Respiratory Sinus Arrhythmia Moderates the Impact of Maternal Prenatal Anxiety on Infant Negative Affectivity

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Running head: PRENATAL ANXIETY, RSA, AND INFANT TEMPERAMENT

Abstract

Maternal prenatal anxiety is associated with infants' temperamental negative affectivity (NA), but it is unclear to what extent children vary in their susceptibility to prenatal influences. We tested a hypothesis that infants' respiratory sinus arrhythmia (RSA), an index of parasympathetic vagal tone and a potential marker of differential susceptibility to environmental influences, moderates the effects of maternal prenatal anxiety on the development of infant NA. Prenatal anxiety was assessed during the last trimester of pregnancy in a low-risk community sample. Infant NA, baseline RSA, and maternal postnatal anxiety were assessed at 8-10 months of infant age. Regression analyses were performed to predict infant NA on the basis of prenatal anxiety, infant baseline RSA, and their interaction (N = 173). Maternal prenatal anxiety and infant RSA interactively predicted infant NA at 8-10 months. Among infants with high RSA, a significant positive association between prenatal anxiety and infant NA was observed, whereas prenatal anxiety did not predict infant NA among infants with low RSA. Vagal tone, as indexed by baseline RSA, may provide a promising marker of differential susceptibility to the long-term effects of varying intrauterine conditions.

Keywords: Prenatal anxiety; infant; temperament; respiratory sinus arrhythmia; differential susceptibility

The developmental origins of phenotypic variation in childhood emotionality can be traced back to as early as the fetal period. The fetal programming hypothesis postulates that during pregnancy, intrauterine conditions related to the health, nutrition, and hormonal transmission of the mother shape the development of the fetus to promote adaptation to the expected conditions of the postnatal environment (Barker, 2003; Glover, 2011). From this perspective, several prospective studies have investigated the effects of maternal stress and anxiety during pregnancy on children's emotional development and psychopathology. There is robust evidence of an association between maternal prenatal stress or anxiety and heightened temperamental negative affectivity in infancy (i.e., frequent expressions of crying and emotional distress to limitations and novel situations; Austin, Hadzi-Pavlovic, Leader, Saint, & Parker, 2005; Bergman, Sarkar, O'Connor, Modi, & Glover, 2007; Davis et al., 2004; Huizink, Medina, Mulder, Visser, & Buitelaar, 2002; Werner et al., 2007) and problem behaviors in later childhood (Gutteling et al., 2005; O'Connor, Heron, Golding, Glover, & the ALSPAC, 2003). Such effects have been shown to hold even when the influence of postnatal maternal stress/anxiety has been taken into account, (e.g., Austin et al., 2005; O'Connor et al., 2003) suggesting a crucial role for prenatal factors in programming the early emergence of phenotypic variation in emotionality. Although heightened negative affectivity may increase the likelihood of later behavior problems, particularly when coupled with low regulatory capacity (Eisenberg et al., 2000), negative affectivity as such is not a maladaptive temperament trait in infancy. Indeed, there is evidence for an important role of temperamental negative affect in conferring susceptibility to environmental influences. Studies testing the *differential susceptibility* model (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011) have shown that infants with heightened negative affectivity are more susceptible than infants with low negative affectivity to both negative and positive environmental influences. Pluess

and Belsky (2009), for example, observed children with high negative affectivity in infancy to exhibit more behavior problems in the context of low-quality childcare, but also fewer problems in the context of high-quality care, as compared to children with low negative affectivity for whom childcare quality had no effect. Therefore, given the role of temperament in potentially moderating the effects of childhood environment on developmental outcomes, understanding the predictors of temperamental negative affectivity in infancy is important.

Relatedly, an important question is whether children vary in their susceptibility to the influence of maternal prenatal factors on postnatal outcomes. In line with the differential susceptibility model, some children may possess biological characteristics that increase developmental plasticity and render such children more susceptible to both risks and advantages provided by varying conditions of the intrauterine environment. Initial studies have provided somewhat mixed evidence of whether genetic variations putatively related to susceptibility to the environment moderate the association between maternal prenatal anxiety and child temperamental negative affectivity. Pluess and colleagues (2011) showed that in a low-risk sample, elevated maternal prenatal anxiety predicted increased temperamental negative affectivity in infants who carried a short allele of the serotonin transporter polymorphism 5-HTTLPR but not in those with two long alleles. No clear evidence of differential susceptibility was found at lower levels of the predictor variable, i.e., significantly lower negative affectivity in the absence of maternal anxiety in short allele carriers. Another large population-based cohort study did not, however, find any evidence of moderation of prenatal effects by the same serotonin transporter genotype (Braithwaite et al., 2013).

While there is mixed evidence of moderation of the prenatal effects on the level of genetic variations, little is known about other biological mechanisms (e.g., physiological or neural functioning) that could provide markers of biological sensitivity to the influence of

maternal prenatal factors. In the present study, we investigated whether infants' cardiac vagal tone, measured as respiratory sinus arrhythmia (RSA), influences the strength of the association between maternal prenatal anxiety and infant temperamental negative affectivity. RSA is the fluctuation in heart rate related to respiratory cycles and it is considered to indicate parasympathetic control of the heart and the sympathetic nervous system (Berntson, Cacioppo, & Quigley, 1993; Porges, 2007). In line with the view of RSA as a "brake" on sympathetic arousal (Porges, 2007), higher baseline RSA (i.e., greater high-frequency heart rate variability during minimal or neutral external stimuli) has been associated with various regulatory functions such as better emotion regulation and executive functioning (Feldman, 2009; Marcovitch et al., 2010; Suess, Porges, & Plude, 1994). In addition, RSA has also been related to physiological and emotional reactivity, with infants higher in RSA shown to be more emotionally reactive (Stifter & Fox, 1990; Stifter & Jain, 1996). As a consequence of higher reactivity, infants with higher RSA may be more likely affected by variations in environmental input (cf. Beauchaine, 2001), which may lead to negative or positive developmental outcomes depending on the quality of the environment. Indeed, recent findings have provided evidence that high RSA is a marker of differential susceptibility to the postnatal environment from very early on in development. Conradt, Measelle, and Ablow (2013) showed, in line with the differential susceptibility model, that in a sample of infants reared in poverty, those with high baseline RSA and a secure attachment relationship (i.e., a proxy for sensitive caregiving environment) expressed the least amount of problem behaviors at 17 months, whereas those with high baseline RSA and insecure (disorganized) attachment showed the highest amount of problem behaviors. Attachment quality was unrelated to the amount of problem behaviors in infants with low baseline RSA. Further support for the role of high RSA as a differential susceptibility factor comes from studies showing that RSA moderates – in a "for better and for worse" manner – the impact of family environment on the

development of externalizing problems and prosocial behavior (Eisenberg et al., 2012; Obradović, Bush, Stamperdahl, Adler, & Boyce, 2010).

The aim of this study, therefore, was to determine whether the effect of maternal prenatal anxiety on infant temperamental negative affectivity is moderated by infants' RSA, a putative physiological marker of sensitivity to environmental influences, in a low-risk community sample. Postnatal maternal anxiety was also taken into account to control for its influence. Infant temperament and RSA, and maternal postnatal anxiety were assessed at 8-10 months of age, as there are notable increases in the expressions of negative affect as infants transition to the second half of the first year (e.g., Braungart-Rieker, Hill-Soderlund, & Karrass, 2010). Although the postnatal assessment of RSA does not reveal whether the same pattern of individual differences in RSA levels extend to the prenatal period, the present findings will reveal whether postnatal RSA is influenced by the maternal anxiety measures and, importantly, whether the effects of maternal anxiety on infant negative affectivity are similar for all infants or contingent on infant RSA. Predictions regarding the direction of the moderation effects were based on recent studies showing that high RSA may act as a differential susceptibility factor to environmental influences (Conradt et al., 2013; Eisenberg et al., 2012; Obradović et al., 2010). Therefore, we predicted that high levels of baseline RSA are related to a) greater infant negative affectivity in the context of elevated maternal prenatal anxiety as compared to infants with low RSA, b) relatively lower infant negative affectivity when maternal prenatal anxiety was low, and c) that the association between maternal prenatal anxiety and infant negative affectivity is weaker or absent in infants with low levels of baseline RSA.

Method

Participants

The final sample consisted of 173 infants who visited the laboratory at approximately 8-10 months of age (mean age = 265 days; *SD* = 23.20; 54% females). The infants who were invited to the laboratory assessments were a randomly selected subsample of the CHILD-SLEEP birth cohort, a prospective longitudinal study (*N* = 1667) investigating the development of normative sleep and sleep-related disorders in infancy and early childhood (Paavonen et al., under review). All infants who visited the laboratory were healthy and full-term (≥ 37 weeks), and predominantly from urban, middle-class Caucasian families. An additional 40 infants were tested but excluded from the analyses due to excessive artefacts in their heart rate data (*n* = 23) or technical difficulties during heart rate recording (*n* = 17). Ethical approval for the study was obtained from the Ethics Committee of the Pirkanmaa Hospital District and informed written consents were given by the parent for participation in the longitudinal study during the last trimester of pregnancy and separately for the experimental procedures upon arrival to the laboratory.

Measures

Maternal anxiety. Mothers filled an abbreviated version (Bieling, Antony, & Swinson, 1998) of the trait scale of the State-Trait Anxiety Inventory (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) prenatally during the last trimester of pregnancy (average gestational week = 34.57, *SD* = 2.65) and postnatally with a set of questionnaires sent at home when the infants were 8 months of age. The 6 items selected from the original questionnaire have been shown to most strongly load on a factor assessing anxiety and worry (Bieling et al., 1998). Responses were given on a 4-point scale ranging from 1 (almost never) to 4 (almost always). The average sum scores of the prenatal and postnatal anxiety measures were 8.84 (*SD* = 2.32; range = 6-18; *N* = 173) and 9.06 (*SD* = 2.55; range = 6-18; *N* = 158), respectively. The internal consistencies for both measures were α = .78.

RSA. During the laboratory assessment, electrocardiography (ECG) was recorded while the infants were looking at a silent 3-min video of a female model assembling a tower of blocks. The video was presented on a 19-inch computer monitor that was surrounded by a black frame to minimize distractions. In the video, the model bore a neutral expression and did not make eye contact. Infants were seated on the parent's lap (in 95% of assessments on the mother's lap) during the recording. The parent was instructed to refrain from speaking unless it was necessary to soothe the infant. ECG was recorded bipolarly with two pre-gelled Ag/AgCl electrodes attached to the midpoint between the collarbone and the nipple on both the left and right chest, and grounded by a third electrode attached above the navel. The ECG data were stored on a computer disk at the sample rate of 1000 Hz using a QuickAmp amplifier and BrainVision Recorder software (Brain Products GmbH, Gilching, Germany). Offline, the data were processed with an in-house, Matlab-based software to identify QRS complexes in the ECG signal and to measure the time between two successive R-peaks (i.e., interbeat intervals, IBIs). After an algorithm-based detection of R-peaks, the data were manually corrected, when necessary, for falsely detected and missing peaks. Files with motion artefacts that required considerable manual editing (i.e., more than 5% of the length of the recording) were excluded. To calculate RSA as an index of vagal tone, the IBI series were first converted to a time-series sampled at 10 Hz by interpolation and filtered using a 241-point optimal finite impulse response digital bandpass filter with a frequency band from .24 to 1.04 Hz, which is the typical frequency band in infant RSA analysis (Bar-Haim, Marshall, & Fox, 2000). RSA was then calculated as the natural logarithm of the variance of the filtered signal. The average RSA for the infants included in the analyses was 3.75 (*SD* = 0.85; range = 1.93-6.88).

Infant negative affectivity. At 8 months of age ($N = 160$), temperament was assessed with the short form of the Infant Behavior Questionnaire-Revised (IBQ-R; Gartstein &

Rothbart, 2003; Putnam, Helbig, Gartstein, Rothbart, & Leerkes, 2014), a widely used parent-report measure of different facets of infant temperament. The 91-item questionnaire measures the occurrence of different infant behaviors within the past two weeks on a scale from 1 (never) to 7 (always). From the IBQ-R, the composite Negative Affectivity (NA) scale was used as the dependent variable in the analyses. NA consisted of the subscales Sadness, Distress to Limitations, Fear, and Falling Reactivity (reverse-scored). The internal consistency of the NA scale was $\alpha = .73$, and the alphas for the subscales ranged from .72 to .83. The average NA for the infants included in the analyses was 3.03 ($SD = 0.73$; range = 1.60-5.25).

Covariates. Educational level (highest completed education on a 5-point scale), monthly net income of the mother, age, alcohol consumption during pregnancy (AUDIT-C; Bush, Kivlahan, McDonell, Fihn, & Bradley, 1998), and postnatal anxiety (at 8 months) were measured with questionnaires and used as mother-related covariates in the analyses. Smoking was not included because none of the mothers included in the analysis reported smoking in the prenatal questionnaire. In initial analyses, prenatal and postnatal depression scores (assessed with the CES-D scale; Radloff, 1977) were also taken into account as covariates. As they did not affect the main outcome of the results, however, they were left out of the final model. From the infant variables, sex and gestational age at birth were used as covariates.

Statistical Analyses

The unadjusted bivariate associations between the continuous variables included in the analyses were first explored with Pearson correlations. For the main question of this study, hierarchical linear regression was used to test the independent and interactive influences of maternal prenatal anxiety and infant RSA in predicting infant negative affectivity. The standardized predictor variables were entered in three steps: the covariates were entered on Step 1, followed by the main effect model including maternal prenatal

anxiety and infant RSA on Step 2, and finally on Step 3 the interaction term of maternal prenatal anxiety and infant RSA was entered. In case of a significant interaction, potential presence of differential susceptibility to the effects of prenatal anxiety was first evaluated by inspecting the form of the interaction plot, i.e., whether there was a crossover interaction suggestive of differential susceptibility (Belsky et al., 2007). More formal evaluation of differential susceptibility was then carried out by analyzing the *regions of significance*, i.e., estimating the standard deviation (*SD*) values of the predictor variable below which and above which the regression lines of the moderator variable begin to differ with respect to the outcome (Bakermans-Kranenburg & van IJzendoorn, 2015; Preacher, Curran, & Bauer, 2006; Roisman et al., 2012). Differential susceptibility is implied when the estimated *SD* values fall within ±2 *SD* of the mean of the predictor (Bakermans-Kranenburg & van IJzendoorn, 2015; Roisman et al., 2012). In addition to the regions of significance analysis, two complementary indicators for evaluating the applicability of the differential susceptibility model were derived (Roisman et al., 2012). *Proportion of interaction* (PoI) provides a metric to evaluate the range of the interaction effects in the data by calculating the proportion of the total interaction that is represented on the right side of the crossover point. In a prototypical differential susceptibility model, this value will be closer to 0.50 rather than extreme values (i.e., 0 or 1, which would indicate that the interaction can only account for either increasing or decreasing values of the predictor). *Proportion affected* (PA), on the other hand, is an estimate of the proportion of cases in the data that are differentially affected by the moderator (i.e., the proportion of cases above the crossover point). Again, support for differential susceptibility is obtained if the value is closer to 0.50 rather than extreme values. Missing data occurred for a maximum of 2.9% for the variables from the prenatal questionnaire and 8.7% for the variables from the 8-month questionnaire. Missing data values were estimated with multiple imputation implemented in IBM SPSS Statistics 21. The pattern of results of the regression

analysis with the original dataset were replicated across five imputed datasets and, therefore, here we report the statistics averaged across the imputed datasets.

Results

The correlations between the continuous study variables are listed in Table 1. Similar to what was observed in the full longitudinal dataset ($r = .17$, $p < .0001$, $N = 1279$), maternal prenatal anxiety was positively correlated with infant NA ($r = .23$, $p < .05$). The results of the regression analysis are depicted in Table 2. In the multivariate regression models, prenatal ($p = .10$) and postnatal anxiety ($p = .07$) were marginally related to infant NA, and girls ($p = .04$) were generally reported to be higher in NA (cf. Gartstein & Rothbart, 2003; Putnam, Gartstein, & Rothbart, 2006). Most importantly, the interaction between RSA and maternal prenatal anxiety in predicting infant NA was significant ($\beta = .19$, $t = 2.34$ $p = .02$). Simple slopes (Aiken & West, 1991) were then estimated to probe the nature of this interaction. As can be observed from Figure 1, at higher level of RSA (+1 *SD* = 4.60), maternal prenatal anxiety significantly predicted infant NA ($\beta = .44$, $t = 3.56$, $p = .001$), whereas at lower level of RSA (-1 *SD* = 2.89) no association between maternal prenatal anxiety and infant NA was observed ($\beta = .05$, $t = .46$, $p = .65$).

TABLE 1, TABLE 2, AND FIGURE 1 ABOUT HERE

Figure 1 shows that the basic assumptions for testing the presence of differential susceptibility are met: a crossover interaction with the regression lines crossing closer to the middle of the distribution of prenatal anxiety and the slope for infants at low levels of RSA being close to zero. Thus, as compared to infants with low RSA, infants with high RSA were reported to express higher levels of negative affectivity when maternal prenatal anxiety was high, but, on the other hand, high RSA was also associated with *lower* levels of negative affectivity when maternal prenatal anxiety was low. The regions of significance analysis provided support for this observation of differential susceptibility, as the lower and upper

bounds of the regions of significance were, respectively, -1.08 and 0.37 *SD*s (i.e., within the conventional limit of ±2 *SD* indicating differential susceptibility; Roisman et al., 2012) This finding indicated that the difference between the low and high RSA regression lines became statistically significant after scores of maternal prenatal anxiety exceeded 0.37 *SD* above (i.e., approximating a score of 9.7) or -1.08 *SD* below (i.e., score of 6.3) the mean of maternal prenatal anxiety. The PoI and PA values were 0.65 and 0.62, respectively, further supporting a differential susceptibility account of the current data.

Discussion

The present results provide initial evidence that the influence of prenatal maternal anxiety on infant temperamental negative affectivity at 8-10 months of age in a low-risk community sample is moderated by infants' vagal tone, indexed by respiratory sinus arrhythmia (RSA). In line with expectations derived from the differential susceptibility model (Belsky et al., 2007), infants with high baseline RSA showed a robust association between prenatal maternal anxiety and postnatal negative affectivity, with the absence of prenatal anxiety related to lower levels, and elevated prenatal anxiety to relatively higher levels of infant negative affectivity at 8-10 months of age. In infants with low baseline RSA, no association between maternal anxiety and infant negative affectivity was observed. We consider the present findings important as they indicate that even within a relatively normative range of prenatal anxiety, vagal tone appears to have a role in whether or not maternal prenatal anxiety influences the postnatal emergence of emotional reactivity.

These results support the view that not all children are similarly affected by varying conditions of the intrauterine environment. Regarding temperament development, findings of the influence of putatively susceptible genetic polymorphisms on the relation between normative variation in maternal prenatal anxiety and postnatal infant negative affectivity have been mixed (Braithwaite et al., 2013; Pluess et al., 2011). As a measure of parasympathetic

functioning that is not determined by any single gene, RSA might offer a more robust marker of biological sensitivity to prenatal influences as compared to single genetic variants. Regarding the potential mechanisms by which vagal tone might confer susceptibility to the environment, high RSA has been associated with higher overall reactivity to external cues (e.g., Beauchaine, 2001; Stifter & Fox, 1990; Stifter & Jain, 1996). Consequently, the large range of autonomic arousal may render infants with high vagal tone relatively more sensitive to transactions with the environment, which could then promote adaptation (i.e., susceptibility) to their particular developmental context (Beauchaine, 2001; Conradt et al., 2013; Porges, 2007). Considering that high RSA has also been linked with increased cortical excitability (Duschek, Wörsching, & Reyes del Paso, 2013), vagal tone can be considered as a biomarker of physiological and neural sensitivity to external cues, and thus susceptibility to various features of the environment.

Although the results showed that the association between maternal prenatal anxiety and infants' negative affectivity is contingent on infants' RSA, it is important to note that the design of the present study included an assessment of RSA only concurrently with the temperament assessment. Therefore, the exact timing of the effects of vagal tone with respect to maternal prenatal anxiety remains unclear. One possibility is that the interindividual variation in RSA observed postnatally in the present sample reflects stable differences in RSA that extend to the prenatal period. If true, it could mean that fetuses with high vagal tone are more responsive to fluctuations in maternal autonomic arousal level associated with elevated anxiety, which could foster the development of a more reactive postnatal phenotype. Some studies have shown changes in fetal heart rate induced by maternal cognitive challenge (DiPietro, Costigan, & Gurewitsch, 2003) and relaxation (DiPietro, Costigan, Nelson, Gurewitsch, & Laudenslager, 2008), but no studies have directly investigated whether such short-term effects of maternal arousal fluctuations on fetal physiology are contingent on fetal

RSA. Future studies should determine the point in development at which clear and stable individual differences in RSA emerge. Although there is evidence that RSA, which has been suggested to be matured by the third trimester (Groome, Loizou, Holland, Smith, & Hoff, 1999), is relatively stable in premature infants at 32-37 weeks of gestational age (Feldman, 2009) and from 4 to 48 months in typically developing infants (Bar-Haim et al., 2000), no studies have tracked the longitudinal stability of RSA within the same sample from late pregnancy through the first year. For more basic measures of heart rate and its variability (beats per minute and standard deviation), stability has been observed from 24 weeks of gestation to 2 weeks and 12 months of postnatal age (DiPietro, Costigan, Pressman, & Doussard-Roosevelt, 2000), pointing to general stability of fetal and infant cardiac measures.

An alternative possibility is that the interindividual variation in RSA emerges postnatally. In this case, the processes triggered by maternal prenatal anxiety, such as epigenetic modification of the sensitivity of the fetal glucocorticoid system (e.g., Conradt, Lester, Appleton, Armstrong, & Marsit, 2013; Glover, O'Connor, & O'Donnell, 2010) could begin to manifest as variation in temperamental negative affectivity only after individual differences in vagal tone have emerged. Yet another possibility is that, given the continuity of maternal anxiety from pregnancy to late infancy (see Table 1), the results could be due to differences in postnatal parenting behavior as a function of maternal anxiety, with infants with high RSA being affected to a greater extent by sensitive and insensitive parenting (cf. Conradt, Measelle, et al., 2013). This interpretation, however, is not fully plausible given that the interaction between prenatal anxiety and RSA was evident even when concurrent anxiety was controlled, indicating that the moderation effects were independent of the mothers' postnatal anxiety. To be able to address these possibilities, it is clear that future prospective studies investigating the role of vagal tone in moderating the effects of the prenatal environment would greatly benefit from direct estimates of fetal RSA.

An intriguing feature of the results is that the outcome measure of the study – temperamental negative affectivity – is in itself an indicator of differential susceptibility to the environment which has been shown to moderate the effects of the quality of early childhood caregiver-child interactions on later social adjustment (e.g., Pluess & Belsky, 2009). It has been argued that the degree of postnatal susceptibility to the environment (e.g., variation in temperamental negative affectivity) is at least partially a function of prenatal experiences, such as maternal anxiety and stress during pregnancy, which have been shown to account for the postnatal temperamental phenotype even when postnatal maternal factors have been controlled (for review, see Pluess & Belsky, 2011). Thus, while the present results were suggestive of prenatal programming of postnatal susceptibility to the environment (indicated by the robust association between prenatal anxiety and infant negative affectivity in the full longitudinal sample), such programming effects appear to be strongest for infants with high cardiac vagal tone, which may be a marker of heightened attunement to external cues and events (cf. Beauchaine, 2001).

Reliance on parental report of infant negative affectivity (instead of measures based on observation) can be considered as a limitation of the present study. Nevertheless, as parental perceptions of infant temperament were moderated by infants' physiology (RSA), it seems clear that the effects observed in this study are not solely explained by the mothers' own anxiety. Second, even though infant RSA was not correlated with prenatal and postnatal maternal anxiety in this study, supporting the use of RSA as an independent predictor, it remains unclear whether other maternal factors could affect the development of individual differences in RSA in utero (although additional analyses showed that maternal prenatal depression also did not correlate with infant RSA). Furthermore, as the maternal prenatal questionnaire measures were administered only during the third trimester, it remains possible that maternal characteristics during earlier phases of pregnancy may have shaped the

development of infant RSA. In addition to measures of fetal RSA, future studies should include a more comprehensive assessment of maternal mood across pregnancy.

In conclusion, the present investigation provided initial evidence for the role of vagal tone in moderating – in a differential susceptibility manner – the influence of maternal prenatal anxiety on the postnatal emergence of temperamental negative affectivity. Important tasks for future studies will be to examine the replicability of the pattern of results in a sample expressing a broader range of prenatal symptoms, and to chart the continuity of child-related susceptibility factors in relation to environmental influences, i.e., whether or not the physiological and temperamental susceptibility factors continue to moderate environmental effects in a differential susceptibility manner beyond infancy and early childhood (cf. Belsky & Pluess, 2013). Coupled with data on maternal well-being already during pregnancy, longitudinal investigations on how vagal tone and temperamental negative affectivity moderate the influence of environmental factors (such as variations in caregiving quality) on social and emotional development in later childhood will result in a more complete and a truly developmental account of children's differential susceptibility to their environments.

Notes

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Tables

Table 1

Correlations Between Continuous Variables

	1	2	3	4	5	6	7	8	9
1 Infant RSA	-								
2 Infant NA	.04	-							
3 Infant GA	.04	-.11	-						
4 Maternal age	-.01	-.12	.04	-					
5 Education	-.04	-.04	.00	.30***	-				
6 Income	-.04	-.04	.03	.26**	.48***	-			
7 Alcohol (pregnancy)	-.05	-.12	-.03	.09	.08	.00	-		
8 Prenatal anxiety	.05	.23*	-.22**	-.08	-.15	-.03	-.01	-	
9 Postnatal anxiety	-.09	.15	-.20**	-.02	-.09	.00	-.05	.64***	-

Note. $N = 173$; $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$

Table 2

Summary of the Regression Analysis Predicting Infant Negative Affectivity

Predictor Variables	*B*	*SE*	*β*	*p*
Step 1 ($F = 2.25$, $p = .04$, $R^2 = .09$)				
Maternal age	-.093	.066	-.13	.16
Education	.004	.064	.01	.95
Income	-.008	.074	-.01	.91
Alcohol consumption	-.071	.051	-.11	.16
Postnatal anxiety	.111	.061	.15	.07
Infant GA	-.057	.058	-.08	.33
Infant sex	-.120	.057	-.16	.04
Step 2 ($F = 2.31$, $p = .03$, $R^2 = .11$)				
Prenatal anxiety	.135	.081	.19	.10
Infant RSA	.039	.057	.05	.50
Step 3 ($F = 2.76$, $p = .006$, $R^2 = .15$)				
Prenatal anxiety × Infant RSA	.141	.060	.19	.02

Note. $N = 173$

Figure Captions

Figure 1. Regression lines illustrating the association between maternal prenatal anxiety and infant negative affectivity at high and low levels of infant RSA (±1 *SD*). The shaded areas represent the regions of significance, i.e., the lower and upper bounds of values for prenatal anxiety outside of which the two regression lines differ significantly.

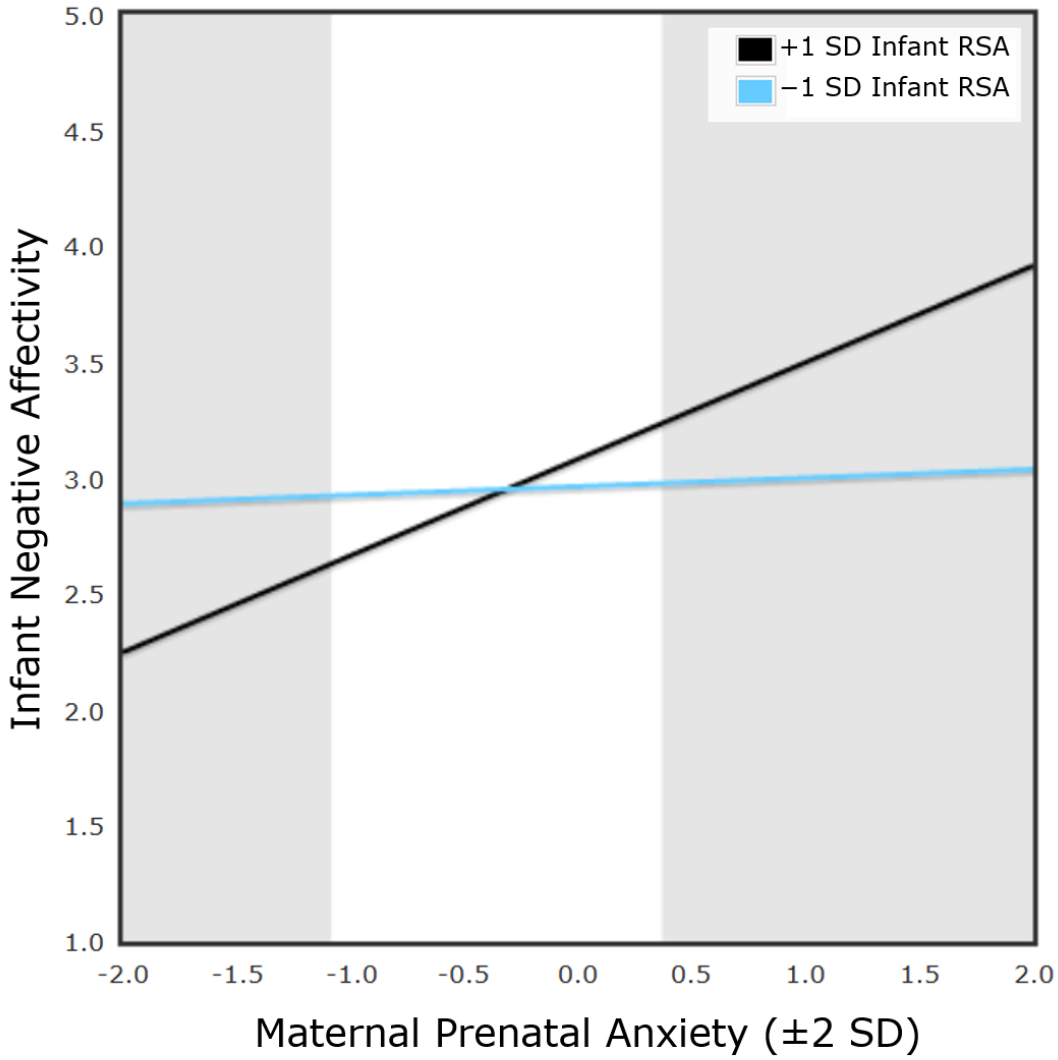
+1 SD Infant RSA
−1 SD Infant RSA
Infant Negative Affectivity
5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
-2.0
-1.5
-1.0
-0.5
0.0
0.5
1.0
1.5
2.0
Maternal Prenatal Anxiety (±2 SD)

FIGURE 1.