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ORIGINAL RESEARCH REPORT

Respiratory sinus arrhythmia during worry forecasts stress-related increases in psychological distress

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Abstract

Respiratory sinus arrhythmia (RSA) has been conceptualized as an index of emotion regulation abilities. Although resting RSA has been associated with both concurrent and prospective affective responses to stress, the impact of RSA reactivity on emotional responses to stress is inconsistent across studies. The type of emotional stimuli used to elicit these phasic RSA responses may influence the adaptive value of RSA reactivity. We propose that RSA reactivity to a personally relevant worry-based stressor might forecast future affective responses to stress. To evaluate whether resting RSA and RSA reactivity to worry inductions predict stress-related increases in psychological distress, an academic stress model was used to prospectively examine changes in psychological distress from the well-defined low- and high-stress periods. During the low-stress period, 76 participants completed self-report mood measures and had their RSA assessed during a resting baseline, free worry period and worry catastrophizing interview. Participants completed another mood assessment during the high-stress period. Results indicated that baseline psychological distress predicted larger decreases in RSA during the worry inductions. Lower resting RSA and greater RSA suppression to the worry inductions at baseline prospectively predicted larger increases in psychological distress from the low- to high-stress period, even after accounting for the impact of baseline distress on RSA. These results provide further evidence that RSA may represent a unique index of emotion regulation abilities in times of stress.

Keywords

Academic stress, autonomic function, emotion regulation, heart rate variability, RSA, worry catastrophizing

History

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Introduction

Respiratory sinus arrhythmia (RSA) has been conceptualized as an autonomic indicator of vulnerability to stress (Porges, 2007; Thayer & Lane, 2009). RSA quantifies oscillations in inter-beat intervals during the respiration cycle (Berntson et al., 1997). Pharmacological blockade studies indicate that RSA is modulated by vagal-dependent cholinergic neurotransmission at the sinoatrial node of the heart (Berntson et al., 1997). Given that the brain stem nuclei regulating cardiac vagal activity are sites of integration of afferent vagal inputs from the viscera and efferent outputs from forebrain and limbic brain regions, RSA is thought to represent an integrated index of emotion regulation abilities (Beauchaine et al., 2007; Porges, 2007; Thayer & Lane, 2009).

Lower resting RSA has been associated with greater psychological distress in cross-sectional studies (Friedman, 2007; Jonsson, 2007; Kemp et al., 2010; Watkins et al., 1998). Resting RSA also predicted future affective responses to stressors in both laboratory (Bornas et al., 2005; Fabes, 1993;)and daily diary studies (Diamond et al., 2011; Fabes & Eisenberg, 1997). In a recent prospective study with cancer patients, lower resting RSA was associated with larger increases in anxiety over the following 12 months (Kogan et al., 2012). This suggests that resting RSA is concurrently and prospectively associated with affective responses to stress.

Some studies indicate that in addition to resting RSA, RSA reactivity to stress is also an independent predictor of affective responses (Fortunato et al., 2013; Gentzler et al., 2009; Utendale, 2013). RSA reactivity represents transient changes in RSA upon exposure to physical or emotional stressors (Frazier et al., 2004; Hamer & Steptoe, 2007; Hofmann et al., 2005; Houtveen et al., 2002; Klinkenberg et al., 2009; O'Donnell et al., 2008; Steptoe & Marmot, 2006; Thayer et al., 1996). Porges' Polyvagal Theory (1995) proposes that greater RSA withdrawal in response to stress is an adaptive response facilitating energy mobilization. Other authors suggest that RSA augmentation during or following an emotional stressor indicates successful emotion regulation (Appelhans, 2006; Butler et al., 2006; Denson, 2011). Longitudinal studies examining the impact of RSA reactivity at baseline on later affective responses indicate that greater RSA reactivity has been prospectively related to both improvement (Gentzler et al., 2009; Greaves-Lord et al., 2010; Rottenberg et al., 2005) and worsening of affective

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symptoms over time (Diamond et al., 2011; Fortunato et al., 2013; Ingjaldsson et al., 2003). Greater RSA withdrawal (and lower RSA augmentation) to different stressors has thus been empirically associated with both adaptive and maladaptive emotional responses.

One complicating factor is that the magnitude of RSA reactivity depends on stable individual differences in negative emotionality as well as state levels of psychological distress. Indeed, greater anxiety and depression were associated with greater RSA suppression during emotional tasks (Fortunato et al., 2013; Graziano & Derefinko, 2013; Hughes & Stoney, 2000; Ottaviani et al., 2009; Pang & Beauchaine, 2012; Shinba et al., 2008; Verkuil, 2009). Furthermore, situational or task demands may also influence whether RSA suppression is adaptive or maladaptive (Porges, 2007). In prior studies, the functional consequences of RSA reactivity depended on the type of stimuli used to elicit these phasic responses. For instance, RSA reactivity to sad movies, but not anger-provoking or happy movies, was selectively associated with internalizing symptoms and future depressive episodes (Fortunato et al., 2013; Rottenberg et al., 2005). These data thus highlight the importance of using prospective designs in combination with emotional stressors to tease apart the impact of baseline distress levels on concurrent RSA reactivity and prospective affective responses to stress.

In accordance with models suggesting that RSA represents an index of emotion regulation capacities (Beauchaine et al., 2007; Porges, 2007; Thayer & Lane, 2009), we propose that RSA reactivity to personally relevant worry inductions might represent an independent predictor of affective responses to future stress. In the present study, two idiographic worry induction tasks targeting personally relevant worry themes were used to test whether RSA reactivity forecast future affective response to stress. Laboratory-based free worry inductions are evidenced to transiently increase negative effect (McLaughlin et al., 2007) and induce RSA suppression (Hofmann et al., 2005; Thayer et al., 1996; Verkuil, 2009). A worry catastrophizing procedure was administered after the free worry period in order to promote an in-depth exploration of personally relevant worry themes and to elicit individual differences in emotion regulation responses (Provencher et al., 2000; Vasey, 1992). These tasks were chosen because they represent a mild emotional stressor, not associated with an actual threat, but leading to transient increases in psychological distress, and, potentially, activating emotion regulation processes. Furthermore, a prospective design encompassing well-defined periods of low and high stress was used to evaluate whether lower resting RSA and greater RSA reactivity would prospectively predict larger affective responses to stress, over and above baseline psychological distress.

Methods

Research design

During the low-stress period, i.e. within the first 4 weeks of the academic semester, participants completed self-report psychological distress measures and participated in two worry induction tasks while their heart rate was recorded. During the high-stress period, i.e. approximately 3 months later during the week prior to final examinations, participants completed another psychological distress assessment.

Participants

Participants were recruited from the institutional Participant Research Pool. Inclusion criteria were being an undergraduate student and being between 18 and 30 years old. Exclusion criteria were reporting an acute or chronic medical condition or taking medication on a regular basis. Participants received course credits in exchange for their participation. This study was approved by the institutional Human Research Ethics Committee, and all participants provided written informed consent prior to participation.

Procedure

Eligible participants completed online questionnaires about their psychological distress using the Depression Anxiety and Stress Scale (see below) in the week prior to the laboratory session. At the beginning of the visit, participants were fitted with a digital interbeat interval recorder. For the resting baseline, participants were instructed to close their eyes and relax as much as possible for 7 min. After the resting baseline, the participants completed the first worry induction task, consisting of a 5-min free worry period. Based on the protocol of Hofmann et al. (2010), participants were told:

> Worry is a chain of negative thoughts, about something that can have negative consequences for you in the future. Typically people worry about something that hasn't happened yet but that could happen in the future, and that is negative. Can you make a list of the things you tend to worry about? Of all worries you mentioned, which do you worry about most often and most intensely about? During the next 5 minutes, I would like you to worry about (the topic you tend to worry most often and most intensely about). Please close your eyes, and try to worry for the complete 5 minutes. If you realize your attention starts wandering off, try to refocus on your worries.

After the free worry period, the worry catastrophizing interview was administered (Provencher et al., 2000; Vasey, 1992). The worry catastrophizing interview is a structured worry task that examines various aspects of the worry process, using a downward arrow technique. Personally relevant worry themes are generated by participants, and then participants are asked ''*what is it about* (the worry theme) *that worries you?*'' For the current study, the most severe worry theme generated by participants in the previous worry task was catastrophized. During the catastrophizing phase of the interview, participants generate a list of possible consequences to the worry by responding to the question ''*If* (the worry theme) *actually happened, what are you afraid would happen next?*'' This question is repeated until the participant can no longer generate any worry consequences. During the next phase of the interview, the experimenter reads each worry consequence and the participant rates the perceived likelihood of each consequence on a scale from 1 (not at all likely) to 100 (extremely likely) as well as the perceived severity of each consequence from 0 (not at all

severe) to 8 (extremely severe). While this task can promote a cognitive reappraisal of the worry theme, it triggers increases in psychological distress among individuals with clinical levels of worry (Vasey, 1992). As a manipulation check, a visual analogue scale ranging from 0 to 100 assessed transient changes in anxiety during the resting baseline, free worry period, and worry catastrophizing interview.

Participants remained seated throughout the resting baseline and worry induction tasks. Laboratory sessions were scheduled between 8:30 to 12:00 to minimize the impact of diurnal variations in RSA (Bonnemeier et al., 2003). Participants were asked to refrain from exercising, smoking, eating and drinking caffeinated beverages at least 2 h prior to the laboratory session (Berntson et al., 1997).

Psychosocial measures

The Depression Anxiety Stress Scales-21 items (Lovibond & Lovibond, 1995) consist of three 7-item scales that assess self-reported negative emotional states of depression, anxiety and perceived stress. The extent to which each statement is endorsed by respondents over the past week is rated on a scale from 0 (*did not apply to me at all*) to 3 (*applied to me very much, or most of the time*). Confirmatory factor analysis indicates that this scale assesses an underlying factor of psychological distress (Henry & Crawford, 2005). Cronbach's α for the total scale was 0.91.

RSA measurement

Participants were fitted with a chest belt hardwired with a digital inter-beat interval recorder (Polar RS800CX; Finland: Kempele). The telemetric inter-beat interval recording device recorded the interval between two successive R-spikes of consecutives QRS complexes using a sampling rate of 1000 samples per second throughout the laboratory session. Recording artifacts were identified and corrected using the CardioEdit software (2007). Artifact correction was performed using integer arithmetic (i.e. dividing or adding intervals between heartbeats to correct missed or spurious R-spike detections). Less than 1% of the beats were edited for each participant. Porges and co-workers (1980) moving polynomial approach was used to extract RSA using the CardioBatch software (2007). A moving polynomial filter was applied to heart rate time series to remove the influence of aperiodic processes and to generate a detrended residualized time series. A bandpassed filter was applied to the detrended time series to extract the variance associated with oscillations in the interbeat intervals across the respiration cycle (0.12–0.40 Hz). The average RSA across each sequential 30-s epoch within each condition was calculated to minimize the impact of violation of the stationarity assumption. The RSA metric is the natural logarithm of the variance of the bandpassed time series. This method provides an optimal assessment of vagally mediated cardiac activity (Lewis et al., 2012).

Averaged RSA was calculated for each experimental phase: resting baseline, free worry and worry catastrophizing. The last 5 min of the resting baseline period and the full 5 min of the free worry period were used for RSA calculation. Given that the length of the worry catastrophizing interview varied across participants from 2 to 5 min, we used the first 2 min of the interview to standardize the induction length across participants[1].Moreover, given that there was a linear decrease in RSA during the worry inductions, RSA reactivity was computed by subtracting baseline RSA from RSA during the worry catastrophizing interview. Therefore, smaller (more negative) reactivity score indicates greater RSA suppression during the worry inductions.

Statistical analysis

Multilevel modeling evaluated within-person trajectories of change in RSA in response to the worry inductions and whether between-person differences in psychological distress impacted the within-person trajectories of change in RSA (Raudenbush & Bryk, 2002). Multilevel modeling allowed us to examine the relationship between the RSA intercept and RSA slope using the estimation of the covariance parameters of the random effects. Multilevel modeling was also used to evaluate whether between-person differences in baseline RSA and RSA reactivity predicted the trajectory of change in psychological distress from the low- to high-stress periods. The covariates included in these models were age, sex, ethnicity, full time student status, as well as time of the day of the heart rate recordings. Restricted maximum likelihood efficiently handled the few missing data (Randenbush & Bryk, 2002). An unstructured covariance better fitted the data. The statistical analyses were performed with SAS 9.3 PROC MIXED (Cary, NC).

Results

The 76 participants had a mean of age of 21.33 (SD = 1.72). The majority of the sample consisted of women (86.8%) and Caucasians (64.5%). Most participants were full-time undergraduate students (92.1%). All participants completed the laboratory-based session and the psychological stress self-report measure during the low-stress period and 68 participants completed the psychological stress questionnaire during the high-stress period. The attrition rate ($n = 8$) was 11%. Participants who did not complete the psychological stress questionnaire during the high-stress period were significantly older than the participants who remained in the study $F\ (74) = 4.31$, $p = 0.04$, but did not differ on other socio-demographic, psychological or cardiovascular characteristics, all $p > 0.16$. RSA data during the resting baseline were lost for one participant because of equipment malfunction. Table 1 presents the Spearman's Rho correlations among baseline RSA, RSA reactivity and psychological distress at the low- and high-stress periods.

Manipulation checks

Table 2 presents the subjective anxiety ratings across the three conditions. Multilevel modeling indicated that there was a significant increase in subjective anxiety across the three time periods of the laboratory session, $\beta(E) = 14.83$ (1.28), t

[1]When the analyses were repeated using the full length of the worry catastrophizing interview, the results were not significantly different from those obtained using the first 2 min of the task. Therefore, only the results obtained when using the first two minutes of the worry catastrophizing interview are presented in the manuscript.

Table 1. Spearman's Rho correlations among resting RSA, RSA reactivity and psychological distress during the low- and high-stress periods.

Variables	1	2	3	4
1. Resting RSA	1.00	−0.57**	0.07	−0.05
2. RSA reactivity		1.00	−0.27*	−0.28*
3. DASS low stress			1.00	0.70**
4. DASS high stress				1.00

RSA, respiratory sinus arrhythmia; DASS, depression anxiety and stress scales.
*$p<0.05$, **$p<0.01$.

Table 2. Means and standard deviations for respiratory sinus arrhythmia (RSA) and self-reported anxiety.

	RSA		VAS Anxiety	
Task	Mean	SD	Mean	SD
Resting	6.70	1.19	14.46	13.37
Free worry	6.53	1.16	36.82	17.96
Worry catastrophizing interview	6.52	0.99	44.45	22.44

RSA, respiratory sinus arrhythmia; VAS, visual analogue scale.

(73) = 11.60, $p<0.001$. Post-hoc tests indicated that anxiety significantly increased from baseline to the free worry task, t (149) = 11.32, $p<0.001$, and from the free worry task to the worry catastrophizing interview, t (149) = 2.86, $p=0.01$.

RSA reactivity to the worry induction tasks

Table 2 presents the raw RSA values across the three experimental conditions. Multilevel modeling indicated that there was a significant reduction in RSA from the resting baseline period to the worry induction tasks, $\beta(E)=-0.09$ (0.04), t (150) = 2.46, $p=0.01$. The curvilinear change term was not significant, $\beta(E)=0.07$ (0.07), t (149) = 1.12, $p=0.26$. Post-hoc tests revealed that there were significant differences between resting RSA and RSA during the free worry period, t (149) = 2.20, $p=0.03$, as well as RSA during the worry catastrophizing interview, t (149) = 2.47, $p=0.01$. However, there was no significant change in RSA between the free worry period and the worry catastrophizing interview, t (149) = 0.27, $p=0.79$. Examination of the change scores revealed that about 50% of the participants experienced RSA suppression from baseline to the worry catastrophizing, while the other 50% had RSA augmentation during the worry inductions (median = −0.03). There was a significant correlation between the RSA intercept and slope, $r=-0.77$, $p<0.001$. Lower RSA intercept was associated with steeper RSA slope during the worry inductions. Figure 1 depicts changes in RSA in response to the worry inductions.

Univariate analysis indicated that psychological distress at baseline was related to changes in RSA during the worry inductions (Table 1). Multilevel modeling revealed that psychological distress in the past week was a significant predictor of the trajectory of change in RSA during the worry inductions, $\beta(E)=-0.006$ (0.002), t (149) = 2.77, $p=0.006$. Individuals with greater psychological distress exhibited greater decreases in RSA during the worry inductions than their less distressed counterparts. Figure 2 illustrates the effect of baseline psychological distress on RSA reactivity to the worry inductions.

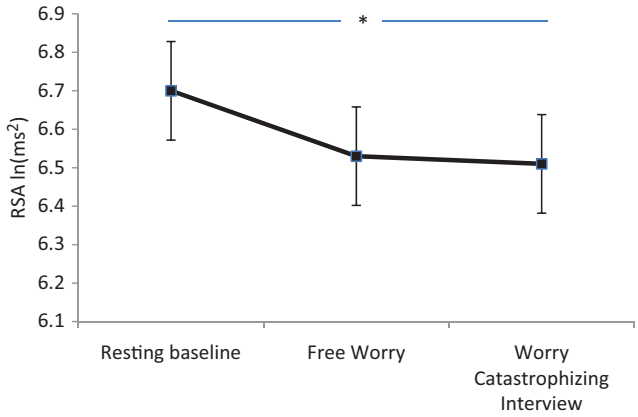

Figure 1. Changes in RSA across the three experimental conditions. Asterisk indicates a significant decrease in RSA from the resting baseline to the catastrophizing interview.

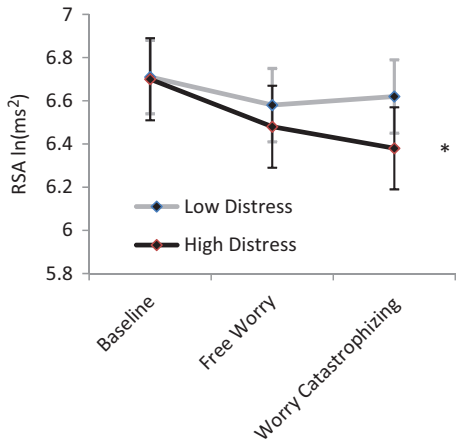

Figure 2. The impact of baseline psychological distress on changes in RSA during the worry inductions. For illustration purposes only, the continuous psychological distress variable was dichotomized using a median split. Asterisk indicates a significant decrease in RSA from the resting baseline to the catastrophizing interview.

Resting RSA and RSA reactivity as predictors of stress-related increase in anxiety

Multilevel modeling indicated that there was a significant increase in psychological distress from the low-stress ($M=10.41$, SD = 8.70) to high-stress (M = 15.12, SD = 10.56) periods, $\beta(E)=4.82$ (0.95), t (67) = 5.09, $p=0.001$. Multilevel modeling evaluated whether between-person differences in resting RSA and RSA reactivity to the worry inductions predicted the trajectory of change in psychological distress from the low-stress to high-stress periods. After adjusting for age, sex, ethnicity, full time status and time of the HR recording, resting RSA, $\beta(E)=-2.05$ (0.94), t (64) = 2.19, $p=0.03$ and RSA reactivity, $\beta(E)=-3.28$ (1.63), t (64) = 2.01, $p=0.05$, were significant predictors of the trajectory of change in psychological distress. Figures 3 and 4 depict changes in psychological distress as a function of resting RSA and RSA reactivity.

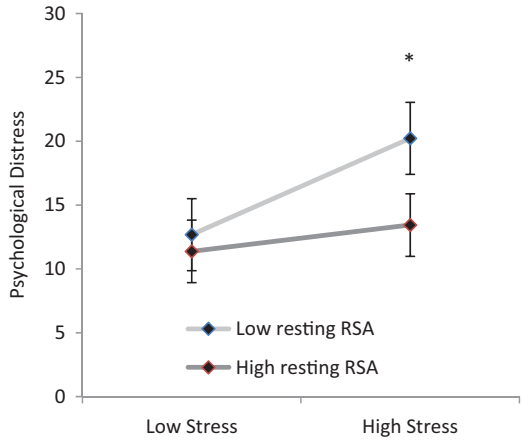

Figure 3. Stress-related changes in psychological distress as a function of resting RSA. For illustration purposes only, the continuous resting RSA variable was dichotomized using a median split. Asterisk indicates a significant mean difference in psychological distress.

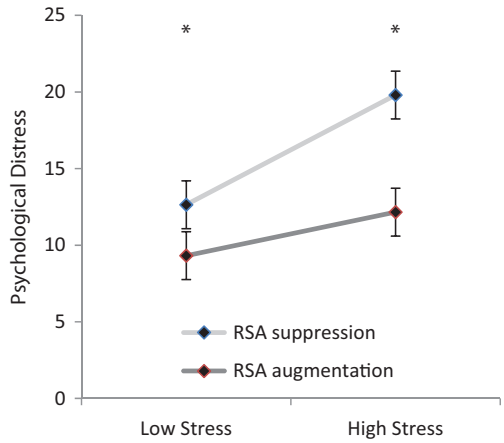

Figure 4. Stress-related changes in psychological distress as a function of RSA reactivity to the worry inductions. For illustration purposes only, the continuous RSA reactivity variable was dichotomized using a median split. Lower reactivity change scores indicated RSA suppression, while greater reactivity change scores indicated RSA augmentation to the procedure. However, baseline psychological distress moderated this effect. Individuals with greater distress experienced larger and longer-lasting RSA suppression in response to the worry inductions than less distressed participants. The worry catastrophizing interview was aimed at exploring in depth the feared consequences of personally relevant worries and evaluating the likelihood and severity of each feared consequence (Provencher et al., 2000). The procedures used during the worry catastrophizing interview activate worry-related threat schemata, but they can also elicit cognitive reappraisal of the worry (Vasey, 1992). In prior studies, the worry catastrophizing interview led to significant increases in subjective anxiety among individuals with clinically levels of worry, but not among healthy participants (Harvey & Greenall, 2003; Provencher et al., 2000; Vasey, 1992). In our study, there was an increase in anxiety during the catastrophizing procedure. It is possible that individuals with greater initial levels of distress might have had more difficulty regulating their emotions while actively exploring personally relevant worrisome thoughts.

Discussion

Results indicated that a free worry period followed by a worry catastrophizing interview led to significant reductions in RSA; these reductions were largest in participants with lower resting RSA. During the low-stress period, psychological distress in the past week moderated the impact of the worry inductions on RSA reactivity such that individuals with greater psychological distress experienced larger reductions in RSA than participants reporting less distress. Further, resting RSA and RSA reactivity prospectively predicted increases in psychological distress from the low- to high-stress period, over and above baseline distress levels. These results suggest that resting RSA and RSA reactivity to personally relevant worry inductions index emotion regulation abilities in times of stress.

In accordance with prior studies, there was a significant decrease in RSA during the free worry period (Hofmann et al., 2005; Thayer et al., 1996). On average, the reduction in RSA was maintained during the worry catastrophizing

Porges (1995) proposes that RSA suppression in response to stress is an adaptive response given that it promotes active engagement in coping with the source of stress. Numerous studies have shown that greater RSA reactivity to diverse stressors is associated with better affective functioning (Gentzler et al., 2009; Rottenberg et al., 2005, 2007). In contrast, other studies indicate that smaller RSA decreases or RSA increases in response to emotional stressors are associated with better emotion regulation and less psychological distress (Butler et al., 2006; Denson, 2011; Fortunato et al., 2013; Ingjaldsson et al., 2003; Libby et al., 2012). Methodological differences in emotional induction methods, RSA quantification (Overbeek et al., 2012), gender differences (Hinnant & El-Sheikh, 2013) and clinical or at-risk status (Graziano & Derefinko, 2013) may account for some of the discrepancies found in the literature.

The type of emotional task may also influence the nature of the relationship between RSA reactivity and future affective outcomes (Porges, 2007). In the current study, the worry inductions forced participants to think about personally relevant threats that have not yet happened. Although the tasks led to subjective increases in anxiety, the participants were not exposed to actual threats. In this situation, vagal withdrawal may not be the most adaptive response given that situational demands requiring energy mobilization or activation of the fight or flight response were not present. We propose that the ability to maintain stable RSA in response to a personally relevant worry-based emotional stressor in the absence of an actual threat might index emotion regulation abilities and therefore forecast anxiety responses to future stressors.

If RSA is an index of emotion regulation, then the relationship between RSA and psychological distress might be larger in times of stress (Thayer & Lane, 2009). Fabes et al. (1997) observed that resting RSA was associated with emotional responses to daily stressors of high or moderate intensity, but not to minor daily annoyances. Furthermore, Hansen et al. (2009) observed that individuals with greater resting RSA maintained an adequate cognitive performance in both the low- and high-stress conditions, while participants

with low-RSA exhibited a decline in performance during the high-stress condition. Our results extend these data and suggest that both resting RSA and RSA reactivity to worry index one's ability to regulate emotional responses in periods of high stress.

Resting RSA was a significant predictor of changes in psychological distress only when RSA reactivity was also included in the model. In accordance with the bivariate correlations, when resting RSA was entered alone in the model, it did not significantly predict changes in psychological distress, $B(SE) = 0.95$ (0.79), $t = 1.19$, $p = 0.24$. This suggests that RSA reactivity might be acting as a suppressor variable that strengthens the relationship between resting RSA and affective response to stress (Paulhus et al., 2004). This is consistent with the results of a recent meta-analysis indicating that covarying resting RSA and RSA reactivity increased the magnitude of the association between RSA reactivity and children's adaptive functioning (Graziano & Derefinko, 2013). This result also emphasizes the importance of considering both resting RSA and RSA reactivity within the same model when predicting affective responses.

A strength of the present study was that the effect of RSA reactivity was evaluated simultaneously on concurrent and prospective psychological distress, thereby allowing us to examine the unique predictive ability of RSA to future affective responses to stress. RSA exhibited a linear decrease during the worry inductions. However, given that the order of the worry tasks was not counterbalanced, we cannot distinguish whether the worry catastrophizing interview was uniquely associated with RSA reactivity, whether the catastrophizing procedure prevented recovery from the free worry period, or whether the mere sustained exposure to worry inductions led to decreases in RSA. Moreover, although the academic stress model encompassing well-defined periods of low and high stress enhanced our ability to detect individual differences in affective responses to stress, these results should be replicated in other samples exposed to stressors that are more chronic and unpredictable in nature and among individuals presenting greater variability in psychological distress.

In summary, resting RSA and RSA reactivity to personally relevant worry inductions forecast stress-related increases in psychological distress. These results provide further evidence that RSA reactivity during worry is a unique index of emotion regulation abilities in times of stress. This suggests that resting RSA and RSA reactivity could facilitate the identification of individuals who are at greater risk of experiencing stress-related increases in psychological distress.

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Declaration of interest

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