domains, see Table 1. The primary purpose of the CFA was twofold: (1) to estimate a robust latent measure of maternal autonomic regulation and (2) to enhance the measurement specificity of a mHRV score by reducing measurement error.

Variable	M	SD	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. HF HRV	32.78	16.2																
2. LFHF ratio	3.19	3.07	-0.76**															
3. DFA alpha	1.19	0.24	-0.89**	0.74**														
4. mHRV Factor Score	0	0.98	0.98**	-0.81**	-0.96**													
5. Maternal depression	5.6	4.4	-0.24*	0.27*	0.16	-0.24*												
6. Maternal anxiety	33.24	10.5	-0.2	0.22	0.1	-0.19	0.75**											
7. Maternal perceived stress	13.06	6.15	-0.15	0.13	0.05	-0.13	0.75**	0.77**										
8. Maternal resting HR	77.22	11.3	-0.41**	0.49**	0.50**	-0.47**	-0.01	-0.04	-0.01									
9. State anxiety	30.76	9.4	-0.1	0.14	0.01	-0.08	0.68**	0.86**	0.74**	-0.02								
10. Income-to-needs	5.31	5.51	-0.17	0.04	0	-0.11	-0.06	0.01	-0.12	-0.26*	-0.04							
11. Maternal age	32.86	5.45	0.07	-0.08	-0.08	0.11	-0.01	-0.03	0.01	-0.27*	-0.05	0.30**						
12. Infant HRV Factor Score	0	0.99	0.27*	-0.19	-0.15	0.25*	-0.14	-0.17	-0.15	0.05	-0.14	-0.21	-0.01					
13. Infant theta-beta ratio	5.62	1.45	-0.30*	0.38**	0.22	-0.30*	0	-0.03	-0.08	0.16	0	0.1	-0.17	-0.34**				
14. Infant frontal alpha asymmetry	0.03	0.15	-0.01	0.15	0.07	-0.06	0.19	0.07	0.19	0.1	0.05	-0.21	0.09	-0.02	0.02			
14. Dyadic physiological synchrony	0.04	0.29	-0.04	0.03	0.07	-0.05	0.13	0.26*	0.22	0.19	0.21	0	0.02	0.27*	-0.14	0.04		
15. Infant age (months)	3.47	0.39	-0.04	0.17	-0.04	-0.04	0.04	0.06	0.09	0.17	0.12	-0.16	0.11	-0.03	0.18	0.12	-0.06	
16. Gestational age (weeks)	39.21	1.23	0.07	-0.17	-0.09	0.08	-0.16	-0.12	0.04	-0.02	0	-0.11	-0.08	-0.08	-0.26*	-0.02	0.05	-0.27**

Table 1. Correlations among variables. Pearson correlations of key variables. Significant correlations in the hypothesized direction between primary variables provided rationale for subsequent regression models.

Maternal HRV (mHRV), anxiety, depression, and perceived stress

For our primary research question, we evaluated associations among mHRV and maternal depression, anxiety, and perceived stress (Table 2). Results demonstrated a significant negative association between mHRV scores with maternal depression and anxiety. There was a trend-level negative association between mHRV scores with maternal perceived stress (See Fig. 1). In other words, mothers with lower mHRV reported significantly higher rates of depression and anxiety. Maternal age, income-to-needs ratio, maternal education, and resting HR were included as covariates but did not show a significant effect. State anxiety was positively associated with all mental health outcomes.

Maternal HRV (mHRV), infant HRV (iHRV), and infant neural markers

Next, we evaluated associations between mHRV scores with infant physiological and neural outcomes. We included infant age, sex, and maternal self-reported mental health using a factor score of the previously evaluated anxiety, depression, and perceived stress (See Table 3). Results demonstrated a significant positive association of

	Depression	Anxiety	Perceived stress
	Std. Beta (se)	Std. Beta (se)	Std. Beta (se)
mHRV	-0.30 (0.09)**	-0.16 (0.07)*	-0.15 (0.09)^
Income-to-needs	-0.06 (0.09)	0.04 (0.06)	-0.05 (0.08)
Maternal Education	-0.15 (0.09)	-0.12 (0.07)	-0.20 (0.08)
Maternal age	0.03 (0.11)	0.06 (0.08)	0.11 (0.09)
Maternal HR	-0.20 (0.1)	-0.07 (0.07)	-0.06 (0.09)
Maternal state anxiety	0.63 (0.06)**	0.84 (0.03)**	0.72 (0.05)**
R ²	0.53	0.76	0.60

Table 2. Maternal mental health outcomes. FDR-Adjusted p -values $^{\wedge} \leq 0.10$; * ≤ 0.05 ; ** ≤ 0.01 ; *** ≤ 0.001 .

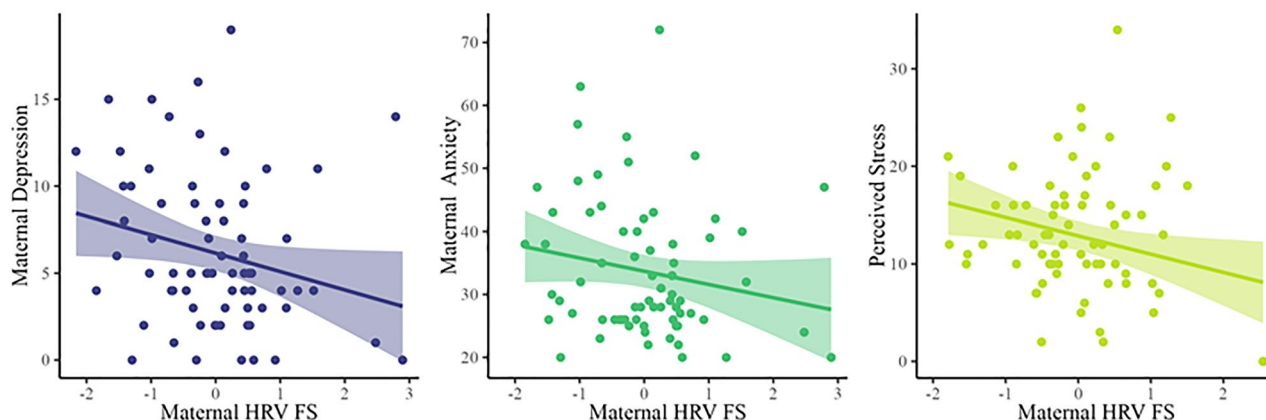


Figure 1. Scatterplots with line of best fit reflecting associations of mHRV to maternal mental health outcomes.

	Infant HRV	Theta/beta ratio	Frontal alpha asymmetry
	Std. Beta (se)	Std. Beta (se)	Std. Beta (se)
mHRV	0.37 (0.12)*	-0.31 (0.13)*	0 (0.15)
Infant age	-0.07 (0.1)	0.15 (0.11)	0.11 (0.12)
Gestational age	-0.13 (0.11)	-0.21 (0.11)^	0.03 (0.12)
Maternal Mental health FS	-0.11 (0.11)	-0.11 (0.11)	0.16 (0.12)
Maternal HR	0.23 (0.12)	-0.02 (0.13)	0.09 (0.15)
R ²	0.15	0.18	0.05

Table 3. Infant physiological and neural outcomes. FDR-Adjusted p -values $^{\wedge} \leq 0.10$; * ≤ 0.05 ; ** ≤ 0.01 ; *** ≤ 0.001 .

mHRV with infant HRV (iHRV) and a significant negative association with infant theta-beta ratio values (See Fig. 2). In other words, for mothers with lower HRV, their infants also showed lower HRV and greater theta power relative to beta power. There were no significant associations with frontal alpha asymmetry. There were also no significant effects of the included covariates in our regression models.

Exploratory dyadic physiological synchrony analysis

We conducted an exploratory analysis to investigate if the coordination of mother-infant HRV may moderate the relationship between mHRV and infant neurophysiology. To calculate physiological synchrony, we applied 0-lag cross-correlations within each dyad between 30-s HF HRV epochs, to estimate synchrony in respiratory sinus arrhythmia (RSA)^{29,53}. We then replicated the infant outcome regressions above but included a main effect term of physiological synchrony and an interaction term of physiological synchrony X mHRV (See full table of results in SI). With iHRV modeled as the dependent variable, there was a main effect of physiological synchrony ($\beta = 0.26$, $p = 0.02$) and an interaction effect of physiological synchrony X mHRV on iHRV ($\beta = 0.25$, $p = 0.02$). Analysis of simple slopes indicated that the positive association between average mHRV and iHRV was amplified among individuals with greater physiological synchrony (mean + 1 SD: $\beta = 0.51$, $p = 0.001$) relative to those with little to no physiological synchrony (mean - 1 SD: $\beta = 0.01$, $p > 0.05$; See Fig. 3a). Further, when the interaction

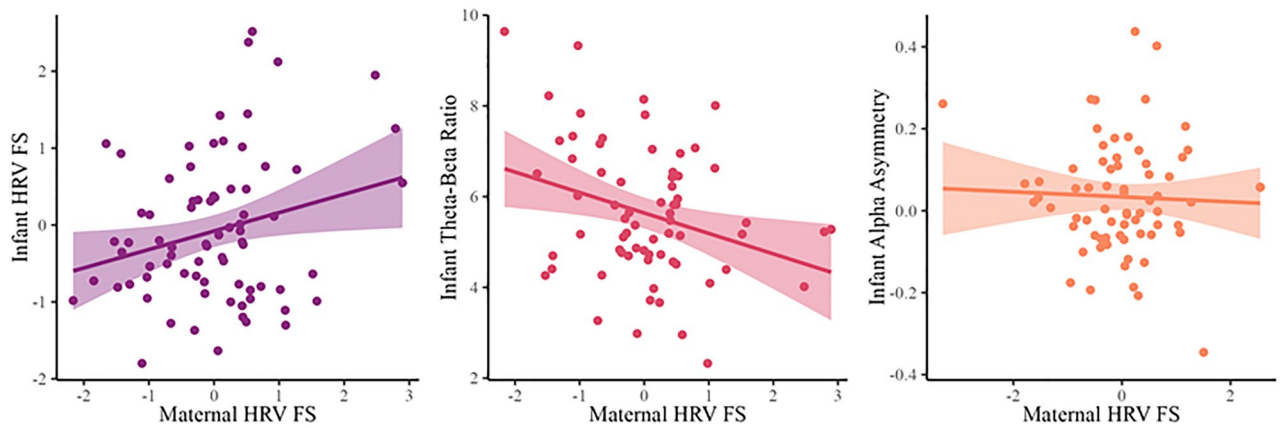


Figure 2. Scatterplots with line of best fit reflecting associations of mHRV to infant physiological and neural outcomes.

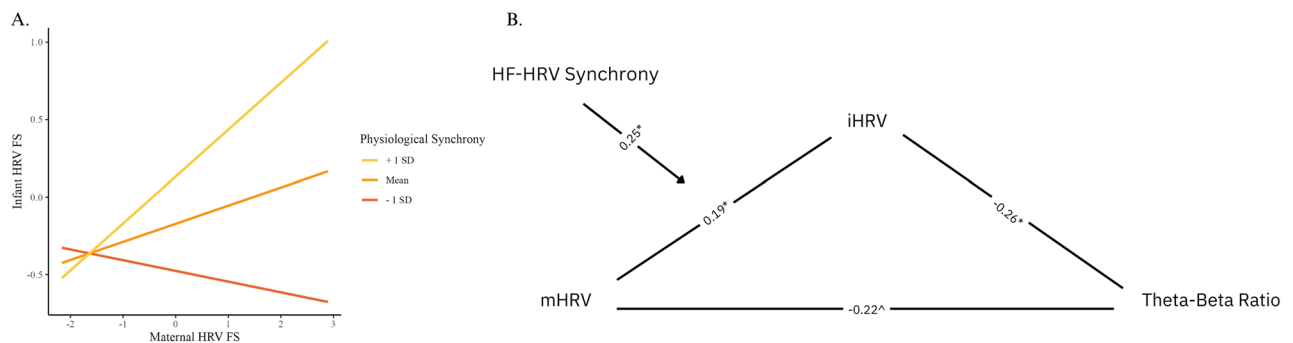


Figure 3. A) Simple slopes plot representing the association of mHRV with iHRV at high (+1 SD above the mean) and low (−1 SD above the mean) levels of physiological synchrony. B) Path model relating mHRV to iHRV, moderated by physiological synchrony, predicting theta-beta ratio.

term was added to the model, the R-squared increased from 0.15 to 0.28, suggesting that an additional 13% of the variance in infant HRV was explained by the physiological synchrony main effect and interaction terms. There were no main or interaction effects of physiological synchrony on infant EEG outcomes.

In a secondary exploratory analysis, our aim was to evaluate a theoretical pathway in which physiological synchrony regulates iHRV, consequently influencing infant neural activity. Specifically, we tested moderated mediation to evaluate if iHRV (conditional upon physiological synchrony) mediated the relationship between mHRV and theta-beta ratio. Our analysis revealed that the interactive effects between iHRV and physiological synchrony played a mediating role in the association between mHRV and the theta-beta ratio ($\beta = -0.06$, CI $[-0.26, -0.003]$; see Fig. 3b). Notably, the mediating role of iHRV was heightened among dyads exhibiting greater positive physiological synchrony (mean +1 SD: $\beta = -0.13$, CI $[-0.52, -0.01]$), compared to those with lower synchrony (mean −1 SD: $\beta = -0.050$, CI $[-0.003, -0.00]$).

Discussion

There is a pressing need to comprehensively understand the physiological underpinnings of maternal mental health during the perinatal period and its impact on infant development. In this study, we investigated maternal heart rate variability (mHRV) in relation to maternal mental health and evaluated associations between mHRV and infant neurophysiological outcomes at 3 months of age using concurrently recorded dyadic HRV and infant EEG. We present novel evidence of an association between an mHRV factor score and maternal self-reports of depression and anxiety symptoms. Additionally, we established an association between mHRV and infant neurophysiology. Finally, we found that dyadic physiological synchrony may amplify the link between mHRV and infant neural function through infant HRV. These findings reinforce mounting evidence highlighting the crucial role of caregiver well-being in infant development^{35,45,54–56} and provide empirical support for a pathway by which caregivers may influence their infant through physiological co-regulation.

The first aim of this study was to evaluate whether a factor score of mHRV, which accounted for shared variance across multiple measures of HRV, was associated with maternal mental health. Controlling for socioeconomic factors and situational anxiety, we found that higher mHRV was correlated with lower levels of depression and anxiety, but not perceived stress. Given that HRV is thought to reflect adaptive autonomic nervous system regulation, caregivers who are physiologically equipped to manage stress in their environment may be less likely

to experience psychological distress^{12,57}. Specifically, it has been posited that stronger ANS regulation may buffer an individual's likelihood of developing mental health conditions such as depression and anxiety^{58,59}. Further, the null association between mHRV and perceived stress is consistent with previous research that has demonstrated a disconnect between physiological stress and perceived stress⁶⁰. In general, this finding adds to a growing body of literature linking physiological stress with mental health^{20,57,61,62}. These findings are aligned with research by Kimmel and colleagues demonstrating that lower prenatal HRV scores were associated with increased prevalence of anxiety disorders during late pregnancy¹⁷. Similarly, these findings also support past research demonstrating differences between HRV reactivity and regulation during caregiver-infant interactions based on symptoms of anxiety and depression^{21,29}. Our findings expand upon the existing literature by linking maternal HRV with normative variation in postpartum mental health experiences within a community sample of mothers.

Prior research has established that maternal psychological distress can interfere with the caregiver-infant relationship and subsequent child development^{36,63–65}. However, relatively little research has looked at the caregiver's physiological states as a predictor of infant neural and physiological function^{45,66,67}. We found a positive relation between mHRV and infant HRV (iHRV), such that mothers with lower group average mHRV were more likely to have infants with lower group average iHRV. Given that infant and caregiver ECG was recorded simultaneously, we explored the possibility that the between-person associations in HRV were modulated by real-time physiological linkage occurring while the infant was seated on the caregiver's lap. We found that physiological synchrony within dyads moderated the association between mHRV and iHRV across dyads. In other words, mothers with low mHRV were more likely to have infants with correspondingly low HRV if they exhibited high physiological synchrony, and vice versa for high mHRV. These findings suggest that physiological co-regulation may underlie the positive correlation observed between average mHRV levels and average iHRV levels across individuals, aligning with prior dyadic physiological synchrony literature^{29,39,68–72}. This finding is particularly notable considering that infants were positioned on their mothers' laps during concurrent recording of dyadic ECG, suggesting evidence of real-time physiological co-regulation potentially facilitated by physical touch^{73,74}. These findings are aligned with prior dyadic synchrony literature demonstrating that experimental manipulation of maternal HRV shows moment-to-moment regulation effects of infant HRV during physical touch³⁹. Moreover, we found evidence of a main effect of physiological synchrony on iHRV, suggesting that greater synchrony is predictive of higher iHRV. This finding may be explained by research suggesting that physiological synchrony captures a critical aspect of the caregiver-infant dynamic, which may drive infant physiological regulation in and of itself^{29,75}.

Our results also demonstrate that higher mHRV scores were correlated with lower infant theta-beta ratios. Mothers who showed greater ANS regulation tended to have infants with increased high-frequency oscillations and lower low-frequency oscillations. Lower theta-beta ratios are neural signatures associated with increased top-down regulation and have been linked to increased attentional control⁴³. Thus, these findings would suggest that children of caregivers with a more regulated physiological stress profile tend to showcase a pattern of neural function associated with greater cognitive control or regulation. Interestingly our regression models linking mHRV to iHRV and theta-beta ratio remained significant after controlling for a host of potentially confounding covariates including maternal mental health. This would suggest that above and beyond perceived psychological experiences, there is utility in measuring caregiver physiological functioning to better understand how caregivers regulate their infant in ways that could affect early trajectories of development^{16,17}. There were no significant associations between mHRV and infant frontal alpha asymmetry. This could possibly be explained by the ontogenetic development of brain specialization that underpins observed differences in alpha asymmetry⁷⁶. Infant brain hemispheres remains relatively unspecialized for the first months of life, particularly in the frontal cortex as this region of the brain follows as more protracted developmental course⁷⁷. Indeed, clearer evidence of cortical specialization emerges around 6-months of age, which may lead to more distinct individual differences in alpha asymmetry emerging at this time^{50,78}.

Interestingly, results did not demonstrate any significant interaction effects between mHRV and physiological synchrony on either infant neural outcome, suggesting that mHRV may not be regulating infant EEG directly via synchrony. This finding led us to conduct an exploratory path analysis evaluating indirect effects of mHRV on theta-beta ratio via the interaction of iHRV and dyadic physiological synchrony. Tests of indirect effects revealed that the association between mHRV and infant theta-beta ratio was mediated by iHRV, conditional upon level of physiological synchrony. To elaborate, for mother-infant dyads with high physiological synchrony, iHRV significantly mediated the relation between mHRV to infant neural function. This could suggest that mothers regulate infant HRV via physiological synchrony, and in turn, infant HRV modulates neural function^{22,79}. Taken together, these findings provide preliminary evidence for a pathway through which maternal autonomic regulation may influence infant neural activity via co-regulation of infant's autonomic nervous system.

Several limitations warrant careful consideration in the context of this study. First, our sample size is relatively modest. To strengthen the robustness of our findings, particularly the more complex moderated mediation analysis, future research should aim to replicate these results with a larger dataset. Furthermore, due to the cross-sectional and observational nature of this investigation, it is crucial to acknowledge that causal directionality cannot be inferred. To address these limitations, future research should also measure contingent associations to explore the interplay between changes in concurrent mHRV and dynamic shifts in maternal mood and emotion. This line of inquiry could provide deeper insights into the intricate relationship between autonomic balance and maternal experiences and behaviors. Additional research is also needed to explore the potential mediating role of infant autonomic function in regulating neural activity. While these questions were not within the scope of present study, future endeavors are warranted to gain a deeper understanding of the interplay between maternal and infant neurophysiology. Lastly, future studies should aim to utilize longitudinal data to advance the field's understanding of how individual differences in maternal autonomic regulation may have enduring impacts on infant development.

The current study sought to investigate maternal autonomic functioning at three months postpartum, an important phase characterized by rapid biopsychosocial changes within caregiver-infant dyads. Our findings provide novel evidence in support of resting maternal heart rate variability as a robust indicator of caregiver mental health. Results also showed that higher maternal heart rate variability corresponded to infant neural EEG profiles associated with heightened attentional control. Moreover, analyses of dyadic physiological synchrony offer evidence for a pathway by which maternal autonomic regulation could impact infant neurophysiology activity through the co-regulation of the infant's autonomic nervous system function. Together, our findings emphasize the importance of assessing the biological underpinnings of caregiver stress during the postnatal period. This knowledge could facilitate the early identification of caregivers in need of additional support due to mental health concerns, highlighting the potential significance of this research in clinical practice.

Methods

Participants

Infants and their mothers were recruited through diverse channels, including community events, family services, healthcare providers, and flyers distributed at local businesses in the New York metropolitan area. These participants were originally enrolled in a larger longitudinal study conducted between May 2018 and December 2019. To be eligible for the present study, participants were excluded if their child was born before 36 weeks gestation, if they had multiple births, or if the child had a developmental disorder. We recruited 104 participants into the study, however, data loss occurred at various stages of the data collection and processing, resulting in a usable sample size of $N = 76$ (See Table 4).

Detailed demographic information for the participants can be found in Table 5. The study was conducted in accordance with the principles outlined in the Declaration of Helsinki, and written informed consent was obtained from a parent or legal guardian before any assessments or data collection occurred. All research procedures were approved by the New York University Institutional Review Board (IRB).

Protocol

Families visited the lab when their infant was approximately 3 months of age. During the visit, ECG was recorded from mother and infant while EEG was simultaneously recorded from the infant. Mothers also completed responses to socio-demographic and mental health questionnaires.

Measures

Family and household characteristics

Questionnaires collected information on demographic factors such as maternal and infant age, race, and ethnicity. Additionally, caregivers provided details regarding their highest level of education completed and the annual household income. To assess the socioeconomic status of the families, the income-to-needs ratio (ITN) was calculated by dividing the total household income by the federal poverty line that corresponds to the number of adults and children in the household.

Maternal mental health

We collected surveys of maternal depression, anxiety, and stress to capture mothers perceived mental health states. Maternal depression was measured using the Edinburgh Postnatal Depression Scale¹⁰⁸ and maternal anxiety was measured using the State-Trait Anxiety Inventory¹⁰⁹. The Edinburgh Postnatal Depression Scale (EPDS) consists of ten items that capture various aspects of mood and emotional well-being. Each item is scored on a four-point Likert scale with higher scores indicating a higher level of depressive symptoms (Cronbach's alpha range between 0.73 and 0.87). The State-Trait Anxiety Inventory (STAI) assesses two aspects of anxiety: state anxiety and trait anxiety. The state anxiety domain measures in-the-moment experiences of temporary anxiety while the trait anxiety domain measures chronic trait-like experiences of anxiety. Item responses are scored on a four-point Likert scale with higher scores indicating greater anxiety (Cronbach's alpha range between 0.86–0.95). For the current analysis we used the STAI trait anxiety total score as a primary outcome. State anxiety was included as a covariate to control for any contextual experiences of temporarily heightened anxiety during experimental testing. Maternal perceived stress was measured using the Perceived Stress Scale (PSS; Cohen et al.¹¹⁰). The PSS is a 14-item questionnaire that asks the respondent to report on their levels of experienced stress in situations during the past month. The perceived stress score was the summed score across all items (Cronbach's alpha > 0.70).

Confirmatory factor analysis (CFA) was used to derive a composite factor score of maternal mental health for use in all infant outcome models. The factor analysis used the total score from the EPDS, STAI trait, and PSS. All variables significantly loaded ($p < 0.001$) on the fully saturated CFA model, indicating strong associations. Fit

	Data loss due to				
	Recruited	Parent declined or infant fussiness	Technical issues	Excessive noise within data	Usable data
Mom ECG	104	8	9	11	76
Infant ECG	104	8	8	4	84
Infant EEG	104	9	6	11	78

Table 4. Data attrition.

	Mean (SD); N (%)
Caregiver age	33 (5)
Caregiver_Ethnicity_Cat	
Hispanic/Latino	37 (50%)
Not Hispanic/Latino	37 (50%)
Unknown	2
Caregiver Race	
American Indian/Alaskan native	2 (2.7%)
Asian	7 (9.5%)
Black/African-American	10 (14%)
Two or more races	29 (39%)
White	26 (35%)
Unknown	2
Relationship status	
Divorced	2 (2.7%)
Living together	11 (15%)
Married	50 (67%)
Single-never married	12 (16%)
Unknown	1
Income-to-needs	5.5 (5.6)
Education (years)	15.7 (3.8)
Gestation	39.25 (1.18)
Infant age (months)	3.48 (0.39)
Infant sex	
Female	26 (34%)
Male	50 (66%)

Table 5. Demographics.

indices suggest excellent fit. (CFI = 1.0, TLI = 1.0, Chi-square = 172.21 $p = 0.00$, RMSEA = 0.00 [90% CI 0.00–0.00], SRMR = 0.00).

EEG data acquisition & processing

Resting EEG data were acquired while infants watched a video of engaging, non-social stimuli (e.g., bubbles, spinning wheel) while seated on their caregivers' laps. The recording room was dimly lit and an experimenter was nearby to soothe the infant with bubbles or a toy if the infant became too fussy. Infants provided between 42 and 600 s ($M = 406$, $SD = 142$) of baseline data. EEG was recorded using a 64-channel HydroCel Geodesic Sensory Net (Electrical Geodesic, Inc., Eugene, OR) and amplifier (Electrical Geodesic, Inc., Eugene, OR; EB NEURO S.p.A., Firenze, Italy). Electrode impedances were kept below 100 K Ω and the sampling rate was recorded at 1000 Hz.

All EEG files were processed in batch using an electroencephalography automated processing platform (BEAPP) software to ensure standardization in data processing and cleaning across all files (See Fig. 5)⁸⁰. Continuous resting EEG files were converted from NetStation format to Matlab format. Data preprocessing was carried out using the Harvard Automated Processing Pipeline for EEG (HAPPE V.1), an automated preprocessing pipeline designed for infant EEG data⁸¹. First, a 1 Hz high-pass and 100 Hz low-pass filter was applied to each EEG recording. Second, the data, which was originally sampled at 1000 Hz was resampled with interpolation to 250 Hz, following guidelines for further HAPPE processing. The third step involved artifact removal and included CleanLine's multitaper approach to removing 60 Hz electrical noise, bad channel rejection, and wavelet-enhanced ICA for artifact rejection with automated component rejection through the Multiple Artifact Rejection Algorithm⁸² in EEGLAB. A subset of spatially distributed electrodes was selected for analysis with MARA: E2, E3, E5, E9, E10, E11, E12, E13, E14, E18, E20, E24, E25, E28, E30, E31, E35, E39, E40, E42, E44, E48, E50, E52, E57, E58, E59, E60 (NetStation Geodesic 64- Channel Net). Bad channels that were initially rejected were repopulated using spherical interpolation to reduce bias in re-referencing and the signal was mean detrended. Finally, each EEG file underwent segmentation into 2-s windows, with each segment subsequently evaluated for residual artifacts. On average, 24 segments were rejected per file ($SD = 10$, range = 0–48). Segment rejection thresholds were determined according to HAPPE's automated rejection criteria⁸¹, which uses amplitude thresholding and assessment of segment likelihood using joint probability calculations. EEG power decomposition was accomplished using Fast Fourier transformation using a multitaper windowing (3 windows) to decompose power into 2-s segments for each channel.

We calculated power in low and high discrete frequency bands, specifically spectral power was computed for Theta (4–6 Hz), Alpha (6–9 Hz), and Beta (13–20 Hz). Relative power at each channel was computed by summing the power within each frequency band, averaging across all segments, and then dividing by the total power

spectrum (2–50 Hz). Two measures of infant neural activity were calculated. First, we computed the theta-beta ratio using whole brain electrodes (See Fig. 4A) to obtain a neural measure indicating the higher abundance of EEG frequencies compared to lower frequencies by dividing relative beta by relative theta. An increased proportion of slow wave (theta) oscillations relative to fast wave oscillations (beta) has shown associations with reduced attentional control during infancy^{83–85}. Furthermore, higher theta-beta ratios during infancy are predictive of lower cognitive control later in childhood^{43,86,87}. We also calculated frontal alpha asymmetry to derive a neural marker of emotion regulation by subtracting log-transformed right frontal hemisphere power values from left frontal hemisphere power values (See Fig. 4B)^{50,88}. Alpha asymmetry during infancy has been correlated with emotion regulation development⁵⁶.

ECG data acquisition & processing

Dyadic ECG data was collected while infants were seated on their mother's laps during infant EEG acquisition using a Physio16 (EGI) device (See Fig. 5). Mothers and infants provided an average of 200 +/- 72 s of resting ECG data (min = 23 s, max = 385 s). Data were edited and processed using the software QRSTool⁸⁹ to remove artifacts and identify heartbeats. R-R intervals were extracted from the processed ECG data. We calculated 3 different indices of heart rate variability that have been associated with autonomic nervous system (ANS) activity^{6,90}.

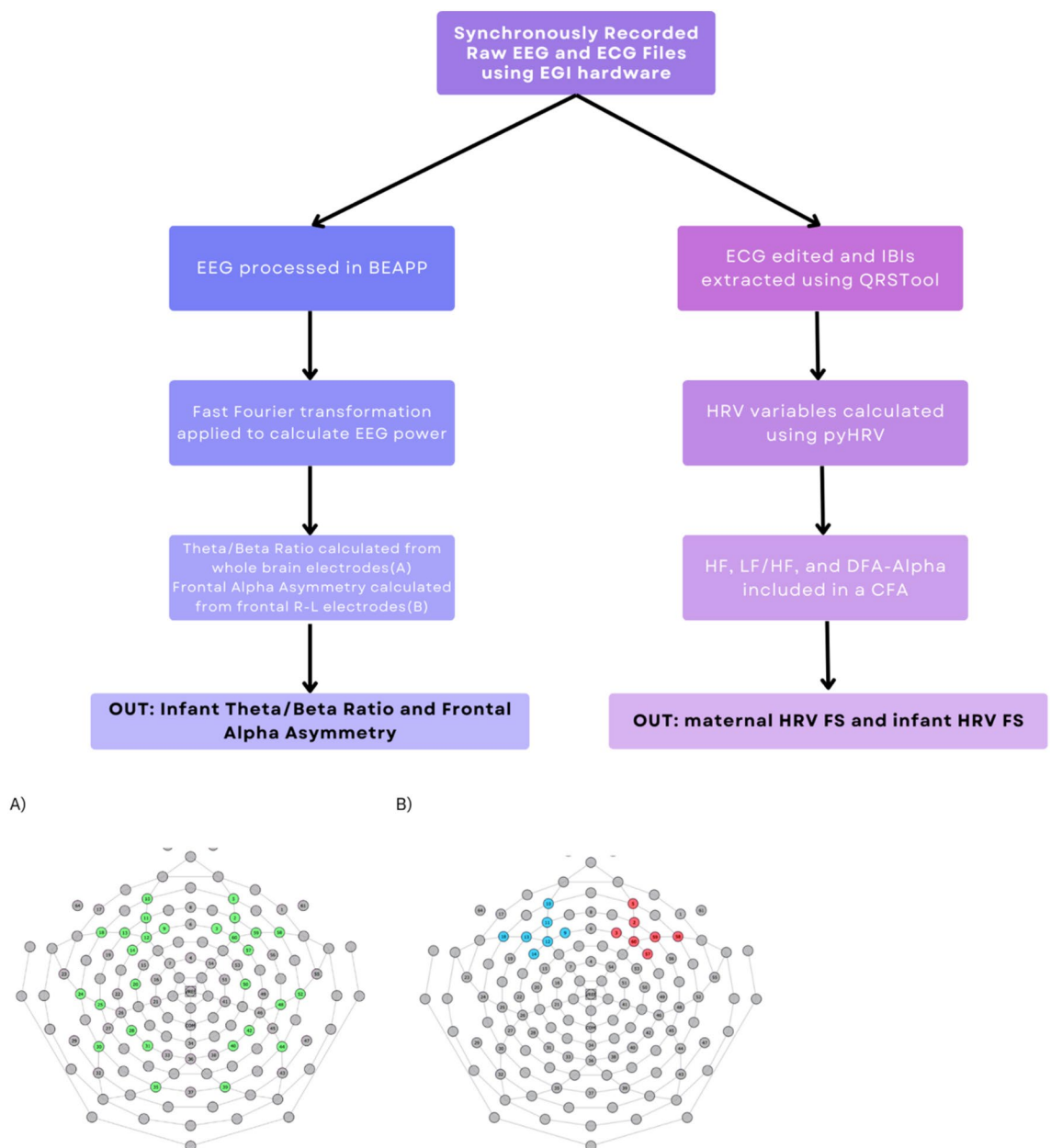


Figure 4. Schematic illustration of the processing pipelines used for ECG and EEG analysis.

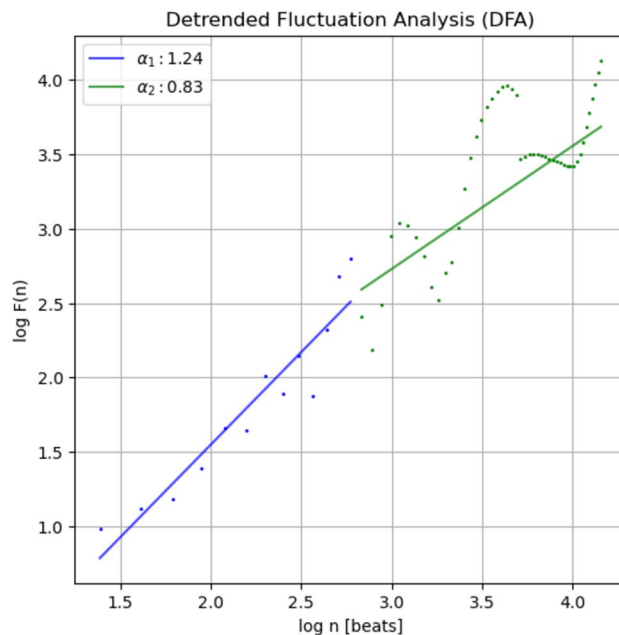


Figure 5. Illustration of a log–log plot depicting the scaling exponent used to calculate DFA alpha 1 from a participant.

All HRV features were calculated with pyHRV⁹¹, a reliable Python signal processing toolbox. Seconds of provided ECG data were not significantly correlated with any of the HRV variables (p 's > 0.21).

Within the frequency domain of the ECG signal, we calculated high-frequency power (HF) and the ratio of low-frequency to high-frequency power (LF/HF) to evaluate the degree to which heart rate is distributed across frequencies^{92,93}. To calculate frequency domain metrics, the RR signal is transformed using fast Fourier transformation to estimate the signal's power spectral density (PSD) using Welch's method. Absolute (sum of power) and log-transformed power was estimated within LF (0.04–0.15 Hz) and HF (0.15–0.40 Hz) discrete frequency bands for mothers and LF (0.04–0.24 Hz) and HF (0.24–1.04 Hz) for infants^{39,92,94,95}. HF power was normalized to the entire power spectrum to correct for skew in the distribution. The LF/HF ratio is calculated by dividing the total absolute power of the LF band by the total absolute power of the HF band and multiplying by 100.

Finally, non-linear quantitative approaches were used to capture the aperiodic dynamics of HRV activity⁹⁶. DFA alpha 1, a scaling exponent, was computed through the following steps:

(1) Integration of the total time series signal; (2) Division of the integrated signal into segments of equal length; (3) Subtraction of a linear least square regression line within each segment to detrend the data; (4) Application of Root-Mean-Square fluctuation analysis to each detrended segment; (5) Repetition of the analysis using varying segment sizes to capture the relationship between fluctuation and time scale For DFA alpha 1, segments ranging from 4 to 16 heartbeats were analyzed (see Fig. 6); (6) Generation of a logarithmic plot to examine this relationship; (7) Fitting a linear regression line to the log–log plot to obtain DFA alpha 1, where the slope represents the scaling exponent. DFA alpha 1 allows us to extract correlations between successive RR intervals over different time scales, offering insights into the underlying physiological mechanisms and regulatory dynamics of the cardiovascular system. Short-term DFA correlations reflect the activity of the baroreceptor reflex⁹⁷. The



Figure 6. CFA of maternal HRV and infant HRV with factor loadings.

baroreceptor reflex is a physiological mechanism that helps regulate blood pressure. In response to changes in blood pressure, the baroreceptor reflex mediates a coordinated response through the ANS. Activation of the baroreceptor reflex leads to inhibition of the sympathetic nervous system and activation of the parasympathetic nervous system.

Using confirmatory factor analysis (CFA), we computed a single latent factor score for heart rate variability (HRV) using the four indices. To assess the goodness of fit of the CFA model, we employed several fit indices, namely the Root Mean Square Error of Approximation (RMSEA), Comparative Fit Index (CFI), Standardized Root Mean Square Residual (SRMR), and Tucker-Lewis Index (TLI). These indices provided valuable information about the adequacy of the model in representing the observed data. All CFA analyses were performed using the Lavaan package in R⁹⁸. Findings indicated that all indicator variables loaded significantly on the HRV factor score ($p < 0.001$), providing support for the validity of the factor structure. The model fit statistics indicated good fit to the data (CFI = 1.00, TLI = 1.00, Chi-square = 186.00 $p = 0.00$, RMSEA = 0.000 [90% CI: 0.00–0.00]; SRMR = 0.000). These results confirm that the four HRV indices were meaningfully associated with the latent HRV factor score. The excellent fit of the model and the significant factor loadings suggest that the HRV factor score provides a comprehensive representation of maternal HRV, encompassing different domains of measurement. We replicated the CFA model using infant HRV indices and found a similar outcome. All indices loaded significantly on the latent HRV factor score and the model fit statistics indicated excellent fit to the data (CFI = 1.00, TLI = 1.00, Chi-square = 230.38 $p = 0.00$, RMSEA = 0.000 [90% CI: 0.00–0.00]; SRMR = 0.000).

Dyadic physiological synchrony analysis

To compute a metric for dyadic physiological synchrony, we calculated HF HRV on 30-s epochs of mother-infant IBI data using the same established pipeline and parameters utilized for the HRV factor score. We chose to calculate HF HRV for the synchrony analysis as HF HRV is associated with Respiratory Sinus Arrhythmia (RSA), which is commonly studied in the context of dyadic physiological synchrony in existing literature^{68,69,99,100}. Physiological synchrony between maternal and infant HRV was assessed for each dyad by computing 0-lag cross-correlations on linearly detrended IBI epochs¹⁰¹ to investigate concurrent changes in parent-infant ANS activity. Only dyads with at least 2 min of usable ECG data were included in the synchrony analysis (5 dyads were removed for having less than 2 min of ECG). Cross-correlations were computed using the tseries package in R¹⁰². The cross-correlation coefficient for each dyad was subsequently utilized as an independent variable in subsequent analysis. Thus, positive cross-correlation coefficients reflect synchrony, whereas coefficients that are 0 or negative reflect a lack of synchrony. Moreover, we refer to higher and lower synchrony values relative to sample averages, where high is +1 SD above the sample average and low is –1 SD below the sample average (Supplementary Table 1).

Data analysis

The overall aim of our analysis is to evaluate the relationship between maternal heart rate variability (HRV) with maternal mental health and infant neurophysiological activity. Our analysis involved several statistical techniques to explore these associations and address our research questions. First, we conducted Pearson correlations to explore the relations between all numeric maternal HRV variables and both maternal and infant outcomes. This analysis allowed us to assess the strength and direction of the correlations between the variables of interest. Next, we constructed multiple regression models to address our primary research questions regarding the relationship between maternal HRV with mental health and infant neurophysiological function at 3 months of age. In the first set of regression models, we included maternal depression, trait anxiety, and perceived stress as dependent variables, while maternal HRV was included as the independent variable. Additionally, we included income-to-needs, maternal age, and maternal resting heart rate (HR), and situational state anxiety as covariates to account for their potential influence. Situational state anxiety was included as a covariate in our first set of models to account for potential short-term experiences of heightened anxiety resulting from testing within an unfamiliar laboratory environment, which could influence HRV readings¹⁰³. In the second set of regression models, we examined the association between maternal HRV factor score with infant HRV factor score and two infant neural outcomes: theta-beta ratio and alpha asymmetry. In the exploratory physiological synchrony analysis, we employed tests of statistical moderation using grand-mean centered mHRV and synchrony scores to compute the interaction term. Significant interactions were probed at one standard deviation below (lower physiological synchrony) and above (higher physiological synchrony) the mean¹⁰⁴. Maternal reported mental health factor scores, maternal resting HR, infant age, and gestational age were included as covariates in all models. Maternal resting HR was included as a covariate in our regression models given the high covariance between HRV and resting HR¹⁶. Statistical mediation tests employed bootstrapping with 5000 samples to generate bias-corrected confidence intervals for indirect effects¹⁰⁵. To increase model parsimony and reduce overfitting, non-significant covariates were removed from the final path analysis.

We screened all regression models for influential outlier data points by examining studentized residuals using the outlierTest function from the car package in R¹⁰⁶. Outliers were identified using a threshold of 3, and the Bonferroni correction method was applied to adjust for multiple comparisons. Only one outlier was identified when maternal anxiety was modeled. This individual was removed from the final model. Full information maximum likelihood (FIML) was used to account for missing data in all analyses, as FIML produces unbiased parameter estimates. All mHRV p -values reported were adjusted for multiple comparisons (3 p -values per family of test) using Benjamini–Hochberg procedure for false discovery rate (FDR) correction¹⁰⁷. All models were run in the R environment.

Data availability

The datasets used and/or analyzed during the current study available from the corresponding author on reasonable request.

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ABA, NHB, and DW wrote the main manuscript text NHB directed the study AH and MZ conducted study procedures and provided manuscript edits All authors reviewed the manuscript.

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