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Associations between respiratory sinus arrhythmia reactivity and internalizing and externalizing symptoms are emotion specific

Christine K. Fortunato,
Department of Human Development and Family Studies, Pennsylvania State University, 110 South Henderson, University Park, Pennsylvania 16802, USA

Lisa M. Gatzke-Kopp, and
Department of Human Development and Family Studies, Pennsylvania State University, 110 South Henderson, University Park, Pennsylvania 16802, USA

Nilam Ram
Department of Human Development and Family Studies, Pennsylvania State University, 110 South Henderson, University Park, Pennsylvania 16802, USA; Max Planck Institute for Human Development, Berlin, Germany

Abstract

Internalizing and externalizing disorders are often, though inconsistently in studies of young children, associated with low baseline levels of respiratory sinus arrhythmia (RSA). RSA is thus considered to reflect the capacity for flexible and regulated affective reactivity and a general propensity for psychopathology. However, studies assessing RSA reactivity to emotional challenges tend to report more consistent associations with internalizing than with externalizing disorders, although it is unclear whether this is a function of the type of emotion challenges used. In the present study, we examined whether baseline RSA was associated with internalizing and/or externalizing severity in a sample of 273 young children (ages 5–6) with elevated symptoms of psychopathology. Following motivation-based models of emotion, we also tested whether RSA reactivity during withdrawal-based (fear, sadness) and approach-based (happiness, anger) emotion inductions was differentially associated with internalizing and externalizing symptoms, respectively. Baseline RSA was not associated with externalizing or internalizing symptom severity. However, RSA reactivity to specific emotional challenges was associated differentially with each symptom domain. As expected, internalizing symptom severity was associated with greater RSA withdrawal (increased arousal) during fearful and sad film segments. Conversely, externalizing symptom severity was related to blunted RSA withdrawal during a happy film segment. The use of theoretically derived stimuli may be important in characterizing the nature of the deficits in emotion processing that differentiate the internalizing and externalizing domains of psychopathology.

L. M. Gatzke-Kopp lmk18@psu.edu.

Keywords

Emotion; Respiratory sinus arrhythmia; Parasympathetic nervous system; Psychopathology; Internalizing; Externalizing

Respiratory sinus arrhythmia (RSA), a marker of parasympathetic control over cardiac arousal, is regularly utilized in the study of emotion reactivity and regulation (Beauchaine, 2001; Beauchaine, Gatzke-Kopp, & Mead, 2007; Oveis et al., 2009; Porges, 2001; Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012). Attenuated baseline RSA is broadly indicative of a decreased capacity to maintain regulatory control over affective arousal and is thought to reflect a general diathesis for pathologically dysregulated emotions (e.g., Beauchaine, 2001; Hinnant & El-Sheikh, 2009; Pine et al., 1998). Research has also indicated that greater RSA withdrawal (increased arousal) in response to an emotional challenge reflects greater attentional engagement with the emotional stimulus and a potential loss of regulatory control over affective arousal (Beauchaine et al., 2007; Calkins, Graziano, & Keane, 2007; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996). This association has been further supported by experimental studies demonstrating that adults instructed to regulate their emotional response exhibit an increase in RSA (Butler, Wilhelm, & Gross, 2006). Although both baseline RSA and RSA reactivity are implicated in dysregulated affective arousal, the results for baseline RSA have been more consistent across a wide range of psychopathologies, at least in adolescent and adult samples (see Beauchaine, 2001).

Whereas lower baseline levels of RSA are thought to reflect capacity for regulating emotional reactivity, and thus a generalized diathesis, it is less clear how changes in RSA in response to a stimulus are associated with psychopathology. Moderate levels of RSAwithdrawal in response to a challenge (reflecting a disengagement of the inhibitory control of the parasympathetic system and a consequent increase in physiological arousal) are considered adaptive and have been associated with positive outcomes, including better academic performance (e.g., Graziano, Reavis, Keane, & Calkins, 2007), executive functioning (e.g., Marcovitch et al., 2010), and social competence (e.g., Blair & Peters, 2003) in preschool- and kindergarten-aged children. However, extreme levels of RSA withdrawal are thought to reflect compromised control and dysregulated affect. Similarly, blunted RSA withdrawal or RSA augmentation (increasing parasympathetic control in response to a challenge, and thus decreasing physiological arousal) is also thought to reflect maladaptive responding. In addition to the level of RSA reactivity, the implications of individual differences in RSA reactivity in relation to symptoms of psychopathology may be informed by the nature of the emotional challenge. Specifically, internalizing symptoms may be associated with greater RSA withdrawal to withdrawal-based emotions (e.g., fear and sadness; Gray, 1982), whereas externalizing symptoms may be associated with greater RSA withdrawal to approach-based emotions (e.g., happiness and anger; Beauchaine, 2001).

Resting RSA as a generalized indicator of regulatory control of affective reactivity

Polyvagal theory suggests that the physical or cognitive capacity to respond to challenges in a rapid and controlled manner is driven, in part, by parasympathetic control of cardiac arousal, indexed via RSA (Berntson et al., 1997; Porges, 2001). In particular, parasympathetic withdrawal of neural cardiac control leads to immediate increases in heart rate and facilitates action readiness through the delivery of oxygen and glucose. Because parasympathetic control is neurally mediated, arousal can be modulated rapidly in response to changing situational demands. Indices of RSA have been proposed to reflect the output of medial prefrontal networks that integrate internal state with external cues in preparing the individual for appropriate environmental engagement (Thayer et al., 2012). As such, changes in RSA are believed to underlie affective responding to challenges (Butler et al., 2006), and higher levels of baseline RSA are proposed to reflect the extent of flexibility with which an individual can respond to changing environmental demands.

Given that low levels of baseline RSA suggest a reduced capacity to respond effectively to situational challenges, it is not surprising that most major psychiatric disorders have been associated with lower levels of resting RSA (see Beauchaine, 2001). However, the majority of researchers who have reported associations between domains of psychopathology and relatively low baseline RSA have assessed adolescents or adults (Beauchaine, Katkin, Strassberg, & Snarr, 2001; Carney et al., 2000; Friedman & Thayer, 1998; Gordis, Feres, Olezeski, Rabkin, & Trickett, 2010; Mezzacappa et al., 1997). The associations are more complicated when considered from a developmental perspective. Studies of early childhood have not revealed associations between baseline RSA and psychological traits as consistently as have studies conducted with older samples, leading some to suggest that reductions in baseline RSA may be an emergent product of dysregulated emotional lability (Beauchaine et al., 2007). Research with infants has indicated that RSA does not demonstrate the individual stability expected of a trait-like measure (Stifter & Fox, 1990; Stifter & Jain, 1996), suggesting that experiential processes continue to shape the functionality of this system early in life, as has been put forward in allostatic load models (e.g., McEwen, 1998). Some researchers have proposed that children reach adult levels of RSA by 5 years of age (Bornstein & Suess, 2000), yet several studies in this age range have not detected associations between baseline RSA and behavior problems, despite reporting findings for task-related RSA reactivity (Calkins et al., 2007; Crowell et al., 2006; Hastings et al., 2008; Skowron et al., 2011). In one longitudinal study in which children were assessed repeatedly across childhood (ages 8–11), decreases in resting RSA were observed among boys exposed to high levels of family stress and who demonstrated greater RSA reactivity to conflict (El-Sheikh & Hinnant, 2011). These findings support the conjecture that low baseline RSA reflects a developmental adaptation over time in response to chronic reactivity, and that such changes continue across childhood.

Since individual differences in baseline RSA may reflect consequences of adaptation to chronic RSA reactivity that are not readily apparent until later in life, individual differences in RSA reactivity to emotional challenges may be more closely associated with risk for the

development of psychopathology in early childhood. This hypothesis remains tenuous, given the dearth of research in early childhood and the fact that many samples have been composed predominantly of high-functioning individuals. Additional work is needed to determine whether associations between baseline RSA and psychopathology are substantiated in early childhood.

RSA reactivity as a specific marker differentiating externalizing and internalizing psychopathology

Unlike the association between lower resting RSA and increased rates of psychopathology in both internalizing and externalizing domains, associations with RSA reactivity are inconsistent across domains. Greater RSA withdrawal in response to negative affect inductions has been associated with increased internalizing symptom severity during the early school years and adolescence (Boyce et al., 2001; Calkins et al., 2007), as well as with parasuicidal behavior in adolescence (Crowell et al., 2005). In contrast, other studies have found either no association between RSA reactivity and externalizing problems in preschool children (Crowell et al., 2006), or less RSA withdrawal (and in some cases, RSA augmentation) to emotional inductions in school-aged children (Boyce et al., 2001; Calkins et al., 2007; Obradović, Bush, Stamperdahl, Adler, & Boyce, 2010). Although the findings are mixed, the general pattern of results suggests that externalizing and internalizing may be associated with opposing profiles of RSA reactivity. In sum, although low levels of baseline RSA are associated with externalizing disorders concurrently and prospectively in children between 6 and 11 years of age (Beauchaine et al., 2001; El-Sheikh, Hinnant, & Erath, 2011), the risks associated with greater RSA withdrawal appear to be more specifically aligned with internalizing disorders across childhood (e.g., Boyce et al., 2001; Monk et al., 2001).

Notably, differences in reactivity across studies and symptom domains may relate to the specific nature of the emotional challenges employed. Although both internalizing and externalizing disorders are often characterized by dysregulated affect, they are also frequently distinguished with regard to which emotion systems are dysregulated. In brief, biologists have theorized that basic affective experiences are a manifestation of neural systems designed to motivate contextually matched behavioral responding (Gray, 1987; McNaughton & Corr, 2009). These motivational systems consist of two networks: approach and avoidance. Approach systems are engaged when the opportunity for reward or the need to overcome an obstacle is present, requiring behavioral activation. Affectively, this is believed to correspond to subjective senses of happiness or joyful anticipation and of anger, respectively. In contrast, avoidance systems are activated when an individual faces uncertainty that could signal risk (McNaughton & Corr, 2004). This system has been largely conceived as generating a subjective state of anxiety, but it may also be associated with the depressive affect associated with withdrawal from the environment.

Externalizing psychopathology has been proposed to reflect disruptions in the neural systems subserving *approach-motivated* emotions (e.g., Beauchaine, 2001; Gray, 1987). Most research has focused specifically on anger in this domain (e.g., Eisenberg et al., 2000; Scarpa & Raine, 1997), although neurobiological models of approach-motivated emotions include both positively (happiness) and negatively (anger) valenced emotions (Carver &

White, 1994; Gray, 1990, 1994; Harmon-Jones & Sigelman, 2001; Watson, 2000). In contrast, internalizing psychopathology is thought to reflect disruptions in neural systems that activate *withdrawal-motivated* emotions (e.g., Beauchaine, 2001; Gray, 1987) including fear and sadness (e.g., Eisenberg et al., 1996; Gray, 1982; Rothbart & Bates, 1998).

The majority of studies examining RSA reactivity to emotion in childhood have utilized stimuli meant to induce withdrawal-motivated emotions, primarily fear and/or sadness (e.g., Beauchaine et al., 2007; Crowell et al., 2006). Therefore, on the basis of neurobiological models, it is not surprising that the risks associated with greater RSA withdrawal are typically associated with internalizing problems (e.g., Boyce et al., 2001; Monk et al., 2001). The lack of association between RSA withdrawal in response to emotion and externalizing symptom severity may be a function of stimuli that are not theoretically aligned with the nature of emotional dysregulation that is hypothesized to contribute to externalizing behavior. That is, some studies examining externalizing symptoms have not attempted to induce approach-motivated emotions that may highlight the specific deficiencies in emotion processing that are relevant to externalizing disorders (e.g., Boyce et al., 2001; Obradović et al., 2010). Studies that have utilized frustration (e.g., Calkins et al., 2007; Hinnant & El-Sheik, 2009)—which is thought to engage approach-motivated anger responding so as to overcome challenge—and happiness conditions (e.g., Calkins et al., 2007) during early and middle childhood have reported associations between externalizing behaviors and RSA augmentation, suggesting that externalizing symptoms may in fact be associated with RSA reactivity only to approach-oriented conditions.

The present study

In the present study, we examined the associations between baseline RSA and RSA reactivity to two withdrawal-oriented (fear, sadness) and two approach-oriented (happiness, anger) emotion inductions in a sample of 273 kindergarten-age children representing the full range of internalizing and externalizing symptom severity. Overall, a motivation-based emotion model suggests that exaggerated RSA withdrawal in response to fear- or sadness-inducing stimuli (withdrawal-motivation emotions) will be specifically associated with internalizing symptoms of psychopathology (e.g., sadness and anxiety), whereas RSA withdrawal in response to anger stimuli (approach-motivation emotion) will be specifically associated with externalizing symptoms of psychopathology (e.g., delinquency and aggression). The hypotheses regarding reactivity to happy stimuli are less clear, given that both internalizing and externalizing disorders are characterized by a lack of positive affect. However, consistent with previous reports (Calkins et al., 2007) and the approach-motivation model, we hypothesized that attenuated RSA withdrawal to positive emotion induction may be especially evident in association with externalizing symptoms.

Method

Participants and procedure

The data for the present analysis were drawn from a larger clinical trial of an intervention program for early-onset aggression. The clinical trial was implemented in cooperation with ten elementary schools in a district serving a low-income urban community in which 78 %

of the students in the catchment area for the district are classified as low-income. The majority of households in the district are headed by a single female (61 %), and only 79 % of the parents are estimated to have a high school diploma. In this region, property crimes are two times as high, and violent crimes are 4.5 times as high, as the statewide averages.

Prior to the study, kindergarten teachers were asked to complete a short screening form assessing aggressive behaviors (the Teacher Observation of Child Adaptation–Revised; Werthamer-Larsson, Kellam, & Wheeler, 1991) for each child in their classroom. From these reports, 339 children (64.3 % male; mean age = 6.03 years, *SD* = 0.37) and their parents were recruited into the study. A total of 207 children from the upper 20 % of the aggression ratings within each classroom were enrolled and randomly assigned to the intervention or control condition. As well, an additional 132 comparison children were recruited from the lowest 20 % of the teacher ratings for aggressive behavior, matched for sex and classroom. Because the present analysis only incorporated data from the preintervention assessment, intervention status will not be considered further. Consistent with the racial demographics of the region, the full sample was 70 % African American, 20 % Latino, 9 % Caucasian, and less than 1 % Asian. Further details can be found in Gatzke-Kopp, Greenberg, Fortunato, and Coccia (2012).

For the present analysis, the complete data on teacher-reported symptoms of psychopathology, as well as physiological reactivity during the emotion induction paradigm, were available for 273 of the 339 children in the sample. This subsample did not differ from the larger sample with respect to race [$\chi^2(2) = 1.57$], sex [$\chi^2(1) = 0.20$], age ($t = 1.15$), or original recruitment category [$\chi^2(2) = 3.18$], all *p*s > .20.

Psychopathology symptoms—The children's levels of internalizing and externalizing problems were measured using teacher responses to the brief version of the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997). Specific to the present inquiry, teachers rated their students' levels of *emotional symptoms* (five items; Cronbach's $\alpha = .80$; e.g., "Many worries or often seems worried") and *conduct problems* (five items; Cronbach's $\alpha = .86$; e.g., "Often fights with other children or bullies them") using a three-point Likert-type scale (0 = *not true*, 1 = *somewhat true*, 2 = *certainly true*). The items for each subscale were summed (range 0–10) and used as indicators of internalizing and externalizing symptoms, respectively. Internalizing ($M = 1.88$, $SD = 2.3$) and externalizing ($M = 2.45$, $SD = 2.78$) symptom scores were both normally distributed across the entire 0–10 range and were moderately correlated, $r = .31$, $p < .001$.

Although children were recruited into the study on the basis of aggressive behavior, recruitment did not follow a clinical threshold model. Rather, the children with the highest relative levels of aggression within each classroom were recruited and matched with children with relatively lower levels of aggression. Because symptom severity was normally distributed in the sample and spanned the full range of the scale, these measures are treated as continuous (rather than nominal) variables.

Emotion induction paradigm—Psychophysiological assessments were conducted in a recreational vehicle that was driven to each school site. Inside the mobile laboratory,

children participated in two tasks, during which electrodermal, cardiac, and electroencephalography (EEG) measures were recorded. Specific to the present study, children participated in a 12-min emotion induction task. Previous work had indicated that movie clips are especially effective in inducing emotion (Schaefer, Nils, Sanchez, & Philippot, 2010). Following previous work that had used an excerpt from the film *The Lion King* in which Simba finds his father dead to induce sadness in children (von Leupoldt, Rohde, Beregova, Thordsen-Sörensen, zur Nieden, & Dahme et al., 2007), stimuli for each of the four emotion categories were drawn from the *The Lion King* in order to maintain consistency across the emotion categories (see also Gatzke-Kopp et al., 2012, and, for EEG data related to this task, Gatzke-Kopp, Jetha, & Segalowitz, 2012). This film has considerable appeal for emotion induction, because it consists of multiple streams of emotion-conveying sensory information that have been documented to successfully induce mood (see Lench, Flores, & Bench, 2011)—namely, facial expression, contextual storyline, and musical score. Furthermore, the target character is a cartoon lion, and thus bypasses concerns over racial matching between the protagonist and participant that might enhance the clips' saliency for some participants and not others (see Roberts & Levenson, 2006). The emotions induced by the four clips were as follows: fear (the hyenas chasing Simba, the main character; ~3 min), sadness (Simba's father dying; ~2 min), happiness (characters singing a joyous song; ~2 min 30 s), and anger (Simba fighting Scar, the evil antagonist; ~2 min). The film clips were presented in chronological order (to avoid confusion about Simba's development over the film). After each emotion clip, a 30-s neutral clip was shown to help reduce carryover effects of the previous emotion. The neutral clip, which did not contain any emotional content, was extracted from the film in chronological sequence between its two flanking emotion clips and portrayed the resolution of the previous clip (e.g., showing that Simba has escaped the hyenas and is fine). The neutral clip was then followed by a 30-s fixation baseline to establish a baseline for the next emotion in the sequence. Presentation and timing (for synchronization with the electrocardiographic [ECG] recordings) were controlled by E-Prime II software (Psychological Software Tools, Inc., Sharpsburg, PA).

Measures

Respiratory sinus arrhythmia—During the emotion induction task, cardiac measures were collected continuously at 500 Hz via the Biolab 2.4 acquisition system (Mindware, Westerville, OH). Three disposable, pre-gelled cardiac electrodes were placed over the child's distal right collar bone, lower left rib, and lower right rib. The ECG data were visually inspected and corrected as necessary by a trained research assistant. Because the validity of RSA may be compromised if participants' respiration frequency exceeds a normal range, respiration was estimated from the impedance wave (measured with an additional four cardiac electrodes) in order to verify that the respiratory frequency within each epoch was within the 0.12–0.40 Hz range established for RSA calculation (Allen, Chambers, & Towers, 2007; Berntson, Quigley, & Lozano, 2007). A peak respiration frequency outside this range resulted in elimination of that epoch from analysis (<1 % of available epochs were removed). For all viable epochs, RSA scores were derived through spectral analysis (fast Fourier transform) of the interbeat interval time series obtained from the ECG following procedures specified by Berntson et al. (1997).

RSA was calculated in 30-s epochs across the task, resulting in a total of 27 epochs, including baseline and the four emotion-inducing conditions. When repeated across multiple epochs, 30 s is an appropriate length of time for valid RSA extraction, allowing for maximum sensitivity of the RSA calculation to dynamic changes across the task (Berntson et al., 1997). Epoch 1 reflects the initial (pretask) baseline, Epochs 2 through 9 reflect the fear condition, Epochs 10 through 14 reflect the sadness condition, Epochs 15 through 21 reflect the happiness condition, and Epochs 22 through 27 reflect the anger condition. Each emotion condition included the fixation baseline epoch that preceded it and the neutral film clip epoch that followed it.

Data analysis

Multiphase latent basis growth curves (see Ram & Grimm, 2007) were used to model nonlinear changes in RSA over the course of the emotion induction task. In brief, this model provides an alternative representation of the change trajectories often modeled via polynomial models (e.g., quadratic or cubic) and is particularly useful for representing complex-shaped trajectories in a parsimonious manner (McArdle & Epstein, 1987; Meredith & Tisak, 1990). Specifically, the conditional five-factor latent-basis growth curve model shown in Fig. 1 was used to model systematic nonlinear changes in RSA across the 27 epochs that were associated with the baseline, fear, sadness, happiness, and anger conditions and how between-person differences in baseline levels and in the extents of change in RSA during the four emotion-inducing conditions were related to differences in internalizing and externalizing problems.

The 27 repeated measures of RSA (series of squares in Fig. 1) were modeled as a function of five latent growth factors (larger circles) and a series of unique/error factors (smaller circles along the bottom, assumed to have homogeneous variance). The factor loadings (arrows connecting larger circles to squares) were estimated from the data (with minimal identification constraints) as in confirmatory factor analysis and are indicative of the shape of the prototypical trajectory of RSA during each segment of the task. The baseline, fear, sadness, happiness, and anger factors are latent growth factors, the individual scores that indicate individuals' baseline levels of RSA (baseline factor) or the extent of their RSA reactivity during each specific emotion. Associations between these factor scores and children's levels of internalizing and externalizing symptoms are captured by the regression parameters connecting the symptom scores (squares at top) with the latent factors.

Note that this modeling framework provides some advantages over a typical analysis of baseline-to-task difference scores (e.g., differences between the average RSA during an emotion block and baseline RSA) in this context. Specifically, the latent-basis model allows us to relax the assumption that each video film clip provided a perfectly homogeneous emotion stimulus. Instead, the model allows for the fact that each video delivered different levels of emotion stimulus across its 2–3 min of view time. Changes in the extents of RSA reactivity produced are captured by the nonlinear shape of the growth curve during each phase of the induction procedure (baseline, fear, sadness, happiness, and anger). However, the model also maintains concordance with experimental procedures in which all of the

children watched the same clips, in that all persons are assumed to follow the same shape trajectory and to differ only in the extent of reactivity.

The model was fit to the data using Mplus 6.2 (Muthén & Muthén, 1998–2012; further details and syntax examples can be found in Ram & Grimm, 2007) with full-information maximum likelihood and standard missing-at-random assumptions (Little & Rubin, 1987). Of specific interest for our research question were the relations between the internalizing and externalizing symptom variables and the five growth factors.

Results

Baseline RSA and externalizing and internalizing symptoms

We first examined the relationship between resting RSA and symptoms of internalizing and externalizing psychopathology. Neither RSA at the initial baseline ($r = -.08$, $p = .27$; $r = .01$, $p = .86$) nor RSA at the baseline preceding the emotion induction task ($r = -.03$, $p = .62$; $r = -.04$, $p = .52$) was significantly correlated with internalizing or externalizing symptom scores, respectively.

Task validation

Prior to the examination of individual differences in the growth trajectories of each emotion, we conducted a series of paired-sample *t* tests to determine whether the emotion induction paradigm was successful in inducing significant RSA reactivity and whether that reactivity was appropriately differentiated between the emotions. The average RSA levels in each condition (pretask baseline, fear, sadness, happiness, anger, and fixation baselines between each emotion clip) were systematically contrasted, and the results are presented in Table 1. As hypothesized, the average RSA was lowest (indicating the highest arousal) during the angry clip (average levels of RSA appear along the diagonal), a value that was significantly different from the average RSA during the happy and sad clips, and marginally ($p = .06$) different from that during the fearful clip (mean differences in RSA are reported in each cell). The fear condition was associated with the next lowest level of RSA, followed by the happiness and sadness conditions, which did not differ from each other. Furthermore, average RSA was consistently higher during the fixation baselines, which followed a 30-s “recovery” or neutral clip prior to onset of the next emotion, than in any of the emotion conditions and did not differ from the initial baseline levels of RSA. This provides strong evidence that children recovered between the emotion-specific clips and that the arousal induced during any given film clip did not carry over into subsequent clips. Additionally, evidence that arousal peaked in the first and last clips strongly suggests that linear effects of time (fatigue, boredom, emotion accumulation, etc.) did not impact the results. Having established the success of the induction paradigm at a general level, we proceeded to describe the specific (nonlinear) patterns of RSA reactivity and their associations with internalizing and externalizing symptoms.

Prototypical RSA reactivity to the emotion-inducing paradigm

Model parameters and fit statistics for the final model are shown in Table 2. As can be seen in the lower rows of the table, the overall fit of the model to the data was good (RMSEA = .

04, CFI = .97, and NNFI = .97; Hu & Bentler, 1999). We will consider the nonlinear changes in RSA and the associations of between-person differences in those changes with internalizing and externalizing symptoms in turn.

The latent-basis factor loadings (upper portion of table) reflect the prototypical pattern or shape of change in RSA across the task. On average, the baseline level of RSA, defined as a flat line (loadings for all 27 repeated measures are constrained to equal 1.00), located at the initial epoch (y_0) and the fixation epochs before and after each film clip, was 6.48 ms^2 (*SE* = 0.06, $p < .05$), with interindividual differences around that level. Consistent with the content of the first video clip, the pattern of factor loadings for the Fear factor (Epochs 2 through 9) reflected that total reactivity was proportioned across the eight segments, with reactivity distributed from 7 % at the initial epoch (fixation cross), to 36 % during the second 30-s epoch, to 73 % during the third epoch, hovering at 68 % during the fourth epoch, to greater and greater proportions up to 100 % at the seventh epoch, and returning back to 36 % during the final 30-s neutral-film segment. On average, the total RSA reactivity (= 100 %) during the fear segment amounted to −0.40 ms^2 (*SE* = 0.06). The latent-basis factor loadings for the other clips followed a similar interpretation.

The nonlinearity of change and the differential reactivity evoked by the different emotional contents of each of the four clips are clearly evident (see the dotted lines in Fig. 2). Observationally, the pattern of change matched the timing of the climax of content within each clip (e.g., the emotional content of the anger clip builds in inverse fashion to the pattern of RSA withdrawal). As expected, average RSA reactivity (withdrawal) during the fear (*M* = −0.40, *SE* = 0.06) and anger (*M* = −0.38, *SE* = 0.06) inductions was more extensive than during the sadness (*M* = −0.18, *SE* = 0.04) and happiness (*M* = −0.30, *SE* = 0.06) inductions. The between-person differences in the extents of RSA reactivity during each of the emotion-eliciting clips were correlated, with *r*s among the factors ranging from .68 to .87, *p*s < .01 (obtained in a model without covariates).[1] That is, children who had greater RSA withdrawal during one of the emotion-eliciting film clips (e.g., fearful) were also likely to have greater RSA withdrawal than their peers during all of the other film clips (e.g., sad, happy, and angry).[2]

Relation between RSA reactivity and internalizing and externalizing symptoms

Our main substantive interest was to determine whether RSA reactivity patterns to specific emotional experiences (fear, sadness, happiness, and anger) were differentially associated with internalizing and externalizing symptoms of psychopathology. Note that race, sex, and age were not significantly related to RSA reactivity and did not moderate the associations with internalizing and externalizing symptoms, and thus, for parsimony of presentation, these variables were not included in the final model.

Internalizing symptoms—As can seen in the bolded rows in Table 2, higher levels of internalizing symptoms were associated with greater RSA withdrawal (increased arousal)

[1]We report correlations here, but note that the corresponding values from the conditional model in Table 2 are covariances.
[2]Follow-up analyses indicated that initial baseline levels of RSA did not moderate the extent of reactivity to emotions or the association between the extents of reactivity to emotion and symptom severity.

during the fear (b = −0.05, SE = 0.02, p = .05; standardized β = −.19, SE = .09, p = .03) and sadness (b = −0.04, SE = 0.02, p = .05; standardized β = −.22, SE = .10, p = .03) inductions. As is shown in Fig. 2A, higher internalizing (i.e., +1.5 *SD*) was associated with greater RSA withdrawal (or a larger downward "stretch" from baseline than the prototypical curve) during the fear and sadness inductions, but was not systematically associated with the magnitude of RSA reactivity during the happiness and anger inductions.

Externalizing symptoms—In contrast to children's internalizing symptoms, higher levels of externalizing symptoms were associated with blunted RSA withdrawal (no or minimal change in arousal) during the happiness induction (b = 0.05, SE = 0.02, p = .02; standardized β = .18, SE = .09, p = .04). Contrary to predictions, no association was found between RSA reactivity during the anger scene and externalizing symptoms. Figure 2B illustrates the blunted RSA withdrawal (or a smaller downward "stretch" from baseline than the prototypical curve) during the happiness condition that was characteristic of individuals higher in externalizing (+1.5 *SD*). Consistent with predictions, no association was found between externalizing severity and the magnitude of RSA reactivity during the fear and sadness inductions.

The interaction between internalizing and externalizing symptoms was also tested in order to evaluate whether associations were a function of total symptom severity, beyond the symptoms in each domain. No relationship was found between the interaction and RSA patterns, suggesting that these associations are indeed domain-specific.

Discussion

In this analysis, we examined how baseline RSA and RSA reactivity to emotional challenges were associated with internalizing and externalizing symptoms in a sample of young children. Contrary to findings in adolescents and adults, we found no evidence that reduced baseline RSA was related to either internalizing or externalizing symptom severity. In line with predictions based on the neurobiological motivation model, we found that internalizing and externalizing symptoms were differentially related to RSA reactivity to withdrawal-motivated and approach-motivated emotions, respectively.

Baseline RSA and internalizing and externalizing symptoms

The co-occurrence between externalizing and internalizing symptoms exceeds the true comorbidity that would occur by chance, strongly suggesting a common diathesis. Low resting baseline RSA has been proposed to reflect a common vulnerability toward dysregulated emotion (Beauchaine, 2001; Kopp & Beauchaine, 2007). Although associations between psychopathology and baseline RSA have been found in studies of adolescents and adults, these associations do not always manifest in studies of young children. For instance, Calkins et al. (2007) found that associations between behavior problems and RSA were confined to laboratory challenge tasks and were not evident at baseline in a sample of 335 five-year-old children. Likewise, no differences were found between baseline measures of RSA in a sample of children 4–6 years old who were diagnosed with ADHD and oppositionality (Crowell et al., 2006). This has led some researchers to postulate that associations between RSA and psychopathology are not readily

detectable until later in childhood, due to a more limited range of functionality at this age truncating individual differences that will become evident as normally developing children expand into greater emotion regulatory capacity (Beauchaine et al., 2007). In line with this perspective, our results suggest that baseline RSA is not associated with the severity of either internalizing or externalizing symptoms (or, in additional follow-up analyses, their interaction) at the age of 6 years. It is possible that patterns of reactivity evident in early childhood will lead to a compensatory adjustment in baseline RSA over time, at which point relatively lower baseline levels may begin to reflect the consequences of psychopathology as well as directly contributing to its continuity. This hypothesis suggests that reactivity profiles are especially important to study in early childhood and that this stage may reflect a developmental window of opportunity for early intervention with high-reactivity children prior to the canalization of trait-like RSA.

RSA reactivity and internalizing or externalizing symptoms

Consistent with the notion that RSA withdrawal facilitates increases in arousal supporting active engagement with a stimulus (Frazier, Strauss, & Steinhauer, 2004; review, Kreibig, 2010), RSA withdrawal was evident across all four emotion inductions used in this study, and the patterns of reactivity were aligned with expected differences in the magnitudes of arousal associated with the different emotions (i.e., anger > fear > happiness > sadness). Consistent with the findings of a recent meta-analysis of mood induction studies (Lench et al., 2011), levels of RSA did not differ significantly between the happiness and sadness conditions. Thus, children appeared, on average, to respond appropriately to the emotion induction paradigm.

In support of the main hypotheses, individual differences in degrees of RSA reactivity to withdrawal-based and approach-based emotions were differentially associated with internalizing and externalizing symptom severity. Moreover, that the interaction between internalizing and externalizing symptoms was not additionally associated with RSA withdrawal in any condition suggests that the associations were not a function of general symptom severity, but coincided with severity of the symptoms in a specific domain. Importantly, these findings are not challenged by the phenomenon of comorbidity, because the patterns of RSA reactivity were not mutually exclusive. In other words, internalizing and externalizing symptoms were not associated with opposing profiles of RSA reactivity, but rather with unique profiles of RSA reactivity that could potentially coexist in a single individual.

Internalizing symptoms—Internalizing symptom severity was associated with more exaggerated RSA withdrawal in response to fear and sadness emotion inductions, but not in response to happiness or anger inductions. These findings support the hypothesis that the association between internalizing symptoms and greater RSA withdrawal is specific to withdrawal-motivation-based emotions. More exaggerated withdrawal of RSA in conditions of fear and sadness may be reflective of heightened attentional engagement with withdrawal-based stimuli in individuals high in internalizing emotionality (Hinnant & El-Sheikh, 2009; Thayer & Lane, 2000) and may place these children in a position where

regulating emotional arousal is more difficult, leading to more intensive emotional experiences.

Externalizing symptoms—Consistent with our hypotheses, externalizing symptom severity was not associated with RSA reactivity to fear and sadness inductions. However, the hypothesis that exaggerated RSA withdrawal would be associated with externalizing severity if examined in response to the approach-based emotion of anger was not supported. Affective reactivity to anger is considered to be characteristic of externalizing disorders, engendering oppositionality, defiance, and retribution types of behavior (Robinson & Wilkowski, 2010). The lack of association in this study may be a function of several factors. First, very little research has incorporated anger, as distinct from fear or other negative affect categories, and it is possible that passive film viewing was not sufficient to induce a subjective sense of anger and that previous studies have used more active conditions of frustration (e.g., Calkins et al., 2007; Hinnant & El-Sheikh, 2009). However, RSA levels were lowest during the anger clip, suggesting that children did respond physiologically and were even lower than in the fear condition. Therefore, it appears that the visual and affective intensity of the anger clip induced robust RSA withdrawal across the entire sample, creating a ceiling effect because of which specific associations with symptom patterns were impossible to detect. It is also possible that RSA withdrawal alone may not be sufficient to explain externalizing symptoms, requiring an assessment of concurrent sympathetic reactivity as a potential moderator of the association between RSA and externalizing behavior (see Beauchaine, 2001; Beauchaine et al., 2007).

Although no association was found between externalizing symptoms and the anger condition, significant effects were found for the happiness condition. In line with previous findings (Calkins et al., 2007), blunted RSA reactivity during the happiness condition was associated with externalizing symptoms in children. Relatively muted RSA reactivity to the happiness condition supports the hypothesis that externalizing psychopathology is characterized by affective reactivity to approach-motivated rather than withdrawal-motivated emotions. These findings highlight the value of utilizing positive emotional conditions in examining emotional reactivity, rather than restricting stimuli to negatively valenced emotions.

The association between externalizing symptoms and blunted reactivity to the happiness condition is consistent with models of conduct problems suggesting pathologically blunted arousal and muted reactivity, particularly to low-intensity stimuli (for a review, see Raine, 2002). This association is also consistent with models of externalizing behaviors as reflecting deficits in approach motivation (see Beauchaine, 2001). Low reactivity to mild-intensity positive affect has been proposed to underlie motivation to engage in high-intensity, sensation-seeking activities and to reduce the effects of mild reinforcements, such as praise, in socializing children's behavior (see Gatzke-Kopp & Beauchaine, 2007). The drive to seek high-intensity experiences could lead to a range of behaviors from risky play (e.g., dangerous stunts on the playground, breaking safety rules, and engaging in dares) to antisocial behaviors (such as bullying) in order to achieve a sense of dominance and control.

Unlike the negative mood clips, which demonstrate an initial decline in RSA that stabilizes for the duration of the clip, the profile for the happiness condition consisted of fluctuation in RSA levels across the duration of the clip. The dynamic profile of RSA response to the happy clip is likely a stimulus-specific function of the nature of the clip used rather than an indication that reactivity fluctuates more in response to happy than to negative emotions. Previous research examining autonomic reactivity to five distinct categories of positive emotion found amusement (most similar to the nature of the clip used in this study) to be associated with the lowest degree of RSA withdrawal (Shiota, Neufeld, Yeung, Moser, & Perea, 2011). This suggests that individual differences in RSA reactivity to amusement may be more difficult to detect when examined as an average across the duration of the emotion. However, using nonlinear growth curves, we were able to detect individual differences in the dynamic reactivity profiles associated with the happy mood clip that would most likely be obscured by averaging across the entire clip, given that the level of positive affect being induced was not necessarily distributed uniformly over the 2.5 min.

Limitations and future directions

It is important to note that although the measures of internalizing and externalizing symptoms were significantly associated with RSA reactivity, they accounted for a relatively small portion of the observed between-person differences ($R^2 < 5$ %). As might be expected, parasympathetic response to affective experience is but one of a multitude of influences on symptoms of psychopathology. However, relations between teacher reports of symptoms and RSA reactivity were on the order of $\beta = .18$ to .22, indicating a reasonable effect, the detection of which may be enhanced by efforts to reduce noise and better account for relevant between-person differences in future studies. For instance, more comprehensive clinical characterization of children's functioning by trained reporters might provide a more precise understanding of RSA reactivity and its relation to the development of mental health than can sole reliance on teacher reports. Furthermore, while the induction procedures did produce RSA reactivity effects of moderate sizes (fear, $d = -0.40$; sadness, $d = -0.18$; happiness, $d = -0.30$; anger, $d = -0.38$), several factors warrant consideration.

The fixed presentation order of the clips may have introduced confounds of timing that, for instance, could have resulted in the lack of significance for the anger clip, which was always presented last. However, RSA levels did not appear to follow a linear function of time, indicating that any confounding introduced by order effects might be complex. In addition, the emotional reactivity to each clip was only assessed from the physiological measures. Because of the young age of our participants, the children were not asked to provide self-reports of their subjective emotional experiences. While the patterns of RSA reactivity across the emotion conditions, as well as the significant associations between RSA reactivity and levels of psychopathological symptoms, suggest that children in this age range are capable of experiencing affect-congruent physiological reactivity to passively viewed scenes, it is not possible to verify that the child responded in an affectively matched manner. For instance, a child demonstrating an exaggerated response to the sad clip might have been experiencing high levels of subjective fear. Although this concern does not compromise the value of the findings reported here, in that such "mismatched" affective responding may underlie vulnerability to internalizing symptoms, future research might consider how facial

coding procedures can be used to identify the specific emotions that each child experiences during such inductions.

Finally, with respect to the induction procedures, blunted reactivity to a passive induction should not be taken as evidence that an individual would not exhibit exaggerated withdrawal in an active, personally relevant emotion-inducing situation. Active emotion induction paradigms (e.g., directed facial action, interactive) may be especially useful for inducing approach-motivated emotions such as anger. Studies that have utilized frustration-inducing laboratory tasks with children have found associations between externalizing symptom severity and RSA augmentation (e.g., Hinnant & El-Sheikh, 2009). Thus, induction techniques that are experienced directly by the participant (e.g., goal blocking) may be more ecologically valid and effective in inducing physiological change than is simple empathic identification with a protagonist depicted in film.

It is also important to emphasize the considerable differences in a range of demographic variables (race, socioeconomic status, and contextual risk factors) between our sample and those in the majority of published studies. While this makes a significant contribution to diversifying the overall literature (e.g., most other samples have been primarily White and middle class), care should be taken in generalizing the findings across studies. For instance, early exposure to chronic stress has been shown to relate to different profiles of physiological function (Lupien, King, Meaney, & McEwen, 2001). Thus, the sample in the present study may be characterized by different physiological profiles than would be typical of the middle and upper class samples used in many studies of RSA. However, assuming that the same mechanisms are invoked by stress exposure across social classes, with only variations in dose, it is possible that greater developmental exposure to stress increases rates of mental health problems through influences on physiological reactivity. This hypothesis will remain tenuous until it is directly tested.

Although the participants in this sample exhibited the full range of developmentally normative to subthreshold to clinical threshold severity in both internalizing and externalizing symptoms, the kindergarteners were younger than many of the samples that have been used in studies of psychiatric disorders, even during childhood. Given the sample age, clinical diagnoses were not established for these participants. However, given the severity of the symptoms and the contextual risk factors evident from the demographics, the probability of manifesting psychiatric diagnostic status is increased for this sample. Although we propose that these participants are representative of individuals at high risk for psychopathology, they may only be representative of a subset of such individuals, warranting caution in extrapolating the findings to clinically recruited samples. All participants were selected on the basis of teacher-reported symptoms of aggressive behavior from a short screening questionnaire. Reliance on teacher report could introduce bias with regard to child race or sex. While the brevity of this scale enabled us to screen all of the kindergarten children within the district over 2 years (n = 1,192), this brevity likely compromises diagnostic specificity. Although we found no statistical evidence of significant skew, kurtosis, or bimodality in the distribution of symptoms, the focus on externalizing behaviors suggests that representation of internalizing severity was likely biased toward comorbidity to a greater extent than would be expected if the selection had focused

exclusively on internalizing symptoms. It is possible that greater representation of “pure” internalizing profiles would yield a different pattern of results. More generally, future work should examine whether and how samples selected in more comprehensive ways could improve the clarity of associations with RSA reactivity in different emotional domains.

Finally, the single-age assessment data used here provide only a snapshot of the between-person association between RSA reactivity to emotions and concurrent symptom severity. The data do not differentiate between children with developmentally stable symptom profiles and children with transient symptoms that might be associated with the transition into school. A key issue for future research will be collecting the longitudinal data necessary for distinguishing and understanding nonnormative and normative developmental symptom patterns and how changes in symptom expression are related to changes in children’s physiology.

The present investigation indicates that an association between RSA reactivity to emotional challenges and symptoms of psychopathology is present as early as 6 years of age, although differences associated with baseline RSA are not. These initial results provide a foundation for further investigation of how reactivity to specific emotions confers risk for early symptoms of childhood psychopathology. Specifically, the results indicate that the motivation framework for examining emotions provides a useful foundation for studying the differentiating features of affective reactivity between internalizing and externalizing domains of psychopathology. Future research may benefit from more extensive representation of affective stimuli in elucidating the nature of dysregulated emotion that contributes to different domains of psychiatric symptoms.

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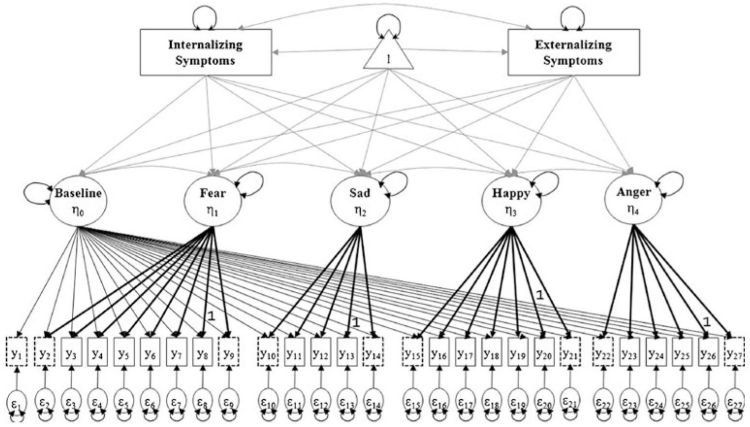

Fig. 1.

Multiphase nonlinear latent-basis modeling of RSA reactivity across the emotion induction paradigm

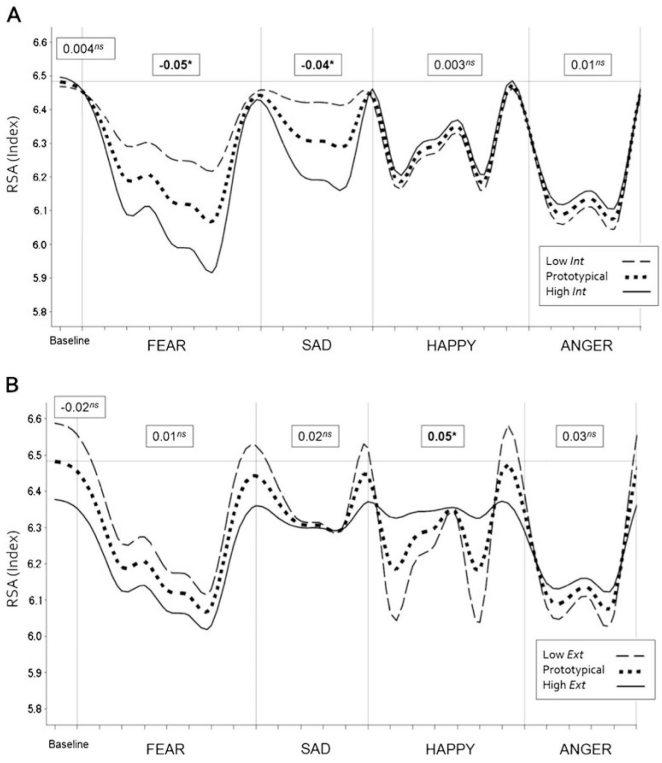

Fig. 2.

Respiratory sinus arrhythmia (RSA) baseline levels and reactivity for prototypical (average) as well as high- and low-internalizing (Int) and externalizing (Ext) symptoms during the emotion-inducing paradigm. High levels of Int are related to greater RSA withdrawal during the fear and sadness conditions (upper panel), while high levels of Ext are related to blunted RSA withdrawal during the happiness condition (lower panel). Boxes contain the parameter estimates for the relationship between RSA reactivity and Int or Ext during each emotion-inducing condition. Low Int and Ext indicate values ≥1.5 *SD*s below average; high Int and Ext indicate values ≥1.5 *SD*s above average. $N = 273$. $^{*}p < .05$; n.s., nonsignificant

Table 1

Contrasts of average RSA levels between each pair of emotion conditions

	Initial Baseline	Fear	Sadness	Happiness	Anger	Fixation Baselines
Initial baseline	**6.40 (1.11)**					
Fear	0.18*	**6.23 (1.13)**				
Sadness	0.06	−0.13*	**6.34 (1.19)**			
Happiness	0.08	−0.10*	0.03	**6.32 (1.11)**		
Anger	0.25*	0.07	0.20*	0.16*	**6.17 (1.12)**	
Fixation baselines	−0.04	−0.24*	−0.10*	−0.16*	−0.33*	**6.45 (1.08)**

* $p < .05$. The bold values on the diagonal are the average RSAs (with standard deviations in parentheses) exhibited across epochs in the different conditions. Higher values indicate more parasympathetic influence and lower levels of arousal. Mean differences between the conditions, evaluated by paired-sample *t* tests, are reported in each cell.

Table 2

Dynamic change in children's RSA reactivity during the emotion-inducing conditions (with standard errors in parentheses), and the relation of these changes to teacher ratings of children's internalizing and externalizing symptoms (with standard errors)

		RSA Reactivity During Conditions Inducing …			
	Baseline	**Fear**	**Sadness**	**Happiness**	**Anger**
Pattern of Change (Latent Basis)					
Factor loadings for RSA	y_1 = 1.00				
	y_2 = 1.00	y_2: .07 (.08)	y_{10}: .22 (.14)	y_{15}: .12 (.09)	y_{22}: .36* (.09)
	y_3 = 1.00	y_3: .36* (.09)	y_{11}: .63* (.14)	y_{16}: .95* (.12)	y_{23}: .96* (.11)
	y_4 = 1.00	y_4: .73* (.09)	y_{12}: .97* (.15)	y_{17}: .72* (.10)	y_{24}: .98* (.11)
	y_5 = 1.00	y_5: .68* (.09)	y_{13} = 1.00	y_{18}: .61* (.10)	y_{25}: .93* (.11)
	y_6 = 1.00	y_6: .88* (.10)	y_{14}: .95* (.15)	y_{19}: .48* (.09)	y_{26} = 1.00
	y_7 = 1.00	y_7: .92* (.10)		y_{20} = 1.00	y_{27}: .08* (.09)
	…	y_8 = 1.00		y_{21}: .09* (.09)	
	y_{27} = 1.00	y_9: .36* (.08)			
RSA Reactivity Factors					
Factor means (*SE*)	6.48*(.06)	−0.40* (.06)	−0.18* (.04)	−0.30* (.06)	−0.38* (.06)
RSA Factor (Co)Variances					
Baseline	.98* (.09)				
Fear	.005 (.05)	.36* (.08)			
Sadness	.02 (.04)	.23* (.06)	.18* (.06)		
Happiness	−.06 (.05)	.28* (.06)	.23* (.05)	.35* (.08)	
Anger	−.06 (.05)	24* (.06)	.20* (.05)	.28* (.06)	.31* (.07)
Children's Symptoms and RSA Reactivity [a]					
Internalizing symptoms	0.004 (0.03)	**−0.05* (0.02)**	**−0.04* (0.02)**	0.003 (0.02)	0.01 (0.02)
Externalizing symptoms	−0.02 (0.02)	0.01 (0.02)	0.02 (0.02)	**0.05* (0.02)**	0.03 (0.02)
R^2	.003 (.007)	.03 (.03)	.04 (.04)	.05 (.04)	.02 (.03)
Fit Statistics					
$\chi^2(406) = 614.61$, $p < .001$, 53 parameters, RMSEA = .04 (Confidence Interval: .04–.05), CFI = .97, NNFI = .97					

N = 273.

[a] Influences of children's behavior on RSA reactivity are indicated by parameter estimates in the growth model's fits to changes in RSA during the different emotion-inducing conditions. Significant reactivity values are bold. Each epoch (y_1, y_2, y_3, etc.) is 30 s in length. The results were not changed when we introduced race, sex, or age as potential covariates and/or moderators, and thus these variables were removed from the final model.

* $p < .05$