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

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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.

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., Rottenberg, Wilhelm, Gross, & Gotlib, 2002; Rottenberg, 2007).

Both developmental psychopathology and biological sensitivity to context models suggest that some individuals may be more biologically sensitive to environmental conditions (e.g., Ellis & Boyce, 2011; Zisner & Beauchaine, in press). Parenting is a key environmental context that is critical to children's development. In particular, the parenting provided when children experience negative emotions may be critical for children's development of emotion regulation skills and subsequent mental health outcomes (Eisenberg, Cumberland, & Spinrad, 1998; Eisenberg et al., 2001). Parental responses to children's negative emotions that are high in invalidation or low in support have been associated with numerous adverse mental health outcomes characterized by poor emotion regulation, including internalizing problems, aggression, and depression (Bilsky et al., 2013; Buckholdt, Parra, & Jobe-Shields, 2014; Crowell et al., 2013; Eisenberg et al., 2001). In the current study, we investigate whether individual differences in parasympathetic regulation of cardiac activity, indexed by resting RSA, may make some children more vulnerable to depressive symptoms in the context of invalidating or unsupportive parental responses to children's negative emotions.

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, Verhulst, & 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.

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, & Whitson, 2001; Katz & Gottman, 1995). By contrast, children with low resting RSA have been found to be particularly sensitive to the effects of childhood maltreatment such that low RSA children who experienced maltreatment showed markedly impaired executive functioning but low RSA children in non-maltreating environments actually displayed better executive functioning than their high RSA peers (Skowron et al., 2013). Similarly, low RSA children may be more sensitive to the effects of protective parenting, with at least one study finding that maternal protectiveness conferred benefits particularly for children with low RSA (Hastings, Kahle, & Nuselovici, 2014). Finally, Hinnant et al. (2015) recently found that the effects of lower resting RSA on externalizing outcomes was strongest for youth also exposed to harsh parenting.

If we wish to understand in which developmental contexts low resting RSA may confer risk for adolescent depression, it may be particularly important to examine parenting in response to youths' negative emotions. Substantial theory and research has suggested that parenting is a key component of the socialization of emotion, including children's ability to understand, experience, express, and regulate their emotions. In particular, parent's reactions to their children's negative emotions provide a salient developmental and interpersonal context in which children learn to understand and regulate their own emotions (Eisenberg et al., 1998). Supportive and validating parental responses to children's negative emotions provide important coaching for effective emotion regulation; supportive responses may include encouraging the healthy expression of emotion, providing assistance with problem-solving, and comforting children to alleviate distress. Low levels of these supportive parental responses to children's negative emotions have been associated with worse emotion

regulation skills, more internalizing symptoms, and more depressive symptoms (Buckholdt et al., 2014; Gottman, Katz, & Hooven, 1996; Thomas, DiLillo, Walsh, & Polusny, 2011).

In contrast with supportive parental responses, invalidating parental responses to children's negative emotions may interfere with the development of adaptive emotion regulation strategies (Crowell et al., 2013; Suveg, Shaffer, Morelin, & Thomassin, 2011). Invalidating responses include minimizing children's emotional experiences and/or punishing children for their experience or expression of negative emotions (Eisenberg et al., 1998; Linehan & Kehrner, 1993). Pervasive invalidation promotes dysfunctional emotion regulation capacities and high levels of invalidating parental responses to children's negative emotions 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, & Silva, 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 inter-beat 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 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 emotions¹.

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 ($B[SE] = .73 [.35]$, $t = 2.04$, $p = .04$).

¹*T*-tests were conducted to examine if there were mean differences in youth-reported parental invalidation or support when mother or father was the chosen parent reported on. There were no significant differences in mean invalidation ($M_{\text{mother}} = 2.51 (.91)$; $M_{\text{father}} = 2.69 (1.20)$; $t = .823$, $p = .412$) or mean support ($M_{\text{mother}} = 5.03 (1.09)$; $M_{\text{father}} = 4.94 (1.28)$; $t = .324$, $p = .717$). Therefore, to maintain overall power for analyses, we elected to conduct all analyses with pooled parental invalidation and support.

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 (1 *SD* below the mean). 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 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, nonsuicidal 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 & Derefinko, 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., Beauchaine, 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., Yaroslavsky et al., 2013). While a meta-analysis found that greater RSA reactivity was associated with fewer internalizing problems among children (Graziano & Derefinko, 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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References

- Bardone AM, Moffitt T, Caspi A, Dickson N. Adult mental health and social outcomes of adolescent girls with depression and conduct disorder. *Development and Psychopathology*. 1996; 8(4):811–829.10.1017/S0954579400007446
- Beauchaine TP. Vagal tone, development, and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology*. 2001; 13(2):183–214.10.1017/S0954579401002012 [PubMed: 11393643]
- Beauchaine TP. Physiological measures of emotion from a developmental perspective: State of the science: Physiological markers of emotion and behavior dysregulation in externalizing psychopathology. *Monographs of The Society For Research In Child Development*. 2012; 77(2): 79–86.10.1111/j.1540-5834.2011.00665.x [PubMed: 25242827]
- Belsky J, Pluess M. Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin*. 2009; 135(6):885–908.10.1037/a0017376 [PubMed: 19883141]
- Bilsky SA, Cole DA, Dukewich TL, Martin NC, Sinclair KR, Tran CV, Maxwell MA. Does supportive parenting mitigate the longitudinal effects of peer victimization on depressive thoughts

- and symptoms in children? *Journal of Abnormal Psychology*. 2013; 122(2):406–419.10.1037/a0032501 [PubMed: 23713500]
- Blandon AY, Calkins SD, Keane SP, O'Brien M. Individual differences in trajectories of emotion regulation processes: The effects of maternal depressive symptomatology and children's physiological regulation. *Developmental Psychology*. 2008; 44(4):1110–1123.10.1037/0012-1649.44.4.1110 [PubMed: 18605838]
- Blumberg SH, Izard CE. Discriminating patterns of emotions in 10- and 11-yr-old children's anxiety and depression. *Journal of Personality and Social Psychology*. 1986; 51(4):852–857.10.1037/0022-3514.51.4.852 [PubMed: 3783429]
- Bosch NM, Riese H, Dietrich A, Ormel J, Verhulst FC, Oldehinkel AJ. Preadolescents' somatic and cognitive-affective depressive symptoms are differentially related to cardiac autonomic function and cortisol: The TRAILS study. *Psychosomatic Medicine*. 2009; 71(9):944–950.10.1097/PSY.0b013e3181bc756b [PubMed: 19834052]
- Buckholdt KE, Parra GR, Jobe-Shields L. Intergenerational transmission of emotion dysregulation through parental invalidation of emotions: Implications for adolescent internalizing and externalizing behaviors. *Journal of Child and Family Studies*. 2014; 23(2):324–332.10.1007/s10826-013-9768-4 [PubMed: 24855329]
- Crowell SE, Baucom BR, McCauley E, Potapova NV, Fitelson M, Barth H, Beauchaine TP. Mechanisms of contextual risk for adolescent self-injury: Invalidation and conflict escalation in mother-child interactions. *Journal of Clinical Child & Adolescent Psychology*. 2013; 42:467–480.10.1080/15374416.2013.785360 [PubMed: 23581508]
- Daughters SB, Gorka SM, Rutherford HV, Mayes LC. Maternal and adolescent distress tolerance: The moderating role of gender. *Emotion*. 2014; 14(2):416–424.10.1037/a0034991 [PubMed: 24364854]
- DeBoard-Lucas RL, Fosco GM, Raynor SR, Grych JH. Interparental conflict in context: Exploring relations between parenting processes and children's conflict appraisals. *Journal of Clinical Child and Adolescent Psychology*. 2010; 39(2):163–175.10.1080/15374410903532593 [PubMed: 20390808]
- Eisenberg N, Cumberland A, Spinrad TL. Parental socialization of emotion. *Psychological Inquiry*. 1998; 9(4):241–273.10.1207/s15327965pli0904_1 [PubMed: 16865170]
- Eisenberg N, Losoya S, Fabes RA, Guthrie IK, Reiser M, Murphy B, Padgett. Parental socialization of children's dysregulated expression of emotion and externalizing problems. *Journal of Family Psychology*. 2001; 15(2):183–205.10.1037/0893-3200.15.2.183 [PubMed: 11458628]
- Ellis BJ, Boyce WT. Differential susceptibility to the environment: Toward an understanding of sensitivity to developmental experiences and context. *Development and Psychopathology*. 2011; 23:1–5.10.1017/S095457941000060X [PubMed: 21262035]
- El-Sheikh M, Harger J, Whitson SM. Exposure to interparental conflict and children's adjustment and physical health: The moderating role of vagal tone. *Child Development*. 2001; 72(6):1617–1636.10.1111/1467-8624.00369 [PubMed: 11768136]
- Fabes, RA.; Eisenberg, N. The coping with children's negative emotions scale - adolescent perception version: Procedures and scoring. Arizona State University; Tempe, AZ: 1998. Available from authors
- Fabes RA, Leonard SA, Kupanoff K, Martin CL. Parental coping with children's negative emotions: Relations with children's emotional and social responding. *Child Development*. 2001; 72(3):907–920.10.1111/1467-8624.00323 [PubMed: 11405590]
- Fabes RA, Poulin RE, Eisenberg N, Madden-Derdich DA. The coping with children's negative emotions scale (CCNES): Psychometric properties and relations with children's emotional competence. *Marriage and Family Review*. 2002; 34(3–4):285–310.10.1300/J002v34n03_05
- Fergusson DM, Horwood LJ, Ridder EM, Beautrais AL. Subthreshold depression in adolescence and mental health outcomes in adulthood. *Archives of General Psychiatry*. 2005; 62(1):66–72.10.1001/archpsyc.62.1.66 [PubMed: 15630074]
- Forbes EE, Fox NA, Cohn JF, Galles SF, Kovacs M. Children's affect regulation during a disappointment. *Biological Psychology*. 2006; 71:264–277. [PubMed: 16115722]

- Fruzzetti AE, Shenk C, Hoffman PD. Family interaction and the development of borderline personality disorder: A transactional model. *Development and Psychopathology*. 2005; 17(4):1007–1030.10.1017/S0954579405050479 [PubMed: 16613428]
- Gentzler AL, Santucci AK, Kovacs M, Fox NA. Respiratory sinus arrhythmia reactivity predicts emotion regulation and depressive symptoms in at-risk and control children. *Biological Psychology*. 2009; 82(2):156–163.10.1016/j.biopsycho.2009.07.002 [PubMed: 19596044]
- Gentzler AL, Rottenberg J, Kovacs M, George CJ, Morey JN. Atypical development of resting respiratory sinus arrhythmia in children at high risk for depression. *Developmental Psychobiology*. 2012; 54:556–567.10.1002/dev.20614 [PubMed: 21960203]
- Gottman JM, Katz LF, Hooven C. Parental meta-emotion philosophy and the emotional life of families: Theoretical models and preliminary data. *Journal of Family Psychology*. 1996; 10(3):243–268.10.1037/0893-3200.10.3.243
- Graziano P, Derefinko K. Cardiac vagal control and children's adaptive functioning: A meta-analysis. *Biological Psychology*. 2013; 94:22–37. [PubMed: 23648264]
- Hastings PD, Kahle S, Nuselovici JM. How well socially wary preschoolers fare over time depends on their parasympathetic regulation and socialization. *Child Development*. 2014; 85:1586–1600.10.1111/cdev.1222 [PubMed: 24527802]
- Hastings P, Nuselovici J, Utendale W, Coutya J, McShane K, Sullivan C. Applying the polyvagal theory to children's emotion regulation: Social context, socialization, and adjustment. *Biological Psychology*. 2008; 79:299–306. [PubMed: 18722499]
- Hayes, AF. PROCESS: A versatile computational tool for observed variable mediation, moderation, and conditional process modeling [White paper]. 2012. Retrieved from <http://www.afhayes.com/public/process2012.pdf>
- Hinnant JB, Erath SA, El-Sheikh M. Harsh parenting, parasympathetic activity, and development of delinquency and substance use. *Journal of Abnormal Psychology*. 2015; 124(1):137–151. [PubMed: 25688440]
- Katz LF, Gottman JM. Vagal tone protects children from marital conflict. *Development and Psychopathology*. 1995; 7(1):83–92.10.1017/S0954579400006350
- Kovacs, M. Children's depression inventory. 2. North Tonawanda, NY: Multi-Health Services Publishing; 2010. (CDI-2) manual
- Kovacs, MK. Childre s depression inventory. Toronto, Canada: Multi-Health, Inc; 1981.
- Kovacs M. The Children Depression Inventory. *Psychopharmacology Bulletin*. 1985; 21:995–998. [PubMed: 4089116]
- Krause ED, Mendelson T, Lynch TR. Childhood emotional invalidation and adult psychological distress: The mediating role of emotional inhibition. *Child Abuse and Neglect*. 2003; 27(2):199–213.10.1016/S0145-2134(02)00536-7 [PubMed: 12615094]
- Lewinsohn PM, Hops H, Roberts RE, Seeley JR, Andrews JA. 'Adolescent psychopathology: I. Prevalence and incidence of depression and other DSM-III—R disorders in high school students': Correction. *Journal of Abnormal Psychology*. 1993; 102(4):517.10.1037/0021-843X.102.4.517
- Light KC, Kothandapani RV, Allen MT. Enhanced cardiovascular and catecholamine responses in women with depressive symptoms. *International Journal of Psychophysiology*. 1998; 28(2):157–166.10.1016/S0167-8760(97)00093-7 [PubMed: 9545653]
- Linehan, MM.; Kehrer, CA. Borderline personality disorder. In: Barlow, DH., editor. *Clinical handbook of psychological disorders: A step-by-step treatment manual*. 2. New York, NY: Guilford Press; 1993. p. 396-441.
- Nelson JA, O'Brien M, Blankson AN, Calkins SD, Keane SP. Family stress and parental responses to children's negative emotions: Tests of the spillover, crossover, and compensatory hypotheses. *Journal of Family Psychology*. 2009; 23(5):671–679.10.1037/a0015977 [PubMed: 19803603]
- Porges SW. Cardiac vagal tone: A physiological index of stress. *Neuroscience And Biobehavioral Reviews*. 1995; 19(2):225–233.10.1016/0149-7634(94)00066-A [PubMed: 7630578]
- Porges SW. The polyvagal theory: Phylogenetic substrates of a social nervous system. *International Journal of Psychophysiology*. 2001; 42(2):123–146.10.1016/S0167-8760(01)00162-3 [PubMed: 11587772]

- Porges SW. The polyvagal theory: Phylogenetic contributions to social behavior. *Physiology and Behavior*. 2003; 79(3):503–513.10.1016/S0031-9384(03)00156-2 [PubMed: 12954445]
- Porges SW. The polyvagal perspective. *Biological Psychology*. 2007; 74(2):116–143.10.1016/j.biopsycho.2006.06.009 [PubMed: 17049418]
- Rechlin T, Weis M, Spitzer A, Kaschka WP. Are affective disorders associated with alterations of heart rate variability? *Journal of Affective Disorders*. 1994; 32(4):271–275.10.1016/0165-0327(94)90091-4 [PubMed: 7897091]
- Rottenberg J, Wilhelm FH, Gross JJ, Gotlib IH. Respiratory sinus arrhythmia as a predictor of outcome in major depressive disorder. *Journal of Affective Disorders*. 2002; 71(1–3):265–272.10.1016/S0165-0327(01)00406-2 [PubMed: 12167527]
- Rottenberg J. Cardiac vagal control in depression: A critical analysis. *Biological Psychology*. 2007; 74(2):200–211.10.1016/j.biopsycho.2005.08.010 [PubMed: 17045728]
- Rottenberg J, Chambers AS, Allen JB, Manber R. Cardiac vagal control in the severity and course of depression: The importance of symptomatic heterogeneity. *Journal of Affective Disorders*. 2007; 103(1–3):173–179.10.1016/j.jad.2007.01.028 [PubMed: 17320191]
- Shannon KE, Beauchaine TP, Brenner SL, Neuhaus E, Gatzke-Kopp L. Familial and temperamental predictors of resilience in children at risk for conduct disorder and depression. *Development and Psychopathology*. 2007; 19(3):701–727.10.1017/S0954579407000351 [PubMed: 17705899]
- Skowron EA, Cipriano-Essel E, Benjamin LS, Pincus AL, Van Ryzin MJ. Cardiac vagal tone and 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*. 2013; 2(2):95–115.10.1037/cfp0000005 [PubMed: 24729945]
- Suveg C, Shaffer A, Morelen D, Thomassin K. Links between maternal and child psychopathology symptoms: Mediation through child emotion regulation and moderation through maternal behavior. *Child Psychiatry And Human Development*. 2011; 42(5):507–520.10.1007/s10578-011-0223-8 [PubMed: 21484417]
- Tan AY, Rehfuess MC, Suarez EC, Parks-Savage A. Nonsuicidal self-injury in an adolescent population in Singapore. *Clinical Child Psychology And Psychiatry*. 2014; 19(1):58–76.10.1177/1359104512467273 [PubMed: 23209310]
- Thayer JF, Friedman BH, Borkovec TD. Autonomic characteristics of generalized anxiety disorder and worry. *Biological Psychiatry*. 1996; 39(4):255–266.10.1016/0006-3223(95)00136-0 [PubMed: 8645772]
- Thomas R, DiLillo D, Walsh K, Polusny MA. Pathways from child sexual abuse to adult depression: The role of parental socialization of emotions and alexithymia. *Psychology of Violence*. 2011; 1(2):121–135.10.1037/a0022469
- Yap MH, Allen NB, Ladouceur CD. Maternal socialization of positive affect: The impact of invalidation on adolescent emotion regulation and depressive symptomatology. *Child Development*. 2008; 79(5):1415–1431.10.1111/j.1467-8624.2008.01196.x [PubMed: 18826533]
- Yaroslavsky I, Rottenberg J, Kovacs M. Atypical patterns of respiratory sinus arrhythmia index an endophenotype for depression. *Development and Psychopathology*. 2014; 26:1337–1352.10.1017/S0954579414001060 [PubMed: 25422965]
- Zisner, AR.; Beauchaine, TP. Psychophysiological methods and developmental psychopathology. In: Cicchetti, D., editor. *Developmental psychopathology*. 3. Hoboken, NJ: Wiley; in press

FIGURE 1. Adolescent depressive symptoms predicted by resting RSA and supportive parental responses to youth negative emotions.

Table 1

Descriptive Statistics and Bivariate Correlations Among Study Variables

	1	2	3	4	<i>M</i>	<i>SD</i>	Range
1. Age	–				12.88	.83	11.35–14.61
2. Depressive Symptoms	.5	–			4.55	4.11	0–19
3. Supportive Responses	.18	–.27**	–		4.99	1.15	1.93–6.78
4. Invalidating Responses	.8	.22*	–.7	–	2.54	.98	1.00–4.89
5. Resting RSA	.9	–.19*	.9	–.2	7.12	.93	4.12–9.97

* $p < .05$.

** $p < .01$.

Table 2

Descriptive Statistics and Comparisons Among Study Variables by Child Sex

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

*
 $p < .05$.