Journal of Emotion and Psychopathology
2025, Vol 1, Issue 2, 478-488
https://doi.org/10.55913/joep.v1i2.61

Emotional Reactivity and Regulation: Associations with Heart Rate Variability During Experimental Worry and Relaxation Inductions

Megan E. Renna[1*], Phillip E. Spaeth[2], Michal Clayton[3], Douglas S. Mennin[3]

[1]*Ohio State University, United States*

[2]*Weill Cornell Medical College, United States*

[3]*Teachers College, Columbia University, United States*

Abstract

Introduction: Emotionality and emotion dysregulation both influence cardiovascular health, putting individuals at a heightened risk for increased morbidity and mortality. Heart rate variability (HRV) is influenced by the frequency and intensity of our emotions (e.g., emotionality) as well as how we regulate our emotions. This study tested whether negative and positive affect covaried with HRV across a baseline resting period along with experimentally induced worry and relaxation conditions to assess the interplay among emotionality, emotion regulation, and cardiovascular function among physically healthy adults. *Methods:* Community participants (n = 85) completed a resting baseline HRV assessment followed by experimentally induced worry and relaxation conditions. Participants completed the Positive and Negative Affect Scale (PANAS) to measure emotionality at baseline and following each experimental condition. HRV was also measured during the worry and relaxation conditions. Linear mixed models were used to assess covariation between affect and HRV across conditions. *Results:* Negative affect was significantly linked to HRV across conditions. As hypothesized, when participants reported higher negative affect, their HRV was significantly lower. In contrast, positive affect did not significantly covary with HRV across the study conditions. *Conclusion:* Findings highlight how negative affect and HRV are linked throughout a lab-based task of emotionality and emotion regulation. These findings provide insight into a potential pathway through which negative emotionality may be linked to poor health and how implementing emotion regulation skills training can mitigate these effects.

Keywords: affect; heart rate variability; worry; relaxation; emotionality; emotion regulation.

Introduction

Heart rate variability (HRV), a measure of fluctuation in the inter-beat intervals of the heart, is considered to be an index of autonomic flexibility, reflecting the balance or imbalance of the sympathetic and parasympathetic branches of the autonomic nervous system (ANS) (Thayer et al., 2010). Autonomic flexibility refers to the ability of the ANS to self-regulate and adapt to physical or psychological distress. HRV is measured via several metrics that represent either time-domain measures (e.g., the amount of variability in measurements of the time between heartbeats), frequency-domain measures (e.g., the distribution of relative signal energy), or non-linear measures (Allen et al., 2007; Shaffer & Ginsberg, 2017). Time-domain measures such as Root mean square of successive RR interval differences (RMSSD) and the standard deviation of interbeat intervals (SDNN) more closely represent

*Corresponding Author: Megan E. Renna ✉ renna.4@osu.edu
Received: 12 June 2023 | Revision Received: 17 January 2024 | Accepted: 25 January 2024
Published by Black Swan Psychological Assessments Pty Ltd
www.emotionandpsychopathology.org | ISSN: 2653-7583

respiratory-linked changes in heart rate compared to other metrics (Allen et al., 2007). In particular, RMSSD accurately represents vagal nerve activity (e.g., parasympathetic nervous system control over heart rate) and is thus well-suited to be used as a biomarker in research and clinical settings (Jarczok et al., 2019).

Low HRV represents autonomic rigidity and is associated with poor general health (Dekker et al., 2000) and a number of adverse cardiovascular outcomes (Appelhans & Luecken, 2006), including increased risk for hypertension (Goit & Ansari, 2016; Schroeder et al., 2003), coronary heart disease (Liao et al., 1997), sudden cardiac death (Maheshwari et al., 2016), and increased mortality following myocardial infarction (Kleiger et al., 1987). Essentially, low HRV represents an inability for the cardiovascular system to flexibly respond to the body and mind's context (e.g., exercise, stress, etc.) – leading to wear and tear over time. In populations without known cardiovascular disease, low SDNN is associated with a 32-45% increase in risk for first cardiovascular event (Hillebrand et al., 2013). Collectively, these physiological changes are implicated in cardiovascular disease (CVD) onset. Relatedly, there is a growing body of evidence highlighting associations between low HRV, adverse cardiovascular health outcomes, and negative emotionality (Kubzansky & Kawachi, 2000; Suls & Bunde, 2005).

HRV serves as a biological proxy of emotional reactivity and regulation (Appelhans & Luecken, 2006). Negative emotions are typically associated with low HRV (Kreibig, 2010). A significant correlation was observed between negative affect and RSA, which remained stable across a 12-month follow-up (Wang et al., 2013). Baseline RSA was positively correlated with positive affect and negatively related to trait neuroticism (Oveis et al., 2009). Both at rest and during an emotion induction, low high-frequency HRV (HF-HRV) has been linked to anxiety (Chalmers et al., 2014; Pittig et al., 2013; Pittig et al., 2018), depression (Kemp et al., 2010), and is generally considered a transdiagnostic biomarker for psychopathology (Beauchaine & Thayer, 2015).

Further investigation of the influence of negative affect and emotional reactivity on HRV, in the context of worry and relaxation, may provide insight into how processes of reactivity and regulation are related. Worry, a characteristic common across several other forms of psychopathology and generally considered as a maladaptive emotion regulation strategy, is best defined as thinking repeatedly about things in the future (Borkovec et al., 1983). The tripartite function of worry suggests this cognitive process alerts individuals to potential threats, prompts awareness to unresolved threatening situations, and prepares individuals for a necessary 'fight or flight' response (Frijda, 1988; Lazarus, 1991). According to Borkovec's model of generalized anxiety disorder (GAD), a psychological disorder characterized by excessive and uncontrollable worrying (Borkovec et al., 2004), worry functions as a means of avoiding the subjective experience and dampening the physiological arousal associated with negative emotionality. However, prior research suggests that worry contributes to the generation and exacerbation of negative affect and is linked to increases in negative affect, physiological activation, and decreased HRV compared to baseline, neutral, and relaxation conditions (Borkovec & Inz, 1990; McLaughlin et al., 2007; Thayer et al., 1996). The Contrast Avoidance Model of worry posits that sustained negative emotion functions to reduce the unpleasantness associated with shifts from positive to negative emotional states (2011). For example, in a study which examined the effects of worry and relaxation on physiological and emotional reactivity, participants with and without a GAD diagnosis were randomly assigned to engage in worry, relaxation, and neutral inductions prior to viewing positive and negative film stimuli (Llera & Newman, 2010). The worry induction resulted in higher levels of negative affect for all participants compared to the neutral and relaxation conditions, which were not statistically different. However, participants in the GAD group, compared to psychologically healthy controls, reported higher levels of negative affect during worry and neutral inductions, compared to non-anxious controls. At the physiological level, participants with a GAD diagnosis had significantly lower HF-HRV during the worry induction than during relaxation, while healthy controls had similar HF-HRV levels across conditions.

Much of the current research examining the impact of psychological processes of anxiety on physical health has focused on worry. This apprehensive state of anticipation of real or perceived threats in the environment causes wear and tear on the body over the long term. Further, the Perseverative Cognition Hypothesis (PCH) conceptualized the role that perseverative processes (e.g., worry) play in exacerbating or maintaining somatic health complaints (Brosschot et al., 2006). The central theory of the PCH links increases in cognitive activation, such as worry, with negative physical health outcomes. Empirical examinations of the physiological implications of perseveration have focused on several biological markers of overall health, including HRV (Allen et al., 2007). Individuals higher in trait worry are more likely to experience lower baseline HRV compared to those less prone to worrying (Brosschot et al., 2007; Lyonfields et al., 1995; Thayer et al., 1996). In response to an experimental stressor task, high trait worriers also demonstrated slower RMSSD recovery (i.e., shifting from lower HRV during the task and returning to baseline RMSSD after the task) compared to low trait worriers (Verkuil et al., 2009). Meta-analytic findings examining the impact of perseveration on HRV and other health-related processes found a significant decrease in HRV throughout experimental manipulations of perseveration in individuals free of psychological or physical diagnoses (Ottaviani et al., 2016). Among correlational studies, there was also a significant association between higher levels of perseverative cognition and decreased HRV (Ottaviani et al., 2016).

While the relationship between negative emotions and HRV are relatively well-established, findings are more limited for the relationship between HRV and positive emotions, with studies varying widely in terms of design and measurement (Kreibig, 2010). In a study of healthy young adults, no significant relations between positive affect and respiratory sinus arrythmia (RSA) emerged (Wang et al., 2013). A recent meta-analysis that assessed the relationship between autonomic nervous system activity and positive emotions found small to null effects for HRV, however the authors noted high heterogeneity between studies in terms of design, emotions measured, and HRV metrics used (Behnke et al., 2022). In contrast to these meta-analytic findings, in a study using an ecological momentary assessment design, higher positive affect predicted higher HRV day-to-day (Määttänen et al., 2021). Additional research examining the association between state- and trait-level position emotions and affect found that emotion dysregulation of positive emotions (e.g., unable to control feelings of love or joy) was associated with higher resting HF-HRV only in a state of high positive affect (Weiss et al., 2021). Researchers interested in the association between positive emotionality and HRV have shown that the type of positive emotion being measured matters. For example, one study found quadratic relationships between RMSSD and contentment, but not excitement (Duarte & Pinto-Gouveia, 2017). Taken together, there is no clear understanding of the association between HRV and positive affect in the context of an emotion regulation paradigm.

Current Study

The current study was a secondary analysis that examined the relationship between affect and HRV across experimentally induced worry and relaxation among adults from the community. RMSSD, positive affect, and negative affect were measured at baseline, following an experimentally induced worry condition, and following an experimentally induced relaxation condition. The worry and relaxation conditions were presented in a fixed order to test how worry promotes emotional and physiological dysregulation while relaxation then returns participants to baseline emotional and physiological functioning. We hypothesized that RMSSD, positive affect, and negative affect would change significantly across conditions. Further, we hypothesized that RMSSD would covary with both positive and negative affect across study conditions in that higher negative affect would be related to lower HRV and positive affect with higher RMSSD.

Methods

Participants

Participants (N = 85) were community members recruited as part of a larger parent study (Renna et al., 2020). Demographics are presented in Table 1. All participants were required to be over 18 years old and be able to read and understand English. Consistent with eligibility requirements of the parent study, exclusion criteria included autoimmune, inflammatory, or cardiovascular diseases, being on a consistent dose of medications shown to alter immunological or cardiovascular functioning, or being diagnosed with bipolar I disorder, alcohol or substance dependence, active psychosis, or a blood injury/injection phobia. Recruitment occurred throughout the New York City area via fliers placed in public spaces.

Affect

The Positive and Negative Affect Scale (PANAS) assessed affect at baseline, following the worry condition, and following the relaxation condition (described below) (Watson et al., 1988). The PANAS yields two 10-item scales, one for positive affect (PANAS-PA) and one for negative affect (PANAS-NA). Items were rated on a 5-point scale and assessed how respondents felt at the present moment. Higher scores on the positive and negative scales indicated higher positive affect and higher negative affect, respectively. In this study, internal consistency was excellent for the PANAS-NA (α = .91) and PANAS-PA (α = .88) subscales.

Covariates

Potential confounds were included based on their theoretical and empirical relationships to affect and HRV (Tsuji et al., 1996). All models adjusted for participants' age, biological sex, race, body mass index (BMI), and medication use.

Heart rate variability measurement

Participants' heart rate variability (HRV) was monitored throughout the experimental portion of the study using the Polar™ Watch system. This ambulatory psychophysiological measurement device collects HRV data via a band with two electrodes placed across the participants' upper abdomen. Electrocardiogram (ECG) sensors on the electrode band measured heart rate by a 1000 Hz sampling rate. Heart rate and respiration data were collected continuously throughout the experimental portion of the data during each discrete segment of the experiment. Data were then uploaded from the device onto a computer for processing and analysis. The root mean square of successive differences between normal heartbeats (RMSSD) was obtained by calculating each successive time difference between heartbeats in milliseconds. Each of the values was then squared. The result was averaged before the square root of the total was obtained. RMSSD metrics was analyzed and derived using CMetX software (Allen et al., 2007). All files were visually inspected for artifacts, which were identified and manually removed prior to calculation of HRV metrics.

Experimental Manipulation

The worry and relaxation conditions were modeled off previous research utilizing worry and relaxation manipulation tasks (Borkovec & Hu, 1990; Borkovec & Inz, 1990; Fisher & Newman, 2013; Llera & Newman, 2010). More information regarding these conditions are reported in the parent study (Renna, Hoyt, et al., 2020). Participants were instructed (via computer prompt) to: "Pick your most worrisome topic and worry about it as intensely as you can in your usual way for the next few minutes. If at any point your mind wanders off track, simply refocus your thoughts back onto your worry topic" (Fisher & Newman, 2013). Participants completed this induction for 10 minutes while receiving a prompt on the computer throughout the induction period to remind them to continue worrying about their most worrisome topic. For the relaxation condition, participants were instructed (via computer prompt) to: "Shift your breathing to your stomach rather than from your chest. Also, slow your breathing rate down to a rate slower than usual but not so slow that it is unpleasant or uncomfortable. You might do this by counting from one to three as you breathe in evenly and then again as you evenly exhale." Participants

completed this induction for 10 minutes while receiving a reminder of the instructions each minute. Participants completed subjective ratings of worry and relaxation at baseline and following both conditions to serve as a manipulation check using the Worry Visual Analogue Scale (WVAS) (Wichelns et al., 2016) and Relaxation Visual Analogue Scale, which was developed for this study.

Procedures

All participants completed written informed consent, and all procedures were approved by the institution's Institutional Review Board (IRB). Following informed consent, participants completed baseline ratings of worry, relaxation, and positive/negative affect. The Polar Watch was attached, and participants then completed a resting baseline for the HRV measurement for five minutes followed by the worry condition for 10 minutes. Immediately following the worry mentation, participants underwent a thirty-minute rest period corresponding to the study design of the parent study (Renna, Hoyt, et al., 2020). Participants then underwent the same sequence of procedures for the relaxation condition, including the thirty-minute wait period. Lastly, participants completed self-report questionnaires to assess their demographics and physical health.

Data Analysis Plan

All analyses were completed within SPSS software version 28. A natural log transformation better approximated normality of residuals for the RMSSD data. We first conducted descriptive analyses including Pearson correlations to assess relationships between all variables at baseline. Growth models with time as the predictor variable and PANAS-NA, PANAS-PA, and RMSSD served as outcome variables to test change in each variable across conditions. We then used linear mixed models using the MIXED command for the primary analyses, which allow explicit modeling of the within-subject correlations due to repeated measurements during each visit. An additional strength of this analytic approach is that it accounts for missing data by maximizing the use of existing data. All analyses controlled for age, race, medication use, and body mass index. Time was modeled as a categorical variable in all models.

Results

Descriptive Statistics

Table 1 provides descriptive statistics and frequencies of all control variables. Overall, participants were relatively young, approximately two-thirds female, and represented a relatively diverse range in ethnicity and race. Bivariate correlations among affect, RMSSD, and each covariate at baseline are in Table 3. There were no significant correlations at baseline among RMSSD, negative affect, and positive affect (*r*s range = -.08 - .10).

Manipulation Check

Results indicated a significant increase in subjective ratings of worry following the worry task for all participants ($t = 9.83$, df = 79, $p < 0.001$). Further, subjective ratings of relaxation significantly increased following the relaxation task compared to baseline ($t = 7.34$, df = 79, $p < 0.001$). In contrast, the worry condition led to a significant reduction in relaxation ($t = 6.06$, df = 79, $p < 0.001$), while the relaxation condition led to a significant reduction in worry across all participants ($t = 14.42$, df = 79, $p < .0001$).

Change across Conditions

Table 2 presents the means and standard deviations of untransformed RMSSD, PANAS-NA, and PANAS-PA during each condition. Growth models revealed that there was a trending change in RMSSD across conditions ($b = 2.57$, $SE = 1.33$, $p = .06$). HRV decreased significantly following the worry condition compared to baseline ($b = -3.13$, $SE = .08$, $p = .02$). In contrast, the relaxation condition significantly increased RMSSD compared to both baseline and the worry condition. PANAS-NA ($b = -1.79$, $SE = .25$, $p < .001$) and PANAS-PA ($b = -2.29$, $SE = .31$, $p < .001$) also changed significantly across conditions. While the worry condition significantly increased negative affect and decreased

positive affect compared to baseline, the relaxation condition reduced negative affect and increased positive affect compared to worry.

Covariance between affect and HRV

PANAS-NA covaried significantly with RMSSD across conditions (b = -.44, SE = .14, p < .01). As PANAS-NA increased across conditions, RMSSD decreased. In contrast, no significant covariance emerged between PANAS-PA and RMSSD across conditions (b = .30, SE = .19, p = .12).

Regarding covariates, age was significantly associated with RMSSD (b = .28, SE = .14, p = .12), indicating that older age was associated with significantly lower RMSSD. BMI, medication use, biological sex, and race were not associated with RMSSD (ps > .25).

Table 1. Demographic characteristics of participants (N = 85)

	Mean (SD)	Number (%)
Age	31.21 (11.82)	
Biological Sex		
Male		32 (37.6%)
Female		53 (62.4%)
Race		
White		31 (36.9%)
Black		16 (19.0%)
Asian American		20 (23.8%)
Hispanic		12 (14.3%)
Mixed Race		4 (4.8%)
Other		1 (1.2%)
BMI	24.76 (5.90)	
Trait Worry	51.19 (14.78)	
Baseline State Worry	37.44 (24.68)	
Baseline State Relaxation	43.62 (24.78)	

Note. SD = standard deviation, BMI = body mass index. Trait worry was measured using the Penn State Worry Questionnaire. State worry and relaxation were assessed using the Worry Visual Analogue Scale and Relaxation Visual Analogue Scale, respectively.

Table 2. Study variable means and standard deviations

	Baseline	Worry	Relaxation
NA	16.49 (6.13)	21.90 (8.06)	13.38 (5.34)
PA	26.52 (7.59)	21.58 (8.52)	22.41 (8.59)
RMSSD	43.77 (24.40)	39.89 (20.12)	53.18 (25.97)

Note. RMSSD = metric of heart rate variability; NA = negative affect; PA = positive affect; BMI = body mass index. RMSSD means and standard deviations represent raw values prior to log transformation during the baseline resting period. NA and PA were measured using the Positive and Negative Affect Schedule (PANAS).

Table 3. Correlations among study variables

	1	2	3	4	5	6	7
1. RMSSD	-						
2. NA	.10	-					
3. PA	-.08	-.07	-				
4. Gender	.03	.04	-.21**	-			
5. Age	-.33**	.01	.35**	-.29**	-		
6. Race	.06	-.02	-.11	-.02	.02	-	
7. BMI	-.11	.01	.10	-.48**	.48**	.04	-

Note. RMSSD = root mean square of successive differences [heart rate variability]; NA = negative affect; PA = positive affect; BMI = body mass index.

Discussion

Earlier research highlights HRV as a proxy for emotional responding (Appelhans & Luecken, 2006). Low HRV also serves as a risk factor for cardiovascular disease (Colhoun et al., 2001; Dekker et al., 2000; Hillebrand et al., 2013), highlighting a potential pathway through which negative affect and associated emotionality relates to poor health. This study tested associations between RMSSD and affect throughout experimentally induced worry and relaxation in a racially and ethnically diverse sample of community-based adults. Changes in RMSSD across conditions showed the expected pattern: RMSSD decreased throughout the worry condition and increased during relaxation. Consistent with study hypotheses, negative affect and RMSSD covaried across study conditions (including baseline, worry, and relaxation conditions). These findings highlight how emotion regulatory strategies, such as worry and relaxation, influence cardiovascular and affective reactivity.

The findings from the current study are in line with several theories underlying emotional reactivity and regulation. Worry contributes to both the generation and exacerbation of negative affect and physiological activation (Borkovec & Inz, 1990; McLaughlin et al., 2007; Thayer et al., 1996). In this study, the worry and relaxation paradigm led to alterations in negative affect in the expected pattern; worry contributed to an increase in negative affect while it was lower following the relaxation condition. Consistent with study hypotheses, these alterations in negative affect also covaried with RMSSD across conditions. The preservative cognition hypothesis (PCH) highlights how processes such as worry can maintain physiological dysregulation, putting individuals at risk for chronic health issues over time (Brosschot, 2010; Brosschot et al., 2006). This study extended past findings showing how worry is associated with decreased HRV (Ottaviani et al., 2016) by examining how negative emotionality (as measured by the PANAS) during and after experimentally-induced regulation (worry and relaxation) alters RMSSD. It is important to note, however, that the relationship between negative affect and HRV is bidirectional, and future research should elucidate the directionality of these relationships using causal experimental designs.

In contrast to our initial hypotheses, positive affect did not covary with RMSSD across conditions. Although research assessing the relationship between positive affect and HRV is varied, a recent meta-analysis highlighted small to null associations between the two across a number of different study paradigms and positive emotions (Behnke et al., 2022). Although the meta-analytic findings largely showed no to small effects, one recent study assessing daily fluctuations between positive affect and HRV did find a significant association between the two (Määttänen et al., 2021). The contrast between cross-sectional or experimental studies with daily diary studies emphasize the idea that the relationship between positive affect and HRV may be particularly sensitive to how and when the two are measured. The null findings from this study may indicate that RMSSD sensitizes to transient changes in positive affect less strongly compared to negative affect, further highlighting the tight link between negative emotional states and disruptions in normal cardiovascular functioning. Indeed, prior research highlights that cardiovascular responses to negative emotions were more prolonged (e.g., slower to return to baseline functioning) following negative emotions compared to after experiencing positive emotions (Brosschot & Thayer, 2003).

Understanding the cardiovascular impact of emotional reactivity and regulation provides insight into how emotionality influences overall health. HRV serves as a risk factor for numerous cardiovascular incidents, including increased risk for mortality (Goit & Ansari, 2016; Liao et al., 1997; Maheshwari et al., 2016; Schroeder et al., 2003). Accordingly, intervening on negative affect, and negative emotionality more broadly, may serve as one avenue to reducing risk for cardiovascular disease and death following a cardiac event among adults. Interventions such as emotion regulation therapy aim to reduce negative emotionality via improved emotion regulation skills (Renna, Fresco, et al., 2020), and prior research has highlighted the health benefits of the treatment (Renna et al., 2021). Further, mindfulness-based interventions that focus on present-moment awareness have been beneficial for improving

cardiovascular health among several samples, including depressed adults and cancer survivors (Bower et al., 2015; Hoge et al., 2018; Pascoe et al., 2017). Although not an explicit goal of mindfulness-based trainings, relaxation, frequently considered an adaptive emotion regulation skill and conceptualized as such in this study, also contributes to improved physical health (Renna, Hoyt, et al., 2020). Given that research has also consistently shown that using adaptive emotion regulation skills can help to mitigate cardiovascular disease risk, training these skills among individuals who may be at risk for CVD can potentially aid in ameliorating the biological risks associated with these conditions, such as HRV, and therefore improve overall health (Appleton & Kubzansky, 2014).

This study had several strengths. The use of three distinct timepoints allowed us to test relationships across the entirety of the experimental paradigm. Further, the ability to test how emotionality related to RMSSD during a period of emotion regulation (e.g., the worry and relaxation conditions) supplied a more nuanced understanding of the conditions under which emotionality and emotion regulation influence cardiovascular functioning. HRV is frequently influenced by respiration; RMSSD, one of many metrics of HRV and the one used in this study, better accounts for respiration than some of other metrics. Limitations include the fact that participants were not followed longitudinally. Further, this analysis was correlational in nature and causality therefore cannot be proven by these data. Although the experimental manipulations of worry and relaxation provided robust changes in positive and negative affect, future research is needed to determine whether these findings generalize to real-world settings. In addition to an individual's baseline stress reactivity potentially impacting results, several cognitive, emotional, and behavioral mechanisms underlying these results can also be explored in future research. Relatedly, although we assessed state-level changes in worry and relaxation before and after the inductions, it is possible that other forms of perseverative cognition, such as rumination, may have simultaneously been induced but was not measured.

Taken together, findings from this study extend prior research on emotionality and HRV by highlighting how RMSSD changes alongside negative affect during an experimentally induced regulation task. Findings from this study offer insight into how reducing negative affect and using adaptive emotion regulation skills can help to mitigate the known cardiovascular disease risks associated with negative emotionality, thus improving longevity and overall health.

Additional Information

Acknowledgements

The authors would like to thank the research assistants of the READ Lab who helped to collect the data associated with this manuscript.

Funding

This study was funded in part through the Dean's Grant for Student Research at Teachers College, Columbia University.

Conflict of Interest

The authors declare that they have no conflicts of interest.

Ethical approval

All participants completed written informed consent, and all procedures were approved by the Institutional Review Board (IRB) at Teachers College, Columbia University and Weill Cornell Medical College.

Data Availability

Data are available upon request to the corresponding author.

Author CRediT Statement

Megan Renna, Ph.D.: data curation, conceptualization, formal analysis, writing – original draft, writing – review & editing, visualization. *Phillip E. Spath, Ph.D.*: data curation, writing – review & editing.

Michal Clayton, M.Phil.: writing – reviewing and editing, formal analysis, visualization. *Douglas S. Mennin, Ph.D.*: supervision, writing – review & editing

References

Allen, J. J., Chambers, A. S., & Towers, D. N. (2007). The many metrics of cardiac chronotropy: A pragmatic primer and a brief comparison of metrics. Biological Psychology, 74(2), 243-262.

Appelhans, B. M., & Luecken, L. J. (2006). Heart rate variability as an index of regulated emotional responding. *Review of General Psychology*, *10*(3), 229-240.

Appleton, A. A., & Kubzansky, L. D. (2014). Emotion regulation and cardiovascular disease risk.

Beauchaine, T. P., & Thayer, J. F. (2015). Heart rate variability as a transdiagnostic biomarker of psychopathology. *International Journal of Psychophysiology*, *98*(2), 338-350.

Behnke, M., Kreibig, S. D., Kaczmarek, L. D., Assink, M., & Gross, J. J. (2022). Autonomic nervous system activity during positive emotions: A meta-analytic review. *Emotion Review*, *14*(2), 132-160.

Borkovec, T., & Hu, S. (1990). The effect of worry on cardiovascular response to phobic imagery. *Behaviour Research & Therapy*, *28*(1), 69-73.

Borkovec, T., & Inz, J. (1990). The nature of worry in generalized anxiety disorder: A predominance of thought activity. *Behaviour Research & Therapy*, *28*(2), 153-158.

Borkovec, T. D., Alcaine, O., & Behar, E. (2004). Avoidance theory of worry and generalized anxiety disorder. *Generalized anxiety disorder: Advances in research and practice*, *2004*, 77-108.

Borkovec, T. D., Robinson, E., Pruzinsky, T., & DePree, J. A. (1983). Preliminary exploration of worry: Some characteristics and processes. *Behaviour Research & Therapy*, *21*(1), 9-16.

Bower, J. E., Crosswell, A. D., Stanton, A. L., Crespi, C. M., Winston, D., Arevalo, J., Ma, J., Cole, S. W., & Ganz, P. A. (2015). Mindfulness meditation for younger breast cancer survivors: a randomized controlled trial. *Cancer*, *121*(8), 1231-1240.

Brosschot, J. F. (2010). Markers of chronic stress: Prolonged physiological activation and (un) conscious perseverative cognition. *Neuroscience Biobehavioral Reviews*, *35*(1), 46-50.

Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research*, *60*(2), 113-124.

Brosschot, J. F., & Thayer, J. F. (2003). Heart rate response is longer after negative emotions than after positive emotions. *International Journal of Psychophysiology*, *50*(3), 181-187.

Brosschot, J. F., Van Dijk, E., & Thayer, J. F. (2007). Daily worry is related to low heart rate variability during waking and the subsequent nocturnal sleep period. *International Journal of Psychophysiology*, *63*(1), 39-47.

Chalmers, J. A., Quintana, D. S., Abbott, M. J., & Kemp, A. H. (2014). Anxiety disorders are associated with reduced heart rate variability: a meta-analysis. *Frontiers in Psychiatry*, *5*, 80.

Colhoun, H. M., Francis, D. P., Rubens, M. B., Underwood, S. R., & Fuller, J. H. (2001). The association of heart-rate variability with cardiovascular risk factors and coronary artery calcification: a study in type 1 diabetic patients and the general population. *Diabetes Care*, *24*(6), 1108-1114.

Dekker, J. M., Crow, R. S., Folsom, A. R., Hannan, P. J., Liao, D., Swenne, C. A., & Schouten, E. G. (2000). Low heart rate variability in a 2-minute rhythm strip predicts risk of coronary heart disease and mortality from several causes: the ARIC Study. *Circulation*, *102*(11), 1239-1244.

Duarte, J., & Pinto-Gouveia, J. (2017). Positive affect and parasympathetic activity: Evidence for a quadratic relationship between feeling safe and content and heart rate variability. *Psychiatry Research*, *257*, 284-289.

Fisher, A. J., & Newman, M. G. (2013). Heart rate and autonomic response to stress after experimental induction of worry versus relaxation in healthy, high-worry, and generalized anxiety disorder individuals. *Biological Psychology*, *93*(1), 65-74.

Frijda, N. H. (1988). The laws of emotion. *American Psychologist*, *43*(5), 349.

Goit, R. K., & Ansari, A. H. (2016). Reduced parasympathetic tone in newly diagnosed essential hypertension. *Indian Heart Journal*, *68*(2), 153-157.

Hillebrand, S., Gast, K. B., de Mutsert, R., Swenne, C. A., Jukema, J. W., Middeldorp, S., Rosendaal, F. R., & Dekkers, O. M. (2013). Heart rate variability and first cardiovascular event in populations without known cardiovascular disease: meta-analysis and dose–response meta-regression. *Europace*, *15*(5), 742-749.

Hoge, E. A., Bui, E., Palitz, S. A., Schwarz, N. R., Owens, M. E., Johnston, J. M., Pollack, M. H., & Simon, N. M. (2018). The effect of mindfulness meditation training on biological acute stress responses in generalized anxiety disorder. *Psychiatry Research*, *262*, 328-332.

Jarczok, M. N., Koenig, J., Wittling, A., Fischer, J. E., & Thayer, J. F. (2019). First evaluation of an index of low vagally-mediated heart rate variability as a marker of health risks in human adults: proof of concept. *Journal of clinical medicine*, *8*(11), 1940.

Kemp, A. H., Quintana, D. S., Gray, M. A., Felmingham, K. L., Brown, K., & Gatt, J. M. (2010). Impact of depression and antidepressant treatment on heart rate variability: a review and meta-analysis. *Biol Psychiatry*, *67*(11), 1067-1074.

Kleiger, R. E., Miller, J. P., Bigger Jr, J. T., & Moss, A. J. (1987). Decreased heart rate variability and its association with increased mortality after acute myocardial infarction. *The American journal of cardiology*, *59*(4), 256-262.

Kreibig, S. D. (2010). Autonomic nervous system activity in emotion: A review. *Biological Psychology*, *84*(3), 394-421.

Kubzansky, L. D., & Kawachi, I. (2000). Going to the heart of the matter: do negative emotions cause coronary heart disease? *Journal of Psychosomatic Research*, *48*(4-5), 323-337.

Lazarus, R. S. (1991). Progress on a cognitive-motivational-relational theory of emotion. *American Psychologist*, *46*(8), 819.

Liao, D., Cai, J., Rosamond, W. D., Barnes, R. W., Hutchinson, R. G., Whitsel, E. A., Rautaharju, P., & Heiss, G. (1997). Cardiac autonomic function and incident coronary heart disease: a population-based case-cohort study: the ARIC Study. *American journal of epidemiology*, *145*(8), 696-706.

Llera, S. J., & Newman, M. G. (2010). Effects of worry on physiological and subjective reactivity to emotional stimuli in generalized anxiety disorder and nonanxious control participants. *Emotion*, *10*(5), 640.

Lyonfields, J. D., Borkovec, T., & Thayer, J. F. (1995). Vagal tone in generalized anxiety disorder and the effects of aversive imagery and worrisome thinking. *Behavior Therapy*, *26*(3), 457-466.

Määttänen, I., Henttonen, P., Väliaho, J., Palomäki, J., Thibault, M., Kallio, J., Mäntyjärvi, J., Harviainen, T., & Jokela, M. (2021). Positive affect state is a good predictor of movement and stress: Combining data from ESM/EMA, mobile HRV measurements and trait questionnaires. *Heliyon*, *7*(2).

Maheshwari, A., Norby, F. L., Soliman, E. Z., Adabag, S., Whitsel, E. A., Alonso, A., & Chen, L. Y. (2016). Low heart rate variability in a 2-minute electrocardiogram recording is associated with an increased risk of sudden cardiac death in the general population: the atherosclerosis risk in communities study. *PLoS One*, *11*(8), e0161648.

McLaughlin, K. A., Borkovec, T. D., & Sibrava, N. J. (2007). The effects of worry and rumination on affect states and cognitive activity. *Behavior Therapy*, *38*(1), 23-38.

Ottaviani, C., Thayer, J. F., Verkuil, B., Lonigro, A., Medea, B., Couyoumdjian, A., & Brosschot, J. F. (2016). Physiological concomitants of perseverative cognition: A systematic review and meta-analysis. *Psychological bulletin*, *142*(3), 231.

Oveis, C., Cohen, A. B., Gruber, J., Shiota, M. N., Haidt, J., & Keltner, D. (2009). Resting respiratory sinus arrhythmia is associated with tonic positive emotionality. *Emotion*, *9*(2), 265.

Pascoe, M. C., Thompson, D. R., Jenkins, Z. M., & Ski, C. F. (2017). Mindfulness mediates the physiological markers of stress: Systematic review and meta-analysis. *Journal of Psychiatric Research*, *95*, 156-178.

Pittig, A., Arch, J. J., Lam, C. W., & Craske, M. G. (2013). Heart rate and heart rate variability in panic, social anxiety, obsessive–compulsive, and generalized anxiety disorders at baseline and in response to relaxation and hyperventilation. *International Journal of Psychophysiology*, *87*(1), 19-27.

Pittig, A., Treanor, M., LeBeau, R. T., & Craske, M. G. (2018). The role of associative fear and avoidance learning in anxiety disorders: Gaps and directions for future research. *Neuroscience & Biobehavioral Reviews*, *88*, 117-140.

Renna, M. E., Fresco, D. M., & Mennin, D. S. (2020). Emotion regulation therapy and its potential role in the treatment of chronic stress-related pathology across disorders. *Chronic Stress*, *4*, 2470547020905787.

Renna, M. E., Hoyt, M. A., Ottaviani, C., & Mennin, D. S. (2020). An experimental examination of worry and relaxation on cardiovascular, endocrine, and inflammatory processes. *Psychoneuroendocrinology*, *122*, 104870.

Renna, M. E., O'Toole, M. S., Fresco, D. M., Heimberg, R. G., & Mennin, D. S. (2021). From psychological to physical health: Exploring temporal precedence throughout emotion regulation therapy. *Journal of Anxiety Disorders*, *80*, 102403.

Schroeder, E. B., Liao, D., Chambless, L. E., Prineas, R. J., Evans, G. W., & Heiss, G. (2003). Hypertension, blood pressure, and heart rate variability: the Atherosclerosis Risk in Communities (ARIC) study. *Hypertension*, *42*(6), 1106-1111.

Shaffer, F., & Ginsberg, J. P. (2017). An overview of heart rate variability metrics and norms. *Frontiers in public health*, *5*, 258.

Suls, J., & Bunde, J. (2005). Anger, anxiety, and depression as risk factors for cardiovascular disease: the problems and implications of overlapping affective dispositions. *Psychological bulletin*, *131*(2), 260.

Thayer, J. F., Friedman, B. H., & Borkovec, T. D. (1996). Autonomic characteristics of generalized anxiety disorder and worry. *Biol Psychiatry*, *39*(4), 255-266.

Thayer, J. F., Yamamoto, S. S., & Brosschot, J. F. (2010). The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. *International Journal of Cardiology*, *141*(2), 122-131.

Tsuji, H., Venditti Jr, F. J., Manders, E. S., Evans, J. C., Larson, M. G., Feldman, C. L., & Levy, D. (1996). Determinants of heart rate variability. *Journal of the American College of Cardiology*, *28*(6), 1539-1546.

Verkuil, B., Brosschot, J. F., de Beurs, D. P., & Thayer, J. F. (2009). Effects of explicit and implicit perseverative cognition on cardiac recovery after cognitive stress. *International Journal of Psychophysiology*, *74*(3), 220-228.

Wang, Z., Lü, W., & Qin, R. (2013). Respiratory sinus arrhythmia is associated with trait positive affect and positive emotional expressivity. *Biological Psychology*, *93*(1), 190-196.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of Personality & Social Psychology*, *54*(6), 1063-1070.

Weiss, N. H., Schick, M. R., Waite, E. E., Haliczer, L. A., & Dixon-Gordon, K. L. (2021). Association of positive emotion dysregulation to resting heart rate variability: The influence of positive affect intensity. *Personality and Individual Differences*, *173*, 110607.

Wichelns, G. A., Renna, M. E., & Mennin, D. S. (2016). Preliminary validation of subjective anchor scales for worry and rumination. *Cognitive Therapy & Research*, *40*(5), 645-660.