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Prenatal Paternal Stress Predicts Infant Parasympathetic Functioning Above and Beyond Prenatal Maternal Stress

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Abstract

Background: Paternal stress is often assessed by maternal report and is posited to influence infant development indirectly by contributing to a mother's stress and experiences during pregnancy. Far less is known about how direct effects of prenatal paternal stress, as described by fathers themselves, are related to an infant's physiological functioning. We assessed fathers' own experiences of stress and examined its direct impact on infant respiratory sinus arrhythmia (RSA), a biological index of self-regulation, at seven months postpartum.

Method: During the third trimester of pregnancy, the UCLA Life Stress Interview was conducted to assess chronic stress in mothers and fathers ($N = 90$). Infant baseline RSA and RSA reactivity in response to the Still-Face paradigm were assessed at seven months postpartum.

Results: Infants of fathers with high prenatal stress showed lower baseline RSA, possibly reflective of poor infant psychophysiological regulation. The predictive role of paternal stress remained significant after controlling for maternal stress.

Conclusions: Our findings provide emerging empirical evidence to support the influence of prenatal paternal stress on infant RSA, highlighting the important role of fathers for child development.

Keywords

Father; prenatal stress; respiratory sinus arrhythmia; infant

The prenatal period is an exquisitely sensitive stage for children's biobehavioural development (Monk & Hane, 2014; Van den Bergh et al., 2018). Assessments of how maternal prenatal stress affects her own mental health and the offspring's developmental outcomes have thus far dominated the field (Van den Bergh et al., 2020). Far less is known

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about how prenatal *paternal* stress can affect infants' biobehavioural outcomes (Brumberg & Shah, 2020; Monk & Hane, 2014).

Fathers' own experience of stress during the prenatal period, independent of maternal stress, may have a unique contribution to early child development. Elevated prenatal paternal stress may limit fathers' ability to engage fully in quality postnatal care (e.g., Davis et al., 2011), further influencing a range of child outcomes, which could include the infant's physiological regulation. Conducting research on prenatal paternal stress not only advances a more comprehensive understanding of the role of prenatal stress in shaping child biobehavioural development but also extends current research on fathering to the perinatal period. Therefore, the overarching goal of this paper is to assess fathers' reported experience of stress during their partner's pregnancy and examine its associations with infant stress physiology at seven months.

Development of the parasympathetic nervous system (PNS) coincides with the development of infant regulatory abilities (Calkins, 2011; Porges & Furman, 2011) and lays the foundation for more sophisticated emotional and behavioural regulation observed across development (Beauchaine, 2001; Brooker & Buss, 2010; Sulik et al., 2015). One widely studied indicator of PNS functioning is respiratory sinus arrhythmia (RSA) which measures variation in heart rate occurring across the respiratory cycle (Porges, 2007). When external stress is minimal, the vagus nerve exerts its inhibitory effect on cardiac output, leading to high resting RSA. High resting RSA has been associated with restorative processes, effective emotion regulation, and executive functioning (Beauchaine, 2001; Feldman, 2009; Porges, 2007). Conversely, low resting RSA is thought to reflect increased stress vulnerability and has been linked with child internalising and externalising problems (Beauchaine et al., 2007; Skowron et al., 2014).

RSA reactivity in the face of environmental challenges also reflects one's coping and regulatory abilities (Porges, 2007). Modest changes in RSA during stressful tasks is a manifestation of the body's shift from using resources on optimizing the efficiency of the heart to focus on attentional and behavioural responses. Both attenuated and excessive RSA withdrawal (i.e., low and high RSA reactivity), on the other hand, may suggest diminished regulatory ability and has been associated with young children's poor self-regulation and social functioning (Beauchaine, 2015; Liew et al., 2011; Schmitz et al., 2011; Sulik et al., 2015).

Infant RSA may be particularly sensitive to the early caregiving environment (Calkins, 2011; Moore & Calkins, 2004; Propper, 2012). Low warmth and negative parenting are related to a smaller RSA decrease in infants during various laboratory challenge tasks (Calkins et al., 2008; Moore et al., 2009). For example, Conradt and Ablow (2010) assessed 5-month-old infants' resting RSA and RSA reactivity over a stressful social task (i.e., still-face paradigm). They found that infants with highly sensitive and responsive mothers have a faster return to baseline RSA following stress, indicating adaptive physiological regulation. The authors argued that a history of sensitive, responsive caregiver-infant interactions may facilitate infants' capacity to utilize their caregivers for soothing, therefore developing self-regulatory abilities.

No research to our knowledge has examined prenatal paternal stress as a predictor of later infant physiological functioning. Research findings of perinatal paternal mental health may provide support for the plausibility of this association, given that mental health is often considered an important source of prenatal stress (Glover, 2011). For example, a prospective, population-based cohort study documented an association between perinatal paternal depression and 2-month-old babies' excessive crying (Van Den Berg et al., 2009). In another study, higher levels of infant difficulties in a father-infant interaction at three months postpartum were found in fathers with higher levels of postnatal depressive symptoms (Parfitt et al., 2013). These findings suggest that exposure to paternal stress may take a toll on their offspring's emotionality and increase in dysregulated behaviours. Given that emotions and behaviours have biological substrates, perhaps the negative effect of paternal prenatal stress may also extend to infant physiological functioning, indexed by RSA.

Fathers' stress during the prenatal period may influence infant physiological functioning in two different ways. On one hand, a father is thought to contribute to child early development indirectly through his partner's stress or mood (Glover & Capron, 2017). Elevated stress can impede a father's involvement and support for his partner during pregnancy and the early postpartum period which may in turn negatively impact infant functioning (Bergman et al., 2007; Stapleton et al., 2012). On the other hand, fathers may have a unique role in shaping early development that is beyond their role in the prenatal support system (Monk & Hane, 2014). This direct effect of fathers on fetal and infant development may be accounted for by epigenetic mechanisms (Rodgers et al., 2013) or fathers' engagement in early postnatal care (Davis et al., 2011). However, there are few studies to date that have evaluated the unique contribution of prenatal paternal stress above and beyond maternal stress (cf. Van den Berg et al., 2009).

Present Study

This paper has two aims. First, we assess fathers' experience of stress during their partner's pregnancy and examine its association with infant physiological functioning at seven months postpartum. We hypothesize that higher prenatal paternal stress will be related to infants' lower baseline RSA and lower RSA reactivity (i.e., less RSA withdrawal). Second, we explore whether paternal stress is uniquely associated with infant RSA above and beyond maternal prenatal stress, or whether the association is accounted for by maternal stress. Given the exploratory nature of this second aim and limited research on paternal prenatal stress, we do not have an a priori hypothesis.

Method

Participants

Data came from 90 pregnant women and their partners prenatally, as well as their babies at seven months postpartum; participants were drawn from a longitudinal study on intergenerational transmission of emotion dysregulation. Women were recruited during or after their second trimester of pregnancy at OB/GYN clinics and via local advertisement (e.g., flyers). They were assessed once prenatally (age $M = 29$ years, range 18 – 40)

and followed up when infants were 7 months old. All women provided written informed consent before every assessment, and their partners provided informed consent digitally before completing online questionnaires and a telephone interview. Maternal eligibility requirements included: English or Spanish-speaking, 18- to 40-years-old, 25 or more weeks into pregnancy, no pregnancy complications (e.g. pre-eclampsia or gestational diabetes), no pregnancy substance use, anticipated singleton birth, and participating hospital delivery. All study procedures were approved by the university's Institutional Review Board. More details about recruitment, study design, eligibility criteria, and assessment procedures can be found in Lin et al (2019).

One hundred sixty-two women were recruited and participated in the parent study. We also asked for their partners; contact information and were able to conduct a life stress interview with 90 partners. All but one was the baby's biological father. Of these 90 families, we obtained RSA data from 62 infants. Table 1 presents demographic information of this sample.

Procedures

After completing questionnaires online, pregnant women came to the research laboratory on campus during their third trimester (between the 26th and 40th week). They participated in a series of behavioural tasks while their physiological activity was collected. We then administered a series of semi-structured interviews, including the UCLA Life Stress Interview (LSI; Hammen et al., 1987). At the end of the prenatal visit, participants were debriefed, compensated, and were asked for permission to contact their partner. We invited the partner to complete a short version of the LSI over the phone and questionnaires online. Prenatal maternal and paternal stress measured via the LSI were used in the present study.

When infants were 7 months old, they and their mothers came into the laboratory to participate in the still-face paradigm and a free-play assessment. Experimenters attached heart rate and respiration monitoring equipment on both mothers and infants. The dyads then watched a 2-minute Baby Einstein video while the infant sat in their mother's lap. Then, infants were placed in a high chair and the experimenters introduced the still-face paradigm (SFP; Tronick et al., 1978). After the normal play episode (2 minutes), mothers were asked to turn their face away for a moment, and then turn back to their infant with a neutral expression for 2 minutes. Mothers were then instructed to look away again for a moment before returning to typical interactions with their infants while remaining seated for another two minutes. The procedure was stopped if the infant became too upset or if the mother requested to stop the procedure.

Measures

UCLA Life Stress Interview (LSI).—Pregnant women's chronic stress was measured by a semi-structured interview– the UCLA LSI (Hammen et al., 1987). Trained interviewers evaluated multiple domains of the participants' lives (i.e., close friendships, partner relationship, co-parenting with baby's father, dating, family relationships with mother, father, and sibling, finances, work status, neighborhood environment, school, and health) in the past six months on a 5-point Likert scale. A score of 1 was indicative of low stress,

whereas a score of 5 reflected high stress. Ratings from all domains were averaged to create a chronic stress score for each participant in the past 6 months.

A shortened version of the LSI (i.e., dating, relationship with sibling, finances, neighborhood, and health domains were removed) was administered to participating fathers via telephone call. The assessed life domains for fathers included close relationships, partner relationship, co-parenting with baby's mother, family relationships with their mother and father, work, and school.

Fathers' Depressive Symptoms.—Fathers completed the Center for Epidemiologic Studies Depression scale (CESD; Radloff, 1977) to report their levels of depressive symptoms prenatally via online questionnaires. The CESD is a 20-item self-report measure assessing various feelings and situations related to depression (e.g., "I felt sad", "I felt lonely", etc.). Participants reported the frequency of these feelings on a Likert-type scale ranging from 0 (*Rarely or none of the time*) to 3 (*Most or all of the time*). A total score is calculated by summing all items (range 0–60). Internal consistency of the CESD in the current sample is .84.

Infant RSA at Seven Months Postpartum.—Baseline RSA was assessed using MindWare equipment and scoring procedures (BioLab software version 3.1). Wireless MindWare mobile devices sampled at 500 Hz (MindWare Technologies Ltd., Gahanna, Ohio, USA). Electrocardiograph data were recorded using a three-lead spot electrode configuration with the negative lead on the baby's right clavicle, the positive lead on the left side of the baby's stomach, approximately under the left rib, and the ground lead on the bottom right stomach, approximately under the right rib. RSA was scored in 30-second epochs by trained research assistants using MindWare's heart rate variability analysis software (version 3.1). This software flagged R peaks within each QRS complex and identified whether the inter-beat intervals were within the expected deviation given surrounding data and expected ranges for an inter-beat interval series. Flagged data points were reviewed by research assistants and corrected when necessary (i.e., misidentified R peak). The heart period time series was then detrended and a Fast Fourier Transformation was used to identify high-frequency variation between .24 and 1.04 Hz (Bar-Haim et al., 2000). Once data were initially cleaned, they were double-checked by a senior investigator, if necessary. Impedance cardiography data were used to extract the respiration signal. These electrodes were placed with the positive current source behind the left ear, the negative current source along the spine immediately below the ribs, the positive sensor on the left sternoclavicular joint, and the negative sensor one inch above the xyphoid process. These data were detrended and used to examine respiration rate and ensure that it fit within the specified frequency for RSA. Epochs with any missing or unusable data were removed, including any RSA values outside the expected range of 1 – 10.

The RSA baseline score was computed by averaging RSA scores across each of the epochs from the Baby Einstein video. Infant RSA reactivity was computed by subtracting the average RSA during the baseline from the average RSA during the still-face episode. Positive difference scores reflect increases in RSA from the baseline to the still-face episode, and negative difference scores reflect decreases in RSA from baseline.

Attrition and Missing Data.

Among the 90 families in which fathers completed the LSI, 62 infants returned to the laboratory at seven months postpartum and provided physiological data. Families with complete physiological data and those with missing data did not differ on demographic variables, including maternal age and education, maternal, paternal, and infant race and ethnicity, or marital status. Results from Little's MCAR test supported that data in this sample were missing at random, as indicated by a nonsignificant chi-square value (χ^2 = 21.51, df = 16, p = .16). Missing values (of the 90 families) were imputed using the MICE package in R and results were pooled from 40 imputations using Rubin's rule (Van Buuren & Groothuis-Oudshoorn, 2011).

Results

Preliminary Analyses.

We first examined the data for normality and outliers (≥ 3 SD from the mean) prior to primary analyses. All variables appeared normally distributed and no outliers were identified. We then evaluated the study variables for demographic effects to determine whether covariates would be needed. The examined demographic variables included maternal age at delivery, household income, paternal, maternal, and infant race, marital status, gestational age at birth, infant birth weight, infant age at the postnatal visit, and infant sex. No associations were significant between these demographic variables and paternal prenatal stress, or infant baseline RSA or reactivity (p_s > .07). Given the increasing body of literature documenting the association between perinatal paternal depression and infant outcomes, we tested whether paternal depressive symptoms during the prenatal period would be associated with paternal stress. Results showed that fathers with more depressive symptoms reported higher levels of stress (r = .25, p < .05); therefore, paternal depressive symptoms were included in later analyses as a control variable. Table 2 includes the descriptive statistics and bivariate correlations among all study variables and covariates. High paternal stress was related to high maternal stress (r = .38, p < .001) as well as lower infant baseline RSA at seven months postpartum (r = − .33, p < .05).

Associations between Paternal Prenatal Stress and Infant Physiological Functioning

Prior to performing each regression analysis, residual scores were examined to ensure conformity with assumptions of multiple linear regression (i.e., normality, linearity, and homogeneity of variance). Two multiple regression models were examined for infant physiological functioning (i.e., baseline RSA and RSA reactivity) at seven months with prenatal paternal depressive symptoms included as a control variable. The model with baseline RSA as the outcome variable (Model 1) was significant, F(2,87) = 5.87, p < .001, R^2 = .122. As shown in Table 3, prenatal paternal stress predicted infant baseline RSA at seven months while controlling for prenatal paternal depressive symptoms. For infants whose fathers were at the same depression level, high prenatal paternal stress was associated with low infant baseline RSA (b = − .70, SE = .28, p = .017). The model with RSA reactivity as the outcome variable (Model 2) was not significant.

We examined whether maternal stress mediated the association between paternal stress and infant parasympathetic functioning. Given that paternal stress was not significantly related to 7-month infant RSA reactivity, we only tested a mediation model with infant baseline RSA as the outcome variable (see Table 3, Model 3). The indirect effect was not significant; therefore, the mediation model was not supported. However, prenatal paternal stress remained a significant predictor of infant baseline RSA levels, even after controlling for maternal stress and paternal depressive symptoms ($b = -.80$, $SE = .30$, $p = .010$).

Discussion

Despite the substantial evidence supporting the effect of maternal stress on early child development (Bergman et al., 2007; Tarabulsy et al., 2014; Van den Bergh et al., 2020), very few studies have explicitly tested the effect of paternal stress on infant physiological functioning. Our study shows that infants of fathers with high prenatal stress showed lower baseline RSA. This result is consistent with the literature documenting associations between greater maternal prenatal adversity and infants' lower baseline RSA (Field et al 2003; Jansson et al., 2010; Jones et al 1998; Ponirakis et al., 1998). Low baseline RSA, reflecting reduced parasympathetic control and infants' potentially adaptive engagement with the environment (Beauchaine, 2001), is considered an indicator of poor regulatory ability. For example, lower baseline RSA has been found to relate to more negative affect and greater arousal in infants (Moore et al., 2009) and more internalising and externalising problems in children and adolescents (Beauchaine et al., 2007; Graziano & Derefinko, 2013; Skowron et al., 2014).

Our findings are also consistent with the broader literature where paternal depression is included in the conceptualization of stress and infants' behavioural outcomes are considered. In fact, a number of studies have shown that prenatal paternal depression has a long reach on children's development (Parfitt et al., 2013; Van Den Berg et al., 2009). For example, Letourneau and colleagues (2019) found that fathers' perinatal depressive symptoms were related to children's greater emotional reactivity and more behavioural withdrawal at 2- or 3-years old. However, it is worth noting that the effect of paternal depressive symptoms on a child's internalising problems in Letourneau and colleagues' (2019) study was only evident when the mothers were also symptomatic. In other words, the effects of paternal and maternal prenatal adversity on children's early development were additive.

Results from the current study, however, suggest an independent effect of paternal prenatal stress. That is, the association between prenatal paternal stress and infant baseline RSA at seven months remained significant even after controlling for maternal stress. Moreover, mediation analysis showed that maternal stress did not account for the association between prenatal paternal stress and 7-month-old infant baseline RSA. These findings are novel in that little research has simultaneously assessed prenatal maternal and paternal effects, making it difficult to evaluate whether paternal effects are unique from maternal effects. Most extant literature seems to implicitly assume that fathers' prenatal contribution to infant development is made indirectly through the mother's stress and mood (Glover & Capron, 2017; Goodman & Gotlib, 1999). For example, the benefits of paternal involvement during pregnancy (e.g., Martin et al., 2007) and partner support (e.g., Stapleton et al.,

2012) on maternal and child functioning are widely documented and highlighted. However, in contrast to the commonly held belief that paternal adversity affects child development indirectly through mothers, we found that paternal prenatal stress had a unique effect on infant baseline RSA and that this paternal effect was not accounted for by elevated maternal stress. These findings are consistent with the literature on fathers and fathering in other developmental stages (i.e., toddlerhood, childhood, and adolescence). This research suggests that applying a mother-centered framework to understanding fathers may fail to capture the unique contributions of fathers to child development (Cabrera & Volling, 2019).

Some substantial epigenetic evidence is available from animal studies to support the associations between adverse pre-conception paternal environment and increased risk of offspring neurophysiological problems (Chan et al., 2018; Rodgers et al., 2013; Yuan et al., 2016). Although retrospective assessment of prenatal stress in the current study captured six months of pregnancy, it may be directly related to fathers' general stress level before and/or at conception. Therefore, our finding of the significant association between paternal prenatal stress and infant baseline RSA could also indicate a genetic or epigenetic programming effect of paternal stress on infant physiological systems. It should be noted that fathers' chronic stress, rather than episodic stress, measured by the LSI was used in our study. Prolonged high levels of chronic stress may have different influence on infant physiological functioning than sporadic, yet significant acute life stressors. The extent to which the number of acute stressors or the severity of episodic life events are associated with infant RSA awaits future research.

Contrary to our hypothesis, paternal prenatal stress was not related to infant RSA reactivity in our study. It could be that infant RSA reactivity was assessed while interacting with the mother, but not the father, during the SFP. Although the SFP is sensitive to the mother-infant relationship, it may not reflect variations in fathers' interactions with the infant. As a result, no significant association was observed between paternal stress and infant RSA reactivity. Moreover, it is important to note that infant RSA reactivity in our study is assessed in the laboratory setting. As a context-specific assessment, infant RSA reactivity in the present study may not be comparable to the reactivity in a stressful father-infant interaction at home.

Our study has several limitations. First, without assessing paternal postnatal stress, we could not evaluate whether fathers' prenatal stress level would predict infant baseline RSA above and beyond their postnatal stress, especially considering the chronicity of paternal stress during the perinatal period (Vismara et al., 2016). As a proxy of an infant's postnatal caregiving environment, maternal everyday stress and parenting stress were tested post hoc for their associations with infant baseline RSA. However, neither of the associations were significant ($p > .17$). Future research that includes measures of paternal postnatal stress and caregiving quality are needed to directly address the question: what is the unique role of paternal prenatal, as compared with postnatal, stress in shaping infant RSA? Relatedly, the lack of measures of postnatal paternal caregiving prevented us from testing postnatal mechanisms that might account for the association between prenatal paternal stress and infant psychophysiology.

Second, although our sample is comparable with or even larger than that of many studies with infant RSA (e.g., Conradt & Ablow, 2010; Moore & Calkins, 2004; Richardson et al., 2019), a larger sample size is desirable for greater statistical power. Third and relatedly, our sample size precluded us from examining whether our findings would differ by infant sex. Bivariate correlations showed that infant sex was not associated with any of the study variables in our study. However, there is increasing evidence to show that female and male infants may be differentially susceptible to prenatal adversities (Dipietro & Voegtline, 2017). Therefore, it is important for future research with larger sample sizes to examine whether the effect of prenatal paternal stress on infant physiology would be different for female and male infants. Lastly, even though the final sample didn't differ from the overall sample on key variables, attrition may have biased our results, and findings from this study may not generalize to other mother-father-infant triads.

This study has a number of important strengths. First, we assessed paternal prenatal stress beyond common mental health symptoms (e.g., depression and anxiety). Additionally, the inclusion of both maternal and paternal stress provides an opportunity to investigate whether paternal effects are unique from maternal effects. More importantly, this study is among the first to investigate the effect of paternal prenatal stress on later infant physiological functioning, particularly parasympathetic reactivity and regulation as indexed by infant RSA. Parasympathetic arousal, as well as the hypothalamic-pituitary-adrenal axis, has been increasingly acknowledged as an important avenue of intergenerational transmission of stress (Rash et al., 2016; Van den Bergh et al., 2020). More studies that examine the prenatal effect of stress on infant PNS functioning are needed to further our understanding of mechanisms underlying prenatal programming.

In conclusion, results of the current study suggest a unique influence of paternal prenatal stress on infant PNS functioning. Infants of fathers with higher prenatal stress showed lower resting RSA. Our findings provide a first foray into understanding the unique role of paternal prenatal stress in shaping early child development. With fathers' increasing involvement in pregnancy and childhood in recent years (Bianchi et al., 2006; Garfield, 2015), it is imperative to investigate fathers' role in the perinatal period. Supporting fathers during this critical period and reducing paternal stress may serve as a means of minimizing the impact of prenatal adversity on child development and could facilitate long-term adaptive outcomes.

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Data Availability Statement:

Part of the data (i.e., infant physiological measure) that supports the findings of the study will be available on the National Institute of Mental Health (NIMH) Data Archive (https://nda.nih.gov/edit_collection.html?id=3240). Other parts of the data are available from the corresponding author upon reasonable request.

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Table 1

Demographic Information of the Study Sample (N = 90)

	n **(%)**	**Mean (*SD*)**	**Range**
Infant Characteristics			
Gestational age at time of delivery (days)	87 (96.7)	275.6 (8.4)	240–289
Infant age at 7-month laboratory visit (days)	61 (67.8)	194.62 (22.2)	156–275
Sex (male)	45 (50.1)		
Race			
Asian, American Indian, or Alaskan Native	3 (3.3)		
White/Caucasian	66 (73.4)		
Black/African American	1 (1.1)		
Multiracial	19 (21.2)		
Ethnicity			
Hispanic/Latino	21 (23.4)		
Paternal Characteristics			
Annual Household Income			
Less than $19,999	10 (10.2)		
$20,000 – $29,999	3 (3.3)		
$30,000 – $39,999	7 (7.8)		
$40,000 – $49,999	6 (6.7)		
$50,000 – $79,999	32 (35.6)		
$80,000 – $99,999	9 (10.0)		
$100,000 and greater	16 (17.8)		
Relationship status (partnered)			
Married	76 (84.4)		
Not married but live-in with the mother	11 (12.2)		
Not married and not live with the mother	3 (3.3)		
Race			
Asian or Pacific Islander	2 (2.2)		
Black/African American	1 (1.1)		
White/Caucasian	58 (64.4)		
Other	7 (7.8)		
Multiracial	3 (3.3)		
Ethnicity			
Hispanic/Latina	10 (11.1)		

Table 2

Descriptive Statistics and Bivariate Correlations Among the Study Variables

	1	2	3	4	M (*SD*)
1. Paternal stress	–				2.20 (.48)
2. Maternal Stress	.38***	–			2.36 (.41)
3. Paternal CES-D[a]	.25*	.21	–		1.65 (.85)
4. 7-month baseline RSA	− .33*	− .01	−.04	–	3.68 (1.00)
5. 7-month RSA reactivity	.10	−. 00	.17	− .27*	− .38 (1.03)

* $p < .05$,

** $p < .01$,

*** $p < .001$

a. CES-D scores are log-transformed

Note. Mean, standard deviation (SD), and bivariate correlations among the study variables are pooled estimates from 40 imputed datasets.

Table 3.

Summary of Multiple Regression Analyses Using Paternal Prenatal Stress Predicting Infant Baseline RSA and RSA Reactivity

	Model 1: 7-month Baseline RSA		Model 2: 7-month RSA reactivity		Model 3: Mediation 7-month Baseline RSA	
	***b* (*SE*)**	***p***	***b* (*SE*)**	***p***	***b* (*SE*)**	***p***
Paternal CESD	.06 (.18)	.737	.19 (.18)	.117	.04 (.18)	.822
Paternal stress	−.70 (.28)	.017	.14 (.27)	.603	−.80 (.30)	.010
Maternal stress					.33 (.35)	.361
R^2 [95% CI]	.122 [.007, .322]		.039 [.006, .196]		.145 [.015, .348]	

Note. b, unstandardized coefficient; *SE*, standard error