Respiratory Sinus Arrhythmia During Episodes of Relived Sadness: The Role of Emotional Intelligence and Affect Intensity

by

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ABSTRACT

This study investigated respiratory sinus arrhythmia (RSA) during relived episodes of sadness as a function of emotional intelligence (EI) and affect intensity. RSA was hypothesized: 1) to be reduced during experienced sadness evidencing vagal withdrawal, 2) to show an inverse association with EI, particularly the managing emotions dimension, affect intensity, and depressive symptomatology. Fifty-six participants were recruited to fill out questionnaires and undergo three phases of psychophysiological recording (baseline, experiential sadness, recovery). Consistent with hypotheses, RSA amplitude was reduced during experiential sadness and magnitude of RSA reduction was inversely associated with both EI and affect intensity. However, magnitude of RSA response was not associated with the emotion management dimension of EI but rather the perceiving emotions dimension and was not associated with depressive symptomatology. Overall, the findings suggest that RSA (cardiac vagal tone) is more indicative of flexible responding to environmental demands rather than explicitly indexing emotion regulation.
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Respiratory sinus arrhythmia during episodes of relived sadness: The role of emotional intelligence and affect intensity

INTRODUCTION

Emotions have been considered the primary motivational system of human beings (Izard, 1991). It is unlikely that even the most mundane of actions occur without some affective component. Thus, emotions play an important role in organizing, motivating, and sustaining behavior.

Emotions can be thought of as real-time, on-line indications of how well we think we are coping with environmental affordances, challenges, demands, and threats (Mathews, Zeidner, & Roberts, 2004). They percolate throughout every area of life and recognition is snowballing that the ability to use and regulate emotions is integral to successful life outcomes (Gratz & Roemer, 2004; Gross, 1998; Gross & Munoz, 1995; Mathews, et al., 2004).

Advances in the study of emotion emphasize that, under the right conditions, they allow us to negotiate environmental challenges in an adaptive manner (Gross, 1998; Mathews, et al., 2004). This suggests that the ability to harness and use emotions for benefit is central to mental health. Indeed, emotion management is implicated in over half of the DSM-IV axis I disorders and all of the DSM-IV axis II disorder. There is causal evidence of a specific role for the inability to use and properly manage emotions in substance abuse (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996), general anxiety disorder (Mennin, Heimberg, Turk, & Fresco, 2002), depression (Coiro & Gottesman, 1996), and borderline personality disorder (Linehan, 1993).

Conceptualizing Emotions

Emotions are multi-component response tendencies which unfold over a relatively short period of time (Fredrickson, 2004). The emotion process begins with an assessment or appraisal
of the personal meaning of an eliciting circumstance and leads to action tendencies, facial expressions, and physiological changes (see Fredrickson & Levenson, 1998). Many historical theories of emotions have postulated that these, often subliminal, characteristic changes conveying emotional information can be inferred through an examination of one’s underlying physiology. With the help of new scientific advancements and a better understanding of neuroanatomy, research has implicated several physiological systems in the regulation and expression of emotions (Porges, 2003). Some of the most promising and influential research linking emotion processes and physiology examines the relationship between the cardiovascular system and the brain (Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996).

The Autonomic Nervous System and Emotion Processing

The emotions experienced while interacting with the environment are associated with varying degrees of physiological arousal (Appelhans & Luecken, 2008). The key system involved in generating and modulating this physiological arousal is the autonomic nervous system (ANS). The ANS is the portion of the nervous system that controls visceral functions of the body. This system innervates smooth and cardiac muscles and glands (Porges, 2003). Functioning primarily at the unconscious level, the ANS is partitioned into the sympathetic and parasympathetic divisions.

The sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS) often work antagonistically to produce varying degrees of physiological arousal. The SNS is an excitatory system that acts to mobilize resources for the purpose of fight or flight activity in the event of environmental stressors. The PNS is an inhibitory system that acts to decrease physiological arousal and promote rest, relaxation and regeneration during moments of safety. Thus, dominance of the SNS is associated with increases in pulse, blood pressure, heart rate, and
arousal while dominance of the PNS is associated with decreases in pulse, blood pressure, heart rate and arousal. The ease with which an individual can transition between high and low states of arousal correlates with how rapidly the ANS acts to vary heart rate (Appelhans & Luecken, 2008). Theories exist linking two physiological regulatory phenomena - respiratory sinus arrhythmia (RSA) and heart rate variability (HRV) - to the experience and regulation of emotions.

1.1 Respiratory Sinus Arrhythmia

Respiratory sinus arrhythmia is a cardio-respiratory phenomenon characterized by heart rate (HR) or R-R peak inter-beat interval (IBI) fluctuations that are in phase with inhalation and exhalation (Grossman & Taylor, 2007). Typically, HR accelerates during inhalation and decelerates during expiration. This waxing and waning of HR due to breathing is believed to represent the functioning of the vagus nerve in regulating cardiophysiology and is often referred to as “vagal tone.” Vagal tone is thought to index parasympathetically mediated activity of the vagus nerve. It has been argued that vagal tone underlies the ability to respond adaptively to emotional provocation, acting as a protective factor against a range of mental health outcomes (Denver, Reed, & Porges, 2007; Porges, et al., 1996). Among other things, this is thought to be because enhanced vagal tone indexes a process of self-management and attention focus that is necessary to modulate emotional state.

Magnitude of RSA is believed to be a linear indicator of vagal tone because the myelinated vagal efferents that synapse on the sinoatrial node (the pacemaker) of the heart have a respiratory rhythm that closely matches RSA (Porges, 2003). The vagus nerve is the 10th cranial nerve which innervates the heart at the sinoatrial node. This cardiac branch of the vagus is a branch of the ANS that helps maintain homeostasis through the regulation of heart rate. In
general, cardiac vagal tone acts to enhance homeostatic functions during states of low environmental demand and regulates cardiac output to support specific behaviors during times of environmental challenges (Porges, et al., 1996). Thus, RSA is believed to index vagal tone which contributes to the ability to respond emotionally to the environment (DiPietro & Porges, 1991; Hofheimer, Wood, Porges, Pearson, & Lawson, 1995). The only time that RSA does not reflect vagal tone seems to be when the body has high levels of inspired and arterial CO₂, such as during intense activity. This confound will not affect the proposed study as participants will not undergo any form of physical activity.

Conceptualizations of RSA as exclusively representing parasympathetic activity derive from the observation that only parasympathetically-mediated cardiac activity has a latency of action quick enough to generate changes in line with respiration. Research has found that the two branches of the ANS rely on two different signalling mechanisms with different temporal effects. Sympathetic influences on heart rate are mediated by transmission of norepinephrine and display a slow course with peak cardiac activation occurring after four seconds and return to baseline after twenty seconds. In contrast, parasympathetic activation is mediated by a faster acting acetylcholine transmission with peak activation occurring after one half second and return to baseline after one second (Berntson, et al., 1997). However, recent research has challenged this view finding that mild sympathetic activity can attenuate RSA (Grossman & Taylor, 2007; Hedman, Tahvanainen, Hartikainen, & Hakumaki, 1995). As discussed later, the present study will report parasympathovagal balance as an index of the relative activation of the SNS and PNS.

1.2 Respiratory Sinus Arrhythmia and Emotions during Childhood

The majority of research examining the relationship between RSA, emotions and health outcomes has examined the construct among infants and children. This is the population from
which many prominent theories of RSA have been generated. These theories have been found reliable and consistent.

Longitudinal evidence has found infant and childhood RSA to be a risk factor for a multitude of adverse mental health outcomes and a protective factor fostering social skills and affect regulation (Eisenberg, et al., 1996; Fabes, Eisenberg, & Eisenbud, 1993). In 1996, Porges and colleagues examined the relationship between vagal tone (RSA) among infants at 7-9 months of age and behavioral problems at age three. Infants who were better able to apply their "vagal brake" (increase vagal tone) during demanding tasks scored lower on social withdrawal, as well as depressive and aggressive behavior at age three. Alternatively, smaller vagal adjustments during demanding tasks among infants were associated with more behavioral problems at age three. This suggests that RSA, the ability to apply the vagal brake in particular, is an important physiological component of appropriate engagement with the environment. Consistent with this notion, larger changes in RSA during social tasks are related to later life adjustment (Hastings, et al., 2008). Furthermore, a better ability to apply the vagal break during social interaction is related to fewer externalizing and internalizing behaviors, better self-regulation across time (Hastings, et al., 2008).

Vagal withdrawal in children is believed to be a physiological strategy promoting sustained attention and active coping behaviors that are mediated by parasympathetic activation (Porges, et al., 1996). Congruent with this notion, children exhibiting greater vagal withdrawal during demanding tasks have shown better state regulation, fewer behavioral problems, and more appropriate emotion regulation during preschool (Calkins, 1997; Calkins & Dedmon, 2000; Calkins & Keane, 2004). In 2004, Calkins and Keane examined the relationship between RSA at age two and task demand as well as behavioral problems at age four. Four year olds were found
to exhibit greater vagal withdrawal suggesting that the RSA suppression response grows more prominent with age. Furthermore, the RSA suppression response was found to vary by type of challenge. Finally, children with stable high RSA at age two were rated by their mothers as more socially skilled and less negatively reactive. These children also exhibited fewer behavioral problems at age four.

One recent study examining the relationship between RSA reactivity, emotion regulation and depressive symptomatology among children found RSA reactivity to be a potential contributor towards children's emotional resilience and adjustment (Gentzler, Santucci, Kovacs, & Fox, 2009). The study found that RSA reactivity in children six to thirteen years of age predicted emotion regulation to dysphoria and depressive symptomatology over time. However, baseline levels of RSA were unrelated to either emotion regulation or depressive symptoms, suggesting that the underlying level of RSA may not have the same importance as children's ability to up and down regulate vagal activity during demanding tasks.

In summary, RSA reactivity during childhood offers a potentially useful tool for predicting problems in behavior, mental health and emotion regulation. Furthermore, a child's ability to apply the vagal brake during demanding tasks appears to reflect the ability to regulate emotions and employ effective coping techniques during stressful environmental circumstances. What remains unclear is exactly how RSA develops and changes over time and which variable might influence this relationship.

1.3 Respiratory Sinus Arrhythmia and Emotions during Adulthood

Research and conceptualizations of RSA in adults is considerably more recent than in children but still illuminates many important characteristics that need be considered. An examination of RSA and emotional responding among female college students revealed that
baseline RSA and changes in RSA index emotion expression and emotion regulation (Butler, Wilhelm, & Gross, 2006). Between-person differences in resting RSA and within-person changes in RSA activity were examined during dyadic communication over an emotion eliciting video clip. Importantly, either no instructions were given (control condition) or participants were asked to employ suppression or reappraisal coping techniques during the communication. Results indicated that higher resting RSA was associated with negative emotional experience and increased levels of negative expressive behavior with less positive expressive behavior which suggests that tonic RSA can be used as an index of emotion expression. Furthermore, RSA increased significantly from baseline to conversation for both the suppressor and the reappraiser coping groups indicating that phasic RSA indexes people's attempts to actively regulate their emotional expressivity.

Another study was able to show that RSA is a marker of flexible responding during highly emotional situations and may serve as a protective factor in vulnerable populations. A study examining rejection sensitivity, hostile conflict behavior and emotion control among 41 undergraduates was able to demonstrate that rejection sensitivity and RSA interact to predict maladaptive conflict behavior (Gyurak & Ayduk, 2008). In this relationship, high RSA was related to reductions in conflict behavior among those high and low in rejection sensitivity indicating that RSA buffers the tendency for those who are rejection sensitive to use conflict behavior. Interestingly, emotion control was found to fully mediate the RSA by rejection sensitivity interaction on conflict behaviour. This suggests that it is the emotion regulating ability of high RSA individuals that diminishes their use of conflict behavior.

Other studies have shown that phasic changes in RSA are sensitive to personally relevant material among adults. Reductions in RSA have been found among participants who are asked to
watch videos of themselves singing and giving a speech while in the presence of other individuals. This suggests that the employment of the vagal brake is related to potentially embarrassing situations (Hofmann, Moscovitch, & Kim, 2006). In support of this finding, the same participants did not show the same vagal withdrawal while preparing for the song and the speech task indicating that RSA may index more emotional content than general anxiety. In line with this, RSA has been found to decrease significantly while watching either a positive or a negative video clip but not during a neutral control (Frazier, Strauss, & Steinhauer, 2004).

Another closely related study found that individuals who exaggerated their GPA during an interview experienced greater increases in their RSA than individuals who did not. Given that being caught in a lie about your GPA during an interview poses a potentially embarrassing situation, this study suggests that magnitude of RSA change may index emotion regulation independent of the direction of effect.

Of interest, one experiment was able to link tonic RSA to positive affect. Individuals who were high in tonic RSA scored higher in positive affect and optimism (Oveis, et al., 2009). This relationship held independent of negative affect. Inconsistent with other experiments, RSA did not change while viewing positive, negative or neutral film clips. However, this study utilized video clips that were two minutes in duration rather than the research standard of five minutes in length. This increase in RSA measurement time would give a more representative RSA spectral value which may affect the results.

Respiratory Sinus Arrhythmia and Emotional Intelligence

If RSA truly indexes emotion processing and emotion regulation then it should show a clear and positive association with emotional intelligence (EI). Although definitions vary, EI is best conceptualized as a form of intelligence used to process and benefit from emotions
(Mathews, et al., 2004; Mayer & Salovey, 2000). From this perspective, EI is composed of mental skills, abilities and capacities. Specifically, emotional intelligence involves the ability to be aware of and perceive emotions, self-generate emotions to assist with thought, understand emotions and the ways in which they are used, and to regulate emotions to promote personal growth (Mayer & Salovey, 1997). Conceptualized in this manner, EI and RSA can both be viewed as relating to an individual’s ability to respond emotionally to environmental demands.

One well validated, reliable and conceptually rich EI test is the Mayer-Salovey-Caruso-Emotional-Intelligence-Test (MSCEIT; (Mayer, Salovey, & Caruso, 2000). The MSCEIT divides EI into four abilities: i) identifying emotions, ii) using emotions to facilitate thought, iii) understanding emotions and iv) managing emotions. The theoretical underpinnings of the MSCEIT imply a hierarchical structure where emotion management would be closer to a general factor of EI than would lower level processes such as emotion identification (Mathews, et al., 2004). Given that the MSCEIT is a performance measure of EI, if higher levels of RSA truly represent an index of emotion regulation process, then higher tonic levels of RSA during baseline and greater changes in phasic RSA during emotional provocations can be hypothesized to be associated with higher scores on global EI and emotion management in particular. Furthermore, it is plausible to suggest that MSCEIT mediates the relationship between RSA and depressive symptomatology in much the same manner as did emotion regulation in a previous study involving depression in children (see Gentzler et al., 2009).

RSA, EI, and Depressive Symptomatology

Depression is a common and debilitating mental health conditions. It is a mood disorder characterized by prolonged periods of sadness, depressive affect, and anhedonia. One of the primary characteristics of depression is dysregulation of emotions. Depressed individuals can be
seen as unable to properly regulate the frequency, intensity, and duration of negative emotions, especially sadness, leading to heightened and prolonged bouts of depressed affect (Gross & Munoz, 1995). If depression is characterized by problems with emotion regulation and sadness than depressive symptomatology should be linked to RSA during emotion provocations that involve experiences of sadness.

*Heart rate variability and measurement of RSA*

Emotion regulation depends on the ability of an individual to adjust physiological arousal on a regular basis (Gross, 1998). In order to perform this task, a flexible autonomic nervous system allowing for rapid generation or modulation of physiological and emotional states is required. Heart rate variability offers a method of capturing flexibility of the ANS by measuring the continuous interplay between the SNS and PNS influences on heart rate (Appelhans & Luecken, 2008). Respiratory sinus arrhythmia is one major component of what is measured as HRV. A large majority of parasympathetically mediated variation in heart rate is produced by RSA (Berntson, et al., 1997), and many researchers report the magnitude of RSA as an index of parasympathetically mediated HRV.

The present study will use spectral analysis of participants’ ECG as a means of obtaining HRV and calculating RSA. The spectral analysis derives HRV by mathematically estimating the variation among a set of temporally ordered R-R intervals known as inter-beat-intervals (IBI's; (Appelhans & Luecken, 2008). In essence, the time between each individual heart beat is derived and used to determine which influence PNS, SNS or a mixture of the two is responsible for generating such a heart beat. The variability, representing varying levels of autonomic activity, is then transformed into frequencies (these frequencies are expressed in hertz, calculated in cycles per second), resulting in the construction of an ECG power spectrum. This process involves
running a mathematical transformation called a Fast Fourier Transformation on successive IBI's to decompose an ECG signal into a series of linear waveforms or frequencies. This process is called power spectral density analysis (Berntson, et al., 1997), and can be performed through the use of commercially based software such as MindWare's HRV program.

The power spectrum measures variance of heart rate derived at different frequencies and contains prominent bands representing the major oscillatory components of HRV thought to reflect underlying autonomic activation (Task Force, 1996). The high-frequency (HF) component occurs at the frequency of adult respiration, 0.15 - 0.40 Hz and reflects RSA (Berntson, et al., 1997). The very-low-frequency (VLF) component ranges between 0.0033 and 0.04 Hz and is an index of SNS activity (McCraty, Atkinson, Tomasino, & Bradley, 2006). Finally, a low-frequency (LF) component occurs within 0.04 and 0.15 Hz. Controversy exists as to whether LF HRV is an index reflecting primarily SNS activation or a mixture of PNS and SNS activation (Task Force, 1996). It is likely that LF HRV indexes activation primarily of the SNS but also of the PNS. Owing to the concern that LF HRV contains PNS influence, many researchers report the LF to HF ratio as a measure of "sympathovagal balance" (Appelhans & Luecken, 2008; Eckberg, 1997; Task Force, 1996). Because SNS and PNS act antagonistically to control heart rate, sympathovagal balance represents relative shifts between SNS and PNS activation.

In summary, HRV can be viewed as an index of central autonomic function with RSA being one prominent component. This means that HRV and RSA are calculated using the same underlying principles and mathematical transformations and that both are suspect to many of the same confounds.

*Potential Confounds of Respiratory Sinus Arrhythmia*
Research has shown that measures of HRV and RSA are sensitive to movement and activity during recording (Grossman & Taylor, 2007; Wilhelm, Pfaltz, Grossman, & Roth, 2006). As such, movement and speech will be monitored and flagged in the present study. Furthermore, RSA is strongly associated with both respiration and metabolism suggesting that physical activity can influence tonic RSA (Grossman & Taylor, 2007). Owing to this consideration, participant’s level of physical activity will be ascertained through the use of the Paffenbarger Physical Activity Index and used as a potential covariate.

There is debate as to whether respiration rate and tidal volume confound measures of RSA. On one hand, Grossman and Taylor (2007) demonstrate that RSA is inversely related to respiration rate and directly related to tidal volume. These researchers recommend the use of paced breathing to an auditory tone as a means of controlling for respiration rate and the use of a within-individual regression approach of regressing participants RSA against measurements of tidal volume as a means of controlling for tidal volume. On the other hand, Denver, Reed and Pories (2007) demonstrated that the relationship between respiration rate and RSA is far from lawful and unlikely to be of concern during psychophysiological recording. Furthermore, Denver and colleagues suggest that the use of a paced breathing exercise can create confounds in both the frequency and amplitude components of RSA. Owing to these considerations, naturally occurring respiration rate and depth will be monitored using a respiratory band and used as a control variable will be performed.

If RSA is indicative of emotional expressivity as suggested by Butler, Wilhelm and Gross (2002) than it should show a prominent relationship with affect intensity. Individuals that are more apt to experience affect intensely should have developed a larger vagal brake response to
compensate for this. Participants level of affect intensity will be measured and used as a potential confounding influence in the present study.

Finally, RSA has shown a relationship with age but not gender. Respiratory sinus arrhythmia changes as a function of age (Craft & Schwartz, 1995), calling for the use of age as a potential covariate in the present study. Unlike age, gender is not considered to be a factor in the measurement of RSA as no research examining RSA has yet to find gender effects. However, the possibility of gender effects will be explored with no expectations.

**Purpose:**

The present study examined the relationship between cardiovascular physiology and emotion responding. Specifically, the relationship between vagal-tone and flexible responding during episodes of re-lived sadness was examined. If vagal tone, measured through RSA, is indicative of flexible cardiovascular responding then we can expect participants to experience vagal withdrawal during experiential sadness recall.

\(H_1:\) There will be an effect of phase on RSA such that vagal withdrawal (reductions in RSA) will be evident during the experiential sadness recall phase, indicating an application of the vagal-brake.

Furthermore, the notion that RSA is indicative of emotion regulation and the ability to respond to the external environment in an emotionally adaptive manner will be tested. The relationship between tonic RSA, phasic RSA, and EI will be used to test this relationship under the following hypotheses.

\(H_2:\) Tonic RSA at baseline will be positively related to EI, consistent with the expectation that high RSA at baseline better equips individuals to respond intelligently to emotional situations.
The magnitude of phasic changes in RSA from baseline to the experiential sadness recall phase will be negatively related to EI such that more emotionally intelligent individuals are better able to apply the vagal brake.

In addition, the theory that flexible responding is inversely related to depression was tested. If, as research and theory suggests, depression is characterized by problems with emotion regulation then depressive symptoms should be linked to RSA during emotion provocations.

$H_4$: Magnitude of RSA changes during emotion provocation will be inversely associated with depressive symptomatology.

Finally, the idea that EI might mediate the relationship between people’s ability to regulate emotions at a physiological level and the experience of depressive symptoms was explored. Being emotionally intelligent is believed to act as a buffer against depression (Mayer, Salovey, & Caruso, 2008). Emotionally intelligent individuals are believed to be better able to regulate their emotions, more likeable and easier to be around, and less likely to experience prolonged bouts of negative affect. Research examining diverse populations supports this idea finding that high emotional intelligence is inversely related to depressive symptomatology and depression per se (Extremera & Fernandez-Berrocal, 2006). In light of these findings, EI was hypothesized to mediate the relationship between RSA and depressive symptomatology.

$H_5$: The relationship between RSA and depressive symptomatology will be significantly reduced when EI is entered as a mediating variable.

METHODS

All necessary ethics forms were submitted for approval to the University of Northern British Columbia’s (UNBC) Research Ethics Board prior to commencing data collection. The
ethics approved informed consent and participant debriefing forms can be found in Appendices E and F respectively.

Participants:

Fifty-six students from the University of Northern British Columbia participated in this experiment. Thirty-five participants recruited through the psychology undergraduate research pool received one bonus credit applied to a psychology course and 21 participants recruited through flyers received $10.00 for their time. There were equal numbers of males and females in the study (28 per cell) and an overall mean age of 26.38 (SD = 9.35, range =18-54).

Apparatus and Materials:

Physiological data were collected using a Dell Optiplex GX620 computer system with Biopac physiological recording hardware running AcqKnowledge 3.7.3 software. For electrocardiogram recordings, participants were connected to three leads using Vermed (Ag/Ag) disposable ECG electrodes attached to each wrist and the right shin. For measures of respiration rate and depth, the respiratory belt TSD201 from Biopac systems was placed around participant’s abdomen. The dependent variables were extracted from the ECG and respiration recordings, respiration rate, respiratory sinus arrhythmia (RSA), heart rate (HR), LF/HF ratio serving as an index of sympathovagal balance, offline using Mindware HRV 2.16 software.

Scales and Questionnaires:

Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT; Mayer, et al., 2000). The MSCEIT is a performance based measure of emotional intelligence designed for adults age 17 and older. The MSCEIT measures four components of EI measured in a hierarchical fashion: i) identifying emotions, ii) using emotions to facilitate thought, iii) understanding emotions and iv)
managing emotions. Participants are required to complete 12 subscales to derive scores on the four branches and an overall score on EI. The scale is administered online and can be written in 30-45 minutes with scoring performed by Multi-Health Systems. Mayer, Salovey et al. found the MSCEIT to possess discriminant and convergent validity and to have adequate reliability (Cronbach Alpha overall = .91, range .76 for the facilitating component to .90 for the perceiving component). Of interest, Mathews, Zeidner and Roberts (2004) criticize the MSCEIT as being more effective for screening emotional stupidity than discriminating levels of EI at the upper ranges (i.e., the emotionally gifted). In spite of this criticism, the MSCEIT is the most popular and well-validated of the EI tests.

_Paffenbarger Physical Activity Index_ (PAI; Paffenbarger, Wing, & Hyde, 1978) (see Appendix A) is a measure of physical activity that is also known as the College Alumnus Questionnaire (CAQ). The PAI quantifies caloric expenditure on a weekly basis by asking participants how many blocks they walk, stairs they climb, and sports, leisure, and recreation time activities that they partake in. The PAI has shown adequate reliability and validity in being related to caloric expenditure, BMI, VO2_max, and the amount of time spent sweating and exercising (http://appliedresearch.cancer.gov/tools/paq/v026.html National Cancer Institute, 2009).

_Inventory of Depressive Symptomatology – Self Report_ (IDS-SR; Rush, Carmody, & Reimitz, 2000) (see Appendix B) is a 30 item self report measure assessing depressive symptomatology. Participants are asked to report on the frequency of several depressive symptoms using a 0-3 scale. The IDS shows excellent psychometric properties as well as construct validity being correlated with the Hamilton Rating Scale for Depression (Rush, Gullion, Basco, Jarrett, & Trivedi, 1996).
Affect Intensity Measure (AIM; Larsen, 1984) (see Appendix C) is a 40 item questionnaire that assesses the intensity with which individuals usually respond to emotional situations. The AIM employs a 6-point Likert scale of agreement/disagreement that has proven both reliable and valid in discerning emotional reactivity among individuals (Larsen & Diener, 1987).

Emotion Reactions Questionnaire (ERQ) (see Appendix D) is a 6-item scale assessing the presence of Ekman’s six basic emotions and the intensity to which they are felt during experiential recall. The ERQ asks participants to report on the six basic emotions using a 0 (no emotion present) to 7 (extremely intense emotion) scale. This scale measures the intensity and impurity of participants re-experienced emotions occurring during experiential recall. Two variables were extracted from this scale: (1) participants rating on subjective sadness was extracted as ERQ Sadness; while (2) participants ratings on the five remaining emotions were summed together and extracted as ERQ Impurity.

Content Analysis of Relived Episodes of Sadness

Descriptions of participants prepared sad events were explored to determine if the objective level of sadness was related to vagal tone response. Two trained lab assistants read transcribed versions of the experienced sadness recall forms and rated the level of sadness that the event would typically bring about. Lab assistants rated sadness on a 0 (no sadness present) to 10 (worst sadness imaginable) scale. Interrater agreement was excellent, \( r = .83 \).

In addition, we analyzed participants’ responses for emotional and cognitive-related content using Pennebaker’s (Pennebaker, Francis & Booth, 2001) Linguistic Inquiry and Word Count (LIWC) program. The LIWC is a computer-based linguistic analysis program designed to infer an individual’s psychological processes, including emotional, social and cognitive
processes, and personal concerns by analyzing textual data from the individual. Pennebaker’s internal dictionary is utilized as the LIWC’s frame of reference encompassing 2,300 words. The LIWC program has been found to be externally valid and internally reliable to other approaches (Pennebaker & Francis, 1996; Pennebaker & King, 1999). Participants’ experienced sadness recall forms were directly transcribed into notepad files and submitted to the LIWC2001 program. Several psychological processes and personal concerns were generated for each participant.

*Procedures*

On a separate occasion before entering the lab, participants were instructed to complete the MSCEIT and an experienced sadness recall form. The MSCEIT was completed online through Multi-Health-Systems while the experienced sadness recall form involved writing about a moment in the participant’s life during which they experienced an intense period of sadness. Relived episodes of sadness were required in enriched detail by having participants recall as much central and cursory detail as possible including: precipitating events, parties involved, feelings experienced, bodily reactions of such feelings, and life changes that arose. The experienced sadness recall form can be found in Appendix F.

Participants reported to the laboratory for a single session. There was a brief habituation period where participants were informed of general reasons why researchers are interested in obtaining psychophysiological information and how those recordings are obtained. To ensure consistency and clarity, the researcher read the study instructions and the informed consent to each participant (see Appendix G). The participants were given the opportunity to ask any questions before signing the consent form. Participants then completed a demographic form.
(Appendix E) followed by the PAI, the IDS-SR and the AIM. Complete listings of the demographics are listed in Table 1.

Obtaining Physiological Recordings

After completing the questionnaires, participants were fitted with ECG electrodes and a respiratory band, and then seated in a comfortable reclining chair that minimized postural changes. Immediately prior to recording, participants were instructed to refrain from talking, falling asleep, performing exaggerated bodily movements or intentionally altering their respiratory patterns.

Once seated in the reclining chair, having all of their questions answered, participants' underwent three recording periods each spanning five minutes. These durations allowed sufficient sampling of heart period for spectral analysis to obtain measures of RSA. The three periods were: baseline recording, experiential recall recording, and recovery. Participants were instructed to clear their minds and relax during baseline and recovery conditions. During the experiential recall period, a protocol was implemented that has been used in this laboratory extensively and for which there is good evidence of induction of the relevant mood state (Prkachin, Mills, Zwaal, & Husted, 2001; Prkachin, Williams-Avery, Zwaal, & Mills, 1999; Rainville, Bechara, Naqvi, & Damasio, 2006).

Experiential emotion inductions were based on the narratives that participants were asked to provide during pre-assessment. While the participants completed questionnaires, the experimenter prepared a synopsis of the experienced sadness recall form that was read to the participant at the start of the experiential recall period. During the description the experimenter encouraged the participant to fall into the designated emotional experience. This was abetted by directing the participant's attention to the stimulus cues and bodily sensations that occurred when
the incident originally took place. The experimenter then allowed the participant to work on generating the emotion him or herself. On one subsequent occasion, the experimenter recued the subject by drawing his or her attention to the stimuli and feelings associated with the event. Participants were debriefed at the end of the study (see Appendix H), given the opportunity to ask any questions, and thanked for their time before leaving the laboratory.

RESULTS

Overview of Analysis:

Upon enrolling in the study, all participants were directed to www.MHSassessments.com where they completed the MSCEIT online through Multi-Health-Systems. Raw data from the MSCEIT was scored by Multi-Health-Systems using expert scoring adjusted for ethnicity. Expert and consensus normative scoring procedures for the MSCEIT are very similar with correlations ranging from .93 to .99, neither procedure having a particular advantage over the other (Mayer, Salovey & Caruso, 2002). The present study utilized expert scoring with the assumption that this technique is less sensitive to influences of time and generalizability. The effect of ethnicity has been found to account for 6.7% of scores in EI (Mayer, Salover & Caruso, 2002). Given that the sample studied was not large enough to test or statistically adjust for ethnicity I elected to have Multi-Health-Systems score the MSCEIT adjusting for ethnicity. Raw scores on the MSCEIT were obtained from Multi-Health-Systems in the form of an Excel spreadsheet.

The demographics form, PAI, IDS-SR, AIM, and ERQ were paper and pencil based; therefore, the data were manually entered into an Excel spreadsheet. The PAI, IDS-SR and AIM were scored using appropriate Excel mathematical functions and the raw data were imported directly into SPSS, Version 18. All psychometric data were screened for potential outliers and missing values. There was no missing data. Inspection of skew, kurtosis, and histograms for the
variables indicated the presence of reasonably normal distributions among variables. Although the PAI and the IDS-SR could not be examined in such a manner, the AIM showed good internal consistency with a Cronbach’s Alpha of .84.

Continuous recordings of ECG and respiration were first scanned for artefacts as recommended by Berntson, Quigley, Jang, and Boysen, (1990). No participants were excluded on these grounds. Next, ECG and respiration recordings from the AcqKnowledge 3.7.3 software were reformatted offline into individual files so that the baseline, experiential recall, and recovery phases were exactly 300 seconds in length. Reformatted data were entered into commercially available software (Mindware HRV 2.16, Mindware, Gahana, OH) to independently extract physiological variables (heart rate, respiration, RSA and LF/HF ratio) in 1-min intervals. The Mindware software flagged abnormalities in the ECG recordings and judgments were made to correct for abnormal beats and artefacts using recommendations from Berntson et al., (1990).

The initial physiological recordings were based on five one minute epochs across three phases (baseline, experiential recall, and recovery). These 15 epochs were examined for outliers using z-score comparisons. Z-scores fell below a cut off value of 3.29, confirming a uniform data set for the variables for the variables heart rate, respiration, and RSA. Low frequency to High Frequency (LF/HF) ratio proved problematic with several outliers falling above the cut off z-score of 3.29. As a result, 20 of the LF/HF one minute epoch scores (2.3%) were omitted to make a uniform data set. These 20 scores were randomly distributed across the participants. The average LF/HF ratio for each participant was calculated in the absence of the outlying epoch.

Congruent with findings from Denver, Reed and Porges (2007), RSA and respiration rate, averaged over the five recording epochs for each phase, showed a negative relationship during
baseline ($r = -0.29, p < 0.05$), experiential sadness ($r = -0.33, p < 0.05$), and recovery ($r = -0.22$, ns). However, respiration rate only accounted for 8% to 10% of the variance in RSA amplitude. Because this level of shared variance was so low it could not be assumed that a significant portion of what is measured as tonic RSA is captured in a mathematical manner by respiration. Therefore, no adjustments were made to tonic RSA. The 15 one minute epochs for heart rate, respiration, RSA, and LF/HF were averaged across their respective phases to make one score for each phase (baseline, experiential recall, and recovery). Analyses were conducted using values averaged across phase.

The manner by which RSA amplitude fluctuates during demanding tasks is indicative of the vagal brake, which coincides with parallel shifts in respiration rate (Grossman & Taylor, 2007). Shifts in RSA amplitude occurring between baseline and experiential recall showed a strong negative association with changes in respiration rate across the same phases, $r = -0.58, p < 0.01$, sharing 34% common variance. Due to this relatively higher amount of shared variance, phasic RSA values were adjusted for respiration. First, RSA was divided by respiration rate for each of the 15 one minute epochs. Next, the one minute epochs were averaged for each phase (baseline, experiential recall, and recovery) and change scores in RSA values adjusted for respiration, $\Delta RSA$, were calculated using the following formula: $\Delta RSA = \text{respiration adjusted } RSA_{\text{Experiential recall}} - \text{respiration adjusted } RSA_{\text{Baseline}}$. Adjusting RSA for respiration in this manner uses the implicit assumption that a significant portion of vagal tone ($\Delta RSA$) is captured in a mathematical manner by respiration. This assumption was deemed appropriate given that there is a prominent theoretical link between vagal tone and changes in respiration rate during mental tasks (Porges, 2003; see Grossman & Taylor, 2007).
Analyses proceeded in the four following steps. First, a series of bivariate correlations were conducted to: (i) ensure that the physiological parameters were behaving as expected; (ii) examine the relationship between physiological parameters and potential demographic variables as confounds; and (iii) determine if and how the psychometric scales were associated with physiological parameters. The bivariate correlation analysis tested hypotheses regarding the associations among RSA, EI, and depressive symptoms. Next, a series of mixed model analyses of variance (ANOVA’s) were run to test differences in physiological parameters occurring across phase. Specifically, one mixed model ANOVA was run for each of RSA, HR, respiration, and LF/HF ratio. There were no a priori hypotheses regarding gender differences; however, having equal number of males and females allowed for this variable to be included in analyses of all ANOVA’s. Then, a one way analysis of co-variance (ANCOVA) was run to test the effect of RSA across phase with all potential confounds held constant. Finally, results for the content analysis of re-experienced sadness are presented.

Demographics

Table 1 displays the overall demographic characteristics of the participants. The majority of participants were younger ($M = 26.38$), and Caucasians (76.8%) having spent several years in formal education ($M = 14.57$).
Table 1

*Demographic Characteristics of the Sample*

<table>
<thead>
<tr>
<th>Descriptor</th>
<th>M</th>
<th>SD</th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (overall)</td>
<td>26.38</td>
<td>9.35</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>28</td>
<td></td>
<td></td>
<td>50.0%</td>
</tr>
<tr>
<td>Female</td>
<td>28</td>
<td></td>
<td></td>
<td>50.0%</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>43</td>
<td></td>
<td></td>
<td>76.8%</td>
</tr>
<tr>
<td>Aboriginal</td>
<td>7</td>
<td></td>
<td></td>
<td>12.5%</td>
</tr>
<tr>
<td>Asian</td>
<td>2</td>
<td></td>
<td></td>
<td>3.5%</td>
</tr>
<tr>
<td>Russian/Guyanese</td>
<td>1</td>
<td></td>
<td></td>
<td>1.8%</td>
</tr>
<tr>
<td>Latin American</td>
<td>1</td>
<td></td>
<td></td>
<td>1.8%</td>
</tr>
<tr>
<td>African American</td>
<td>1</td>
<td></td>
<td></td>
<td>1.8%</td>
</tr>
<tr>
<td>Portuguese</td>
<td>1</td>
<td></td>
<td></td>
<td>1.8%</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>25.39</td>
<td>6.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Years of Education</td>
<td>14.57</td>
<td>1.96</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note: N = 56, 28 males, 28 females*

*Descriptive statistics*

Descriptive statistics for the psychometric scales and the physiological variables are presented in Table 2.
## Table 2

*Descriptive Statistics of the Sample*

<table>
<thead>
<tr>
<th>Descriptor</th>
<th>$M$</th>
<th>$SE$</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAI</td>
<td>4312.71</td>
<td>391.38</td>
<td>91</td>
<td>12280</td>
</tr>
<tr>
<td>IDS-SR</td>
<td>13.66</td>
<td>1.16</td>
<td>0</td>
<td>39</td>
</tr>
<tr>
<td>AIM</td>
<td>141.34</td>
<td>2.16</td>
<td>101</td>
<td>185</td>
</tr>
<tr>
<td>EI</td>
<td>0.52</td>
<td>0.0083</td>
<td>0.27</td>
<td>0.61</td>
</tr>
<tr>
<td>Strategic EI</td>
<td>0.51</td>
<td>0.0091</td>
<td>0.31</td>
<td>0.66</td>
</tr>
<tr>
<td>Experiential EI</td>
<td>0.53</td>
<td>0.011</td>
<td>0.21</td>
<td>0.63</td>
</tr>
<tr>
<td>EI Managing emotions</td>
<td>0.42</td>
<td>0.009</td>
<td>0.24</td>
<td>0.53</td>
</tr>
<tr>
<td>EI Understanding emotions</td>
<td>0.60</td>
<td>0.015</td>
<td>0.33</td>
<td>0.81</td>
</tr>
<tr>
<td>EI Using emotions</td>
<td>0.46</td>
<td>0.01</td>
<td>0.16</td>
<td>0.56</td>
</tr>
<tr>
<td>EI Perceiving emotions</td>
<td>0.58</td>
<td>0.017</td>
<td>0.26</td>
<td>0.73</td>
</tr>
<tr>
<td>ERQ_Sadness</td>
<td>4.64</td>
<td>0.19</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>HR Baseline (BPM)</td>
<td>71.31</td>
<td>1.16</td>
<td>56.2</td>
<td>93.6</td>
</tr>
<tr>
<td>HR Experiential Recall (BPM)</td>
<td>74.9</td>
<td>1.27</td>
<td>59.6</td>
<td>98.2</td>
</tr>
<tr>
<td>HR Recovery (BPM)</td>
<td>70.6</td>
<td>1.08</td>
<td>56.4</td>
<td>92</td>
</tr>
<tr>
<td>Respiration Baseline (BPM)</td>
<td>13.59</td>
<td>0.53</td>
<td>5.8</td>
<td>22.4</td>
</tr>
<tr>
<td>Respiration Experiential Recall (BPM)</td>
<td>15.54</td>
<td>0.53</td>
<td>7</td>
<td>27.2</td>
</tr>
<tr>
<td>Respiration Recovery (BPM)</td>
<td>13.3</td>
<td>0.51</td>
<td>6.4</td>
<td>22.6</td>
</tr>
<tr>
<td>RSA Baseline (m/s²)</td>
<td>6.67</td>
<td>0.17</td>
<td>3.58</td>
<td>10.06</td>
</tr>
<tr>
<td>RSA Experiential Recall (m/s²)</td>
<td>6.13</td>
<td>0.16</td>
<td>3.28</td>
<td>9.18</td>
</tr>
<tr>
<td>RSA Recovery (m/s²)</td>
<td>6.56</td>
<td>0.17</td>
<td>3.09</td>
<td>9.31</td>
</tr>
<tr>
<td>$\Delta$RSA (m/s² adjusted for respiration)</td>
<td>-0.11</td>
<td>0.03</td>
<td>-0.76</td>
<td>0.36</td>
</tr>
<tr>
<td>LF/HF Baseline</td>
<td>1.46</td>
<td>0.17</td>
<td>0.06</td>
<td>6.13</td>
</tr>
<tr>
<td>LF/HF Experiential Recall</td>
<td>1.81</td>
<td>0.19</td>
<td>0.27</td>
<td>7.62</td>
</tr>
<tr>
<td>LF/HF Recovery</td>
<td>1.76</td>
<td>0.18</td>
<td>0.25</td>
<td>5.99</td>
</tr>
</tbody>
</table>

*Note: N = 56, 28 males, 28 females; PAI = Paffenbargar Physical Activity Index; IDS-SR = Inventory of Depressive Symptomatology – Self Report; AIM = Affect Intensity Measure; EI = Emotional Intelligence; ERQ = emotion reaction questionnaire, HR = Heart Rate; RSA = Respiratory Sinus Arrhythmia; LF/HF = Low Frequency to High Frequency Ratio.*
Analysis of the Association between Variables

Bivariate correlations were run in order to determine the relative association between variables before further analyses were performed. Three groups of bivariate correlations were performed: an analysis check examining the associations among the physiological variables; a search for cofounding variables between the physiological variables and the demographic variables; and a final correlation testing the association between RSA and the psychometric variables. Table 3 displays the bivariate correlations for the physiological variables tested. As would be expected, heart rate values showed strong positive associations across the three phases (see Table 2). However, heart rate was not associated with respiration and was negatively associated with RSA only during baseline and recovery. Also as expected, respiration rates showed moderate relationships across the three phases and with RSA. Finally, RSA values showed strong positive associations among themselves across the three phases. Of note, baseline levels of RSA were negatively associated with changes in RSA occurring during experiential sadness, $r = -.39, p < .01$, indicating that participants with a high level of tonic RSA amplitude exhibit greater vagal tone responsiveness.
Table 3

*Inter-Correlations between Physiological Variables*

<table>
<thead>
<tr>
<th></th>
<th>HR 2</th>
<th>HR 3</th>
<th>Respiration 1</th>
<th>Respiration 2</th>
<th>Respiration 3</th>
<th>RSA 1</th>
<th>RSA 2</th>
<th>RSA 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR 1</td>
<td>.75**</td>
<td>.93**</td>
<td>.2</td>
<td>.09</td>
<td>.21</td>
<td>-.56**</td>
<td>-.50*</td>
<td>-.49**</td>
</tr>
<tr>
<td>HR 2</td>
<td>.72**</td>
<td>.11</td>
<td>.05</td>
<td>.18</td>
<td>-.28*</td>
<td>-.53**</td>
<td>-.30*</td>
<td></td>
</tr>
<tr>
<td>HR 3</td>
<td>.24+</td>
<td>.11</td>
<td>.24+</td>
<td>-.57**</td>
<td>-.53**</td>
<td>-.58**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Respiration 1</td>
<td></td>
<td></td>
<td>-.40**</td>
<td>-.74**</td>
<td>-.29*</td>
<td>-.05</td>
<td>-.19</td>
<td></td>
</tr>
<tr>
<td>Respiration 2</td>
<td></td>
<td></td>
<td>.36**</td>
<td>-.03</td>
<td>-.33**</td>
<td>-.04</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Respiration 3</td>
<td></td>
<td></td>
<td>-.17**</td>
<td>-.01</td>
<td>-.22</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>RSA 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.65**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RSA 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.72**</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note: N = 56, 28 males, 28 females; + = p < .10, * = p < .05, ** = p < .01; HR = Heart Rate; RSA = Respiratory Sinus Arrhythmia; 1 = Baseline; 2 = Experiential Recall; 3 = Recovery.*

Table 4 displays the bivariate correlations between the physiological variables and the demographic characteristics. Heart rate was not associated with age or gender. However, a lower resting heart rate was associated with a higher metabolism and education indicated by higher scores on time spent in formal education and a greater PAI value. Females, younger participants, and those with a lower body mass index tended to have a higher respiration rate. Finally, RSA showed a strong negative association with age but failed to evidence a relationship with PAI.
Table 4

Association between Physiology and Demographic Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Gender</th>
<th>Age</th>
<th>BMI</th>
<th>Education</th>
<th>PAI</th>
</tr>
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<tbody>
<tr>
<td>HR 1</td>
<td>.15</td>
<td>-0.4</td>
<td>-0.3</td>
<td>-.24+</td>
<td>-.26+</td>
</tr>
<tr>
<td>HR 2</td>
<td>.22</td>
<td>.03</td>
<td>.09</td>
<td>-.16</td>
<td>-.20</td>
</tr>
<tr>
<td>HR 3</td>
<td>.21</td>
<td>.05</td>
<td>-.01</td>
<td>-.20</td>
<td>-.28</td>
</tr>
<tr>
<td>Respiration 1</td>
<td>.25+</td>
<td>-.23+</td>
<td>-.34*</td>
<td>-.09</td>
<td>-.10</td>
</tr>
<tr>
<td>Respiration 2</td>
<td>-.07</td>
<td>-.20</td>
<td>-.16</td>
<td>-.15</td>
<td>.02</td>
</tr>
<tr>
<td>Respiration 3</td>
<td>.27*</td>
<td>-.31*</td>
<td>-.52**</td>
<td>-.07</td>
<td>.05</td>
</tr>
<tr>
<td>RSA 1</td>
<td>.00</td>
<td>-.42**</td>
<td>.02</td>
<td>.01</td>
<td>.08</td>
</tr>
<tr>
<td>RSA 2</td>
<td>.00</td>
<td>-.40**</td>
<td>-.05</td>
<td>.00</td>
<td>.06</td>
</tr>
<tr>
<td>RSA 3</td>
<td>-.05</td>
<td>-.48**</td>
<td>.04</td>
<td>-.07</td>
<td>.04</td>
</tr>
<tr>
<td>Δ RSA</td>
<td>.23+</td>
<td>.05</td>
<td>-.13</td>
<td>.06</td>
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</table>

Note: N = 56, 28 males, 28 females; + = p < .10, *= p < .05, **= p < .01; HR = Heart Rate; RSA = Respiratory Sinus Arrhythmia; 1 = Baseline; 2 = Experiential Recall; 3 = Recovery; Δ RSA = respiration adjusted changes in RSA amplitude.

Table 5 displays the bivariate correlation between RSA and psychometric variables. RSA during experiential recall showed a strong negative relationship with affect intensity ($r = -.44, p < .01$), indicating that individuals with a propensity to experience affect intensely have lower resting RSA amplitude. Furthermore, changes in RSA between baseline and experiential recall showed a negative relationship with affect intensity ($r = -.28, p < .05$). In other words, those who experience affect more intensely have greater reductions in vagal tone indicating parasympathetic withdrawal suggestive of a lesser ability to apply the vagal brake. More importantly, tonic RSA did not evidence the theorized relationship with EI or depressive symptomatology. However, changes in RSA between baseline and experiential recall showed the predicted negative relationship with the perceiving emotions dimension of emotional intelligence ($r = -.31, p < .05$). Furthermore, the pattern of change in RSA between baseline and experiential...
recall tended to evidence a negative relationship with experiential emotional intelligence and overall emotional intelligence.

Table 5

*Bivariate Correlations between RSA and Psychometric Variables*

<table>
<thead>
<tr>
<th></th>
<th>RSA 1</th>
<th>RSA 2</th>
<th>RSA 3</th>
<th>Δ RSA</th>
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<td>.10</td>
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<td>-.07</td>
<td>.02</td>
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<tr>
<td>EI Understanding emotions</td>
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<td>-.16</td>
<td>.00</td>
<td>-.14</td>
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<tr>
<td>EI Using emotions</td>
<td>-.04</td>
<td>-.15</td>
<td>-.02</td>
<td>-.06</td>
</tr>
<tr>
<td>EI Perceiving emotions</td>
<td>.14</td>
<td>-.05</td>
<td>.15</td>
<td>-.31*</td>
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<td>ERQ Sadness</td>
<td>-.12</td>
<td>-.22+</td>
<td>-.18</td>
<td>.04</td>
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</tbody>
</table>

Note: N = 56, 28 males, 28 females; + = p < .10, * = p < .05, ** = p < .01; AIM = Affect Intensity Measure; IDS-SR = Inventory of Depressive Symptomatology – Self Report; EI = Emotional Intelligence; ERQ = Emotion Reaction Questionnaire; RSA = Respiratory Sinus Arrhythmia; 1 = Baseline; 2 = Experiential Recall; 3 = Recovery; Δ RSA = change in RSA adjusted for respiration.

*Analysis of Physiological Changes Attributable to Experienced Sadness Induction*

Four mixed model ANOVA’s were run to test the following four hypotheses: (1) RSA amplitude will be reduced, indicative of applying the vagal brake, during experiential recall of sadness relative to baseline and recovery; (2) heart rate will increase during experiential recall of sadness relative to baseline and recovery; (3) respiration rate will increase during experiential recall of sadness relative to baseline and recovery; and (4) HF/LF ratio will be lower, suggesting greater sympathetic activation, during experiential recall of sadness relative to baseline and recovery.
Analysis of RSA

A 2 (Gender) by 3 (Phase; baseline, experiential recall, recovery) mixed model ANOVA was conducted on RSA to test the hypothesis that vagal tone is implemented during experiential sadness. Initial examination of the analysis indicated the assumption of sphericity was violated ($\chi^2 = 17.26 p < .01$), therefore the Greenhouse-Geisser correction factor was used in the results reported.

Within-Group Effects: The main effect of phase was significant, $F(1.56, 108) = 11.53$, $MSE = 0.40, p < .01, \eta^2 = .18$. Figure 1 shows the overall pattern of RSA. Follow up pairwise comparisons, adjusted with the Holms-Bonferroni correction to control for family wise error, revealed that RSA was significantly lower during experiential sadness recall relative to baseline, $M_{\text{diff}} = 0.54, SEM = 0.14, p < .01$, and recovery, $M_{\text{diff}} = 0.43, SEM = 0.13, p < .01$. RSA did not differ between baseline or recovery, $M_{\text{diff}} = 0.11, SEM = 0.08, p > .05$.

Between-Groups Effects: The main effect of Gender was not significant, $F(1, 54) = 0.02$, $SEM = 0.20, p > .05, \eta^2 = .00$. The observed power to detect the effect was low at .05

Figure 1

Main Effect of Respiratory Sinus Arrhythmia ($m/s^2$) and Associated Standard Error
Analysis of Respiration

A 2 (Gender) by 3 (Phase; baseline, experiential recall, recovery) mixed model ANOVA was conducted on respiration to test the hypothesis that respiration increases during experiential sadness recall. Initial examination of the analysis indicated the assumption of sphericity was violated ($\chi^2 = 12.02 \ p < .01$), therefore the Greenhouse-Geisser correction factor was used in the results reported.

*Within-Group Effects:* The main effect of phase was significant, $F(1.66, 108) = 12.39$, $MSE = 8.83, \ p < .01, \eta^2 = .17$. Figure 2 shows the overall pattern of respiration rate. Follow up pairwise comparisons, adjusted with the Holms-Bonferroni correction to control for family wise error, revealed that respiration rate was significantly elevated during experiential sadness recall relative to baseline, $M_{\text{diff}} = 1.95, \ SEM = 0.57, \ p < .01$, and recovery, $M_{\text{diff}} = 2.25, \ SEM = 0.57, \ p < .01$. Respiration rate did not differ between baseline or recovery, $M_{\text{diff}} = 0.31, \ SEM = 0.38, \ p > .05$.

*Between-Groups Effects:* The main effect of Gender was not significant, $F(1, 54) = 1.82$, $SEM = 30.41, \ p > .05, \eta^2 = .03$. The observed power to detect the effect was low at .26.

Figure 2:

*Main effect of Respiration (BPM) and Associated Standard Error*
Analysis of Heart Rate

A 2 (Gender) by 3 (Phase; baseline, experiential recall, recovery) mixed model ANOVA was conducted on heart rate to test the hypothesis that heart rate increases during experiential recall of sadness. Initial examination of the analysis indicated the assumption of sphericity was violated ($\chi^2 = 34.06, p < .01$), therefore the Greenhouse-Geisser correction factor was used in the results reported.

Within-Group Effects: The main effect of phase was significant, $F(3.6, 108) = 18.52$, $MSE = 16.15, p < .01, \eta^2 = .25$. Figure 3 shows the overall pattern of heart rate. Follow up pairwise comparisons, adjusted with the Holms-Bonferroni correction to control for family wise error, revealed that heart rate was significantly elevated during experiential sadness recall relative to baseline, $M_{\text{diff}} = 3.59, SEM = 0.86, p < .01$, and recovery, $M_{\text{diff}} = 4.31, SEM = 0.90, p < .01$. Heart rate did not differ between baseline or recovery, $M_{\text{diff}} = 0.72, SEM = 0.43, p > .05$.

Between-Groups Effects: The main effect of Gender was not significant, $F(1, 54) = 2.43$, $SEM = 194.04, p > .05, \eta^2 = .04$. The observed power to detect the effect was low at .33.

Figure 3

Main Effect of Heart Rate (BPM) and Associated Standard Error
**Analysis of LF/HF Ratio**

A 2 (Gender) by 3 (Phase; baseline, experiential recall, recovery) mixed model ANOVA was conducted on respiration to test the hypothesis that LF/HF ratio increases during experiential sadness recall. Initial examination of the analysis indicated the assumption of sphericity was upheld ($\chi^2 = 3.91, p > .05$).

*Within-Group Effects:* The main effect of phase was not significant, $F(2, 108) = 2.16$, $MSE = 0.92$, $p > .05$, $\eta^2 = .04$. Figure 4 shows the overall pattern of LF/HF ratio.

*Between-Groups Effects:* The main effect of Gender was not significant, $F(1, 54) = 1.92$, $SEM = 3.51$, $p > .05$, $\eta^2 = .04$. The observed power to detect the effect was low at .28.

**Figure 4**

*Pattern of LF/HF ratio and Associated Standard Error*

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**Analysis of RSA Controlling for Confounds**

A 3 (Phase; baseline, experiential recall, recovery) one way repeated measures ANCOVA was conducted to test the effect of phase on RSA as well as its interaction with the covariates age
and AIM. Initial examination of the analysis indicated the assumption of sphericity was violated ($\chi^2 = 12.43, p < .01$), therefore the Greenhouse-Geisser correction factor was used in the results reported.

**Within-Group Effects:** RSA was found to be related to the covariate age, $F(1, 53) = 14.48$, $MSE = 2.95$, $p < .01$, $\eta^2 = .22$ and tended to be related to the covariate AIM, $F(1, 53) = 3.08$, $MSE = 2.95$, $p < .10$, $\eta^2 = .06$. After controlling for age and AIM, the main effect of phase was significance, $F(1.65, 106) = 4.02$, $MSE = 1.42$, $p < .05$, $\eta^2 = .07$, indicating that the main effect of phase on RSA cannot be accounted for by differences in age or affect intensity.

**Analysis of RSA as a Function of Affect Intensity**

A 3 (Phase; baseline, experiential recall, recovery) by 2 (affect intensity) mixed model ANOVA was conducted to better understand the influence of affect intensity on the effect of RSA. The AIM was dichotomized using a median split and entered into the ANOVA as a between subject variable. Initial examination of the analysis indicated the assumption of sphericity was violated ($\chi^2 = 13.21, p < .01$), therefore the Greenhouse-Geisser correction factor was used in the results reported.

**Within-Group Effects:** The main effect of phase was significant, $F(1.64, 108) = 13.09$, $MSE = 0.35$, $p < .01$, $\eta^2 = .20$. Follow up pairwise comparisons, adjusted with the Holms-Bonferroni correction to control for family wise error, revealed that RSA was significantly lower during experiential sadness recall relative to baseline, $M_{diff} = 0.54$, $SEM = 0.14$, $p < .01$, and recovery, $M_{diff} = 0.43$, $SEM = 0.13$, $p < .01$. RSA did not differ between baseline or recovery, $M_{diff} = 0.11$, $SEM = 0.08$, $p > .05$.

**Between-Groups Effects:** The main effect of affect intensity exhibited a trend towards significance, $F(1, 54) = 3.12$, $SEM = 3.73$, $p = .08$, $\eta^2 = .06$. The observed power to detect the
effect was low at .41. The influence of affect intensity on the phase by RSA effect is shown in Figure 5. An analysis of the means and standard error scores indicates that the high affect intensity group scored significantly lower during experiential recall. This was confirmed through a t-test, $t = 3.38$, $SEM = .29$, $p < .01$.

Figure 5

*Effect of Affect Intensity on RSA with Associated Standard Error*

![Graph showing the effect of affect intensity on RSA with associated standard error.](image)

*Content Analysis of Relived Episodes of Sadness*

Bivariate correlations were conducted to fully appreciate the re-lived episodes of sadness and their relationship to subjective sadness, objective sadness, affect intensity and emotional intelligence. Subjective and objective ratings of sadness were not associated with tonic or phasic values of RSA, all $r's < .11$, all $p's > .10$. However, use of descriptive sadness in the experienced recall form assessed through the LIWC tended to show a negative association with $\Delta$ RSA, $r = -.24$, $p > .10$, and a positive association with resting RSA, $r = .26$, $p < .10$. This pattern of results suggests that participants who more vividly described the sadness associated with their re-lived
event had a relatively high level of tonic RSA amplitude that shifted dramatically towards parasympathetic withdrawal during experiential recall.

Table 6 displays the bivariate correlations between ratings of sadness and psychometric variables. ERQ sadness showed a positive association with ERQ impurity, $r = .27, p < .05$, indicating that subjective ratings of sadness were higher when emotions other than sadness occurred during experiential recall. As would be expected, the AIM showed a positive association with ERQ sadness, $r = .29, p < .05$, indicating that those who experience affect more intensely rated their subjective level of experienced sadness higher.

Objective ratings of sadness were not related to subjective experiences of sadness, see Table 6. However, objective sadness was positively associated with LIWC sadness, $r = .31, p < .05$, and emotional intelligence, $r = .29, p < .05$, suggesting that more emotionally intelligent participants selected more sad events and described such events more vividly. Adding to this, emotional intelligence was negatively associated with ERQ impurity, $r = -.38, p < .01$, demonstrating that more emotionally intelligent individuals were better able to re-experience the pure sensations of sadness associated with an event. Of interest, every dimension of emotional intelligence showed this similar pattern of associations. For ease of convenience only the pattern of associations between overall emotional intelligence are presented.
Table 6

*Inter-Correlations between Ratings of Sadness and Psychometric Variables*

<table>
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<th>ERQ Impurity</th>
<th>Objective Sadness</th>
<th>LIWC Sadness</th>
<th>AIM</th>
<th>EI</th>
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<td>ERQ Impurity</td>
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<td></td>
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<td>LIWC Sadness</td>
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<td>AIM</td>
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<td></td>
<td>-.02</td>
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<td></td>
</tr>
</tbody>
</table>

Note: N = 56, 28 males, 28 females; + = p < .10, * = p < .05, ** = p < .01; ERQ = emotion reactions questionnaire; ERQ Impurity = sum of all ERQ affect ratings besides sadness; LIWC = Linguistic Inquiry Word Count; AIM = Affect Intensity Measure; EI = Emotional Intelligence.

DISCUSSION

The purpose of this investigation was to further our understanding of cardiovascular physiology and emotion responding. Specifically, the present study examined the relationship between vagal tone (RSA), experiences of sadness, and psychological constructs of affect intensity, EI, and depressive symptomatology. Important findings to emerge were: (a) RSA amplitude was reduced during experiential sadness recall relative to baseline and recovery phases; (b) participants displaying high EI and who were prone to experience affect intensely exhibited greater reductions in RSA amplitude during experiential sadness; (c) tonic level of RSA was not related to EI or depressive symptomatology; (d) physiological parameters of HR and respiration but not LF/HF ratio behaved as would be expected; (e) changes in respiration rate between baseline and experienced sadness confounded similar changes in RSA; (f) tonic RSA amplitude was inversely related to age; and (g) participants writing events that scored high on
subjective content of sadness tended have higher resting RSA amplitude, experienced greater vagal tone responsiveness, and wrote about events scored higher on objective ratings of sadness. Each of these points is commented upon in turn.

Supporting the first hypothesis, reductions in RSA amplitude were evident during experiential sadness recall indicative of a shift in cardiac activation towards parasympathetic withdrawal. One of the most consistent and robust findings in the HRV and RSA literature is that RSA amplitude is reduced during demanding tasks indicating a withdrawal in vagal-tone that is believed to represent flexible responding and self regulation (Appelhans & Leuken, 2006; Butler et al., 2006; Gyurak & Ayduk, 2008; Hoffmann et al., 2006). The finding of this experiment is directly in line with this body of research showing that episodes of relived sadness are a significant stressor, reducing cardiac vagal tone, and necessitating a regulatory response before tonic cardiac activation can be restored.

Of interest, high levels of tonic RSA amplitude coincided with greater vagal tone responses during experiential recall. Three possible interpretations could explain this finding. First, those with high resting RSA amplitude might have prepared sadder events causing an increased effectiveness of the experienced sadness recall. This interpretation does not seem likely given that subjective experiences and objective ratings of sadness were in no way related to tonic or phasic RSA amplitude. Second, it is possible that people with high resting RSA amplitude experience emotions more intensely. This interpretation was only partially supported. Participants higher in affect intensity did experience greater changes in RSA amplitude during experiential recall; however, those same participants who experienced greater affect intensity were found to have lower, rather than higher, levels of tonic RSA amplitude. Finally, the third interpretation is that people with high resting RSA amplitude experience greater vagal
withdrawal indicating that they are responding in a more adaptive manner but this effect is
masked by a suppressor variable, such as affect intensity. This interpretation seems most likely
given that participants with high resting RSA amplitude were less prone to experience affect
intensely, prepared events that scored equally high in objective sadness as did those with low
tonic RSA amplitude, experienced greater vagal withdrawal, and still recovered in similar
lengths of time.

The second hypothesis was not supported. Tonic levels of RSA were not related to EI
however, phasic changes in RSA amplitude were negatively related to EI supporting the third
hypothesis. Individuals scoring higher in emotional intelligence, particularly on the perceiving
emotions dimension, displayed greater vagal withdrawal suggesting that their ANS is more
reactive in part because they are hyper-sensitive to the emotional cues of others. This
relationship is even more profound considering that affect intensity was inversely associated with
phasic changes in RSA amplitude. Participants who were prone to experience affect intensely
also exhibit greater changes in RSA amplitude during experienced sadness and this effect was
orthogonal to that of emotional intelligence. These findings suggest that implementation of vagal
tone indicates emotional responding with larger amplitude changes occurring in participants who
perceive and experience affect more intensely.

The fourth hypothesis was not supported. Vagal tone responsiveness during experienced
sadness was in no way related to depressive symptomatology. This null relationship could be the
result of an improper measurement tool used to assess depression rather than a true null finding
between depression and vagal tone. Depression in this study was measured through depressive
symptomatology which may not relate to vagal tone in the same manner that depressed affect is
believed to. Additionally, most of the participants in this study had very few depressive
symptoms, thus scoring low in severity for depression. It is assumed that autonomic
dysregulation leads to depression, but perhaps the reverse is true where depression results in
autonomic dysregulation, a theory that could only by tested by examining vagal tone
responsiveness in a sample of depressed patients. Regardless, hypothesis five involved a
mediation model between EI, vagal tone, and depressive symptomatology. This hypothesis was
not tested because no relationship was found between depressive symptomatology and EI or
vagal tone responsiveness.

With the exception of the LF/HF ratio, the physiological parameters all behaved as
expected supporting the content validity of RSA as a measure of vagal tone in this study.
Sadness is a negative emotion that stresses the body, prompting a sympathetic response.
Congruent with this expectation, experienced sadness resulted in an increase in participants’
heart rate and respiration rate evidencing sympathetic arousal. Although not statistically
significant, changes in LF/HF ratio that occurred during experiential recall evidenced the
appropriate shift towards greater LF activation suggesting greater sympathetic arousal. The null
finding for the LF/HF ratio by phase effect is likely the result of inadequate power to detect a
moderate effect of \( \eta^2 = .04 \). This study only had a power of 0.41 to detect this effect which is far
below the recommended 0.80 level indicating that this finding is truly inconclusive.

Findings from this study speak to the present debate surrounding the role of respiration in
measurement of vagal tone. Denver, Porges and Denver (2007) theorize that vagal tone and
respiration do not share enough common variance to confound measures of one another while
Grossman and Taylor (2007) argue that vagal tone, in particular changes in vagal tone during
demanding tasks, is confounded by modest changes in respiration rate and depth. The findings
from this experiment suggest that both authors are correct on different aspects of this debate. Our
results suggest that tonic levels of RSA do not co-vary with respiration, sharing only 10% common variance, while phasic changes in RSA do co-vary with associated changes in respiration rate, sharing 34% of common variance. Although vagal tone and respiration rate are only weakly associated at rest, implementation of the vagal brake is significantly confounded by associated changes in respiration rate and should be adjusted accordingly.

Tonic levels of RSA amplitude were negatively associated with age indicating that RSA amplitude is naturally reduced during the process of aging. This finding is consistent with previous research (Craft & Schwartz, 1995) that suggests that age should be controlled as a potential covariate when examining tonic levels of RSA. However, phasic changes in RSA were not associated with age suggesting that cardiac vagal tone responses to sadness do not change as a function of age.

Finally, participants who prepared events that scored high for subjective content of sadness had higher resting RSA amplitude, experienced greater vagal tone responsiveness, and wrote about events that scored higher on objective sadness. Incorporating all of the findings from this experiment, participants who experience affect intensely, and who intelligently perceive emotions in others likely wrote about events that were high in objective and content sadness. Reliving such events would cause them to experience greater vagal withdrawal during experiential recall. The only evidence in contrary to this theory was the finding that participants who experienced affect intensely showed lower resting RSA amplitude and yet subjective content of sadness was greater in the experiential recall forms of those exhibiting higher resting RSA amplitude. It must be acknowledged that the relationship between vagal tone responsiveness, emotional intelligence, affect intensity, and experienced sadness may exist in a greater degree of complexity than the present experiment was able to capture.
LIMITATIONS

Despite these novel findings, it is important to recognize that the associations found here came from observations within a laboratory setting using a moderately homogenous group. For further validation this study should be repeated in various settings with a wider range of individuals in order to permit generalizability of these results. In other words, it still needs to be determined whether emotional intelligence and affect intensity have such profound effects on vagal tone during episodes of sadness that occur in everyday life. Further, it would be valuable to determine whether an induction of a different negative emotional state such as anger, or a positive emotional state such as gratitude, optimism, or joy would alter the pattern of vagal tone responsiveness and its combined effects with emotional intelligence and affect intensity. This study examined the relationship between heart rate variability (RSA) and experiential sadness recall. What it did not examine was explicit coping mechanisms used to regulate affect. For instance, it is unclear what the participants were actually thinking about in the five minute recovery phase or if they applied some form of coping mechanism during experienced sadness recall. It would be valuable information to know whether the high emotionally intelligent and affective participants employed a differential coping strategy or none at all in relation to the low emotionally intelligent and affective.

Depressive symptomatology was not found to relate to vagal tone responsiveness or emotional intelligence. This null finding could be due, in part, to the time of the school year during which testing occurred. Depressive symptomatology taps into physiological complaints that would be reported in higher volume during stressful parts of the semester (e.g., during mid-terms). Using a university population, it would be interesting to know if depressive
symptomatology fluctuates in relationship to time of semester and if these fluctuations could in part explain these null results.

IMPLICATIONS

The findings of this study help to contrast two perspectives on HRV. Given that there is central nervous system control over parasympathetic influences in heart rate, some have suggested that HRV is a marker for high levels of self-regulation abilities and capacities (Thayer & Lane, 2000). Then again, the HRV measurement procedures most directly bear witness to individual differences in cardiovascular flexibility, which support higher levels of flexible responding to environmental provocations (Appelhans & Luecken, 2006; Ode, Hilmert, Zielke, & Robinson, 2010). Flexible responding could support adaptive self-regulation but need not do so. Our results speak to HRV as flexible responding.

Vagal responsiveness was a function of affect intensity and the intelligent perception of emotions. Participants more prone to detecting the emotional cues of others and who experienced affect in a more intense manner exhibited greater vagal withdrawal during experienced sadness. These participants were better able to conjure the felt experience associated with the original event. Interestingly and contrary to expectations, vagal responsiveness was not a function of the intelligent regulation of emotion. Together, these findings suggest that HRV measures responsiveness to environmental demands rather than regulatory capacity per se. This does not imply that HRV should not be used as a proxy measure of emotional regulation. On the contrary, HRV should be used as a proxy measure of emotion regulatory capabilities only when such measures are taken: (a) during conditions where participants are asked to consciously self-regulate their mood; (b) when affect intensity is measured and controlled for; and (c) when measures of vagal tone occurring during task demands are adjusted for associated changes in
respiration. Researchers can no longer assume that vagal tone reflects self-regulation but instead must test the construct using a greater degree of specificity.

Vagal responsiveness was found to reflect emotional responding and may be more applicable to the study of disorders characterized by dramatic shifts in affect (e.g., bipolar disorder, borderline personality disorder) than it is to depression. Vagal responsiveness to the emotion sadness did not index depressive symptoms among non-depressed participants. Future research should examine the link between vagal tone and depressive affect rather than depressive symptoms and should perform such a task by sampling a depressive population to test if these constructs are definitively related.

Vagal tone during experiential recall was related to the intelligent perception of emotions in others. This finding has interesting implications suggesting that HRV may index empathy to emotions conveyed through facial expressions. The perceiving emotions dimension of the MSCEIT assesses the accuracy of people’s ability to detect emotions conveyed through facial expressions. Finding that vagal tone responsiveness is greater among participants who more accurately perceive emotions in others offers support to the theory that RSA forms a biological basis for empathy.

Theoretical conceptualizations of empathy suggest that the affective states of others are best understood by activation of neural networks usually involved in the processing of one's own affective states (see Decety & Lamm, 2006). It is interesting to speculate that the cardiovascular system operates in a similar manner. Vagal tone of empathic individuals then would operate by emulating the emotional tone of others. Further support for this theory arises from the relationship between affect intensity and vagal tone. Individuals who experience affect more intensely had lower resting vagal tone suggesting a lower capacity to respond adaptively to
environmental demands (Butler, Wilhelm & Gross, 2006). In addition to this, those who experienced affect intensely exhibited more pronounced vagal tone responses. An argument could be made that people who experience affect intensely are prone to respond dramatically to emotional material, in part, due to empathic neural connections. This argument coincides with findings that affect intensity, measured through the AIM, indexes the use of personalizing and empathic cognitions (Larsen, Diener & Cropanzano 1987).

The idea that RSA forms a biological basis for empathy is an interesting theory that would suggest that empathy is a skill that can be trained in much the same way as one trains their cardiovascular system to respond during exercise. A great deal more research should be performed in order to test the merit of this theory.
References


Appendix A

Paffenbarger Physical Activity Index (PAI)

Name _______________________ Date _______________

PLEASE ANSWER THE FOLLOWING QUESTIONS BASED ON YOUR AVERAGE DAILY PHYSICAL ACTIVITY HABITS FOR THE PAST YEAR

1. How many stairs did you climb up on an average day during the past year?

__________ stairs per day (1 flight or floor=10 stairs)

2. How many city blocks or their equivalent did you walk on an average day during the past year?

__________ blocks per day (12 blocks = 1 mile)

3. List any sports, leisure, or recreational activities you have participated in on a regular basis during the past year. Enter the average number of times per week you took part in these activities and the average duration of these sessions. Include only time you were physically active (that is, actual playing or activity time).

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<td></td>
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<td></td>
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Appendix B

INVENTORY OF DEPRESSIVE SYMPTOMATOLOGY (SELF-REPORT)
(IDS-SR)

FULL NAME _____________________________________________ TODAY'S DATE _______________

Please circle the one response to each item that is most appropriate to how you have been feeling over the past 7 days.

1. Falling asleep
   0. I never took longer than 30 minutes to fall asleep
   1. I took at least 30 minutes to fall asleep, less than half the time (3 days or less out of the past 7 days)
   2. I took at least 30 minutes to fall asleep, more than half the time (4 days or more out of the past 7 days)
   3. I took more than 60 minutes to fall asleep, more than half the time (4 days or more out of the past 7 days)

2. Sleep during the night
   0. I didn't wake up at night
   1. I had a restless, light sleep, briefly waking up a few times each night
   2. I woke up at least once a night, but I got back to sleep easily
   3. I woke up more than once a night and stayed awake for 20 minutes or more, more than half the time (4 days or more out of the past 7 days)

3. Waking up too early
   0. Most of the time, I woke up no more than 30 minutes before my scheduled time
   1. More than half the time (4 days or more out of the past 7 days), I woke up more than 30 minutes before my scheduled time, but I got back to sleep eventually
   2. I almost always woke up at least one hour or so before my scheduled time, but I got back to sleep eventually
   3. I woke up at least one hour before my scheduled time, and couldn't get back to sleep

4. Sleeping too much
   0. I slept no longer than 7–8 hours/night, without napping during the day
   1. I slept no longer than 10 hours in a 24-hour period including naps
   2. I slept no longer than 12 hours in a 24-hour period including naps
   3. I slept longer than 12 hours in a 24-hour period including naps

5. Feeling sad
   0. I didn't feel sad
   1. I felt sad less than half the time (3 days or less out of the past 7 days)
   2. I felt sad more than half the time (4 days or more out of the past 7 days)
   3. I felt sad nearly all the time

6. Feeling irritable
   0. I didn't feel irritable
   1. I felt irritable less than half the time (3 days or less out of the past 7 days)
   2. I felt irritable more than half the time (4 days or more out of the past 7 days)
   3. I felt extremely irritable nearly all the time

7. Feeling anxious or tense
   0. I didn't feel anxious or tense
   1. I felt anxious (tense) less than half the time (3 days or less out of the past 7 days)
   2. I felt anxious (tense) more than half the time (4 days or more out of the past 7 days)
   3. I felt extremely anxious (tense) nearly all the time

8. Your state of mind in response to good or desired events
   0. I was in a better state of mind which lasted for several hours when good events occurred
   1. I was in a better state of mind but I didn't feel like my normal self when good events occurred
   2. I was in a better state of mind only somewhat to a rather limited range of desired events
   3. My state of mind wasn't better, even when very good or desired events occurred in my life

9. State of mind in relation to the time of day
   0. There was no usual relationship between my state of mind and the time of day
   1. My state of mind often related to the time of day because of my circumstances (e.g., being alone, working)
   2. In general, my state of mind was more related to the time of day than to my circumstances
   3. My state of mind was clearly and predictably better or worse at a particular time each day

9A. Was your state of mind typically worse in the morning, afternoon or evening? (Circle the one that applies, if any)
9B. Were variations to your state of mind attributed to your circumstances? (yes or no) (circle one)

10. Your state of mind
    0. My state of mind was normal
    1. I was sad, but this sadness was pretty much like the sadness I would feel if someone close to me died or left
    2. I was sad, but this sadness was a little bit different from the sadness I would feel if someone close to me died or left
    3. I was sad, but this sadness was very different from the type of sadness associated with grief or loss
Please complete either 11 or 12 (not both)

11 Decreased appetite
   0 There was no change in my usual appetite
   1 I ate somewhat less often or smaller amounts of food than usual
   2 I ate much less than usual and only by forcing myself to eat
   3 I rarely ate within a 24-hour period, and only by really forcing myself to eat or when others persuaded me to eat

12 Increased appetite
   0 There was no change in my usual appetite
   1 I felt a need to eat more frequently than usual
   2 I regularly ate more food than usual and/or greater amounts of food than usual
   3 I felt driven to overeat both at mealtime and between meals

Please complete either 13 or 14 (not both)

13 Decreased weight (within the last 14 days)
   0 There was no change in my weight
   1 I feel as if I've had a slight weight loss
   2 I've lost 2 pounds (about 1 kilo) or more
   3 I've lost 5 pounds (about 2 kilos) or more

14 Increased weight (within the last 14 days)
   0 There was no change in my weight
   1 I feel as if I've had a slight weight gain
   2 I've gained 2 pounds (about 1 kilo) or more
   3 I've gained 5 pounds (about 2 kilos) or more

15 Concentration/decision making
   0 There was no change in my usual capacity to concentrate or make decisions
   1 I occasionally felt indecisive or found that my attention wandered
   2 Most of the time, I found it hard to focus or to make decisions
   3 I couldn't concentrate well enough to read or I couldn't make even minor decisions

16 Perception of myself
   0 I saw myself as equally worthwhile and deserving as other people
   1 I put the blame on myself more than usual
   2 For the most part, I believed that I caused problems for others
   3 I thought almost constantly about major and minor defects in myself

17 View of my future
   0 I had an optimistic view of my future
   1 I was occasionally pessimistic about my future, but for the most part I believed things would get better
   2 I was pretty certain that my immediate future (1-2 months) doesn't hold much promise of good things for me
   3 I saw no hope of anything good happening to me any time in the future

18 Thoughts of my own death or suicide
   0 I didn't think of suicide or death
   1 I felt that life was empty or wondered if it was worth living
   2 I thought of suicide or death several times a week for several minutes
   3 I thought of suicide or death several times a day in some detail, or I made specific plans for suicide or actually tried to take my life

19 General interest
   0 There was no change from usual in how interested I was in other people or activities
   1 I noticed that I was less interested in people or activities
   2 I found I had interest in only one or two of the activities I used to do
   3 I had virtually no interest in the activities I used to do

20 Energy level
   0 There was no change in my usual level of energy
   1 I got tired more easily than usual
   2 I had to make a big effort to start or finish my usual daily activities (for example shopping, homework, cooking or going to work)
   3 I really couldn't carry out most of my usual daily activities because I just didn't have the energy

21 Capacity for pleasure or enjoyment (excluding sex)
   0 I enjoyed pleasurable activities just as much as usual
   1 I did not feel my usual sense of enjoyment from pleasurable activities
   2 I rarely got a feeling of pleasure from any activity
   3 I was unable to get any pleasure or enjoyment from anything
22 Interest in sex (please rate interest, not activity)
   0 I was just as interested in sex as usual
   1 My interest in sex was somewhat less than usual
   or I didn’t get the same pleasure from sex as I
   used to
   2 I had little desire for or rarely derived pleasure
   from sex
   3 I had absolutely no interest in or derived no
   pleasure from sex

23 Feeling more sluggish than usual
   0 I thought, spoke, and moved at my usual pace
   1 I found that my thinking was more sluggish than
   usual or my voice sounded dull or flat
   2 It took me several seconds to respond to most
   questions and I was sure my thinking was more
   sluggish than usual
   3 I was often unable to respond to questions
   without forcing myself

24 Feeling restless (agitated, not relaxed, fidgety)
   0 I didn’t feel restless
   1 I was often fidgety, wringing my hands, or needed
   to change my sitting position
   2 I had sudden urges to move about and was quite
   restless
   3 At times, I was unable to stay seated and needed
   to pace around

25 Aches and pains
   0 I didn’t have any feeling of heaviness in my arms
   or legs and didn’t have any aches or pains
   1 Sometimes I got headaches or pains in my
   stomach, back or joints but these pains were only
   temporary and they didn’t stop me from doing
   what I needed to do
   2 I had these sorts of pains most of the time
   3 These pains were so bad they forced me to stop
   what I was doing

26 Other bodily symptoms
   0 I didn’t have any of these symptoms: heart
   pounding fast, blurred vision, sweating, hot and
   cold flashes, chest pain, palpitations, ringing in my
   ears, or shaking
   1 I had some of these symptoms but they were mild
   and were only temporary
   2 I had several of these symptoms and they
   bothered me quite a bit
   3 I had several of these symptoms and when they
   occurred I had to stop doing whatever I was doing

27 Panic/phobia symptoms
   0 I had no panic attacks or specific fears (phobias)
   (such as animals or heights)
   1 I had mild panic attacks or fears that didn’t
   usually change my behaviour or stop me from
   functioning
   2 I had significant panic attacks or fears that forced
   me to change my behaviour but didn’t stop me
   from functioning
   3 At least once a week, I had panic attacks or
   severe fears that stopped me from carrying on my
   daily activities

28 Constipation/diarrhea
   0 There was no change in my usual bowel habits
   1 I had intermittent constipation or diarrhea which
   was mild
   2 I had diarrhea or constipation most of the time but
   it didn’t interfere with my day-to-day functioning
   3 I had constipation or diarrhea for which I took
   medicine or which interfered with my day-to-day
   activities

29 Sensitivity to others
   0 I didn’t feel easily rejected, slighted, criticized
   or hurt by others at all
   1 I occasionally felt rejected, slighted, criticized
   or hurt by others
   2 I often felt rejected, slighted, criticized or hurt
   by others; but these feelings had only slight effects
   on my relationships or work
   3 I often felt rejected, slighted, criticized or hurt
   by others and these feelings impaired my
   relationships and work

30 Feeling weighted down/physical energy
   0 I didn’t experience the physical sensation of
   feeling weighted down and without physical
   energy
   1 I occasionally experienced periods of feeling
   physically weighted down and without physical
   energy, but without a negative effect on work,
   school, or activity level
   2 I felt physically weighted down (without physical
   energy) more than half the time (4 days or more
   out of the past 7 days)
   3 I felt physically weighted down (without physical
   energy) most of the time, several hours a day,
   several days a week

Thank you
Range 0-84 Score
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Appendix C

Affect Intensity Measure (AIM)

DIRECTIONS:
The following questions refer to emotional reactions to typical life-events. Please indicate how YOU react to these events by placing a number from the following scale in the blank space preceding each item. Please base your answers on how YOU react, not on how you think others react or how you think a person should react.

<table>
<thead>
<tr>
<th>Never</th>
<th>Almost Never</th>
<th>Occasionally</th>
<th>Usually</th>
<th>Almost Always</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>

1. ______ When I accomplish something difficult I feel delighted or elated.
2. ______ When I feel happy it is a strong type of exuberance.
3. ______ I enjoy being with other people very much.
4. ______ I feel pretty bad when I tell a lie.
5. ______ When I solve a small personal problem, I feel euphoric
6. ______ My emotions tend to be more intense than those of most people
7. ______ My happy moods are so strong that I feel like I’m in heaven.
8. ______ I get overly enthusiastic.
9. ______ If I complete a task I thought was impossible, I am ecstatic.
10. ______ My heart races at the anticipation of some exciting event.
11. ______ Sad movies deeply touch me.
12. ______ When I’m happy it’s a feeling of being untroubled and content rather than being zestful and aroused.
13. ______ When I talk in front of a group for the first time my voice gets shaky and my heart races.
14. ______ When something good happens, I am usually much more jubilant than others.
15. ______ My friends might say I'm emotional.
16. ______ The memories I like the most are of those times when I felt content and peaceful rather than zestful and enthusiastic.
17. ______ The sight of someone who is hurt badly affects me strongly.
18. ______ When I'm feeling well it's easy for me to go from being in a good mood to being really joyful.
19. ______ "Calm and cool" could easily describe me.
20. ______ When I'm happy I feel like I'm bursting with joy.
21. ______ Seeing a picture of some violent car accident in a newspaper makes me feel sick to my stomach.
22. ______ When I'm happy I feel very energetic.
23. ______ When I receive an award I become overjoyed.
24. ______ When I succeed at something, my reaction is calm contentment.
25. ______ When I do something wrong I have strong feelings of shame and guilt.
26. ______ I can remain calm even on the most trying days.
27. ______ When things are going good I feel "on top of the world."
28. ______ When I get angry it's easy for me to still be rational and not overreact.
29. ______ When I know I have done something very well, I feel relaxed and content rather than excited and elated.
30. ______ When I do feel anxiety it is normally very strong.
31. ______ My negative moods are mild in intensity.
32. When I am excited over something I want to share my feelings with everyone.
33. When I feel happiness, it is a quiet type of contentment.
34. My friends would probably say I'm a tense or "high-strung" person.
35. When I'm happy I bubble over with energy.
36. When I feel guilty, this emotion is quite strong.
37. I would characterize my happy moods as closer to contentment than to joy.
38. When someone compliments me, I get so happy I could "burst."
39. When I am nervous I get shaky all over.
40. When I am happy the feeling is more like contentment and inner calm than one of exhilaration and excitement.
Appendix D

**Emotional Reactions Questionnaire (ERQ):**

The following questions are designed to gauge the basic emotions that you experienced during the recall period along with their associated intensity. Please take a few minutes to mark down the intensity of each emotion below using the scale provided as a reference guide.

<table>
<thead>
<tr>
<th>No Emotion</th>
<th>Weak Intensity Emotion</th>
<th>Intense Emotion</th>
<th>Extremely Intense Emotion</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

**Surprise:**

0 1 2 3 4 5 6 7

**Happiness:**

0 1 2 3 4 5 6 7

**Sadness:**

0 1 2 3 4 5 6 7

**Fear:**

0 1 2 3 4 5 6 7

**Disgust:**

0 1 2 3 4 5 6 7

**Anger:**

0 1 2 3 4 5 6 7
Appendix E

Demographics

Instructions: Complete the following by checking the appropriate boxes and filling in the blanks.

1. Gender
   - Male
   - Female

2. How old are you? (in years)
   ____________________________

3. What is your height? (in cm or inches)
   ____________________________

4. What is your weight? (in Kg or lbs.)
   ____________________________

5. What is your ethnic/cultural heritage?
   - White
   - Chinese
   - Black
   - Filipino
   - Latin American
   - Korean
   - Japanese
   - Arab
   - Metis
   - Status Aboriginal
   - Nonstatus Aboriginal
   - Southeast Asian (e.g., Cambodian, Vietnamese, etc.)
   - South Asian (e.g., East Indian, Pakistan, Sri Lankan, etc.)
   - Other: ____________________________

6. What is the highest level of school that you have completed? (Check one answer and provide the levels attained or the number of years completed)
   - Some high school
     ____________________________
     Grade _ completed
   - Finished high school
     ____________________________
     # ____ years completed
   - Some college or university or post secondary
     ____________________________
   - Finished college/university
     ____________________________
   - Don't know/not applicable
     ____________________________
Appendix F

University of Northern B.C.
Cardiovascular Psychophysiology Lab

Experiential Sadness Recall Form

Please read these instructions:

One of the tasks we would like to perform on the next test day is to interview you about an experience in your life that made you very sad. The point of this is to have you relive the experience, your thoughts, actions and feelings to the greatest extent possible. In order to do this, we need to ask you to identify an incident that happened to you, during which you felt sad and about which we can talk to you.

We would like you to take some time and recall the experience in your life during which you felt the most intense sadness that you have ever felt. We would like you to select an event that involves another person and still makes you sad to think about it. We would also like this incident to evoke a strong and pure emotion. By pure, we mean that you experienced a lot of sadness and little of any other emotion, for example, anger or fear. Please remember that if you feel a particular incident is too traumatic or painful to recall, or if you really feel that you would not be able to talk about it, consider recalling some other incident that will elicit the same emotion. If you are now ready to proceed, please write a description of an incident below.

Where were you at the time? What could you see, hear and smell around you?

________________________________________________________________________

________________________________________________________________________

________________________________________________________________________

________________________________________________________________________

Who/what was it that caused you to become so sad? How did it/he/she enter the scene? What did it/he/she look like? What were they wearing?

________________________________________________________________________

________________________________________________________________________

________________________________________________________________________

What happened that made you so sad? What did he/she do or say to you?

________________________________________________________________________
How did you react? What did you do or say in return?


What were you thinking to yourself throughout the incident?


Did your body respond in any way? Did your heart start beating faster? Did your muscles tense up?


What other aspects of the situation or object/person added to your sadness?


Did the way you react also add to your sadness?
Appendix G

Informed Consent Form

Primary Researchers: Dr. Ken Prkachin
Josh Rash B.Sc.(Hon)

Consent Form – Physiological responses during emotion provocation and mental health

This consent form is part of the process of informed consent. It should give you the basic idea of what the research is about and what your participation will involve. If you would like more detail about something mentioned here, please do not hesitate to ask. Please take the time to read this form carefully and to understand any of the accompanying information.

Purpose and Objectives of the Study: The purpose of the research project is to better understand how the cardiovascular system responds during emotions. Throughout this project we hope to improve understanding of the physiological basis of mental health.

Possible Benefits: Your participation provides you an opportunity to learn about current research in psychology, and methods used by psychologists to study health. Your participation also provides an opportunity to have an impact on the development of knowledge.

Risks: This study involves recalling, experiencing and re-living periods of sadness from your past. Recalling periods of sadness can be emotionally uncomfortable. IF YOU FEEL THAT YOU DO NOT WANT TO PARTICIPATE FURTHER, OR, AFTER BEGINNING PARTICIPATION, YOU DO NOT WANT TO CONTINUE YOUR PARTICIPATION, PLEASE LET THE RESEARCHER KNOW. YOU WILL NOT BE PENALIZED FOR DISCONTINUING YOUR PARTICIPATION. The study will require, at most, one and one-half hours of your time.

Procedure: A series of questions will be asked at the beginning of the study to ensure that you are a suitable candidate to participate. Following this, you will be asked to fill out several questionnaires during which the experimenter will read the episode of recalled sadness that you were previously instructed to bring with you. Psychophysiological recording will commence following completion of the questionnaires.

For psychophysiological recording, you will be fitted with disposable ECG electrodes placed on each wrist and the right shin as well as a respiratory chest band placed around the thorax. Recordings will entail sitting in a comfortable chair and remembering the sad experience.

Withdrawal from the Study: You are under no obligation to participate in this study, and once the study has begun you may terminate your participation at any time without penalty. If for any reason you feel that you are unable to participate or wish to end your participation once the experiment has begun, please feel free to inform the researcher of your wishes.

Confidentiality and Anonymity: Your confidentiality and anonymity, including your data and any storage medium, will be protected at all times (by participant number not by names) in a secured filing cabinet in a locked lab. That is, your data will not be identified by name. The researchers will safeguard and store the data, results, and associated materials. Confidential and anonymous data
will be stored indefinitely owing to the consideration that future research discoveries can prompt new forms of analyses that may be applied to already existing data. Bearing this in mind, aggregate results will not be used for research falling outside of the parameters of the present study.

Use: The data collected in this experiment is intended to be used for a publication in a research article and/or conference presentation. Only aggregate (all participants combined) data will be reported, so there is no chance that you could be personally identified.

Questions: At the completion of the study, you will receive a description of the experiment. If you have any questions regarding this study, including the results or to discuss the potential of adverse reactions, please contact Dr. Ken Prkachin (kmprk@unbc.ca or 960-6633), Psychology Program, University of Northern British Columbia. Complaints or concerns regarding this study may also be directed to the Office of Research at UNBC (contact the Office of the Research at 960-5820 or via e-mail at reb@unbc.ca).

Consent:

I, __________________________, have read the above description and agree to participate. A copy of this form has been given to me for my records. The procedure and its possible risks have been explained to me by __________________________ and I understand them. I understand that I am free to withdraw from the study at any time without penalty of any type.

_________________________________________  ________________
Signature                            Date

_________________________________________
Researcher
Appendix H

Primary Researchers: Dr. Ken Prkachin
Josh Rash

Information Debriefing Sheet

Thank you for participating in one of the Health Psychology Laboratory’s research studies. The study that you have participated in is concerned with how the cardiovascular system works to influence mental and emotional health in humans. This experiment is concerned with the role of the autonomic nervous system (ANS) in sadness. The parasympathetic and the sympathetic branches of the ANS act to continuously adjust heart rate to meet the demands of the environment. The sympathetic branch is an excitatory system that acts to increase physiological arousal while the parasympathetic branch is an inhibitory system that acts to decrease physiological arousal. Thus, dominance of the sympathetic branch is associated with increases in pulse, blood pressure, heart rate, and arousal while dominance of the parasympathetic branch is associated with decreases in pulse, blood pressure, heart rate and arousal. Specifically, the purpose of the research project is to determine (1) if the ability to regulate the influence of the sympathetic and parasympathetic branches during emotional provocations is related to depressive symptoms, and (2) to test the influence of emotional regulation on autonomic functioning and depressive symptoms.

This is a relatively new area of research, but understanding the role of cardiovascular functioning in mental health is becoming an area of great scientific interest. Previous research has shown that cardiovascular functioning can be measured in a way that meaningfully reflects autonomic activity that has been associated with emotion regulation and temperament in children. Other lines of research have used grade school children to show that cardiovascular functioning is associated with depressive symptoms. For excellent reviews of this research area you can refer to the following source:


Again, thank you for your participation in this project – your contribution is greatly appreciated. If you have any questions regarding any aspect of the project, or would like more information about the results of the study, please feel free to contact Dr. Ken Prkachin (kmprk@unbc.ca).

Should you require assistance dealing with sensitive issues please stop by or contact the University of Northern British Columbia’s Counseling Centre by phone at (250) 960-6369.