Knepp et al., *Cogent Psychology* (2015), 2: 1038896
http://dx.doi.org/10.1080/23311908.2015.1038896

cogent psychology

COGNITIVE SCIENCE & NEUROSCIENCE | RESEARCH ARTICLE

The impact of trait worry and emotion regulation on heart rate variability

Michael M. Knepp[1]*, Erin R. Krafka[2] and Erika M. Druzina[1]

Received: 29 January 2015
Accepted: 31 March 2015
Published: 28 April 2015

*Corresponding author: Michael M. Knepp, Department of Psychology and Neuroscience, University of Mount Union, 1972 Clark Avenue, Alliance, OH 44601, USA
E-mail: kneppm@mountunion.edu

Reviewing editor:
Peter Walla, University of Newcastle, Australia

Additional information is available at the end of the article

Abstract: High anxiety and poor emotion regulation have been found to function as independent causes of stress to the autonomic nervous system. The aim of this study was to further explore how these factors may interact to control heart rate variability. Fifty college students took part in a three-part cardiac recording session followed by questionnaires on trait worry and emotion regulation. An interaction for trait worry and emotion reappraisal was found on two markers of heart rate variability. Low trait worriers with high emotion reappraisal had higher vagal tone than the other three groups. This finding was across all three phases with no specific reactivity or recovery difference. Emotion suppression was not found to significantly impact vagal tone. The negative impacts of trait worry and emotion regulation on heart rate variability were found in this sample of healthy college-aged individuals. Specifically, high trait worry could have a deleterious effect on parasympathetic control of the heart. Emotion regulation skills meanwhile can function as a buffer to stress and a reliance on sympathetic control.

Subjects: Anxiety in Adults; Behavioral Medicine; Psychological Science

Keywords: trait worry; emotion regulation; emotion suppression; heart rate variability; cardiovascular reactivity

ABOUT THE AUTHORS

Michael M. Knepp is an assistant professor in the Department of Psychology and Neuroscience at the University of Mount Union. He is the director for the undergraduate neuroscience major and runs the Psychophysiology and Neuropsychology Undergraduate Laboratory. He recently chaired a symposium on conducting research at smaller teaching-priority universities.

Erin R. Krafka is a second-year master of Public Health student at Wright State University. Her primary public health and research interests include studying health psychology, promoting healthy behaviors among individuals with disabilities and chronic conditions, improving childhood nutrition and reducing childhood obesity, particularly in underserved areas.

Erika M. Druzina is a senior undergraduate student at the University of Mount Union. Her major studies include neuroscience and with a minor in chemistry. Her interests are geared toward preventative care, as in the study regarding vagal tone and temperament, which can link to novel advances and treatment plans for cardiovascular health.

PUBLIC INTEREST STATEMENT

This paper examined the relationship how anxiety and emotion regulation can influence cardiovascular system functioning. Reduced variation in the beat to beat changes of the heart is associated with higher cardiovascular mortality. Previous research has found that trait and clinical anxiety are linked with decreases in heart rate variability. In this study, we explored how emotion regulation might be involved with the anxiety–cardiovascular system association. The findings indicate that the group that was lower anxiety and used more emotion reappraisals had the highest amounts of heart rate variability which would be cardioprotective. There were no significant findings related to the relationship between anxiety and emotion suppression with heart rate variability.

cogent psychology

1. Introduction

Clinical manifestations of anxiety have been theoretically related to an autonomic nervous system imbalance (Mitchell & Shapiro, 1991). This imbalance can be expressed in numerous forms and characterized by heart rate variability analysis. An elementary understanding of the autonomic nervous system views it as antagonistic and reciprocal (e.g. as sympathetic control increases, parasympathetic control decreases, and vice versa). A more complex explanation of the activity of this system has been proposed, which better characterizes its modes of control (Berntson, Cacioppo, & Quigley, 1991). Overall, this model of autonomic nervous system control describes nine modes of activity allowing for the variability seen in the heart rate control system including concordant and discordant activation and inhibition of the two branches. This pattern is one aspect of the cardiovascular system that exhibits such complex patterns or autonomic flexibility. Related to this project, reduced flexibility can be a predictor of future anxiety (Greaves-Lord et al., 2010). Autonomic flexibility directly relates to cardiovascular health and has been termed cardiac vagal control of the heart (for review, see Friedman, 2007). In particular, Saul (1990) noted that the parasympathetic component of heart rate has better control over the cardiac system due to a faster transmission rate.

Heart rate variability has been studied with regard to anxiety, utilizing individuals with clinical disorders, including panic disorder and generalized anxiety disorder. Patients with panic disorder have exhibited lower levels of vagal heart rate control when compared with normal control participants (Friedman, 2007). Furthermore, Friedman and Thayer (1998) have previously hypothesized that the low vagal tone and reduced heart rate variability found in patients with panic disorder are indicative of limited psychophysiological flexibility. Individuals with generalized anxiety disorder have exhibited shorter cardiac interbeat intervals and lower high-frequency spectral power across all task (baseline, relaxation, worry) conditions (Thayer, Friedman, & Borkovec, 1996). Fear and anxiety can be thought of as a continuum ranging from the early state variable of fear, through the trait-level anxiety onto the clinical stages of a disorder (Plutchick, 1990). This link between generalized anxiety disorder and decreased vagal control of the heart emerges as another reason to investigate the relationship between worry and the cardiovascular system.

1.1. Worry and the cardiovascular system

Worry as an independent variable in cardiovascular reactivity studies has produced results mirroring those of studies using patients with generalized anxiety disorder. In relation to generalized anxiety disorder, worrisome thinking has been found to be related with phasic reductions in overall vagal tone (Lyonfields, Borkovec, & Thayer, 1995). Other research has shown the existence of slower recovery of blood pressure due to worry, as well as emotional stress during rumination periods (Glynn, Christenfeld, & Gerin, 2002). Meanwhile, students who were lower in brooding following a three-day written emotional disclosure task had a higher ambulatory blood pressure at a two-week follow-up (O'Connor, Ashley, Jones, & Ferguson, 2014). Persistent and intrusive worry has been more broadly linked with multiple cardiac risk factors, such as phasic and tonic heart rate elevations, low overall heart rate variability, and poor cardiac vagal control of the heart (Lyonfields et al., 1995; Thayer et al., 1996; Thayer, Friedman, Borkovec, Johnsen, & Molina, 2000). Both basic and applied researches have found this relationship between worry and cardiac function. Starting with basic research, lab-induced worry has been found to elicit increased heart rate and reduced heart rate variability when compared with baseline (Hofmann et al., 2005). As a task variable, Thayer et al. (1996) found state worry to be linked with shorter interbeat intervals, smaller mean successive differences for those intervals, and lower levels of high-frequency spectral power when the worry phase was compared with the relaxation and baseline periods. Furthermore, greater respiratory sinus arrhythmia suppression during worry induction has been found to be predictive of larger distress increases from low- to high-stress task periods (Gouin, Deschênes, & Dugas, 2014).

In applied research, increased heart rate with decreased heart rate variability was found during real-life situational worry in ambulatory recording studies of waking and sleep periods (Brosschot, Van Dijk, & Thayer, 2007; Pieper, Brosschot, van der Leeden, & Thayer, 2007). Kubzansky et al. (1997) found higher risks for nonfatal and fatal cardiovascular disease in men reporting higher levels of

social worry when compared to low-worrying men. The Kubzansky research group suggested that worry could also be investigated through a moderated or mediated relationship with traditional cardiovascular risk factors, such as smoking.

1.2. Research on emotion regulation

As its own variable, emotion regulation has been linked to cardiovascular disease risk (for review see, Appleton & Kubzansky, 2014). Another example found that directed facial expressions and tasks that reconstruct various emotions produce changes in the autonomic nervous system (Levenson, Ekman, & Friesen, 1990). Voluntarily constructed facial configurations produced the subjective emotions associated with those configurations and when subjects produced facial configurations that most closely resembled the associated emotion expression, the autonomic differences between emotions was most noticeable (Levenson et al., 1990). Kreibig, Wilhelm, Roth, and Gross (2007) indicated distinct physiological response patterns for neutral, sadness, and fear relating these ideas to the idea of conservation–withdrawal behavioral responses. Emotion suppression has been found to have both reactivity and recovery period consequences when dealing with a cognitive stressor (Quartana & Burns, 2010). Lemaire, El-Hage, and Frangou (2014) found, however, that emotion suppression during a negative image paradigm was an adaptive emotion regulation mechanism with regard to cardiovascular activation. Previous research has also found that respiratory sinus arrhythmia could be used as an internal marker of self-regulation (Pu, Schmeichel, & Demaree, 2010). Finally, when compared with high cognitive reappraisers, individuals with low appraisal had worse cardiovascular response patterns, such as decreased cardiac output (Mauss, Cook, Cheng, & Gross, 2007).

Emotion regulation has been used previously with worry as both a task variable and a trait when exploring cardiovascular deficits. Individuals with social anxiety increased cardiac efficacy by reappraising stress arousal as a potential positive tool (Jamieson, Nock, & Mendes, 2013). Gramzow, Willard, and Mendes (2008) argued that academic exaggeration was an adaptive emotion regulation strategy to decrease worry. Academic exaggeration in that study resulted in increased respiratory sinus arrhythmia. Finally, Aldao and Mennin (2012) found that regulation strategies were a benefit to healthy controls but a hindrance to those with generalized anxiety disorder with regard to cardiac flexibility.

While anxiety and emotion regulation have impacted the cardiovascular system both as personality traits and laboratory tasks, previous research has not explored whether or not these factors interact with each other. Based on the previously listed research, anxiety results in autonomic dysregulation and poor emotion regulation strategies have been associated with poorer cardiovascular system function. Yet, it has not been determined if these traits are truly functioning as separate inputs or if one particular combination of worry and emotion regulation is more cardioprotective or hyperreactive. By further examining the interaction between these factors, it would be possible to determine how emotion regulation strategies can be used to further protect individuals with high and low worry levels.

1.3. Hypotheses

Based on previous research, higher trait worry has predicted decreased heart rate variability and increased autonomic dysregulation. Poor emotion reappraisal and increased suppression of emotions are also potential risk factors for cardiovascular issues. While previous research has not explored the interaction between worry and emotion regulation, the hypotheses of this study built from these previous works. This interaction was expected to impact both markers of vagal tone: root mean square of successive differences (rMSSD) and high-frequency power. In particular, high trait worriers who reported low reappraisal of emotions were expected to have decreased vagal tone at baseline, during the stress task, and in the following recovery phase, while low trait worriers with high emotion reappraisal scores would have had the best levels of vagal tone. In addition, low trait worriers that do not suppress emotions were expected to have increased vagal tone across all three phases, while high trait worriers who indicated emotion suppression regulation strategies would have had the lowest vagal tone.

2. Method

2.1. Participants

Fifty college students (36 women; *M* age = 19.88) were recruited from psychology courses at a private Midwestern university to participate in a study of the cardiovascular system. The sample was predominantly Caucasian (90%). Participants were recruited utilizing the SONA research system to allow for anonymity and each student received academic credit for their participation. At signup through the online system, students were asked to refrain from alcohol and caffeine for 12 hours before the study. While there was no pre-test inclusion criterion, the demographic questionnaire included questions on physical and mental health to use as exclusions for data analysis. No students reported any physical health issues that were of concern for this study and no medications listed resulted in subject exclusion. No students withdrew during the course of the study. Written informed consent was obtained prior to participation and the study protocol was approved by the university's IRB.

2.2. Apparatus and materials

2.2.1. Psychophysiological recording

Researchers recorded the electrocardiogram (ECG) to a computer hard drive using a BIOPAC MP36 system (BIOPAC Systems Inc., Goleta, CA) sampled at 1,000 Hz. Physiological signals were acquired through three disposable, pre-gelled electrodes (BIOPAC Systems Inc., Goleta, CA) using a limb lead II placement. The interbeat interval time series for each subject was analyzed with the Kubios HRV Analysis Software 2.0 for Windows developed by The Biomedical Signal Analysis Group at the University of Kuopio in Finland. This software was also used to correct for any artifact within the ECG signal. The first dependent variable acquired through this software was the time-domain measure of rMSSD. The second measure was high-frequency power. The cut-offs used for the high-frequency power bandwidth were .15–.40 Hz. A fast Fourier transform was used in this study.

2.2.2. Penn State Worry Questionnaire

The Penn State Worry Questionnaire was used to assess the cognitive component of anxiety, worry (Meyer, Miller, Metzger, & Borkovec, 1990). The Penn State Worry Questionnaire consists of 16 items using a five-point Likert scale approach measuring for trait worry. Sample items on this scale include "I've been a worrier all my life" and "When I am under pressure I worry a lot." Previous research has indicated a potential cut-off at 45 in a clinical sample, with a cut-off near 62 in a college student sample on the scale of 80 (Behar, Alcaine, Zuellig, & Borkovec, 2003). Reliability of the Penn State Worry Questionnaire within this study was acceptable (α = .70).

2.2.3. Emotion Regulation Questionnaire

The Emotion Regulation Questionnaire is a 10-item scale that examines inner emotional experience and emotional expression with two subscales: reappraisal and suppression (Gross & John, 2003). This questionnaire used a seven-point Likert scale and includes questions with both positive and negative valances. The reappraisal scale included statements such as "When I want to feel less negative emotion, I change the way I'm thinking about the situation." The suppression scale contained statements similar to "I keep my emotions to myself." Previous research has indicated acceptable to good internal consistency (α = .75–.82 for reappraisal; α = .68–.76 for suppression) and scale independence across four samples (mean r = −.01; Gross & John, 2003). Within this study, the emotion reappraisal subscale had good reliability (α = .85), while the emotion suppression scale's consistency was acceptable (α = .74).

2.2.4. Adult Temperament Questionnaire

The Adult Temperament Questionnaire is an extension of a previous Physiological Reactions Questionnaire originally developed by Derryberry and Rothbart (1988). The 77-item short form was used in this study which consists of a seven-point Likert scale and contains four main factors: negative affect,

extraversion/surgency, effortful control, and orienting sensitivity. The Adult Temperament Questionnaire has strong convergence with the Big Five scales as well (α = .64–.74; Evans & Rothbart, 2007). The four main factors along with 13 different subscales were examined in this study.

2.2.5. Depression Anxiety Stress Scale

The short form of the Depression Anxiety Stress Scale (DASS) was administered as a manipulation check to ensure that state conditions did not influence the results (or to control any significant impacts). The short form is a 21-item measure which instructed students to respond to the items only regarding their experiences in the last week (S. H. Lovibond & P. F. Lovibond, 1995). The scale utilized a four-point Likert scale with seven items for each subscale. Sample items include "I couldn't seem to experience any positive feeling at all," "I felt I was close to panic," and "I found myself getting agitated." The DASS anxiety and depression scales has been found to correlate α = .81 and α = .74 with the Beck Anxiety Inventory and Beck Depression scales, respectively (P. F. Lovibond & S. H. Lovibond, 1995).

2.3. Procedure

Upon receiving written consent of participation, a research assistant applied the three electrodes following the lead II placement on the limbs and connected the participant to the recording system. Participants sat in a padded desk chair facing a computer screen throughout physiological recording with arms placed on the desk at heart level. The first recording phase was a three-minute vanilla baseline period. The participant sat comfortably in the chair while a segment of a multicultural documentary, *Powaqqatsi: Life in Transformation* (Reggio, 1988) was played. This video has been used as a vanilla baseline in previous cardiovascular studies (Vella & Friedman, 2007; Waldron, Wilson, Patriquin, & Scarpa, 2015). The neutral stimulus was used to control for any possible preservation confounds from reading the consent form about a cognitive task. The second phase was a serial subtraction task. Participants started at 3,000 and attempted to count backward aloud by seven as fast as they could with as few errors as possible. This cognitive stressor lasted for three minutes before the research assistant stopped the participant. The final recording phase was a three-minute recovery period. The participant was instructed to sit in the chair comfortably with their eyes closed while attempting to not think about anything in particular.

Following the cardiovascular recording, electrodes were removed and the participant was allowed to work on the questionnaires at his or her own pace. A demographics questionnaire was presented first followed by the Penn State Worry Questionnaire, and the Emotion Regulation Questionnaire. Following the questionnaires of note, the study continued with the DASS (S. H. Lovibond & P. F. Lovibond, 1995) and the Adult Temperament Questionnaire (Evans & Rothbart, 2007) which were included as potential exploratory analyses. A final questionnaire page asked participants about their laboratory experience including the perceived difficulty and the stress level of the cognitive task. Following participation, students were debriefed about the purpose of the research study and allowed time to ask questions about the questionnaires. Overall, the laboratory study was completed in 45 minutes.

2.4. Data analysis

Data from the various questionnaires were transformed and high–low groups were created through a median split which involved controlling for gender. While there are issues with median splits, the trait worry groups created in this study (high worry M = 61.0, SD = 7.3; low worry M = 44.4, SD = 10.8) had scores mirroring participants recruited in Knepp and Friedman (2008). Manipulation checks were run on state conditions from the DASS and on the difficulty and stress of the cognitive stressor to eliminate any confounds to the trait hypotheses. These manipulation checks were to ensure that the cognitive task did function as a laboratory stressor and to ensure that state conditions did not bias the study. Had these factors significantly impacted rMSSD or high-frequency power across the study, they would have been controlled for in the final analyses. Table 1 includes the means and standard deviations for each questionnaire demonstrating the overall levels of worry, emotion reappraisal, emotion suppression, and state levels of depression, anxiety, and stress. A 2 (worry) × 2 (reappraisal) × 3 (pre, task, post) mixed repeated measures design was used to examine both markers of vagal tone. A similar 2 (worry) × 2 (suppression) × 3 (pre, task, post) mixed repeated measures design was also run on

Table 1. Questionnaire Means and SDs for full sample (*N* = 50)

	M	SD
Penn state worry score	52.16	12.31
Emotion reappraisal	29.48	6.13
Emotion suppression	14.22	5.08
State depression	3.26	3.87
State anxiety	3.20	2.70
State stress	5.96	3.91

the other element of emotion regulation. The Greenhouse-Geisser correction for sphericity was used for within-subjects analyses. Follow-up analyses of the moderation effects were explored using regression analysis.

3. Results

3.1. Manipulation checks

For state conditions, there was no relationship between state anxiety, state stress, or state depression and any of the parasympathetic variables (p values > .10). There was no relationship between how difficult or stressful a participant rated the serial subtraction task and any of the dependent variable outcomes (p values > .10). Finally, high and low trait worriers did not differ on either how difficult or stressful they felt the cognitive stress task was (p values > .10). The task did significantly impact both heart rate and heart rate variability. For example, heart rates were higher (M = 86.8, SD = 13.7) during the task phase when compared to the baseline (M = 75.0, SD = 13.5) and recovery periods (M = 75.5, SD = 13.7, F (1.3, 62.8) = 162.4, p < .001).

3.2. Trait worry and emotion regulation

In the first analysis of trait worry and emotion regulation, a two-way interaction was found for worry and emotion reappraisal on rMSSD (F (1, 36) = 4.238, p < .05) across all phases of the study. This interaction showed that low trait worriers who were also in the high emotion reappraisal group had significantly higher rMSSD than the other three groups. There were no other interactions or main effects on rMSSD (p values > .10). Figure 1 illustrates this two-way interaction. To further explore the interaction between trait worry and emotion reappraisal, regression analyses were run on each epoch. For the baseline period, the moderation effect had a significant impact on rMSSD (β = .41, p < .02). The effect was significantly impacted rMSSD during recovery epoch (β = .32, p < .05) but only had a trend toward significance during the task period (β = .31, p = .053).

A similar two-way interaction was found between worry and reappraisal on high-frequency power (F (1, 36) = 5.119, p < .05). As with rMSSD, low trait worriers who were also in the high emotion reappraisal group had significantly greater high-frequency power than the other three groups across all three phases of the study. Figure 2 displays the interaction of worry and emotion reappraisal on high-frequency power. There were no further interactions or any main effects on high-frequency power. When examining emotion regulation, trait worry, and their outcomes on heart rate, there were no significant interactions or main effects (p values > .10). With the follow-up analysis, the interaction significantly impacted baseline levels of high-frequency power (β = .34, p < .05). This effect on high-frequency power was also significant during the task (β = .34, p < .05) and recovery period epochs (β = .32, p < . 05).

With the relationship between trait worry and emotion suppression, there were no interaction effects on any of the vagal tone markers (p values > .10). With between-subjects main effects, there were two trends toward significance with Penn State Worry groups. In these cases, the trend was that low trait worriers had higher rMSSD (F (1, 43) = 3.677, p < .075) when compared to high trait worriers

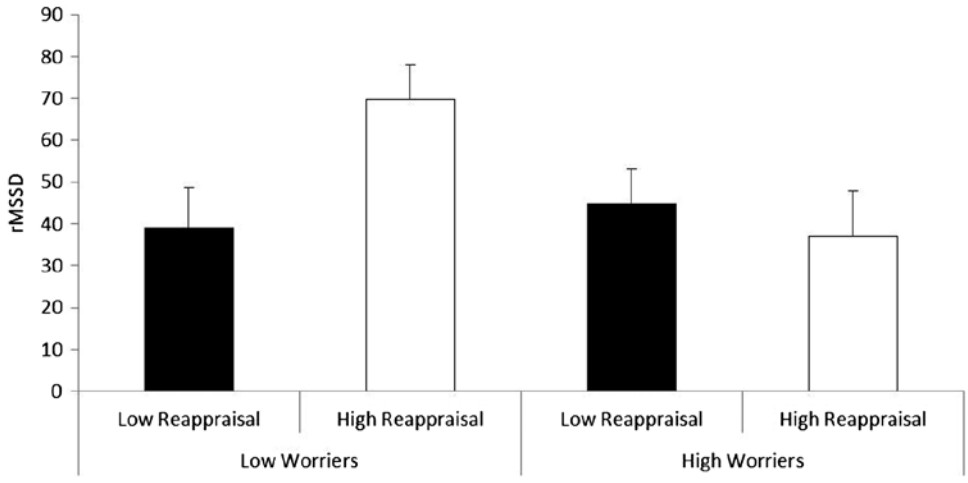

Figure 1. Interaction between trait worry and emotion reappraisal on rMSSD.

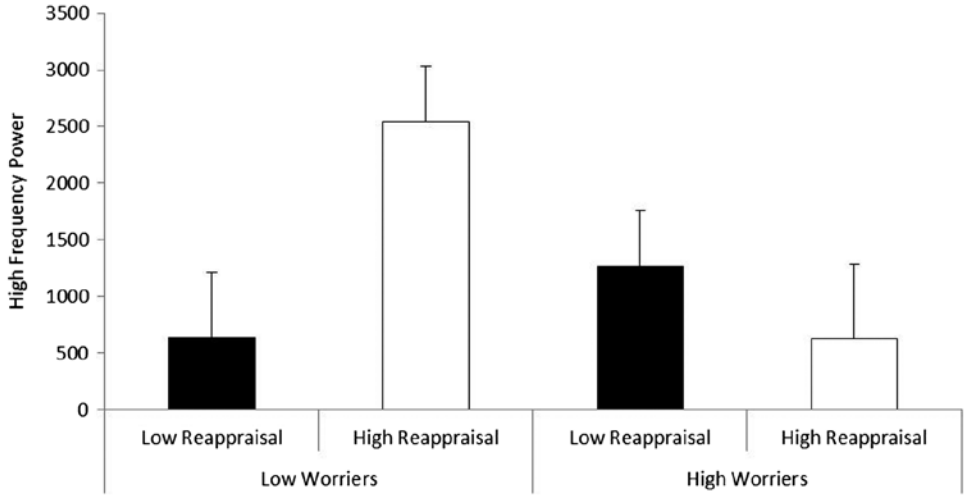

Figure 2. Interaction between trait worry and emotion reappraisal on high-frequency power.

across the phases. There were no worry or emotion suppression main effects related to high-frequency power. Finally, as with emotion regulation, there were no interaction or main effects relating trait worry and emotion suppression to difference in heart rate across the phases of the study (*p* values > .10)

4. Discussion

Within the investigation of trait worry and emotion regulation, the primary hypotheses were partially supported. This study found that low trait worriers who are high in emotion reappraisal had the most rMSSD and high-frequency power across all three phases of the study. However, there was more limited support for emotion suppression influences on the cardiovascular system (Quartana & Burns, 2010). This study did not support previous research by Santucci et al. (2008), which had noted that maladaptive emotion regulation strategies predicted low vagal tone, while adaptive emotion regulation did not predict vagal tone outcomes. In both situations, high trait worriers had lower parasympathetic control of the heart. This finding supports the previous research from both the trait worry research field (Lyonfields et al., 1995) and the clinical realm of generalized anxiety disorder (Thayer et al., 1996); however, it did not support Knepp and Friedman (2008) which noted a difference in chronic high heart rate, not heart rate variability. Aldao and Mennin (2012) was supported in that emotion reappraisal was a further benefit to the low worriers.

Examining the low and high groups for worry, this sample mirrored the non-clinical group findings of Knepp and Friedman (2008) which had similar high and low Penn State Worry Questionnaire group means. The cut-off points within the groups also mirrored previous work using clinical samples (Behar et al., 2003). Aldao, Nolen-Hoeksema, and Schweizer (2010) note the moderating effects of clinical versus normative samples of emotion regulation research. Furthermore, within this study, poor reappraisal was not a further detriment to high worriers. Relating to the stressor task in particular, individuals with low trait worry and high emotion reappraisal came into the lab with higher vagal tone and were able to keep higher parasympathetic control during the stressor task. This finding demonstrated an autonomic flexibility in these individuals which can function as a barrier to daily stress weakening the cardiovascular system.

4.1. Limitations

The primary limitation within this study was the utilization of a split-half analysis, which could have weakened how different the various low and high groups were. Using trait worry as an example, Knepp and Friedman (2008) had split their worry groups based on previous control and generalized anxiety disorder means using online questionnaire administration with laboratory follow-ups. While the split-half analysis in this study was not ideal, within the trait worry domain, the low group was similar to previous control groups, while the high group was just slightly below previous disorder groups. These values are similar to control group and clinical group averages found in the previous publication. All other major independent variables were also statistically different (all p values < .001). A secondary limitation was the use of a college student sample. With an average age slightly under 20 years, these high trait worry students may not have had enough time yet to experience the deleterious effects related to parasympathetic control of the heart. In this way, an older, clinical sample may be more likely to see a main effect for trait worry in addition to the trait worry by emotion reappraisal interaction.

4.2. Benefits and future research

The primary benefit of this particular study is that it combined various adaptive and maladaptive cognitions whereas much of the previous work in the field has not. Previously, physiological outcomes have been investigated within the realms of anxiety (Friedman, 2007; Mussgay & Rüddel, 2004; Pittig, Arch, Lam, & Craske, 2013), specific emotions (Stephens, Christie, & Friedman, 2010), and emotion regulation (Mauss et al., 2007); however, there had not been an attempt to find any intersections in these fields. This particular study noted that lower worriers with high emotion reappraisal are the particular group with high parasympathetic control of the heart. This interaction might be what drives the findings of previous works that only study anxiety and emotion regulation. It may be that it is less likely to find high emotion reappraisers in generalized anxiety disorder groups; which is why these patients consistently have lower vagal tone when compared with controls. It may also be that low and high trait worriers impact the findings of emotion-specific studies by populating one group or the other. One particular explanation for the cardioprotective nature of a low worry–high emotion reappraisal group could be cortical control of function. Wittling, Block, Genzel, and Schweiger (1998) supported the idea that vagal control of the heart is under control of the left hemisphere. Furthermore, Ahern and Schwartz (1985) argued for differential lateralization of emotionality with increased relative left hemispheric activation for positive emotions. In this way, it might be that left hemispheric activation by positive emotionality, decreasing worry, is driving increased vagal control of the heart.

There are various clinical implications for this study. Primarily, this study found that low worriers with high emotion reappraisal had better vagal control of the heart compared with the other three groups. In this way, neither low worry nor high emotion reappraisal on its own was protective. One clinical implication as it relates to this study is that the non-optimal groups would be at greater risk for coronary heart disease (Kubzansky et al., 1997). Aldao et al. (2010) noted the issues with rumination as a poor emotion regulation strategy; rumination as found in anxiety and worry often has an issue with clinical cardiac ailments (Brosschot, Gerin, & Thayer, 2006). Within generalized anxiety disorder, emotional difficulties have been noted as well as a decrease in mindfulness behavior (Roemer et al., 2009). From these issues, Mennin, Heimberg, Turk, and Fresco (2002) recommend that

an emotion regulation perspective could assist clients with generalized anxiety disorder. Furthermore, mindfulness training might be a clinical route to assist both emotion regulation deficiencies and anxiety–worry issues (Hofmann, Sawyer, Witt, & Oh, 2010; Jha, Stanley, Kiyonaga, Wong, & Gelfand, 2010). Finally as a societal note, Gluskin (2012) in his discussion of hooligan behavior, covered issues of negative inputs as a concern for processing information sources. Lang, Newhagen, and Reeves (1996) found that negative videos in news stories increase the negative emotional impact of the story and the cognitive processing of the message; yet, Grabe, Zhou, Lang, and Bolls (2000) found that while tabloids increase arousal and attention they do not impact memory and recall of this information. From this study, it would be the lack of long-term rumination from worry or decreased emotion reappraisal that would result in cardiovascular issues.

Expanding from this study, future research could take this trait worry and emotion regulation interaction and explore its impacts on the sympathetic nervous system. In particular, a new line of research exploring the cardiovascular system through blood pressure outcomes and impedance cardiography would test whether or not trait worry and emotion regulation interact on more pure sympathetic nervous system markers such as pre-ejection time. Emotion suppression might not have related with vagal tone, but it could still have negative outcomes for cardiovascular health if it interacts with high trait worry to result in a chronically overative sympathetic nervous system. Future research in this domain could also move forward with looking at interactions among temperament and emotion regulation as is being conducted with children. Finally, across all of these components, it would be ideal to see how these heart rate variability markers such as the standard deviation of RR intervals, coefficient of variation of RR intervals, rMSSD, SDNN, pNN50, and high-frequency power work within trait worry, emotion regulation, and temperament to impact long-term cardiovascular health.

Funding
The authors received no direct funding for this research.

Competing interests
The authors declare no competing interests.

Author details
Michael M. Knepp[1]
E-mail: kneppm@mountunion.edu
ORCID ID: http://orcid.org/0000-0003-3334-8065
Erin R. Krafka[2]
E-mail: krafka.8@wright.edu
ORCID ID: http://orcid.org/0000-0001-7270-4136
Erika M. Druzina[1]
E-mail: druzinem@mountunion.edu
[1] Department of Psychology and Neuroscience, University of Mount Union, 1972 Clark Avenue, Alliance, OH 44601, USA.
[2] Center for Global Health, Wright State University, 3123 Research Blvd., Suite 200, Kettering, Dayton, OH 45420-4006, USA.

Citation information
Cite this article as: The impact of trait worry and emotion regulation on heart rate variability, Michael M. Knepp, Erin R. Krafka & Erika M. Druzina, *Cogent Psychology* (2015), 2: 1038896.

References

Ahern, G. L., & Schwartz, G. E. (1985). Differential lateralization for positive and negative emotion in the human brain: EEG spectral analysis. *Neuropsychologia, 23*, 745–755. http://dx.doi.org/10.1016/0028-3932(85)90081-8

Aldao, A., & Mennin, D. S. (2012). Paradoxical cardiovascular effects of implementing adaptive emotion regulation strategies in generalized anxiety disorder. *Behaviour Research and Therapy, 50*, 122–130. http://dx.doi.org/10.1016/j.brat.2011.12.004

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review, 30*, 217–237. http://dx.doi.org/10.1016/j.cpr.2009.11.004

Appleton, A. A., & Kubzansky, L. D. (2014). Emotion regulation and cardiovascular disease risk. In J. Gross (Ed.), *Handbook of emotion regulation* (pp. 596–612). New York, NY: Guilford Press.

Behar, E., Alcaine, O., Zuellig, A. R., & Borkovec, T. D. (2003). Screening for generalized anxiety disorder using the Penn State Worry Questionnaire: A receiver operating characteristic analysis. *Journal of Behavior Therapy and Experimental Psychiatry, 34*, 25–43. http://dx.doi.org/10.1016/S0005-7916(03)00004-1

Berntson, G. G., Cacioppo, J. T., & Quigley, K. S. (1991). Autonomic determinism: The modes of autonomic control, the doctrine of autonomic space, and the laws of autonomic constraint. *Psychological Review, 98*, 459–487. http://dx.doi.org/10.1037/0033-295X.98.4.459

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*, 113–124. http://dx.doi.org/10.1016/j.jpsychores.2005.06.074

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*, 39–47. http://dx.doi.org/10.1016/j.ijpsycho.2006.07.016

Derryberry, D., & Rothbart, M. K. (1988). Arousal, affect, and attention as components of temperament. *Journal of Personality and Social Psychology, 55*, 958–966. http://dx.doi.org/10.1037/0022-3514.55.6.958

Evans, D. E., & Rothbart, M. K. (2007). Developing a model for adult temperament. *Journal of Research in Personality, 41*, 868–888. http://dx.doi.org/10.1016/j.jrp.2006.11.002

Friedman, B. H. (2007). An autonomic flexibility-neurovisceral integration model of anxiety and cardiac vagal tone. *Biological Psychology, 74*, 185–199. http://dx.doi.org/10.1016/j.biopsycho.2005.08.009

Friedman, B. H., & Thayer, J. F. (1998). Autonomic balance revisited: Panic anxiety and heart rate variability. *Journal of Psychosomatic Research, 44*, 133–151. http://dx.doi.org/10.1016/S0022-3999(97)00202-X

Gluskin, E. (2012). An argument regarding the nature of hooligan behavior. *Dialogues in Philosophy, Mental and Neuro Sciences, 5*, 51–53.

Glynn, L. M., Christenfeld, N., & Gerin, W. (2002). The role of rumination in recovery from reactivity: Cardiovascular consequences of emotional states. *Psychosomatic Medicine, 64*, 714–726.

Gouin, J.-P., Deschênes, S. S., & Dugas, M. J. (2014). Respiratory sinus arrhythmia during worry forecasts stress-related increases in psychological distress. *Stress, 17*, 416–422. http://dx.doi.org/10.3109/10253890.2014.949666

Grabe, M. E., Zhou, S., Lang, A., & Bolls, P. D. (2000). Packaging television news: The effects of tabloid on information processing and evaluative responses. *Journal of Broadcasting and Electronic Media, 44*, 581–598. http://dx.doi.org/10.1207/s15506878jobem4404_4

Gramzow, R. H., Willard, G., & Mendes, W. B. (2008). Big tales and cool heads: Academic exaggeration is related to cardiac vagal reactivity. *Emotion, 8*, 138–144. http://dx.doi.org/10.1037/1528-3542.8.1.138

Greaves-Lord, K., Tulen, J., Dietrich, A., Sondeijker, F., van Roon, A., Oldehinkel, A., … Huizink, A. (2010). Reduced autonomic flexibility as a predictor for future anxiety in girls from the general population: The TRAILS study. *Psychiatry Research, 179*, 187–193. http://dx.doi.org/10.1016/j.psychres.2009.04.014

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85*, 348–362. http://dx.doi.org/10.1037/0022-3514.85.2.348

Hofmann, S. G., Moscovitch, D. A., Litz, B. T., Kim, H.-J., Davis, L. L., & Pizzagalli, D. A. (2005). The worried mind: Autonomic and prefrontal activation during worrying. *Emotion, 5*, 464–475. http://dx.doi.org/10.1037/1528-3542.5.4.464

Hofmann, S. G., Sawyer, A. T., Witt, A. A., & Oh, D. (2010). The effect of mindfulness-based therapy on anxiety and depression: A meta-analytic review. *Journal of Consulting and Clinical Psychology, 78*, 169–183. http://dx.doi.org/10.1037/a0018555

Jamieson, J. P., Nock, M. K., & Mendes, W. B. (2013). Changing the conceptualization of stress in social anxiety disorder: Affective and physiological consequences. *Clinical Psychological Science, 1*, 363–374. http://dx.doi.org/10.1177/2167702613482119

Jha, A. P., Stanley, E. A., Kiyonaga, A., Wong, L., & Gelfand, L. (2010). Examining the protective effects of mindfulness training on working memory capacity and affective experience. *Emotion, 10*, 54–64. http://dx.doi.org/10.1037/a0018438

Knepp, M. M., & Friedman, B. H. (2008). Cardiovascular activity during laboratory tasks in women with high and low worry. *Biological Psychology, 79*, 287–293. http://dx.doi.org/10.1016/j.biopsycho.2008.07.002

Kreibig, S. D., Wilhelm, F. H., Roth, W. T., & Gross, J. J. (2007). Cardiovascular, electrodermal, and respiratory response patterns to fear-and-sadness-inducing films. *Psychophysiology, 44*, 787–806. http://dx.doi.org/10.1111/psyp.2007.44.issue-5

Kubzansky, L. D., Kawachi, I., Spiro, A., Weiss, S. T., Vokonas, P. S., & Sparrow, D. (1997). Is worrying bad for your heart? A prospective study of worry and coronary heart disease in the normative aging study. *Circulation, 95*, 818–824. http://dx.doi.org/10.1161/01.CIR.95.4.818

Lang, A., Newhagen, J., & Reeves, B. (1996). Negative video as structure: Emotion, attention, capacity, and memory. *Journal of Broadcasting and Electronic Media, 40*, 460–477. http://dx.doi.org/10.1080/08838159609364369

Lemaire, M., El-Hage, W., & Frangou, S. (2014). Reappraising suppression: Subjective and physiological correlates of experiential suppression in healthy adults. *Frontiers in Psychiatry, 5*(571), 1–9.

Levenson, R. W., Ekman, P., & Friesen, W. V. (1990). Voluntary facial action generates emotion-specific autonomic nervous system activity. *Psychophysiology, 27*, 363–384. http://dx.doi.org/10.1111/psyp.1990.27.issue-4

Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: Comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories. *Behaviour Research and Therapy, 33*, 335–343. http://dx.doi.org/10.1016/0005-7967(94)00075-U

Lovibond, S. H., & Lovibond, P. F. (1995). *Manual for the Depression Anxiety Stress Scales* (2nd ed.). Sydney: Psychology Foundation.

Lyonfields, J., Borkovec, T. D., & Thayer, J. F. (1995). Vagal tone in generalized anxiety disorder and the effects of aversive imagery and worrisome thinking. *Behavior Therapy, 26*, 457–466. http://dx.doi.org/10.1016/S0005-7894(05)80094-2

Mauss, I. B., Cook, C. L., Cheng, J. Y., & Gross, J. J. (2007). Individual differences in cognitive reappraisal: Experiential and physiological responses to an anger provocation. *International Journal of Psychophysiology, 66*, 116–124. http://dx.doi.org/10.1016/j.ijpsycho.2007.03.017

Mennin, D. S., Heimberg, R. G., Turk, C. L., & Fresco, D. M. (2002). Applying an emotion regulation framework to integrative approaches to generalized anxiety disorder. *Clinical Psychology: Science and Practice, 9*, 85–90.

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the penn state worry questionnaire. *Behaviour Research and Therapy, 28*, 487–495. http://dx.doi.org/10.1016/0005-7967(90)90135-6

Mitchell, J. H., & Shapiro, A. P. (1991). The confusing syndromes of cardiovascular autonomic imbalance: Cardiovascular dysautonomias. In A. P. Shapiro & A. Baum (Eds.), *Behavioral aspects of cardiovascular disease* (pp. 75–102). Hillsdale, NJ: Lawrence Erlbaum.

Mussgay, L., & Rüddel, H. (2004). Autonomic dysfunctions in patients with anxiety throughout therapy. *Journal of Psychophysiology, 18*, 27–37. http://dx.doi.org/10.1027/0269-8803.18.1.27

O'Connor, D. B., Ashley, L., Jones, F., & Ferguson, E. (2014). Maladaptive rumination moderates the effects of written emotional disclosure on ambulatory blood pressure levels in females. *Health Psychology and Behavioral Medicine, 2*, 1067–1077. http://dx.doi.org/10.1080/21642850.2014.973881

Pieper, S., Brosschot, J. F., van der Leeden, R., & Thayer, J. F. (2007). Cardiac effects of momentary assessed worry episodes and stressful events. *Psychosomatic Medicine, 69*, 901–909. http://dx.doi.org/10.1097/PSY.0b013e31815a9230

Pittig, A., Arch, J. J., Lam, C. W. R., & 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*, 19–27. http://dx.doi.org/10.1016/j.ijpsycho.2012.10.012

Plutchick, R. (1990). Emotions and psychotherapy: A psychoevolutionary perspective. In R. Plutchick & H. Kellerman (Eds.), *Emotion: Theory, research, and experience: Vol. 5. Emotion, psychopathology, and*

psychotherapy (pp. 3–41). San Diego, CA: Academic Press.
Pu, J., Schmeichel, B. J., & Demaree, H. A. (2010). Cardiac vagal control predicts spontaneous regulation of negative emotional expression and subsequent cognitive performance. *Biological Psychology, 84*, 531–540. http://dx.doi.org/10.1016/j.biopsycho.2009.07.006
Quartana, P. J., & Burns, J. W. (2010). Emotion suppression affects cardiovascular responses to initial and subsequent laboratory stressors. *British Journal of Health Psychology, 15*, 511–528. http://dx.doi.org/10.1348/135910709X474613
Reggio, G. (Director). (1988). *Powaqqatsi: Life in transformation* [Motion picture]. USA: MGM/United Artists.
Roemer, L., Lee, J. K., Salters-Pedneault, K., Erisman, S. M., Orsillo, S. M., & Mennin, D. S. (2009). Mindfulness and emotion regulation difficulties in generalized anxiety disorder: Preliminary evidence for independent and overlapping contributions. *Behavior Therapy, 40*, 142–154. http://dx.doi.org/10.1016/j.beth.2008.04.001
Santucci, A. K., Silk, J. S., Shaw, D. S., Gentzler, A., Fox, N. A., & Kovacs, M. (2008). Vagal tone and temperament as predictors of emotion regulation strategies in young children. *Developmental Psychobiology, 50*, 205–216. http://dx.doi.org/10.1002/(ISSN)1098-2302
Saul, J. P. (1990). Beat-to-beat variations of heart rate reflect modulation of cardiac autonomic outflow. *News in Physiological Science, 5*, 32–37.
Stephens, C. L., Christie, I. C., & Friedman, B. H. (2010). Autonomic specificity of basic emotions: Evidence from pattern classification and cluster analysis. *Biological Psychology, 84*, 463–473. http://dx.doi.org/10.1016/j.biopsycho.2010.03.014
Thayer, J. F., Friedman, B. H., & Borkovec, T. D. (1996). Autonomic characteristics of generalized anxiety disorder and worry. *Biological Psychiatry, 39*, 255–266. http://dx.doi.org/10.1016/0006-3223(95)00136-0
Thayer, J. F., Friedman, B. H., Borkovec, T. D., Johnsen, B. H., & Molina, S. (2000). Phasic heart period reactions to cued threat and nonthreat stimuli in generalized anxiety disorder. *Psychophysiology, 37*, 361–368. http://dx.doi.org/10.1111/psyp.2000.37.issue-3
Vella, E. J., & Friedman, B. H. (2007). Autonomic characteristics of defensive hostility: Reactivity and recovery to active and passive stressors. *International Journal of Psychophysiology, 66*, 95–101. http://dx.doi.org/10.1016/j.ijpsycho.2007.03.014
Waldron, J. C., Wilson, L. C., Patriquin, M. A., & Scarpa, A. (2015). Sexual victimization history, depression, and task physiology as predictors of sexual revictimization: Results from a 6-month prospective pilot study. *Journal of Interpersonal Violence, 30*, 622–639. http://dx.doi.org/10.1177/0886260514535258
Wittling, W., Block, A., Genzel, S., & Schweiger, E. (1998). Hemisphere asymmetry in parasympathetic control of the heart. *Neuropsychologia, 36*, 461–468. http://dx.doi.org/10.1016/S0028-3932(97)00129-2

***Cogent Psychology* (ISSN: 2331-1908) is published by Cogent OA, part of Taylor & Francis Group.**