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BRIEF REPORT

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Infant respiratory sinus arrhythmia and maternal depressive symptoms predict toddler sleep problems

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Abstract

This study examined the direct and interactive effects of infants' respiratory sinus arrhythmia (RSA) and maternal depressive symptoms (MDS) during the first 6 months of life in the prediction of children's sleep problems at age 18 months. Participants included 156 children and their mothers who were followed from 3 to 18 months of age. At ages 3 and 6 months, infants' cardiac activity was recorded at rest and during the still-face paradigm, a mother–child social challenge task, and estimates of infant baseline RSA (RSAB) and RSA withdrawal (RSAW) were calculated. Mothers reported about their depressive symptoms at 3, 6, and 18 months, and about infants' sleep problems at age 18 months. Less RSAW and higher levels of MDS predicted more sleep problems at age 18 months. Additionally, RSAB moderated the link between MDS and children's sleep problems such that MDS were related to more sleep problems only for infants with high levels of RSAB. Results illustrate the importance of RSA as both a direct predictor and a moderator of maternal influences in the prediction of early sleep problems.

KEYWORDS
maternal depression, mother–infant relations, respiratory sinus arrhythmia, sleep/wake

1 | INTRODUCTION

Sleep problems in early childhood are reported by approximately 20–40% of parents (Mindell, Kuhn, Lewin, Meltzer, & Sadeh, 2006) and have been associated with substantial health burdens for families, including decreased maternal wellbeing, sleep problems at school-entry age, and poor child mental and physical health in childhood (Martin, Hiscock, Hardy, Davey, & Wake, 2007; Simard, Nielsen, Tremblay, Boivin, & Montplaisir, 2008). Although it is theorized that sleep, arousal, affect, and attention are closely intertwined in a dynamic biological regulatory system (Dahl, 1996), little is known about how early psychophysiological functioning is related to sleep problems. Research on school aged children suggests that respiratory sinus arrhythmia (RSA; an index of parasympathetic nervous system [PNS] functioning) is associated with both the amount and quality of children's sleep (Elmore-Staton, El-Sheikh, Vaughn, & Arsiwalla, 2012; El-Sheikh & Buckhalt, 2005). However, these processes have not yet been examined during infancy.

The current study is the first to examine longitudinal links between RSA during infancy, maternal depressive symptoms (MDS), and sleep in early toddlerhood. This developmental period is crucial for identifying risk for disrupted sleep because early sleep problems are predictive of on-going sleep problems in childhood and maladaptive parental bedtime behaviors may continue over time in reaction to these sleep difficulties (Simard et al., 2008). Therefore, we examined the direct associations between infant RSA and sleep, as well as the interactions between RSA and maternal depressive symptoms (MDS), a well-established risk factor for infant sleep disturbance (e.g., Teti & Crosby, 2012), in the prediction of sleep in early toddlerhood.

1.1 | RSA AND SLEEP IN CHILDHOOD

The autonomic nervous system (ANS), made up of the sympathetic branch (SNS) and parasympathetic branch (PNS), supports behaviors involved in maintaining attention and responding to novel stimuli and environmental challenge. The extant literature has found that pathways

of the PNS play a key role in the regulation of state, motor activity, attention, and emotion (Calkins, Propper, & Mills-Koonce, 2013). Parasympathetic control of the heart is assessed via the influence of the vagus nerve (Porges, 1992) and the resulting variability in heart rate, which occurs at the frequency of breathing (RSA). High baseline RSA (RSAB) may reflect a greater propensity to organize physiological resources to respond appropriately to environmental challenge (Porges, 1992). Research on children's physiological regulation of emotion and attention has focused primarily on RSA withdrawal (RSAW), or the decrease in RSA (from baseline) that occurs during challenging contexts. RSAW is thought to support physiological regulation because this decrease is accompanied by an increase in heart rate and other functions that allow an individual to mobilize, attend to the environment, and employ the resources necessary to deal with challenge (Calkins & Keane, 2009). Greater infant RSAW during challenge has been related to better state regulation, higher soothability, and greater attentional control (Degangi, Dipietro, Greenspan, & Porges, 1991; Huffman et al., 1998).

Less efficient physiological regulation during the day can disrupt the process of going to sleep, which involves a sharp decrease or cessation of vigilance (Dahl, 1996). A series of studies by El-Sheikh and colleagues (Elmore-Staton et al., 2012; El-Sheikh & Buckhalt, 2005; El-Sheikh, Erath, & Bagley, 2013) supports the links between RSA functioning during the day and sleep in older children. For example, reduced RSAW (smaller decrease in RSA from baseline) during a challenging attention task was associated with more severe sleep problems, implying that less effective PNS regulation may, in part, underlie sleep disturbances (El-Sheikh & Buckhalt, 2005; El-Sheikh et al., 2013).

The link between RSAB and sleep is less well-established. While some studies have not clearly demonstrated direct links between RSAB and sleep in school-aged children (El-Sheikh & Buckhalt, 2005; El-Sheikh, Erath, & Keller, 2007, El-Sheikh et al., 2013), one study on preschool children found that lower RSAB was associated with lower sleep efficiency (Elmore-Staton et al., 2012). Although these findings provide evidence for links between RSA and sleep, further research is needed in order to clarify the nature of these relations.

1.2 | RSA, MATERNAL DEPRESSIVE SYMPTOMS, AND SLEEP

Childhood sleep patterns develop within a complex matrix of biological and environmental influences (Sadeh & Anders, 1993). Links between environmental risk factors and infant sleep problems have been established, including the well-documented risk factor of maternal depressive symptoms [MDS] (Sadeh, Tikotzky, & Scher, 2010). Elevated MDS are associated with more childhood sleep problems (Bayer, Hiscock, Hampton, & Wake, 2007; Warren, Howe, Simmens, & Dahl, 2006), and these links can be explained by factors such as more dysfunctional maternal cognitions about infants' sleep and specific maternal nighttime behaviors (Teti & Crosby, 2012). However, little is known about how children's physiological functioning may modify these links.

The biological sensitivity to context hypothesis (BSC; Boyce & Ellis, 2005) suggests that children who are more physiologically reactive to contextual influences are likely to be more susceptible to both adverse and enriching environmental experiences in the course of their development. For example, children with high RSAB (an indicator of the propensity to be physiologically reactive) or high RSAW (a marker of heightened physiological reactivity) show the lowest adaptive functioning when exposed to adverse environmental conditions such as disorganized attachment relationships, maternal emotional distress, and harsh parenting compared to children with low RSAB and RSAW. However, they show the highest level of adaptive functioning when exposed to low-family adversity and secure attachment relationships (Conradt, Measelle, & Ablow, 2013; Obradović, Bush, Stamperdahl, Adler, & Boyce, 2010). To date, only one study has examined this notion in the prediction of sleep. In a sample of school-aged children, RSAW (during a social stress task) interacted with MDS to predict sleep measures over time. Consistent with the BSC, hypothesis higher levels of MDS were related to less time spent in sleep during the night only for children with high RSAW, and not for children with low RSAW (Keller, Kouros, Erath, Dahl, & El-Sheikh, 2014). Additional research is needed to further unpack the complex interactions between children's physiological functioning, maternal behaviors, and child sleep during infancy.

1.3 | THE CURRENT STUDY

The goal of the current study was to examine the associations between children's RSA, MDS, and later sleep problems. We add to extant the literature by examining these links during *infancy*, a time that is highly important for establishing healthy sleep patterns (Sadeh et al., 2010). Based on previous research with older children (Elmore-Staton et al., 2012; El-Sheikh & Buckhalt, 2005; El-Sheikh et al., 2013) we hypothesized that lower RSAB and RSAW, both of which have been associated with less effective regulation of emotion and attention (Calkins, 1997; Degangi et al., 1991; Feldman, 2009; Huffman et al., 1998) would be associated with elevated sleep problems in toddlerhood, as would more severe MDS during infancy. Furthermore, we hypothesized that RSAB and RSAW would interact with MDS in predicting later sleep problems. Based on the BSC hypothesis (Boyce & Ellis, 2005), and prior research examining RSA as a marker of BSC (Conradt et al., 2013; Obradović et al., 2010) we expected that infants with higher RSAB (i.e., greater capacity for reactivity to contextual influences) and greater RSAW (i.e., greater reactivity in response to a specific challenge) would exhibit the highest level of sleep problems when exposed to elevated MDS, and the lowest level of sleep problems when exposed to low levels of MDS.

2 | METHODS

2.1 | Participants

Participants in this study were a convenience subsample of The Durham Child Health and Development Study (DCHDS), a longitudinal study of 206 socioeconomically and racially diverse families living in and around a mid-sized city in North Carolina. Families were recruited when their infants were 3 months of age from a largely urban community via fliers and postings at birth and parenting classes, as well

as through phone contact via birth records. The study included only full-term (>37 + 0 weeks) infants born without significant medical complications.

The subsample used in the current study included families that participated in the 3, 6, and 18 months data collection points (N = 156). Observational, cardiac, and self-report data were collected during home (3 months) and laboratory visits (6 and 18 months). In this subsample, 52% of the children were female, 54% were African American (46% were European American), and approximately 50% of the sample was low income (below 200% of the poverty level). This subsample did not differ significantly from the complete sample on any of these variables.

2.2 | MEASURES

2.2.1 | Infant respiratory sinus arrhythmia (RSA)

Baseline (3 and 6 months)

At the start of the assessments, the experimenter placed two disposable pediatric electrodes on the infant's chest. The electrodes were connected to a preamplifier, from which the output was transmitted to a heart interbeat interval (IBI) monitor (Mini Logger; Mini-Mitter/Respironics, Bend, OR) for R-wave detection. The infant wore a smock with a large pocket in which the monitor was placed. Once the monitor was securely in place and the infant was acclimated and in a calm state (approximately 5 min after electrode placement), the infant was seated the mother's laps and the mother was asked not to interact with the infant for 2–4 min so that stimulation was minimized and IBI could be measured during a neutral and calm state (baseline).

Withdrawal (3 and 6 months)

Following the baseline assessment, RSA was collected during the Still Face Paradigm (SFP; Tronick, Als, Adamson, Wise, & Brazelton, 1978). A full description of the procedure can be found in Propper et al. (2008). In summary, infants' were seated in a car seat and three 2-min episodes occurred which consisted of *normal* (mothers played with their babies as they normally would); *still-face* (mothers looked at their children without any facial movements or vocalizations); *reunion* (mothers could respond to their infants in any way that they felt was appropriate). Following previous research (Moore & Calkins, 2004; Propper et al., 2008), difference scores were computed by subtracting still-face episode RSA from baseline RSA, such that sign indicated direction of change, with positive values indicating greater RSAW. We were only interested in the still-face episode for the current analyses because it reflects RSA reactivity in response to a social stressor during which infants must regulate their own arousal without the help of their mothers.

The cardiac data files were edited by two reliable researchers using MXEdit software (Delta Biometrics, Bethesda, MD). Porges' (U.S. Patent No. 4,510,944, 1985) method of calculating RSA was used, in which a moving polynomial filter (with band-pass filter set to 0.24–1.04 Hz, the frequency of spontaneous respiration in infants) is used to remove frequencies lying outside a normal physiological range, and the estimate of RSA is reported in units of $\ln(ms)^2$. RSA was calculated every 15 s from the baseline and the still-face periods. This epoch duration is typical for studies of short-duration tasks (Huffman et al., 1998). The mean of the epochs from 3 to 6 months was used to represent RSAB and RSAW during infancy.

2.2.2 | Maternal depressive symptoms

Mothers reported depressive symptoms during the 3, 6, and 18 months assessments using the Brief Symptom Inventory (BSI; Derogatis & Melisaratos, 1983), a validated self-report measure consisting of 53 items. The six items of the depression scale define a spectrum of depressive symptoms such as "feeling lonely" in the preceding 7 days, and are rated on 5-point Likert scale from 0 (not at all) to 4 (extremely). Ratings across the six items were averaged to create score for each time point with higher scores indicating more severe MDS (α ranged between .72–.81). The mean MDS score from 3 and 6 months (scores ranged between 0 and 2.08) was used to represent MDS during infancy.

2.2.3 | Toddler sleep problems

Mothers reported about their children's sleep problems during the 18 months assessment using the sleep items from the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2000). The seven CBCL items define a spectrum of sleep problems, such as "has trouble getting to sleep," and "wakes up often at night," that are rated on a three point scale ranging from 0 (not true) to 2 (very or often true). The CBCL sleep composite score has previously shown convergent validity with other established parent-reported sleep measures such as the Children's Sleep Habits Questionnaire (CSHQ; Owens, Spirito, & McGuinn, 2000) total score and the sleep disturbance index of the Sleep Disorders Inventory for Students (SDIS; Luginbuehl, Bradley-Klug, Ferron, Anderson, & Benbadis, 2008; Becker, Ramsey, & Byars, 2015). Further, CBCL sleep items were associated with several other sleep measures. For example, "trouble sleeping" has been related to sleep latency assessed by both diary and actigraphy (Gregory et al., 2011). The seven items were summed to create a sleep problems score (α = .70), with higher scores indicating more severe sleep problems.

2.2.4 | Covariates

Consistent with previous reports based on the DCHDS data set (e.g., Moore et al., 2009; Wagner, Propper, Gueron-Sela, & Mills-Koonce, 2016) the covariates included in the analysis were child sex, child race, and income to needs ratio. These covariates were chosen based on the diverse nature of the sample which over sampled for poverty and African American race. Child's race and sex were reported by the child's primary caregiver at the time of recruitment. Family income-to-needs ratio was determined using the mother's report of the total family yearly income at the 3 and 6 months' assessments, the size of the family, and the 2003 federal poverty guidelines. Children's exact ages at the 3, 6, and 18 months' assessments were also included as covariates to account for random variation in scheduling and participant availability.

2.2.5 | Missing data

Among the 156 families in the subsample, 13% (n = 20) were missing cardiac data from both time points and 3% (n = 5) were missing maternal depressive symptoms data from age 6 months. Reasons for missing cardiac data included heart rate monitor problems or failure, too much artifact in the heart rate data due to movement or removal of equipment, or the infant could not complete the task due to extreme fussiness. Children with missing data did not differ from children without missing data in terms of family income group or race. However, males (n = 17 of 22) had significantly more missing data than females (n = 5 of 22), λ^2 = 8.76, p = .003. Multiple imputation (MI) was used to impute missing data in the RSAB and RSAW variables (Schafer, 1997; Schafer & Graham, 2002). A total of 20 imputed datasets were generated using SPSS 23. All models were estimated using the imputed data. Coefficients and standard errors are reported from the pooled estimates and the simple slopes and regions of significance for significant interactions were obtained from the complete case model.

3 | RESULTS

3.1 | Descriptive statistics

Table 1 presents the bivariate correlations, means, and standard deviations for all study variables. MDS during infancy were significantly related to toddler sleep problems at age 18 months (r = .17, p = .03), and the correlation between infant RSAW and toddler sleep problems approached significance (r = −.16, p = .06).

3.2 | Hierarchical OLS regression model

A series of hierarchical OLS regression models (Table 2) indicated that after taking into account the effects of covariates (step 1), both RSAW (B = −.55, p = .04) and MDS (B = 1.64, p = .02) significantly predicted sleep problems at age 18 months (step 2). In step 3, a significant interaction was observed between RSAB and MDS (B = 1.58, p = .05). All other interaction terms were non-significant and were trimmed from the model. The final regression model accounting for 18% of the variance in toddler sleep problems (see Table 2).

The significant interaction between infant RSAB and MDS was probed at high (+1 *SD*) and low (−1 *SD*) levels of infant RSAB (Figure 1). The positive association between MDS and children's sleep problems was significant only for children who had high levels of RSAB during infancy (simple slope = 3.52, t = 3.42, p = .001), and not for children who had low levels of RSAB (simple slope = .17, t = .15, non-significant [*ns*]). To examine whether this interaction was consistent with the BSC hypothesis, simple slopes for high (+1 *SD*) and low (−1 *SD*) levels of MDS were estimated revealing that the association between infant RSAB and child sleep problems was significant only under high (simple slope = .81, t = 2.17, p =.03) and not under low levels of MDS (simple slope = −.57, t = 1.43, *ns*). Regions of significance (RoS) analysis indicated that when MDS scores were high (above .26; approximately ¾ SD above the mean of the centered MDS variable) children with high RSAB had significantly higher levels of sleep problems than children with low RSAB.

4 | DISCUSSION

This study examined the direct and interactive effects of infants' RSA and maternal depression on sleep problems in toddlerhood, proposing that higher baseline RSA and greater RSA reactivity would represent a biological marker of sensitivity to the parenting environment (Boyce & Ellis, 2005; Obradović et al., 2010). Findings augment extant literature by demonstrating the important role of PNS functioning in infancy for children's sleep. Consistent with past research suggesting that less RSA reactivity may indicate less effective regulation (Degangi et al., 1991; El-Sheikh & Buckhalt,

TABLE 1 Bivariate correlations between study variables

	1	2	3	4	5	6	7	8	9	10	11
1. Child sex (0 = Male)	–	.07	−.00	−.05	.05	.06	−.03	.01	−.19**	−.00	.07
2. Child race (0 = European American)		–	−.30****	−.05	.50	−.17**	.08	−.21**	−.05	.01	.09
3. Family income-to-needs ratio			–	.02	−.00	.08	−.00	.03	−.22***	−.16**	−.10
4. Infant age 3m assessment				–	−.01	−.05	.13	.02	.02	.03	.03
5. Infant age 6m assessment					–	−.15*	.07	−.06	−.04	−.02	.04
6. Infant age 18m assessment						–	.09	.22*	.04	.06	−.08
7. Infant RSAB							–	.18**	.04	.08	.00
8. Infant RSAW								–	.18**	.14	−.16*
9. Infancy MDS									–	.59****	.17**
10. MDS 18m										–	.10
11. Toddler sleep problems											–
Mean			2.99	3.69	7.30	19.00	3.50	.43	.25	.26	2.33
SD			2.52	.48	.79	1.12	.84	.73	.32	.45	2.31

*p < .10, **p < .05, ***p < .01, ****p < .001. RSAB, baseline respiratory sinus arrhythmia, RSAW, respiratory sinus arrhythmia withdrawal, MDS, maternal depressive symptoms, 3m = 3 months, 6m = 6 months. Reported correlation coefficients are pooled estimates from the MI analysis (N = 156).

TABLE 2 Hierarchical OLS regression model predicting toddler sleep problems at age 18 months

	Step 1 B (*SD*)	Step 2 B (*SD*)	Step 3 B (*SD*)
Child sex	.15 (.38)	.38 (.38)	.33 (.38)
Child race	.19 (.40)	−.02 (.41)	−.02 (.41)
Family income-to-needs	−.08 (.08)	−.04 (.08)	−.05 (.08)
Infant age 3m assessment	.08 (.38)	.04 (.38)	−.07 (.38)
Infant age 6m assessment	.18 (.24)	.13 (.23)	.15 (.23)
Infant age 18m assessment	−.12 (.17)	−.10 (.17)	−.12 (.17)
Concurrent MDS (18m)	.14 (.44)	−.30 (.52)	−.41 (.52)
Infant RSAB (3m, 6m)		.13 (.23)	.08 (.23)
Infant RSAW (3m, 6m)		−.79 (.28)***	−.80 (.29)***
Infancy MDS (3m, 6m)		1.79 (.75)**	1.65 (.75)**
RSAB * infancy MDS			1.58 (.80)**
R^2	.02	.14**	.18**

*$p < .10$, **$p < .05$, ***$p \leq .01$. RSAB, baseline respiratory sinus arrhythmia; RSAW, respiratory sinus arrhythmia withdrawal; MDS, maternal depressive symptoms; 3m, 3 months; 6m, 6 months; 18m, 18 months. Reported regression coefficients and standard deviations are pooled estimates from the MI analysis ($N = 156$).

2005), we found that less RSAW (i.e., infants who did not exhibit a decrease in RSA) during maternal unresponsiveness in the SFP was associated with more sleep problems in toddlerhood. This finding implies that infants' inability to adaptively regulate physiological functioning in the context of the mother–child relationship may be associated with aberrant sleep patterns.

The task of going to sleep involves a naturally occurring separation from the mother, in which the child is required to separate physically and emotionally from the parent (Anders, 1994; Scher, 2008). Therefore, one explanation for these findings may be that that infants who do not show physiological regulation when dealing with distress caused by maternal unavailability may also experience difficulties in

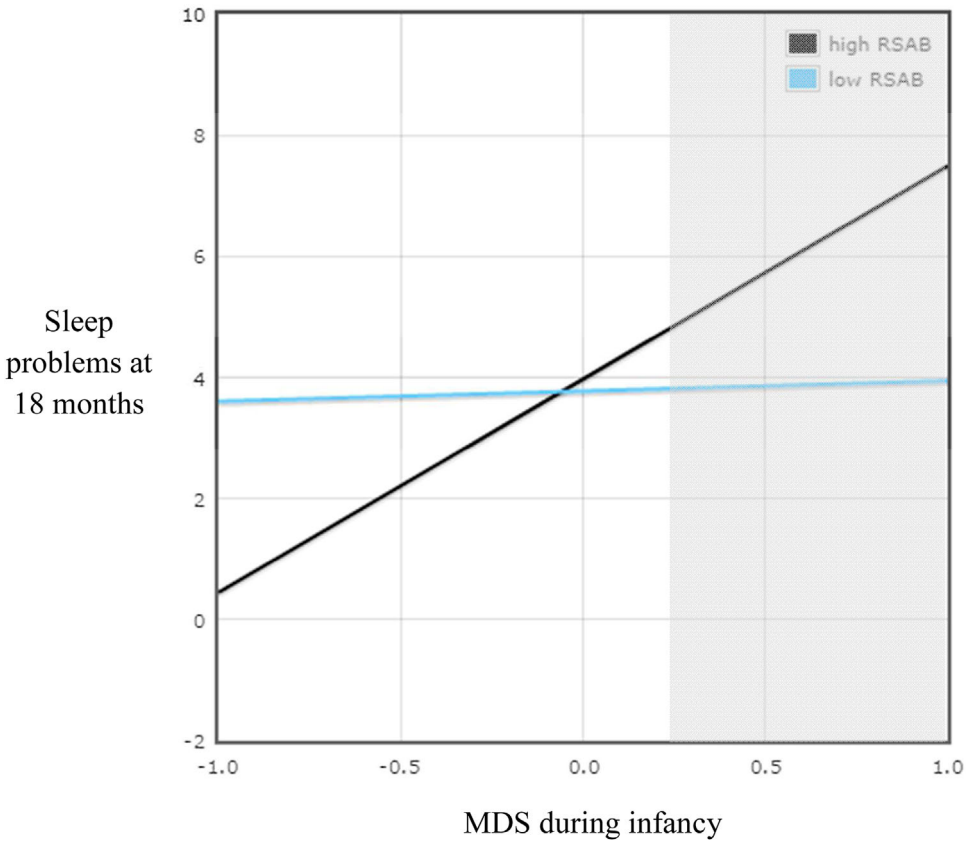

FIGURE 1 RoS analysis for the interaction between RSAB and infancy MDS on children's sleep problems at age 18 months. The shaded area represents the RoS: the values of MDS for which there is a significant difference in sleep problems between infants with high and low levels of RSAB

regulating distress caused by the separation from their mothers during bedtime and through the night, which may lead to an inability to transition to sleep. However, it may also be that infants who did not exhibit RSAW in the still-face paradigm did not experience distress in this situation. An important next step will be to examine whether infants' RSAW during bedtime is related to the quality and quantity of their sleep, and to include behavioral measures of infants' distress.

Contrary to our hypothesis, we did not find a direct effect of infant RSAB on toddler sleep problems. However, RSAB did moderate the relation between MDS and later sleep problems, such that infants with higher RSAB appeared to be more susceptible to the negative effects of MDS (leading to increased sleep problems) than children with lower RSAB. Results support the notion that physiological regulation of arousal may impact sleep problems (Dahl, 1996) such that children who are more physiologically reactive may be more strongly affected by environmental adversity compared to children with lower physiological reactivity (Boyce & Ellis, 2005). It may be that because RSAB represents the *propensity* to be physiologically reactive to environmental challenge (Porges, 1992), in the absence of an adverse environment RSAB does not necessarily indicate heightened physiological reactivity throughout the day. However, children with higher RSAB may become highly reactive when exposed to continuous interactions with a mother that is experiencing depressive symptoms, and this heightened reactivity may in turn compromise their sleep quality and/or quantity. These findings support the view that infants with high RSAB may have an early disposition for heightened physiological reactivity, which under negative environments may lead to the consolidation of maladaptive coping strategies that portend adjustment problems later in development (Conradt et al., 2013).

Findings from this study were partially consistent with the BSC perspective. Although infants with high RSAB showed more severe sleep problems than other infants when exposed to adverse contextual experiences (i.e., heightened MDS), they did not have less severe sleep problems when exposed to low MDS, as predicted by the BSC hypothesis. The BSC hypothesis suggests that children with heightened physiological reactivity are more susceptible to benefit-conferring features of the environment (Obradović et al., 2010). In the current study, there may have been a dose effect in which low-maternal MDS did not necessarily involve the presence of a nurturing parenting environment that activated biological susceptibility to positive and enriching environments. Future studies should further examine the roles of infants' both RSAB and RSAW across adverse and supportive parenting contexts.

Results from this study should be considered in light of several limitations. First, our measure of sleep problems was based on maternal report and did not include objective measures of child sleep, such as actigraphy. Self-report of both MDS and child sleep could be influenced by reporter bias such that mothers with heightened depressive symptoms may experience their children's sleep problems as more severe. However, the non-significant concurrent association between mothers' depressive symptoms and child sleep at age 18 months implies that depression did not bias mothers' reports on child sleep. Second, we did not obtain measures of sleep at ages 3 and 6 months or RSA at age 18 months. A repeated measures cross-lagged model could allow examination of the bidirectional relations between RSA and sleep. Third, information about prenatal depression and use of psychotropic medication was not collected because the sample was a community sample, which was at low risk for clinical depression. Given the possible effects of in utero exposure to maternal depression and antidepressants on infant sleep (Suri, Lin, Cohen, & Altshuler, 2014), it is important to include prenatal assessments of maternal depression in future research to understand their link to postnatal depression and infant sleep problems. Finally, in light of recent findings that demonstrate interactive effects between RSAW and skin conductance in the prediction of children's sleep (Erath & El-Sheikh, 2015), it is important to consider the sympathetic branch of the ANS as well. Despite these limitations, results suggest that individual differences in RSA have direct and interactive effects on sleep, and highlight the importance of examining physiological functioning towards a better understanding of sleep in early childhood.

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