Examining Biological Vulnerability in Environmental Context: Parenting Moderates Effects of Low Resting Respiratory Sinus Arrhythmia on Adolescent Depressive Symptoms

ABSTRACT: Polyvagal theory suggests that parasympathetic regulation of cardiac function, indexed by resting respiratory sinus arrhythmia (RSA), may be a marker of emotion regulatory capacity and associated with youth psychopathology. Contemporary models of psychopathology suggest that the effects of biological vulnerability may be moderated by developmental context. The aim of the present study was to examine whether parenting, particularly parental responses to youth’s negative emotions, moderated the effects of resting RSA on depressive symptoms among early adolescents. We examined resting RSA, depressive symptoms, and parental responses to youth negative emotions among 120 adolescents aged 11–14 years (M = 12.86, SD = .85; 52.5% female). Resting RSA and lack of supportive parenting interacted to predict youth depressive symptoms, such that low resting RSA predicted more depressive symptoms only in the context of low levels of supportive parental responses to youth’s negative emotions. By contrast, high resting RSA buffered the effects of low supportive parenting on youth depressive symptoms. These findings highlight the importance of understanding joint contributions of biological vulnerability and developmental context on youth depression outcomes. © 2015 Wiley Periodicals, Inc. Dev Psychobiol 9999: 1–10, 2015.

Keywords: RSA; depression; parenting; sensitivity to context; adolescence

INTRODUCTION

Parasympathetic regulation of cardiac activity, indexed by respiratory sinus arrhythmia (RSA), has been identified as a potential physiological biomarker of emotion regulatory capacity (Beauchaine, 2001, 2012; Porges, 1995, 2003). High resting RSA is associated with better emotion regulation and fewer mental health problems among youth (see Beauchaine, 2012). Low resting RSA, on the other hand, has been suggested as a potential risk factor for psychopathology in general and depression in particular (Yaroslavsky, Rottenberg, & Kovacs, 2014). An association between low resting RSA and depression has been found in several studies, but effects are generally small and there have been multiple studies that fail to find such a link (see, e.g.,
Lower resting RSA has been found to be associated with higher internalizing symptoms among both preschoolers (Hastings et al., 2008) and children (Forbes, Fox, Cohn, Galles, & Kovacs, 2006). Gentzler et al. (2012) and Shannon et al. (2007) both found that older children and young adolescents (ages 5–14) with lower resting RSA self-reported more depressive symptoms. Similarly, studies have also found RSA-depression associations among adults (Light, Kothandapani, & Allen, 1998; Rechlin, Weis, Spitzer, & Kaschka, 1994). However, research on resting RSA and depression is mixed. Several studies have failed to find an association between resting RSA and depression (Bosch, Riese, Ormel, Verhults, & Oldehinkel, 2009; Rottenberg, Chambers, Allen, & Manber, 2007). One study actually found that higher resting RSA was associated with more, rather than less, sadness (Rottenberg et al., 2002). In a critical meta-analysis, Rottenberg (2007) found that across 13 studies there was a small to moderate effect of resting RSA on depression ($d = 0.33$), with more than half of studies finding nonsignificant relationships.

One explanation for inconsistent findings may be that resting RSA only confers risk for depression under certain conditions. Current theory on autonomic vulnerability factors for psychopathology suggests that such risk factors “... may help specify which children are most vulnerable to developing disorders in contexts of liability and risk [emphasis added].” (Zisner & Beauchaine, in press, p. 7). Specifically among developmental samples, it may be important to examine the role of environmental context in understanding the relationship between resting RSA and depression.

**Parasympathetic Regulation and Depression**

Polyvagal theory suggests that parasympathetic influence on cardiac activity may be an important contributor to the regulatory control of arousal, representing a higher order of regulation compared to sympathetic influences (Porges, 2001, 2007). Thus, polyvagal theory suggests that parasympathetic regulation of cardiac activity may be a useful index of the psychological capacity for self-regulation in general and emotion regulation in particular. Respiratory sinus arrhythmia (RSA) is a measure of the high frequency variability in heart rate across the breathing cycle and is a noninvasive index of parasympathetic regulation. Under baseline (resting) conditions, parasympathetic influence should be high and thus higher resting RSA indicates greater physiological flexibility and ability to adapt when faced with environmental stressors (Porges, 1995, 2007). Consistent with theory, high resting RSA in youth has been associated with lower negative emotionality, more adaptive emotion regulation, social competence, and lower levels of internalizing and externalizing symptoms (see Beauchaine, 2001, 2012 for reviews; see also Gentzler, Santucci, Kovacs, & Fox, 2009; Thayer, Friedman, & Borkevec, 1996).

Some research suggests that low resting RSA may also confer biological vulnerability to depression. Lower resting RSA has been found to be associated with higher internalizing symptoms among both preschoolers (Hastings et al., 2008) and children (Forbes, Fox, Cohn, Galles, & Kovacs, 2006). Gentzler et al. (2012) and Shannon et al. (2007) both found that older children and young adolescents (ages 5–14) with lower resting RSA self-reported more depressive symptoms. Similarly, studies have also found RSA-depression associations among adults (Light, Kothandapani, & Allen, 1998; Rechlin, Weis, Spitzer, & Kaschka, 1994). However, research on resting RSA and depression is mixed. Several studies have failed to find an association between resting RSA and depression (Bosch, Riese, Ormel, Verhults, & Oldehinkel, 2009; Rottenberg, Chambers, Allen, & Manber, 2007). One study actually found that higher resting RSA was associated with more, rather than less, sadness (Rottenberg et al., 2002). In a critical meta-analysis, Rottenberg (2007) found that across 13 studies there was a small to moderate effect of resting RSA on depression ($d = 0.33$), with more than half of studies finding nonsignificant relationships.

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**Understanding Biological Risk in Environmental Context**

The biological sensitivity to context model of psychopathology suggests some children are more biologically sensitive to the effects of their developmental environments (e.g., Ellis & Boyce, 2011). Such children may be biologically vulnerable to developing mental health problems when exposed to adverse or high-risk developmental contexts, but may show evidence of resiliency and even flourishing in protective developmental contexts. Recent research has indicated that children with high resting RSA may be protected from developing mental health problems even when exposed to adverse developmental contexts such as maternal depression (e.g., Blandon, Calkins, Keane, & O’Brien, 2008) or marital conflict (e.g., El-Sheikh, Harger, & Whitsone,
have been associated with disorders of emotion regulation including borderline personality disorder, nonsuicidal self-injury, and depression (Krause, Mendelson, & Lynch, 2003; Yap, Allen, & Ladouceur, 2008). In sum, supportive and validating responses from parents guide children in learning how to label, control, and understand their emotions and emotional reactions. However, consistent lack of support and/or invalidation promotes dysfunctional emotion regulation capacities, which ultimately undermines the child’s development of effective emotion regulation strategies and may lead to adverse mental health outcomes such as depression.

Understanding the biological and environmental processes involved in depression is particularly salient in early adolescence. Rates of depression increase dramatically between ages 13 and 18, and the strongest predictor of later depressive diagnoses is early depressive symptoms (Bardone, Moffitt, Caspi, Dickson, & Dickson, 1996; Fergusson, Horwood, Ridder, & Beautrais, 2005; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993). However, there is very little empirical data examining RSA as predictor of depressive symptoms among early adolescents and no studies, to our knowledge, that examine RSA in the context of parental responses to youth’s negative emotions.

The Current Study

The purpose of the current study was to clarify the role of individual differences in low resting RSA on depressive symptoms among early adolescents, and to examine whether the effect of low resting RSA on depressive symptoms is moderated by developmental context, specifically parental responses to youth negative emotions. We hypothesized that the effect of low RSA on depressive symptoms would be strongest for youth experiencing high parental invalidation or low parental support.

METHODS

Participants

Participants were 120 youth ranging in age from 11 years to 14 years (M = 12.88, SD = .84, 52.5% female). The majority of youth were Caucasian (79.1%), with smaller percentages identifying as Asian–American (8.2%), African–American (1%), Native American (1%), and biracial or multiracial (10.9%). The sample was predominantly middle to upper middle class, with a mean household income of over $75,000. Only 6.5% of families reported annual household incomes below $35,000. Most children lived with both biological parents (76.3%), with smaller percentages being raised jointly.
by divorced parents (20.1%) or single, widowed, or foster parents (3.6%).

Procedure

All procedures employed in this study were approved by the university Institutional Review Board. The study involved school-based screening followed by a laboratory visit. Youth were invited to participate in the school-based screening if they were (1) 11 to 14 years old at the time of screening; (2) in 5th to 8th grades; and (3) if they and one parent were sufficiently fluent in English to complete study questionnaires. Parents provided consent and youth assent for screening. As the purpose of the broader study was to identify premorbid pathways to adolescent-onset depression, youth and a parent were invited to the laboratory visit if the youth’s self-reported depressive symptoms at the screening visit were below the clinical cutoff. Approximately 88% of screened youth were eligible to participate in the laboratory visit.

Laboratory visits were conducted by a team of two trained experimenters. Visits lasted approximately 2.5 hr, during which time youth and parent completed a series of tasks. Only procedures related to the current report are summarized. Youth completed self-reported measures on a desktop computer. Then youth participated in a 4-min resting baseline period seated at the computer while viewing relaxing nature scenes on the computer screen.

Youth were paid $20 and received a small gift for participation in the study; parents were paid $50. In all, 125 youth participated in the laboratory visit. Data from five children were excluded from analyses because of missing/outlier physiological data (described in more detail below), yielding the final sample N of 120.

Measures

**Depressive Symptoms.** Youth depressive symptoms were assessed with the Children’s Depression Inventory (CDI-2; Kovacs, 2010). The CDI-2 is a 28-item self-report inventory that inquires about the presence of depressive symptoms within the past two weeks; it is normed for use with youth aged 8 to 17. Each item contains three statements; participants were asked to select the statement that best described them in the previous 2 weeks. Total scores on the CDI can range from 0 to 54, with higher scores indicating more severe depressive symptoms. The CDI has repeatedly demonstrated excellent internal consistency (alpha reliability ranges from .80 to .87), test–retest reliability, and predictive and construct validity, especially in community samples (Blumberg & Izard, 1986; Kovacs, 1981, 1985). The CDI-2 was administered at the screening visit to determine eligibility for the study; only youth with CDI scores below 15 at the time of screening were invited to the laboratory visit (88% of youth were eligible for the laboratory visit). At the laboratory visit, which occurred 2–6 months following the screening visit, the CDI-2 was administered again; this is the depressive symptom score used in the current analyses. The internal consistency of the CDI-2 at the laboratory visit was adequate (.79).

**Respiratory Sinus Arrhythmia (RSA).** Youths’ cardiac activity was recorded throughout a 4-min seated resting baseline. All recordings occurred in the same laboratory suite with standardized temperature and lighting. Participants were asked to refrain from use of caffeine and stimulant medication for 36 hr prior to the laboratory session, and oral confirmation of their adherence to this protocol was obtained from both parent and youth upon arrival. Only two youth were currently using other medications (1 SSRI and 1 antihistamine) and medication use was controlled for in subsequent analyses. Disposable pre-gelled Ag/AgCl electrodes were placed on their chests and abdomens using a Lead II placement. Electrocardiograph (ECG) data were acquired continuously using Biopac MP150 Data Acquisition Unit (Goleta, CA) and sampled at 1000 Hz. ECG data were processed offline using Mindware Technologies HRV 3.0.10 analysis program (Gahanna, OH). Data were visually inspected for movement artifacts or incorrect placement of markers by the automated scoring algorithm and corrected as needed by trained research assistants. The resulting interbeat interval time series was subjected to a fast Fourier transformation by the Mindware software, and power in the respiratory frequency band (.15–.40 Hz) was derived from the spectral density function. Respiration rates were examined and all fell within the expected range, therefore we elected not to control for respiration. RSA values were extracted in 30 s epochs. The average RSA value across the 4 min of the resting baseline was used to create a single Resting RSA score. Approximately 4% of the sample (N=5) had missing, incomplete, or inaccurate RSA data that could not be recovered using standard scoring techniques, due to excessive movement artifacts or technical problems (e.g., loosening leads). Altogether, there was RSA data available for 120 children. Range and mean value for resting RSA were consistent with published literature for community developmental samples (see Table 1; Zisner & Beauchaine, in press).

**Supportive and Invalidating Parental Responses to Youth Negative Emotions.** Parental responses to their children’s negative emotions were assessed with the adolescent self-report form of the Coping with Children’s Negative Emotions Scale – Adolescent Perception Version (CCNES-AP; Fabes & Eisenberg, 1998; Fabes, Poulin, Eisenberg, & Madden-Derdich, 2002). The CCNES-AP is a self-report measure in which adolescent’s respond to 12 hypothetical situations in which they experience distress (e.g., “When I get down because I had a bad day, my parent usually . . . “). Youth indicated the likelihood that their parent would display each of the six possible responses to the situation ranging from 1 (very likely) to 7 (very likely). The measure yields six subscales: problem-focused reactions (e.g., “helps me think of things to do to get my problem solved”), emotion focused reactions (e.g., “tries to get me to think of good things that happened”), expressive-encouragement reactions (e.g., “listens to me talk about my feelings”), minimization reactions (e.g., “tells me that I really have nothing to be sad about”), punitive reactions (e.g., “tells me to straighten up
emotions, and negatively correlated with resting RSA of invalidating parental responses to youth negative symptoms were positively correlated with higher levels of depressive symptoms.

Table 1. Variable means, standard deviations, and correlations for continuous variables are provided in the mean.

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>M</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Age</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>12.88</td>
<td>.83</td>
<td>11.35–14.61</td>
</tr>
<tr>
<td>2. Depressive Symptoms</td>
<td>.5</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>4.55</td>
<td>4.11</td>
<td>0–19</td>
</tr>
<tr>
<td>3. Supportive Responses</td>
<td>.18</td>
<td>– .27**</td>
<td>–</td>
<td>–</td>
<td>4.99</td>
<td>1.15</td>
<td>1.93–6.78</td>
</tr>
<tr>
<td>4. Invalidating Responses</td>
<td>.8</td>
<td>.22*</td>
<td>– .7</td>
<td>–</td>
<td>2.54</td>
<td>.98</td>
<td>1.00–4.89</td>
</tr>
</tbody>
</table>

*p < .05.
**p < .01.

and stop sulking around the house”), and distress reactions (e.g., “becomes obviously uncomfortable when s/he sees I am feeling down”). Based on previous research, two composite scales, supportive (problem-focused, emotion-focused, expressive encouragement) and nonsupportive/invalidating (minimization, punitive, distress) responses were calculated as averages of the subscales (DeBoard-Lucas, Fosco, Raynor, & Grych, 2010; Fabes, Leonard, Kupanoff, & Martin, 2001; Fabes et al., 2002; Nelson et al., 2009). Youth were allowed to choose the parent reported on with a prompt reading: “On the following items you will be asked how your parent would respond in each situation. To do this, you need to pick one parent, and complete the items about them. Please indicate below which parent you are going to answer the questions about.” Most youth chose to report on their mother (82.1%) but some chose to report on their father (19.9%). Cronbach’s alphas in the current sample for supportive and invalidating composite scales were .95 and .89, respectively.

Statistical Analysis

All data analyses were performed in SPSS 21.0. The PROCESS macro for SPSS (Hayes, 2012) was utilized to separately examine the hypotheses that resting RSA and parental supportive and invalidating responses to children’s negative emotions would interact to predict depressive symptoms. The PROCESS macro yields coefficient and standard error estimates for the predictor, moderator, and interaction term and is intended for use in moderation analyses that can be represented by a single regression coefficient. PROCESS estimates simple slopes at the sample mean of the moderator, as well as one standard deviation above and below the mean.

RESULTS

Variable means, standard deviations, and bivariate correlations for continuous variables are provided in Table 1. Variable means, standard deviations, and t-tests by sex are provided in Table 2. Depressive symptoms were positively correlated with higher levels of invalidating parental responses to youth negative emotions, and negatively correlated with resting RSA and supportive parental responses to youth negative emotions.

The PROCESS macro for SPSS (Hayes, 2012: Model 1) was used to conduct moderation analyses. Depressive symptoms were entered as the dependent variable. Resting RSA was entered as the independent variable and parental responses were included as the moderator. Two moderation models were tested, including one examining supportive parental responses and one examining invalidating parental responses as moderators. Child sex was entered as a covariate in both models. PROCESS mean-centered resting RSA and parental responses prior to analysis.

Results for the first model indicated that there was no main effect of Resting RSA ($B[SE] = 1.42 [1.12]$, $t = 1.28, p = .20$) or of Invalidating Parental Responses ($B[SE] = .66 [2.97]$, $t = .22, p = .83$). The Resting RSA x Invalidating Parental Responses term was also not significant ($B[SE] = .25 [0.41], t = .60, p = .55$).

Results for the second model indicated that with child sex, Supportive Parental Responses, and Resting RSA in the model, there was a significant main effect of Resting RSA on youth depressive symptoms ($B[SE] = –4.60 [1.93], t = –2.39, p = .02$). There was also a significant main effect of Supportive Parental Responses on youth depressive symptoms ($B[SE] = –6.20 [2.60], t = –2.38, p = .02$). The Resting RSA x Supportive Parental Responses interaction term was also significant ($B[SE] = .73 [.35], t = 2.04, p = .04$).

To interpret this interaction, we examined the effects of resting RSA on youth depressive symptoms at high levels of supportive parental responses (1 SD above the mean) and low levels of supportive parental response.
As expected, lower resting RSA only significantly predicted higher depressive symptoms in the context of low supportive parental responses to child negative emotions ($B[SE] = -1.75 [.61]$, $t = -2.87, p = .005$). Lower resting RSA was unassociated with depressive symptoms in the context of high supportive parental responses to child negative emotions ($B[SE] = -.12 [.53]$, $t = -.23, p = .82$). Results are presented visually in Figure 1.

**DISCUSSION**

Despite extensive evidence implicating low resting RSA as a biomarker of deficient emotion regulatory capacity, data on the association between resting RSA and depression specifically is mixed (Rottenberg, 2007). Beauchaine and colleagues have emphasized that the utility of autonomic physiology indices may be in specifying which children are most vulnerable to developing disorders when exposed to developmental contexts of liability and risk (Beauchaine, 2001; Zisner & Beauchaine, in press). Our study found that, consistent with this biological sensitivity to context model, low resting RSA was only associated with depressive symptoms among adolescents in the context of low supportive parenting. This finding significantly advances our understanding the specific developmental contexts in which low resting RSA may confer risk for depressive disorders.

Low resting RSA indexes biological emotion regulatory capacity and a strong body of literature links low resting RSA with internalizing symptoms, externalizing symptoms, poor emotion regulation skills, and depression among youth (see Zisner & Beauchaine, in press). However, developmental experiences may provide opportunities for youth to develop other manifest emotion regulation skills that may compensate for this biological vulnerability; by contrast, adverse developmental experiences may exacerbate biological risk. Parental responses to children’s negative emotions are a key context in which children develop emotion regulation skills. Extensive research suggests that parental responses to children’s negative emotions which encourage the expression of these emotions and facilitate problem-solving provide a supportive environment for children to develop adaptive emotion regulation skills (see, e.g., Eisenberg et al., 1998), which in turn set the stage for adaptive mental health development. By contrast, parents who fail to provide such supportive responses to children’s negative emotions and/or who provide punitive, dismissive, or minimizing responses have children who develop maladaptive emotion regulation skills and are more likely to experience mental health problems such as depression (Krause et al., 2003; Yap et al., 2008). Thus, we expected that parental responses to children’s negative emotions might be an important developmental context that would moderate the direct effect of resting RSA on depression outcomes.

Study results support the premise that low resting RSA may only confer risk for depressive symptoms in the context of receiving poor parental support when experiencing negative emotions. Without considering parenting as a moderator, low resting RSA was only very modestly correlated with depressive symptoms and inconsistently associated with depressive symptoms in regression analyses. These results support other studies finding that resting RSA-depression relationships may be strongest when other environmental or

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Table 2. Descriptive Statistics and Comparisons Among Study Variables by Child Sex

<table>
<thead>
<tr>
<th></th>
<th>Girls ($N = 63$)</th>
<th>Boys ($N = 57$)</th>
<th>T-test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>12.81 (.85)</td>
<td>12.94 (.83)</td>
<td>.86</td>
</tr>
<tr>
<td>Depressive Symptoms</td>
<td>5.45 (4.61)</td>
<td>3.77 (3.63)</td>
<td>2.22*</td>
</tr>
<tr>
<td>Supportive Responses</td>
<td>5.17 (1.00)</td>
<td>4.79 (1.26)</td>
<td>1.76</td>
</tr>
<tr>
<td>Invalidating Responses</td>
<td>2.49 (.95)</td>
<td>2.72 (1.00)</td>
<td>1.67</td>
</tr>
<tr>
<td>Resting RSA</td>
<td>7.14 (1.00)</td>
<td>7.09 (.86)</td>
<td>.26</td>
</tr>
</tbody>
</table>

*p < .05.
contextual moderating variables are taken into account (Blandon et al., 2008; El-Sheikh et al., 2001).

A full biological sensitivity to context model might suggest that youth with low resting RSA may be more sensitive to both adverse and protective environmental contexts, with these youth doing poorly under adverse conditions but perhaps faring better than their non-biologically sensitive peers under protective environmental contexts. However, our results suggested that low resting RSA was only associated with depression in the adverse context of low supportive parenting; in the protective context of high supportive parenting there was no association between RSA and depression, suggesting that low RSA youth were not reporting fewer depressive symptoms than their high RSA peers in similarly supportive contexts.

Interestingly, low parental support was a significant moderator of the resting RSA-depression relationship while high parental invalidation was not. While parental invalidation was modestly but significantly correlated with higher youth depressive symptoms, with covariates in the regression models this direct relationship was no longer significant nor did parental invalidation significantly moderate the effects of RSA on depressive symptoms. One explanation is that this was a community sample generally premorbid for clinically significant mental health problems and for which clinically significant depression at screening was an exclusion factor. Thus, there may have been a restricted range for the type of severe parental invalidation observed among more clinical samples. Although the mean and range for youth reported parental invalidation in our sample was comparable to that observed in other community samples of adolescents (see, e.g., Daughters, Gorka, Rutherford, & Mayes, 2014; DeBoard-Lucas et al., 2010; Suveg et al., 2011), the range of scores on the parental invalidation measure was more narrow than that observed on the parental support measure. In addition, parental invalidation has been most consistently linked with poor emotion regulation skills, non-suicidal self injury, and borderline personality disorder among clinical samples, which are outcomes less likely to be observed among a community sample of early adolescents (2005; Linehan & Kehrer, 1993; Tan, Rehfuss, Suarez, & Parks-Savage, 2014).

The current results should be interpreted with attention to the limitations of our sample and design. On the one hand, these findings are consistent with a growing body of literature on RSA and depression, which suggests that low resting RSA may only confer risk for depression in some contexts. The presence of moderating variables such as parental socialization of emotion may help explain prior mixed findings on the relationship between RSA and depression. The role of developmental context on the RSA-depression association among early adolescents is particularly salient, as this is a key developmental period for the emergence of emotion regulation skills that might offset the risk associated with low resting RSA. However, examining these relationships among community samples generally premorbid for clinical significant depression limits our ability to generalize these findings to clinical samples, as does examining them cross-sectionally and with entirely youth self-report of parenting and symptoms. Several of the resting RSA-depression nonfindings have been among adult samples with diagnosable Major Depressive Disorder, which suggests that RSA-depression relationships may vary by age or clinical status (see, e.g., Rottenberg et al., 2002). It will be important to follow this sample over time to examine the role of resting RSA and parenting in predicting increases in depressive symptoms and the emergence of depressive disorders over time.

Additionally, while this study focused on resting RSA, it is important to note that an increasing body of empirical evidence suggests that RSA reactivity may also be an important index of emotion regulation (Graziano & Derefniko, 2013). Individual differences in the ability to regulate vagal tone may be indexed by examining changes in RSA from baseline to challenging or stressful tasks, with vagal withdrawal hypothesized to facilitate regulatory processes critical for effective attentional, behavioral, and emotional control. Although blunted RSA reactivity has been found to be associated with disorders of emotion regulation such as externalizing problems (e.g., Beaufrechaine, 2001), the pattern of relationship between RSA reactivity and internalizing problems such as depression is mixed. Some researchers suggest that blunted RSA reactivity may be a correlate of depressive disorders representing the blunted emotional reactivity characteristic of the disorder (see, e.g., Yaroaslavsky et al., 2013). While a meta-analysis found that greater RSA reactivity was associated with fewer internalizing problems among children (Graziano & Derefniko, 2013), the pattern of findings varied as a function of both sample (clinical versus community) and methodology (how stressful the task). Given the emergent research on RSA reactivity and internalizing problems such as depression, future research may consider the joint effects of resting RSA and RSA reactivity on depression outcomes among youth as well as how these biological effects may be moderated by environmental context.

Finally, the current study was limited by its reliance on self-report data from the same informant (the child).
In the future, it will also be important to include more sophisticated measurement of environmental risk, particularly observed parenting in response to youth negative emotions.

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quality of parenting show concurrent and time-ordered associations that diverge in abusive, neglectful, and non-maltreating mothers. Couple and Family Psychology: Research and Practice, 2(2), 95–115. DOI: 10.1037/cfp0000005


