



Virginia Commonwealth University
VCU Scholars Compass

Theses and Dissertations

Graduate School

2013

Heart Rate Variability as a Moderator of Trauma Writing Outcomes

Alison Eonta

Virginia Commonwealth University

Follow this and additional works at: <http://scholarscompass.vcu.edu/etd>

 Part of the [Clinical Psychology Commons](#)

© The Author

Downloaded from

<http://scholarscompass.vcu.edu/etd/3236>

This Dissertation is brought to you for free and open access by the Graduate School at VCU Scholars Compass. It has been accepted for inclusion in Theses and Dissertations by an authorized administrator of VCU Scholars Compass. For more information, please contact libcompass@vcu.edu.

HEART RATE VARIABILITY AS A MODERATOR OF TRAUMA WRITING OUTCOMES

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of
Philosophy at Virginia Commonwealth University

By: ALISON EONTA, M.S.
Virginia Commonwealth University, 2010
B.A. University of Virginia, 2005

Director: Scott R. Vrana, Ph.D.
Professor of Psychology
Department of Psychology

Virginia Commonwealth University
Richmond, Virginia
September, 2013

Acknowledgement

I wish to thank several people for their help and support in the completion of this dissertation. First and foremost, I would like to thank Dr. Scott Vrana for his tireless support, encouragement, and advice throughout every step of this process. I feel very grateful and fortunate to have had you as an advisor throughout graduate school. I would like to thank Andrea Konig for bringing me on board this project and serving as a great friend and colleague. I would also like to thank Drs. David Coogan, Bruce Rybarczyk, Michael Southam-Gerow, and Shawn Utsey for their thoughtful comments throughout the proposal and defense stages of this dissertation. Many thanks go out to all past and present members of the Vrana lab; discussing research ideas with all of you was always stimulating. Finally, I would like to extend my gratitude to Stephanie Dyal for all of her data collection help.

I would also like to thank my husband, Doug Sechler, and my entire extended Eonta-Czajkoski family for all their love and support, not just as it relates to this dissertation, but to all aspects of my life.

Table of Contents

	Page
Acknowledgement.....	ii
List of Tables.....	vii
List of Figures.....	viii
Abstract.....	ix
Introduction.....	1
Literature Review.....	3
Overview of Trauma Writing.....	3
Response Training.....	9
Heart Rate Variability.....	11
Basics and theory.....	11
HRV frequency bands and their interpretation.....	15
High frequency HRV: Parasympathetic control and emotion regulation.....	16
Low frequency HRV: Sympathetic control and task load.....	18
Very low/ultra low frequency HRV.....	20
Measurement.....	20
Analysis.....	20
Data treatment.....	22
Demographic variables.....	23
Gender.....	23
Ethnicity.....	23

Age.....	24
HRV and outcome variables.....	24
PTSD.....	24
Depression.....	26
Physical illness.....	27
Heart rate and skin conductance.....	27
Statement of Problem.....	28
Statement of Hypotheses.....	29
Method.....	30
Experimental Overview.....	30
Participants.....	31
Design and Procedure.....	32
Self-Report Measures.....	36
Physiological Measures.....	38
Data Reduction and Screening.....	39
Data Analysis Plan.....	41
Results.....	44
Demographics.....	44
Attrition.....	44
Preliminary Data Screening.....	47
Baseline Data.....	48
Hypothesis 1.....	52
Hypothesis 2.....	52

Hypothesis 3.....	53
Hypothesis 4.....	57
Hypothesis 5.....	57
Hypothesis 6.....	60
Discussion.....	61
Hypothesis 1.....	61
Hypothesis 2.....	65
Hypothesis 3.....	65
Hypothesis 4.....	68
Hypothesis 5.....	68
Hypothesis 6.....	69
Limitations and Future Directions.....	70
List of References.....	73
Appendices.....	83
A Training Protocol	83
B Writing Instructions	93
C Demographic Questionnaire	96
D Davidson Trauma Scale	97
E Short Version of the Davidson Trauma Scale	99
F Center for Epidemiological Studies-Depression Scale (CES-D).....	101
G The Pennebaker Inventory of Limbic Languidness (PILL).....	103
H Self-Assessment Manikin (SAM).....	105
I Hypothesis 3 Regression Table.....	106

J	Hypothesis 4 Regression Table.....	107
K	Hypothesis 5 Regression Table.....	108
L	Hypothesis 6 Regression Table.....	110
Vita.....		111

List of Tables

	Page
Table 1. Demographic Information.....	45
Table 2. Heart Rate and Skin Conductance Means (and Standard Deviations) at Baseline and During Writing Period.....	49
Table 3. Baseline Log RSA Means (and Standard Deviations).....	50
Table 4. Questionnaire Data at Baseline and at One-Month Follow-Up.....	51
Table 5. PTSD Symptom Severity at Post-Writing Session One and Session Three.....	52
Table 6. Pearson Product-Moment Correlations Between Baseline Log RSA and Baseline Measures.....	53

List of Figures

	Page
Figure 1. Attrition Rates.....	46
Figure 2. Moderation of the Effect of Trauma Condition on Follow-Up Depression Symptoms by Log RSA.....	54
Figure 3. Moderation of the Effect of Trauma Condition on Post-Writing Session Three PTSD Symptom Severity by Log RSA.....	56
Figure 4. Moderation of the Effect of Training Condition on Follow-Up Physical Illness Symptoms by Log RSA for Trauma Writers Only.....	59

Abstract

HEART RATE VARIABILITY AS A MODERATOR OF TRAUMA WRITING OUTCOMES

By: Alison Eonta, M.S.

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy at Virginia Commonwealth University

Virginia Commonwealth University, 2013.

Major Director: Scott R. Vrana, Ph.D.
Professor of Psychology
Department of Psychology

Writing about personal traumatic experiences is associated with beneficial effects on physical and psychological symptoms compared with writing about emotionally neutral events. However, not everyone benefits from trauma writing to the same extent. The present study hypothesized that the effectiveness of trauma writing may be moderated by emotion regulation, as indexed by respiratory sinus arrhythmia (RSA). Research also shows that greater physiological reactivity is predictive of better trauma writing outcomes. Given the importance of physiological output in emotional processing, response training was developed and found to increase appropriate physiological reactivity. Because higher RSA is thought to indicate a more flexible response style including processing both emotional and physiological cues, it was

hypothesized that trauma writers with higher resting RSA who received response training (as opposed to stimulus or no training) would have the best outcomes. It was also predicted that higher resting RSA would be related to lower baseline levels of depression, posttraumatic stress disorder (PTSD), physical illness symptoms, heart rate, and skin conductance.

In the current study, participants wrote for 20 minutes on three occasions about a personal traumatic event (n = 113) or a neutral topic (n = 133) and received response training (n = 79), stimulus training (n = 84) or no training (n = 83). Heart rate and skin conductance were recorded in sessions one and three during a 10-minute baseline, 20-minute writing, and 10-minute recovery period. Self-reported trauma symptoms were assessed in each session. At baseline and one month after completing the sessions, participants filled out assessments of depression, PTSD, and physical illness symptoms.

As predicted, participants with higher resting RSA who wrote about a trauma had greater reductions in symptoms of PTSD at post-writing session three. Higher resting RSA was also related to lower levels of resting heart rate and skin conductance. No relation was found between RSA and baseline symptoms of depression, PTSD, and physical illness. This study also found no effect of resting RSA as a moderator of response training outcomes.

Heart Rate Variability as a Moderator of Trauma Writing Outcomes

Research has shown that writing about a personally relevant trauma is associated with improved physical and psychological health (Sloan & Marx, 2004b). People who write about a neutral topic, conversely, do not seem to experience similar benefits to psychological and physical health (Sloan & Marx, 2004b). While general trends in the research seem to indicate that trauma writing is beneficial, some studies find no beneficial effects of trauma writing (Batten, Follette, Hall, & Palm, 2002; Kloss & Lisman, 2002; Stroebe, Stroebe, Schut, Zech, & van den Bout, 2002) or even find adverse effects (Gidron, Peri, Connolly, & Shalev, 1996). Since not everyone is benefitting from trauma writing, it is important to investigate both the mechanisms by which trauma writing works, and the individual factors that influence trauma writing outcomes, as trauma writing is a potentially beneficial clinical treatment for people who have experienced a traumatic event.

Heart rate variability (HRV) is generally seen as an index of emotion regulation (Thayer & Lane, 2000) and may play a role in moderating the beneficial effects of trauma writing. Higher levels of HRV are associated with greater emotional and behavioral flexibility, and tend to be associated with better health outcomes, both physical and mental (Berntson et al., 1997). Given these findings, HRV may moderate outcomes of a trauma writing intervention, especially given the fact that trauma writing has been found to be emotionally arousing (Sloan & Marx, 2004b).

There may also be ways to increase an individual's therapeutic response to trauma writing. When confronted with personally relevant traumatic stimuli, people with greater physiological reactivity experience beneficial physical and mental health outcomes (Beckham, Vrana, May, Gustafson, & Smith, 1990; Epstein, Sloan, & Marx, 2005; Foa & Kozak, 1986;

Lang, Melamed, & Hart, 1970; Sloan, Marx, & Epstein, 2005). To further enhance physiological reactivity in response to imagery exposure, Lang and colleagues (Lang, Kozak, Miller, Levin, & McLean, 1980) developed a response training protocol that helps participants focus on their physiological sensations and reactions. In response training, response-oriented details such as actions or somatic responses are emphasized. While response training has been shown to increase physiological reactivity in response to trauma-relevant images (Lang et al., 1980; Lang, Levin, Miller, & Kozak, 1983; Miller, Levin, Kozak, Cook, Mclean, & Lang, 1987), no studies have yet investigated response training's applicability to written trauma-relevant cues. Further, because people with high HRV display more flexibility in their emotional responding, they may be prepared to benefit more from response training.

The current study examines whether HRV moderates the effects of trauma writing on psychological and physical health symptoms in that higher resting levels of HRV are expected to be associated with better physical and mental outcomes following a trauma writing intervention. In addition, higher resting levels of HRV will be associated with lower baseline symptoms of physical and mental distress (Berntson, et al., 1997). Higher resting levels of HRV are also expected to be associated with lower resting heart rate and skin conductance as well as more appropriate across-session physiological reactivity in response to the trauma writing intervention, such as elevated heart rate and skin conductance during the first writing session.

To date, only one study has looked at resting HRV and trauma writing (O'Connor, Allen, & Kaszniak, 2005). A better understanding regarding which individuals benefit most from trauma writing and why would allow clinicians to better select and tailor trauma writing interventions for maximal psychotherapeutic benefit. In this study, a diverse group of male and female college students were asked to write on three occasions about either a personally relevant

traumatic event or a neutral topic. Participants also received response training, stimulus training (this form of training focuses on stimulus details such as colors and sizes of objects), or no training, and physiological reactivity was recorded at baseline, during writing, and during a recovery period. Symptoms of depression, PTSD, and physical illness were assessed at the initial session and at one-month post-writing. PTSD symptoms were also assessed post-writing at each session.

In order to further investigate the connections between trauma writing and emotion regulation, the relevant literature on trauma writing and response training will be reviewed. This will be followed by more in-depth information about HRV, specifically the theory behind HRV, the interpretation of different HRV frequency bands, methodological issues pertaining to HRV, the relationship between HRV and demographic variables, and the relationship between HRV and outcome variables of interest.

Literature Review

Overview of Trauma Writing

Research has shown that writing about a personally traumatic event is beneficial to both physical and mental health (Pennebaker & Seagal, 1999; Sloan & Marx, 2004b). The “writing paradigm” was developed by Pennebaker (Pennebaker & Seagal, 1999). Typically, participants are asked to recall a traumatic event and write about it on three to seven occasions for 15-20 minutes per occasion. Participants are asked to write about their trauma with as much emotion and feeling as possible (Pennebaker, 1997). In laboratory settings, the potential benefits of trauma writing have been studied among diverse nonclinical and clinical samples, including people with posttraumatic stress disorder (PTSD) and bereavement. In clinical settings, trauma writing has also been used as a form of exposure therapy for posttraumatic stress disorder

(PTSD), both as a standalone treatment (van Emmerik, Reijntjes, & Kamphuis, 2013) and as an important component in multi-component treatment packages like Cognitive Processing Therapy (Resick et al., 2008).

Smyth's (1998) meta-analysis of trauma writing studies reviewed thirteen trauma writing studies conducted on healthy, nonclinical samples. Smyth (1998) found moderate effect sizes ($d = 0.47$) in symptom improvement for participants who disclosed traumatic events compared to participants who wrote about neutral topics. Among the thirteen studies that were reviewed, tracked symptoms included visits to health centers, self-reported physical symptoms, blood pressure and heart rate, and anxiety and affect, suggesting that trauma writing is linked to beneficial physical health, physiological, and psychological outcomes. These three symptom clusters will be reviewed below. Because the current study uses a sample of nonclinical college students, this review will focus on studies involving this group.

Physical health benefits were associated with trauma writing in a number of studies using college students. Fewer visits to physicians were reported two months, six months, and 1.4 years post-writing among people who wrote about a trauma, compared to people who wrote about neutral topics (Pennebaker & Beall, 1986; Pennebaker, Colder, & Sharp, 1990; Pennebaker & Francis, 1996; Pennebaker, Kiecolt-Glaser, & Glaser, 1988). Fewer self-reported physical illness symptoms have also been observed at one and two months post-trauma writing, compared to people who wrote about neutral topics (Epstein et al., 2005; Sloan & Marx, 2004b; Sloan, et al., 2005). Improvements in immune functioning have also been observed among trauma writers, but not among neutral writers (Booth, Petrie, & Pennebaker, 1997).

Additionally, there appear to be physiological differences occurring across sessions. College students who wrote about a traumatic event had reductions in heart rate, skin

conductance, and cortisol levels across writing sessions, while students who wrote about a neutral topic did not experience decreases in these physiological markers (Epstein et al., 2005; Petrie, Booth, Pennebaker, Davison, & Thomas, 1995; Sloan, Marx, Epstein, & Lexington, 2007).

Trauma writing has also been shown to have a number of psychological effects. Directly after writing, college students who wrote about trauma reported more fear, anxiety, and depression, compared to participants who wrote about a neutral event (Greenberg, Wortman, & Stone, 1996). Across sessions, however, participants who wrote about trauma reported greater decreases in unpleasantness, negative mood, and arousal relative to participants who wrote about a neutral topic (Sloan et al., 2005; Sloan & Marx, 2004b). Between one and four months post-writing, participants who wrote about a traumatic event reported greater reductions in post-traumatic symptom severity, depressive symptoms, avoidant behavior, overall distress, and intrusive thoughts compared to participants who wrote about a neutral topic (Park & Blumberg, 2002; Sloan et al., 2005; Sloan & Marx, 2004b).

While most studies have found that trauma writing is associated with numerous physical and mental health benefits, this finding is not universal. Three studies in the literature report no benefits following a trauma writing intervention. In a study using a nonclinical undergraduate student sample, Kloss and Lisman (2002) found no self-reported reductions in physical or psychological health symptoms following a trauma writing intervention. Two other studies that found no benefits to trauma writing involved clinical samples. In a study of trauma writing using a sample of women with a history of childhood sexual abuse, no psychological or physical benefits were observed following trauma writing (Batten et al., 2002). In a study by Stroebe and colleagues (2002), trauma writing yielded no physical or psychological benefits among bereaved

individuals immediately following a loss or 6-months after the trauma writing intervention was completed. Only one study was found in which adverse effects were reported following the trauma writing intervention. In a study by Gidron and colleagues (1996) involving a sample of people with PTSD, participants who wrote about their most severe trauma (and were also asked to orally describe their trauma, which was a deviation from the Pennebaker writing paradigm), actually had increased visits to health care providers and increased avoidance symptoms at five weeks post-writing, compared to participants who had written about a neutral topic.

Sloan, Marx and colleagues (2007) were critical of the studies that found that trauma writing yielded no benefits (Batten et al., 2002; Kloss & Lisman, 2002; Stroebe et al., 2002) or were associated with adverse outcomes (Gidron et al., 1996). Sloan, Marx and colleagues (2007) attributed the study outcomes to the researchers' deviations from the traditional Pennebaker writing paradigm. It is also possible that these studies did not find overall benefits to trauma writing because individual differences moderated response to the trauma writing intervention (Sloan & Epstein, 2005).

Overall, trauma writing, compared to neutral writing, seems to be beneficial to physical and psychological health, particularly across sessions. The reasons for these benefits may be partially explained by the bioinformational theory of emotions (Lang, 1979). According to bioinformational theory, memory networks are made up of three different types of "information units": stimulus units, response units, and meaning units. Stimulus units consist of representations and descriptions of events. Response units consist of behaviors, physiological readiness for action, and emotional, subjective language. Meaning units consist of objective, declarative language. The emotional memory network as a whole becomes activated when some

external input activates enough of those information units. For the memory network to be completely accessed, all three types of information units must be activated.

Lang suggests that stimulus and meaning units are easier for people to activate than response units (Lang, 1984). For example, Lang (1984) points out that people's self-reported distress levels in reaction to certain events may not correspond with their physiological reactions, suggesting that the memory network is not being fully accessed. This has implications for exposure treatment. Numerous imagery studies have found that participants who demonstrate greater heart rate reactivity at the beginning of exposure treatment have better outcomes than participants who do not experience as much initial physiological reactivity (Beckham et al., 1990; Foa & Kozak, 1986). As discussed earlier, writing about a traumatic event has also been shown to increase initial physiological reactivity and result in habituation of physiological symptoms across sessions and more favorable treatment outcomes over time (Epstein et al., 2005; Sloan & Marx, 2004b; Sloan et al., 2005). These findings are consistent with bioinformational theory, and suggest that participants who are better able to access the complete memory network, including the response units, are better able to process and modify the memory network and achieve symptom relief.

Bioinformational theory would predict that activation of the complete memory network is associated with how beneficial trauma writing will be to different participants. The response units within the memory network play a key role, as response units that involve physiological and emotional responses may be more difficult to activate than the stimulus and meaning units of the memory network. Given the relative difficulty of accessing response units as well as the key role the response units play in memory network activation, it is possible that trauma writing will be more effective for people who have higher levels of emotion regulation, especially because

the traditional instructions for the Pennebaker writing paradigm emphasize that participants write about the trauma with as much emotion and feeling as possible (Pennebaker, 1997).

One proposed physiological correlate of emotion regulation is heart rate variability (Porges, 1995a). HRV is a measure of the variation in the time intervals between heart beats. As will be reviewed in more detail below, higher levels of HRV, indicating greater variability in the interbeat intervals of the heart, are associated with better physical and mental health outcomes as well as greater emotion regulation, whereas lower levels of HRV are associated with poorer physical and mental health outcomes and inferior emotion regulation.

To date, there have been only two studies that have looked at HRV and trauma writing. Both studies focused on respiratory sinus arrhythmia (RSA), which is a naturally occurring form of HRV that occurs as a result of the breathing cycle. Both studies conceptualized RSA as being a moderator of written disclosure outcome and a physiological correlate of emotion regulation (O'Connor et al., 2005; Sloan & Epstein, 2005). In the Sloan and Epstein study (2005), a mixed-gender, mixed-ethnicity sample of college students ($N = 94$) wrote for 20 minutes on three consecutive days. Sloan & Epstein (2005) found that, for trauma writers but not for neutral writers, higher RSA during the first writing session significantly predicted improvements in depression symptoms and physical health symptoms at follow-up one month later. Across session RSA was found to be stable for both the neutral and trauma writers. Sloan and Epstein (2005) interpreted their findings to mean that the benefits of trauma writing are further enhanced for participants with better emotion regulation ability.

The other study that looked at emotion regulation and trauma writing was conducted by O'Connor and colleagues (2005). A sample of bereaved individuals ($N = 29$), the majority of whom were white and female, wrote about either the death of a loved one or a neutral topic for

20 minutes on three separate occasions. Both writing groups experienced significant across-session decreases in depression, anxiety, and grief symptoms, with no differences between trauma and neutral writers. However, resting RSA moderated the influence of the written disclosure intervention, as trauma writers with the highest RSA showed the most improvement in mood at a one-month follow-up session. As expected, elevated resting RSA did not predict outcomes for the neutral writers, whose writing topics were largely free from emotional content. These findings suggest that people with better emotion regulation (i.e. higher RSA) make better use of the emotions elicited by the trauma writing intervention, whereas people with poorer emotion regulation do not receive as much benefit from trauma writing (O'Connor et al., 2005).

Taken together, the two studies that look at both emotion regulation (as indexed by resting RSA (O'Connor et al., 2005) or RSA during the first writing period (Sloan & Epstein, 2005)) and trauma writing found that people with better emotion regulation receive greater benefits from trauma writing, as seen by improved psychological and physical health, compared to trauma writers with poorer emotion regulation. Emotion regulation does not seem to affect symptom outcomes for neutral writers. Given these findings, it seems that individual differences in emotion regulation are moderating the effectiveness of trauma writing interventions. In the next section, response training will be reviewed as a potential tool that can help participants further enhance their physiological and emotional responses to trauma writing.

Response Training

Lang's bioinformational theory posits that a memory network can only be fully activated and modified when three different types of information units (stimulus, meaning, and response) are engaged. In order to amplify physiological responding in imagery research and to test bioinformational theory, researchers have developed ways in which individuals can better access

a memory network's response units. Response training, developed by Lang and colleagues (1980), consists of a brief interaction between a participant and a response trainer. Response trainers read scripts aloud to the participants, who are asked to imagine the situation being described and then describe the situation to the trainer. As participants describe the situation, trainers provide reinforcement in the form of verbal encouragement whenever participants use response language to describe the situation. Response language could include actions as well as somatic, physiological, and perceptual responses that the participant describes within the context of the imagined situation. To gauge the effectiveness of response training, it was compared to "stimulus training," in which the trainer provided encouraging verbal feedback only when the participant described stimulus details such as colors, textures, and sizes of objects.

Studies by Lang and his colleagues have found that response training amplified heart rate reactivity in situationally-appropriate ways (Lang et al., 1980; Lang et al., 1983), compared to stimulus training. Additionally, response training has been shown to amplify the appropriate physiological reactivity of participants with relatively poor imagery ability in response to personalized emotional scenes (Miller et al., 1987). Without such training, these participants tend to have difficulty accessing their emotional memory networks when presented with standard imagery scripts, but when they receive response training and are presented with personalized emotional scenes, physiological reactivity is strengthened (Miller et al., 1987). To date, the physiological effects of response training have been investigated using heart rate reactivity, skin conductance, and ocular activity, among other measures (Lang et al., 1980; Lang et al., 1983; Miller et al., 1987).

It is thought that response-trained individuals with better emotion regulation (i.e., higher HRV) will experience an appropriate, enhanced physiological response (i.e., increased heart rate

and skin conductance) to an initial trauma writing activity, indicating that the memory network is becoming more fully activated; however, response-trained individuals with poorer emotion regulation are not expected to display such enhanced physiological reactivity to the trauma writing activity.

In order to better understand emotion regulation, however, one must have a better understanding of heart rate variability. The next section includes information about the theories of HRV, how different HRV frequency bands are interpreted, and different methodological issues to consider when looking at HRV.

Heart Rate Variability

Basics and theory. Heart rate variability refers to the variation in the interbeat intervals of the heart and provides information about the functioning of the autonomic nervous system, which controls actions of the heart. The autonomic nervous system is comprised of two branches: the sympathetic nervous system and the parasympathetic nervous system. An oft-used analogy to describe the stress response conceptualizes the body's autonomic nervous system as a car, with its two branches being cast as the pedals. The sympathetic nervous system plays the role of the accelerator pedal (which, as Friedman (2007) puts it, may be “sticky” when confronted with a feared stimulus), while the parasympathetic nervous system functions as the brake pedal, which, when responding to stress, may malfunction.

When the body is under stress, it responds by activating the sympathetic nervous system, which releases norepinephrine and epinephrine – hormones that increase heart rate and blood pressure (Yehuda, McFarlane, & Shalev, 1998). Among other bodily reactions, activation of the sympathetic nervous system also induces other “fight or flight” responses such as dilated pupils, increased heart rate and force of contraction, and inhibited digestion. The parasympathetic

nervous system complements the sympathetic nervous system by offering a “rest and digest” response associated with, among other bodily processes, a slowing down of heart rate and enhanced digestion.

Historically, researchers emphasized the sympathetic effects on the cardiovascular system. As the 1990’s progressed and newer methods of measuring bodily processes were developed and disseminated, it became easier to analyze sympathetic *and* parasympathetic processes in more sophisticated ways (Berntson et al., 1997). Rather than attributing a measure like heart rate totally to one system over the other, contributions of both systems were considered. Additionally, the concept of homeostasis (usually viewed as a stable equilibrium) was questioned. Whereas strict homeostasis models may have viewed any instability or changes as being inherently negative, new models and theories emphasized the flexible, adaptive nature of variability (Friedman, 2007).

Porges’ polyvagal theory has proven influential in trying to make sense of the function of cardiac parasympathetic control (Porges, 1995a). Taking an evolutionary perspective, Porges focused on the vagus nerve. In humans, the vagus nerve is the tenth cranial nerve. It begins in the brain (the nucleus ambiguus, specifically) and connects to the cardiac sinoatrial node (Porges, 1995b) as well as other visceral organs. These visceral organs play large roles in emotion expression and communication, and include the soft palate, pharynx, larynx, esophagus, and facial muscles (Porges, 1995b).

The vagus nerve slows heart rate and inhibits sympathetic activity. This vagal pathway is used to conserve energy outflow, meaning that parasympathetic activity tends to be highest when the body is at rest (especially in non-REM sleep). When a “challenge” is presented, however (be it a cognitive problem, a situation that demands increased attention, or a major physical threat),

parasympathetic activity decreases (Porges, 1995b). As such, the vagus nerve (as a parasympathetic mechanism) allows mammals to have greater flexibility and responsivity to environmental conditions than other creatures. By focusing on the pathway and function of the vagus nerve, polyvagal theory, in short, connects the evolutionarily adaptive self-regulatory functions of the parasympathetic nervous system to social functioning and emotional expression and regulation (Porges, 1995b).

The neurovisceral integration model (developed by, among others, Friedman, Lane, and Thayer) builds on the biological-emotional connections put forth in Porges' polyvagal theory. The neurovisceral integration model incorporates a number of functions (attentional regulation, classical conditioning, affective information processing, and behavioral and physiological flexibility) and structures (central nervous system, and the cardiovascular system) into one large network (Thayer & Lane, 2000; Sack, Hopper, & Lamprecht., 2004). Under this model, people respond to events in their environment with emotions, which serve to direct behavior toward helping the person meet a particular goal. When emotions are properly functioning as self-regulatory responses to environmental cues, the person can meet environmental changes in a flexible, adaptive fashion. By extension, when a person chooses one particular response, it also means that the person is inhibiting another less suitable response (Thayer & Lane, 2000).

At this point, the neurovisceral integration model draws from a dynamical systems perspective to help conceptualize how, exactly, a person carries out the goal-directed behavior prescribed by their chosen response. The dynamical systems perspective (which grew out of chaos theory (Friedman, 2007)) views an organism as being made up of many different informational circuits that work together in a controlled way (Friedman, 2007; Thayer & Lane, 2000). These interconnected "circuits" are characterized by a complexity and flexibility which, at

first glance, may make them seem random and haphazard, when in fact they are healthy and orderly (Friedman, 2007). In this sense, an emotional response is not something that is decided upon by some central processor and then carried out in a linear fashion; rather, the emotional response is reflective of a “distributed system” (Thayer & Lane, 2000). This terminology comes from the field of computer science, and refers to a set of autonomous processes (the informational circuits, in this case) that interact with each other and with the demands of the environment in order to meet a shared goal. As Thayer and Lane (2000) put it, “the emotional response ‘emerges’ from the interaction” rather than gets passed down.

One network located within the central nervous system (which consists of the brain and spinal cord) that is implicated in goal-directed behavior is the Central Autonomic Network (CAN) (Friedman, 2007). It is part of an “internal regulation system” (Friedman, 2007) that controls functions such as visceromotor responses, neuroendocrine responses, and behavioral responses. The CAN is comprised of a number of brain structures, including but not limited to the cingulate cortex, the amygdala, the hypothalamus, the nucleus ambiguus, etc. (Thayer & Lane, 2000). The CAN facilitates autonomic output through sympathetic and parasympathetic neurons, which stimulate the actions of the heart through the stellate ganglion (a collection of sympathetic nerve cells) and the vagus nerve (made up of parasympathetic fibers). The interaction of both the vagus nerve and the stellate ganglion while inputting into the cardiac sinoatrial node is the source of variability in heart rate (Friedman, 2007; Thayer & Lane, 2000). The heart can then give information back to the CAN if necessary (Thayer & Lane, 2000). In short, the CAN incorporates structures and functions of the central nervous system with structures and functions of the autonomic nervous system when selecting, inhibiting, and organizing responses (Friedman, 2007; Thayer & Lane, 2000).

Most recently, Thayer and colleagues published a review article of neuroimaging studies that looked at the link between HRV and cerebral blood flow (Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012). They found that the amygdala and the medial pre-frontal cortex (mPFC), which are involved in perceptions of threat and safety, are also associated with HRV. Thayer and colleagues believe that this finding supports the idea that HRV may index important emotional functions associated with adaptability and health.

When people are not able to regulate their emotional responses, people are less able to interact with their environments in efficient manners (Thayer & Lane, 2000). Thayer and Lane (2000) view inefficiency as a precursor to dysregulation and, in more severe cases, mental and physical pathology. Emotion regulatory processes are seen as negative feedback loops (Sack et al., 2004). The purpose of a negative feedback loop is to help keep a system flexible. The CAN is functioning optimally and efficiently when its functional and structural components are more loosely associated – this allows for better flexibility in adapting to changing environmental conditions (Friedman, 2007; Thayer & Lane, 2000). When the emotion regulatory processes (in this case, the negative feedback loops) are disrupted, however, the emotion *dysregulation* that arises may result in the development of a positive feedback loop (Sack et al., 2004). Positive feedback loops are associated with rigidity, a diminished ability to adapt to changes in one's environment, as well as potentially negative mental and physical health outcomes (Thayer & Lane, 2000). Thus, people who are better able to regulate their emotions (as indexed by having higher HRV) may be able to better adapt to changes in their environment while optimizing the functioning of the CAN.

HRV frequency bands and their interpretation. In mammals, there is a great deal of naturally occurring variability in the time elapsed between heartbeats (Rottenberg, 2007). By

assessing the amount of time between heart beats, or by focusing on certain frequencies of the heart rate waves, researchers can better understand the contributions of parasympathetic and sympathetic activity to the heart. At any given time, both parasympathetic activity (which slows HR) and sympathetic activity (which accelerates HR) work in tandem to regulate heart rate.

In keeping with a dynamical systems perspective and the neurovisceral integration model, HRV is one example of a physiological marker that is inherently (and healthily) variable in nature. Certainly, when viewed long-range, there exist expected, stable ranges of HRV, but moment-to-moment, HRV (as its moniker suggests) is quite adaptable and flexible. Certain predictable HRV frequencies correspond with particular bodily processes and psychological phenomena (Friedman, 2007). Though there is debate regarding exactly how many frequency bands exist and what they represent (Berntson et al., 1997), in the following section more information will be given about high frequency, low frequency, and very low/ultra low frequency HRV.

High frequency HRV: Parasympathetic control and emotion regulation. Common interbeat intervals in healthy human adults fall into the 0.15 Hz to 0.4 Hz band (Berntson et al., 1997), known as the high frequency band. High frequency HRV levels are generally the most widely studied. When one breathes, there is naturally-occurring variation in heart rate. In a normal breathing cycle, the pulse quickens with inspiration and slows with expiration of breath. This pattern is known as respiratory sinus arrhythmia, or RSA. RSA patterns commonly fall within the high frequency band, highlighting the parasympathetic influence on the high frequency HRV band.

When focusing on parasympathetic control of the heart, it is important to realize that some parasympathetic activity may be occurring outside of the traditional limits of the high

frequency band. For some people, a noticeable percentage of breaths may be occurring at frequencies below 0.15 Hz (about nine breaths/minute) or above 0.4 Hz (about 24 breaths/minute), especially if a task involves stress or exercise – conditions that alter respiration patterns (Berntson et al., 1997). Different research groups prefer to use different ranges when studying RSA; for example, Sahar, Shalev, and Porges (2001) as well as Allen, Chambers, and Towers (2007) believe that the 0.12 – 0.40 Hz range best encompasses RSA activity.

The most widely accepted interpretation of HRV is that high frequency HRV (in particular, RSA data) can serve as an index for vagal (or parasympathetic) control of the heart (Berntson et al., 1997). Unfortunately, it is very difficult to validate HRV measures at the level of autonomic control, as the direct measurement of autonomic cardiac nerve activity is not currently possible in living human subjects (Berntson et al., 1997). In order to approximate such a direct measurement, pharmacological methods have been used to block certain functions of the heart. When, for example, the sympathetic functions of the heart are being blocked, it is possible to observe (in this “missing piece”) what this function is contributing, and how the rest of the autonomic nervous system acts in the absence of the influence of this branch.

Pharmacological blockade studies have generally yielded findings that indicate the dominant role the parasympathetic branch has over the sympathetic branch in terms of heart rate regulation. In these pharmacological studies, almost two thirds of the acceleration in heart rate in response to physiologically arousing stimuli was due to the withdrawal of parasympathetic control (Sack et al., 2004).

In their neurovisceral integration model, Thayer & Lane (2000) suggest that high frequency HRV can also serve as an index of a person’s ability to self-regulate, or control one’s thoughts, actions, and emotions. Thayer and colleagues (2012) point out that that the enhanced

regulation associated with greater HRV applies to both positive and negative affect, as a lack of emotion can also signify pathology. Thayer and Lane (2000) theorize that elevated high frequency HRV levels indicate greater self-regulation and adaptability, and less high frequency HRV indicates poor self-regulation and decreased adaptability to changing environmental factors. In other words, people with higher vagal tone (i.e. higher RSA or high frequency HRV) have greater vagal control of their hearts, which allows these individuals a greater ability to react to their environment and physiologically and emotionally regulate after encountering a stressor (Porges, 1991).

Low frequency HRV: Sympathetic control and task load. A low frequency band exists between 0.05 Hz and 0.15 Hz (Berntson et al., 1997). This range can also be referred to as “medium frequency HRV” (Friedman, 2007). In one of the livelier debates in the HRV literature, researchers disagree about which autonomic processes are being represented within this frequency (Berntson et al., 1997). The general consensus is that low frequency HRV likely has both sympathetic *and* parasympathetic origins.

RSA (a parasympathetic process) can fall within the low frequency band if individuals are taking relatively few breaths per minute (less than nine, generally) (Berntson et al., 1997). The main physiological influence at the low frequency level, however, is believed to stem from blood-pressure regulation processes (Berntson et al., 1997; Friedman, 2007; Friedman & Thayer, 1998). Using a simulation of the baroreflex, which is the body’s response pattern related to short-term blood pressure control, Wesseling and Settels (1985) demonstrated that the Mayer-Hering rhythm (occurring at 0.1Hz) is related to the baroreflex and explains more than half of HRV in the low frequency band.

Unlike with the high frequency band, however, sympathetic processes are also thought to contribute to low frequency HRV, and some researchers view low frequency HRV as being mostly reflective of sympathetic control. Sympathetic activity, for example, plays a role in blood pressure regulation (Friedman, 2007). However, it is generally agreed that, while sympathetic activity may play a greater role at this low frequency, parasympathetic influences are also coming into play (Berntson et al., 1997). Therefore, it is more difficult to use low frequency HRV as an index of any one sympathetic or parasympathetic phenomenon (as is usually done with vagal control of the heart in the high frequency band).

Because the parasympathetic contribution to the low frequency band is somewhat unclear, some researchers choose to report on the ratio of low frequency to high frequency power (LF/HF) to assess sympathetic-parasympathetic balance, with greater LF/HF ratios indicating greater levels of sympathetic control (Cohen et al., 2000; Friedman, 2007; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). This LF/HF ratio is generally accepted as a better estimate of sympathetic activity than just using low frequency measures alone.

Interpretations of low frequency HRV go beyond the strictly physiological. For example, research has shown that HRV decreases as a function of invested mental effort, especially in tasks where working memory is involved, like mental arithmetic, memory search, counting, or planning (Fairclough & Mulder, 2012). The low frequency band is most sensitive to manipulations of task load, perhaps because it is less affected by respiration than the high frequency band. With increased task load comes a cluster of other physiological symptoms: increased BP and HR, decreased blood pressure variability (BPV), breathing amplitude, and

baroreflex sensitivity – this pattern is also called the “defensive reaction type” (Fairclough & Mulder, 2012).

Very low/ultra low frequency HRV. Some variability in interbeat intervals also occurs at “very low” or “ultra low” frequencies (0.003 – 0.05 Hz) (Berntson et al., 1997). These lower frequencies are not studied as much as the higher, more typical frequencies, and are thought to reflect thermoregulatory cycles, plasma resin activity, circadian rhythms, hormonal regulation, and metabolic regulation (Berntson et al., 1997; Friedman, 2007; Friedman & Thayer, 1998). Little research has been done on very low/ultra low frequency HRV.

Measurement. As mentioned earlier, the advent of more sophisticated psychophysiological measurement techniques has made it easier to assess HRV. The first step toward acquiring useable HRV data usually involves obtaining raw cardiac signal data. Generally, interbeat intervals are derived from an electrocardiogram (ECG) (Rottenberg, 2007). Though an ECG is the preferred method by which to obtain the raw cardiac signal data, HRV can also be derived from continuous blood pressure recordings or a photoplethysmographic record (which measures the volume of blood flow optically by calculating differences in light absorption) (Berntson et al., 1997). Unlike ECG, however, these methods often lack sharp peaks, making it difficult to obtain clear interbeat intervals.

Analysis. Researchers have a number of options when measuring HRV: time-domain analyses, frequency domain analyses, and non-linear techniques. The current study will use time-domain analyses because interbeat intervals are being recorded. Time domain analyses are based on interbeat intervals. When measuring HRV using IBI data, it is very important that data be clean. Missing and corrected values can have a significant impact on the conversion of IBI data to HRV (Berntson and Stowell, 2003), and one of the leading conversion programs, CMetX,

does not allow for any missing IBI values (Allen et al., 2007). Descriptive statistics are often used to run time domain analyses, obtaining measures like ranges, standard deviation, and variance (Berntson et al., 1997). Widely used time domain variables include SDNN, or the standard deviation of interbeat intervals. Another popular time domain measure is the square root of the mean squared difference of successive interbeat intervals, or RMSDD. Another approach is to count the number of successive interbeat interval pairs that vary by more than 50 ms (NN50). Once calculated, NN50 can then be used to calculate pNN50, which is the proportion of NN50 divided by the total number of interbeat intervals in the researcher's time frame of interest. Another time domain approach is to calculate the standard deviation of the average interbeat intervals over short periods of time (SDANN; Berntson et al., 1997; Henry, Minassian, Paulus, Geyer, & Perry, 2010). The current study operationalizes HRV as being the natural log of the variance of the interbeat interval time series filtered at the .12-.40 Hz range, thus yielding Log RSA (Allen et al., 2007).

In time domain analyses, each interbeat interval of interest is treated as an independent data sample. The drawback to these descriptive time-domain statistics is that they do not independently provide information about the different frequency components of HRV; if researchers wish to investigate certain frequencies, these frequencies must be filtered out before time-domain analyses are applied. The current study, for example, focuses on respiratory sinus arrhythmia (RSA) because it is an index of emotion regulation, so the high frequency respiration band (.12-.40Hz) must first be selected before the natural log of the variance of the interbeat interval series is taken (Allen et al., 2007).

RSA is one of the most common metrics used to measure high frequency HRV. Because it is linked to respiration, however, some researchers believe that it is not enough to just report

RSA, and that rate of respiration (also called tidal volume) must also be tracked (Berntson et al., 1997; Ritz, Alatupa, Thons, & Dahme, 2009; Lewis, Furman, McCool, & Porges, 2012). Other researchers, however, do not believe that respiratory parameters exert an undue influence on RSA, and argue that RSA alone is a sufficient measure of high frequency HRV (Allen et al., 2007; Denver, Reed, & Porges, 2007). Given the recent research of Denver and colleagues (2007), RSA in the current study was obtained through interbeat interval measurement alone, without measuring rate of respiration as well.

Data treatment. Regardless of the statistical method being employed, it is important to reduce the “noise” that may be present in the R-wave. One of the best preventative methods is using accurate, high-quality recording methods. Even with the best recording equipment, however, it is almost inevitable that there will be some artifacts contaminating the ECG, often attributable to movements made by human subjects. To identify outliers in the interbeat interval series, it is recommended that the data be inspected both visually, using a graphical representation of the R-waves, and using an algorithm (Berntson et al., 1997).

Heart period rhythms vary, so it is recommended that researchers measure multiple interbeat cycles (Berntson et al., 1997). When HRV is measured for longer periods of time, however, variability naturally tends to be greater. Researchers must try to strike an appropriate balance between choosing heart rate periods that have a long enough duration to yield representative data across multiple interbeat cycles, and avoiding prolonging the duration of measurement, thus introducing excess variability into the data. Specifically, it is recommended that recordings last at least one minute (if focusing on HF bands) or two minutes (for LF bands). Also, it is recommended that recording periods not stretch beyond five minutes (Task Force of the European Society of Cardiology and the North American Society of Pacing and

Electrophysiology, 1996). The Task Force (1996) acknowledges that it can be difficult for subjects to maintain stable mental states across five-minute periods. Therefore, it is possible to split up the five-minute time period into more stable one to two-minute time periods (depending on the frequency band of interest) that can then be compared to each other to evaluate stability over time.

Demographic variables. A variety of demographic variables can affect HRV. Because many studies that look at HRV are composed of diverse individuals, the demographic variables of gender, ethnicity, and age will be addressed.

Gender. It is believed that gender affects HRV, but studies have found mixed and inconclusive results. Some studies do not find resting HRV differences between men and women (Li et al., 2009; Sloan et al., 1998), while other studies by Evans and Koskinen find that women have higher HRV than men (as cited in Li, 2009). Still other studies find that men have higher HRV than women (De Meersman & Stein, 2007; Wang, Thayer, Treiber, Snieder, 2005). In the De Meersman and Stein study, however, men's elevated HRV only appeared to manifest during the day. At night, no significant differences in HRV were observed between young men and women, and gender differences in HRV decreased as people aged, and seemed to disappear by the time people were 50 and older (De Meersman & Stein, 2007). Clearly, more research is needed to establish normative HRV levels in men and women, particularly if gender differences could be fluid across age and time of day.

Ethnicity. Little has been written about HRV and ethnicity, and of what has been written, results are mixed. Some studies have found that African Americans have higher resting HRV than Caucasians (Li et al., 2009; Wang et al., 2005). However, in a recent study by Choi and colleagues (2006), African Americans displayed lower levels of both high frequency HRV

and low frequency HRV. When age was also taken into account, it was found that parasympathetic functioning decreased more gradually over time for Caucasian participants, whereas the African American participants were experiencing declining parasympathetic activity at sharper rates at younger ages – Choi et al. (2006) called this trend “premature aging” of the autonomic nervous system.

Age. As people age, they are at greater risk for cardiovascular disease and death (De Meersman & Stein, 2007). Myocardial infarctions and heart failure are associated with significant, long-term reductions in parasympathetic control and, by extension, high frequency HRV. In their review of vagal changes across the lifespan, De Meersman & Stein (2007) point out that parasympathetic control tends to decrease through a person’s 50’s; after the age of 60 parasympathetic control continues to decrease, but the rate of decline becomes more stable. Overall, increased age is associated with decreased HRV (Cohen et al., 1997). Greater caution should also be exercised when interpreting HRV in elderly samples. As people age, they tend to have greater incidences of abnormal heartbeats (making HRV difficult to interpret and perhaps subject to extensive interpolation) as well as increasingly random heart rate patterns (which may artificially inflate variability) (De Meersman & Stein, 2007).

HRV and Outcome Variables. In the current study, a variety of outcome variables will be investigated. The following section provides a review of the literature detailing relationships between HRV and PTSD, depression, physical illness symptoms, heart rate, and skin conductance.

PTSD. There has not yet been significant research conducted with PTSD samples that investigates HRV, and no meta-analyses exist that examine the relationship between HRV and PTSD. Two studies (Blechert, Michael, Grossman, Lajtman, & Wilhelm, 2007; Cohen et al.,

1997) compared the resting HRV levels of people with a diagnosis of PTSD and healthy controls, but no manipulations were run. Both studies found that people with PTSD had lower resting HRV than the control group.

Two other studies by Cohen and colleagues (1998; 2000) incorporated tasks into their design, assessing for both resting HRV and HRV reactivity to tasks. In the Cohen et al. (1998) study, participants with PTSD were asked to recount the traumatic “triggering event” that led to their PTSD symptoms, while a non-PTSD control sample was asked to describe a stressful negative life event. No HRV changes were observed between baseline and the trauma recall task for participants with PTSD, but researchers found that, for the non-PTSD sample, low frequency HRV rose and high frequency values fell while describing the stressful negative life event. At baseline, people with PTSD had lower resting HRV than people without PTSD.

Cohen and colleagues (2000) also compared baseline HRV and HRV responses to recollection tasks among people with PTSD, people with panic disorder, and healthy controls. They found that people with PTSD had, as expected, lower resting HRV compared to a healthy control group. During the recollection task (during which people with PTSD were asked to describe their traumatic “triggering event”), participants with PTSD did not show significant changes in low frequency or high frequency activity compared to baseline, as did people with panic disorder and the healthy controls (both groups experienced lowered high frequency and higher low frequency activity while patients with panic disorder recalled panic attacks and normal controls recalled stressful negative life events).

The current study investigates resting HRV, and these four studies (Blechert et al., 2007; Cohen et al., 1997; Cohen et al., 1998; Cohen et al., 2000) all found that participants with a clinical diagnosis of PTSD had lower resting HRV than healthy controls.

Depression. Recent meta-analyses were reviewed to investigate the link between HRV and depression (Kemp et al., 2010; Rottenberg, 2007). Rottenberg's (2007) article included two meta-analyses: one with relatively physically healthy participants that included 13 studies, for a total of 312 clinically depressed participants and 374 participants who were not depressed. His second meta-analysis included participants who were "cardiovascularly compromised" and was made up of six studies, with 262 clinically depressed individuals, and 334 participants who were not depressed. These meta-analyses focused only on high frequency HRV (Rottenberg, 2007). Kemp et al.'s (2010) meta analysis contained 18 studies, which included a total of 673 clinically depressed participants and 407 nondepressed control participants. This meta-analysis had a slightly different focus than Rottenberg's, as it focused only on individuals who did not have cardiovascular disease and included studies that focused on aspects of HRV beyond just the high frequency band.

Both meta-analyses found that clinical depression is associated with reduced HRV. The overall effect of depression on high frequency HRV in the Rottenberg (2007) meta-analysis conducted with relatively physically healthy participants was $d = 0.332$ (95% CI, $d = 0.179$ - 0.485), indicating a small to medium effect size. Rottenberg's meta-analysis with cardiovascularly compromised participants had a similar, small effect for depression on high frequency HRV: $d = 0.280$ (95% CI, $d = 0.126$ - 0.434). The Kemp et al. (2010) meta-analysis used Hedges' g to calculate effect size separately for different frequencies and ratios of HRV. For high frequency HRV the effect of depression was small: Hedges' $g = -.210$ (95% CI, $g = -.396$ to $-.024$). Additionally, Kemp et al. (2010) found that people with more severe depressive symptoms also tend to have lower HRV, relative to other depressed individuals.

Because the current study sampled nonclinical college students, three other studies were found that included nonclinical individuals and examined the relationship between HRV and depressive symptoms (Fagundes, Diamond, & Allen, 2012; Hopp et al, 2013; Yaroslavsky, Bylsma, Rottenberg, & Kovacs, 2013). For these studies, the relationship was less clear-cut. Fagundes and colleagues' (2012) sample consisted of healthy 14-year-old children, for whom there was no relationship between RSA and depression at baseline. Yaroslavsky and colleagues' (2013) sample was all-female and included both healthy controls and women who had childhood-onset depressive disorder. On average, the women with childhood-onset depressive disorder had three previous depressive episodes, and 24% were currently in a depressive episode. No differences in resting RSA were found between the healthy controls and the participants with childhood-onset depression, and the entire sample's resting RSA was not significantly related to depressive symptoms. Hopp and colleagues (2013) studied a community sample of adults who reported a stressful event within the past eight weeks and found that greater resting RSA corresponded to lower baseline depression levels.

Physical illness. Two review articles found that heart rate variability was associated with poorer health outcomes (Haensel, Mills, Nelesen, Ziegler, & Dimsdale, 2008; Thayer & Lane, 2007). The review by Thayer and Lane (2007) found that, independent of other risk factors, decreased vagal function was associated with all-cause mortality. In the same review, decreased vagal function was also associated with cardiovascular disease risk factors such as obesity and smoking, as well as inflammation. Haensel and colleagues reviewed 13 articles and found that, overall, HRV was inversely related to inflammatory markers (2008).

Heart rate and skin conductance. Greater resting high frequency HRV appears to be related to lower resting heart rate (HR). Numerous studies have supported this finding. For

example, both Allen and colleagues (2007) and Hibbert, Weinberg, and Klonsky (2012) found that greater resting respiratory sinus arrhythmia was related to lower resting HR. The relationship between resting HRV and resting skin conductance (SC) has been less thoroughly researched, however. SC is generally thought to be an index of sympathetic nervous system activity, as well as emotional lability (Boucsein, 1992). However, the only study that could be found investigating the relationship between resting HRV and resting SC yielded no significant relationship between them, and consisted of a sample of 14-year-olds (Diamond, Fagundes, & Cribbet, 2012).

Statement of Problem

Recent review articles and studies involving trauma writing have shown consistency in their findings. People who write about a personally relevant traumatic event tend to have better health outcomes compared to people who write about a neutral topic (Pennebaker & Seagal, 1999; Sloan & Marx, 2004b; Smyth, 1998). However, not all studies find that trauma writing is associated with beneficial outcomes (Batten et al., 2002; Gidron et al., 1996; Kloss & Lisman, 2002; Stroebe et al., 2002). Individual differences in emotion regulation may account for some of the variation in trauma writing findings, in addition to further explaining the mechanisms by which trauma writing is effective. The objectives of this study were to: 1) investigate the relationship between resting HRV (an index of emotion regulation) and pre-writing symptoms of depression, PTSD and physical illness, as well as baseline physiological levels; 2) determine how resting HRV may moderate the effects of trauma writing and training protocol (response, stimulus, or none) on symptoms of physical illness, depression, and PTSD; and 3) explore the expectation that higher resting levels of emotion regulation will be associated with enhanced physiological reactivity in response to the first trauma writing intervention.

Statement of Hypotheses

The following hypotheses were presented:

1. Participants with greater high frequency HRV will have lower baseline levels of physical illness, PTSD, and depression symptomatology.
2. Participants with greater high frequency HRV will have lower resting HR and SC.
3. Among trauma writers, resting high frequency HRV will moderate the effects of trauma writing on physical illness, PTSD, and depression symptoms at one-month follow-up, with greater resting high frequency HRV being associated with better symptom outcomes at follow-up. Also, among trauma writers, resting high frequency HRV will moderate the effects of trauma writing on the change in PTSD symptoms from post-writing session one to post-writing session three, with greater across-session reductions in PTSD symptoms expected among trauma writers with greater resting high frequency HRV. Among neutral writers, resting levels of high frequency HRV will not play a moderating role.
4. Among trauma writers, participants who display greater resting high frequency HRV will display appropriately enhanced physiological trends across sessions; HR and SC reactivity is expected to be elevated in response to the first trauma writing session. Neutral writers will not experience enhanced physiological reactivity in response to the initial writing activity.
5. Trauma writers who receive response training and have greater resting high frequency HRV will demonstrate the best outcomes on physical illness, PTSD, and depression symptoms at one-month follow-up compared to trauma writers in the stimulus training or no training groups. Similarly, trauma writers who receive

response training and have greater resting high frequency HRV are expected to have the greatest symptom reductions in PTSD symptoms from post-writing session one to post-writing session three compared to trauma writers in the stimulus training or no training groups.

6. Trauma writers who receive response training and have greater resting high frequency HRV are expected to show the most elevated HR and SC reactivity in response to the first trauma writing session, compared to trauma writers in the stimulus training or no training groups.

Method

Experimental Overview

Participants were undergraduate students enrolled in an introductory psychology class. Within two weeks, participants attended three lab sessions, and one month later they completed a packet of follow-up questionnaires. Participants were randomly assigned to one of six possible groups (3 Training Condition X 2 Trauma Condition). In the first session, participants filled out demographic questionnaires as well as questionnaires about physical illness symptoms, PTSD symptoms, and depression symptoms. Participants were trained (response, stimulus, or no training), baseline physiological levels were recorded, and then participants were told to write for 20 minutes about either a personally traumatic event or a neutral topic. Physiological reactivity was recorded during writing and a recovery period. In the second and third sessions participants again wrote about their assigned topic for 20 minutes, though physiological levels were only assessed during the third session. One month after the third session, participants were mailed a follow-up packet that included questionnaires about physical health symptoms, depressive symptoms, and PTSD symptoms.

Participants

In constructing the study, a power analysis was conducted using 0.80 power, a trauma writing effect size of 0.23 (Smyth, 1998), and an alpha level of 0.05, which indicated that approximately 30 participants per group should be sufficient to find an effect, resulting in a total sample size of 180 (Cohen, Cohen, West, & Aiken, 2003). A power analysis of 0.80 power, an imagery response training effect size of 0.35 (Lang et al., 1983), and an alpha level of 0.05 found that approximately 14 participants per group would be sufficient to find an effect, resulting in a total sample size of 84 (Cohen et al., 2003). These two sample sizes were averaged, obtaining a total sample size of 132 participants. Attrition rates in previous trauma writing studies have ranged from 4% (Sloan & Marx, 2004a) to 10% (Brown & Heimberg, 2001). To account for a higher estimated attrition rate (10%), participants were enrolled until 145 completed the study. To further increase statistical power, the protocol was administered to an additional 101 participants (N = 246). In total, 195 participants completed all three writing sessions and sent back the one-month follow-up questionnaires.

All participants (N = 246) in the current study were recruited through the Virginia Commonwealth University (VCU) Department of Psychology Research Pool, also referred to as “SONA”, which consists of approximately 1,500 undergraduates per fall, spring, and summer semesters. Participants were compensated with course credit, as is typical in trauma writing studies (Kloss & Lisman, 2002; Pennebaker & Beall, 1986; Smyth, True & Souto, 2001). It is estimated that approximately 55.8-84.5% of undergraduate students have experienced at least one event of sufficient intensity to qualify as a potential traumatic stressor, and 33% have experienced four or more such events of a traumatic nature (Smyth, Hockemeyer, Heron, Wonderlich & Pennebaker, 2008; Vrana & Lauterbach, 1994), so a non-clinical undergraduate

sample was used, as has been the case with other studies of trauma writers (see review by Sloan & Marx, 2004b).

Design and Procedure

Approval was sought from the appropriate Institutional Review Board (IRB), and all participants in the study provided informed consent. In advertisements for this study, participants were told that they would be writing about their lives, and that they would receive course credit as compensation. Participation in the study was voluntary and was not required as part of participants' psychology courses. Once participants expressed interest in participating in the study via the online SONA pool, they were contacted individually by a researcher who helped schedule three sessions and answered any basic questions the participants had regarding the study. If participants were able to schedule all three sessions within a two week period of time, their participation in the study was confirmed.

After participants expressed interest in the current study and were successfully scheduled for three lab-based sessions, participants were randomly assigned to one of six groups in a 3 Training Condition (response training, stimulus training, and no training) X 2 Trauma Condition (trauma, neutral) design. Full participation in the study consisted of attending three lab-based sessions within a two-week time period, and completing and mailing back a packet of follow-up questionnaires approximately one month after session three was completed.

At the beginning of session one, participants were welcomed to the lab and signed an informed consent form. Participants then completed several questionnaires, including a demographic questionnaire, the Self-Assessment Manikin (SAM), the Center for Epidemiological Studies-Depression Scale (CES-D), the Pennebaker Inventory of Limbic Languidness (PILL), and the long form of the Davidson Trauma Scale (DTS-Long form).

Completion of these questionnaires usually took at least 30 minutes, giving participants time to acclimate to the lab setting before any physiological measures were taken. It was only at this point in the study that individual participant's experiences began to differ based on the condition to which the participants were randomly assigned.

Participants in the response training condition spent approximately 45 minutes with a researcher who led the training session (see Appendix A for training protocol). For response trained participants, the training session consisted of a researcher teaching them diaphragmatic breathing techniques and reading four action-oriented scripts aloud (Lang, 1977). These scripts contained references to behavioral and physiological responses without mentioning emotional responses. Examples of behavioral and physiological responses can be found in phrases such as, "Your neck and shoulder muscles are tense and stiff," or, "You breathe deeply." After the researcher read each script, participants were prompted to imagine the script and describe each script's imagery to the researcher. Participants received reinforcement (verbal praise by the researcher) when they described behavioral and physiological imagery. If, in a participant's description to the researcher, no such behavioral and physiological imagery was present, participants were encouraged by the experimenter to include these types of imagery details in descriptions of the scripts that were still to come.

As in the response training condition, participants in the stimulus training condition also spent about 45 minutes with a researcher, who trained participants in diaphragmatic breathing techniques and read four action-oriented scripts aloud (Lang, 1977). Unlike in the response training condition, however, the four scripts in the stimulus training condition contained references to stimulus detail without mentioning physiological or behavioral responses. Examples of stimulus detail are included in phrases such as, "The texture of the page with the

color picture is smooth and glossy,” or “One of your friends drops a red hat.” After reading each script, the researcher prompted participants to imagine the script and describe its imagery aloud. Participants received verbal praise from the researcher when their imagery descriptions included descriptive detail. If, in a participant’s description to the researcher, no such descriptive detail was present, participants were encouraged by the experimenter to include this type of descriptive imagery in descriptions of the scripts that were still to come.

Participants in the no training condition received training in diaphragmatic breathing techniques from a researcher, but unlike participants in the response and stimulus training conditions, these participants received no imagery training from a researcher.

After participants received response, stimulus, or no training, the researcher placed electrodes below the participant’s lowest left rib and the right clavicle to assess IBIs. Electrodes filled with lubricant were also attached to the palm of participants’ nondominant hand to assess skin conductance. Participants were not told what measures the electrodes would be assessing, but were informed that the electrodes were recording the “signals that your body produces all of the time.” Participants were encouraged to use the diaphragmatic breathing technique they had just been taught to help them relax their bodies and minds. As participants relaxed, the researcher continuously recorded IBIs for a 10-minute baseline period.

After the 10-minute baseline period, the researcher provided writing instructions to participants (see Appendix B for writing instructions). These writing instructions differed based on writing condition (trauma writing vs. neutral writing). Participants in the trauma writing condition were asked to write about the most traumatic or distressing situation they had experienced in their entire life. Participants were instructed that this traumatic event should match the one identified earlier on the DTS-Long form. Participants were encouraged to include

as many details as possible about their emotions and feelings. Participants in the neutral writing condition were asked to write about how they spent a typical day. Participants were told not to include details about their emotions or opinions, but instead to keep their writing as factual as possible.

Prior to beginning to write, participants in the response training and the stimulus training group were told to draw from the imagery training they had received earlier while they wrote about their assigned topic. Participants in the no training group received no such instructions. Participants began writing when the researcher left the room. As participants wrote, the researcher continuously recorded IBIs during the 20-minute writing period. After the writing period was over, the researcher collected the participants' writing and administered the SAM. After participants completed the SAM, the researcher asked participants to relax, using the diaphragmatic breathing technique, for ten minutes. As participants relaxed, the researcher recorded IBIs during this 10-minute recovery period. Participants filled out the short form of the Davidson Trauma Scale (DTS-Short form), and then the researcher removed the electrodes. Participants were thanked for coming and reminded of their next two lab-based appointments. The total time required for session one was usually about two hours.

At the second session, participants were greeted by a researcher and asked to fill out the SAM. The researcher then gave participants the same writing instructions they had received in their first writing session. As in the first session, participants wrote for 20 minutes; however, no physiological data were recorded during the second session. After the writing portion of the session, participants completed the SAM. Participants were asked to relax, using the diaphragmatic breathing technique, for ten minutes. Following the relaxation period, participants

filled out the DTS-Short form. The researcher then reminded participants of their third and final session. Session two usually lasted about 45 minutes.

At the third and final lab session, participants filled out the SAM. As in session one, a researcher then attached electrodes below their lowest left rib and right clavicle in order to measure IBI. Participants were asked to sit quietly and use diaphragmatic breathing skills during a 10-minute baseline period. The researcher then provided participants with the same writing instructions as in sessions one and two. While participants wrote for 20 minutes, the researcher recorded IBI. The researcher then collected the participant's writing and administered the SAM. The researcher then instructed the participant to sit quietly and use diaphragmatic breathing skills for a 10-minute post-writing recovery period. Following the recovery period, participants filled out the DTS-Short form. The researcher then removed the sensors, and participants were reminded to fill out a packet of follow-up questionnaires that they would receive in the mail in one month. Participants' mailing addresses were confirmed, and the researcher explained that participants would be debriefed by e-mail upon the completion of the study. Session three usually lasted about one hour.

One month after completing session three, participants were mailed a follow-up packet of questionnaires that contained the CES-D, the DTS-Long form, and the PILL. Researchers estimated that the packet should take approximately five to ten minutes to complete. Participants who successfully completed and mailed back the packet of questionnaires were entered into a random drawing for one of three \$100 cash prizes to be distributed at the end of every semester.

Self-Report Measures

All participants completed a demographic questionnaire (see Appendix C). A number of additional self-report measures were also administered during the course of the study.

Davidson Trauma Scale (DTS): The long version of the DTS (Davidson, 1996) (see Appendix D) was administered to participants at session one and as part of the follow-up packet. The DTS measures both severity and frequency of PTSD symptoms that the participant experienced over the past week. The DTS consists of 17 items that correspond to the DSM-IV diagnostic criteria for PTSD. The DTS has been shown to have good test-retest reliability ($r = 0.86$) and internal consistency ($r = 0.99$) (Davidson et al., 1997). During sessions one, two and three, participants also filled out the short version of the DTS. The short, state version of the DTS consists of 13 items that correspond to a PTSD diagnosis based on criteria B, C, and D in the DSM-IV (see Appendix E), and was developed by the Beckham lab to measure within-session PTSD symptoms. When filling out this questionnaire, participants were asked to consider the symptoms they had experienced since they stopped writing. Though frequency symptoms were collected for both the long and short versions of the DTS, they are not being reported in this study, as these results were highly correlated with the severity symptoms but were not as strong. Thus, only severity symptoms for the long and short versions of the DTS will be investigated and reported.

Center for Epidemiological Studies-Depression Scale (CES-D): The CES-D (Radloff, 1977) (see Appendix F) was administered during session one and as part of the follow-up packet of questionnaires. The CES-D consists of 20 items that assess depression symptoms over the past week. The 20 items make up six scales that reflect different depressive symptoms: depressed mood, feelings of guilt and worthlessness, feelings of helplessness and hopelessness, psychomotor retardation, loss of appetite, and sleep disturbance. High internal consistency has been reported for this measure, with Cronbach's alpha ranging from .85 to .90 (Radloff, 1977). This measure has been shown to have good construct validity (Radloff, 1977), including in an

exclusively female sample (Knight, Williams, McGee, & Olaman, 1997). Additionally, test-retest reliability and internal consistency were not found to vary among different ethnic groups or language groups (Roberts, Vernon, & Rhoades, 1989). This measure is intended to be used by the general population, making it a good fit for the participants in the current study.

The Pennebaker Inventory of Limbic Languidness (PILL): Participants completed the PILL (Pennebaker, 1982) (see Appendix G) at session one and as part of the follow-up questionnaire packet. It consists of 54 items addressing the frequency, rated on a five-point Likert scale, of various physical symptoms and sensations such as “sneezing spells” and “swollen joints”. High internal consistency has been reported for this measure, with Cronbach’s $\alpha = 0.91$. The two-month test-retest reliability was 0.83 ($N = 177$), and the PILL correlated moderately with the Hopkins Symptom Checklist ($r = 0.48, P < 0.01$) (Pennebaker, 1982).

Self-Assessment Manikin (SAM): Participants filled out the SAM (Bradley & Lang, 1994) (see Appendix H) at sessions one, two, and three. The SAM is a pictorial assessment tool that assesses self-reported pleasure (or valence) and arousal. Two rows of drawings appear; on the top row, five figures appear with expressions ranging from happy to unhappy, and on the bottom row, five figures are drawn to represent arousal states ranging from excited to calm. Nine dots appear below the drawings, spaced at regular intervals that span the row of drawings. Participants select both a pleasure dot and an arousal dot that best correspond with how they are currently feeling. In previous studies, emotional and somatic responses to emotionally evocative stimuli co-varied with the valence and arousal dimensions on the SAM (Bradley, Greenwald, Petry, & Lang, 1992). Though data from the SAM were collected, they are not being reported for this study, as no hypotheses pertain to the SAM.

Physiological Measures

Researchers collected physiological data at sessions one and three, recording physiological data continuously for a 10-minute baseline period before the writing period, for a 20-minute writing period, and for a 10-minute post-writing recovery period.

Heart Rate (HR): To record electrocardiograph (ECG) signal, researchers attached sensors below the participant's right clavicle and lowest left rib. A ground electrode was placed on the participant's forearm. Adhesives were applied to the sensors and a medical gel was applied to the sensors to help ensure that ECG was properly and accurately recorded. In order to extrapolate a measure of heart rate, interbeat intervals (IBI) were obtained. A Coulbourn S75-01 Hi Gain Bioamplifier amplified and filtered the ECG signal, and the R-wave peak of the ECG signal was used to signal a computer-based timer, which recorded the time between each R-wave in milliseconds, thus yielding IBI. Off-line, the IBI data were converted to HR, as measured in beats per minute.

Heart Rate Variability (HRV): As mentioned earlier, this study uses time-domain analyses to measure HRV. Because time-domain analyses are based on IBI data (Berntson et al., 1997), collection of IBI data for purposes of measuring HRV took place in the same manner as with HR. Off-line, the IBI data were converted to HRV.

Skin Conductance (SC): SC was recorded using a Coulbourn S71 - 22 Skin Conductance Coupler that applied a constant .5 V across two standard Ag-AgCl electrodes. Participants were asked to wash their hands with tap water (no soap), and two electrodes filled with lubricant were attached to participants' nondominant palms, as the researchers did not wish for the physical movement associated with writing to influence SC. SC was sampled at 10 Hz with a 12-bit analog-digital converter and converted off-line to μmhos .

Data Reduction and Screening

Heart Rate (HR): Off-line, the IBI data were hand-screened in order to detect and correct any artifacts that were present. The IBI data were then inputted into a computer program that converted the IBI data to HR in beats per minute. Though HR data were collected at both sessions one and three, for the purposes of this study only session one HR at baseline and during the writing period were used in analyses. Preliminary examinations of the data indicated that the most stable measure of baseline HR was the mean of the last five minutes of the baseline period of session one, for which 233 participants had valid HR data. The most stable measure of HR during the session one writing period was determined to be the mean of minutes 3-8 of the session one writing period, for which 230 participants had valid HR data.

Heart Rate Variability (HRV): As noted in the Literature Review, when using time-series data that measures HRV from IBI data, it is very important that the IBI data be clean and stable. Even one missing or corrected IBI value can make HRV incalculable (Berntson & Stowell, 2003). Off-line, the IBI data were hand-screened in order to detect and correct any artifacts that were present. The hand-corrected IBI data were then reduced in time. As mentioned in the Literature Review, the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996) recommends that high frequency HRV data be studied in 1-5 minute “chunks.” In this study, researchers decided to look at the two-minute period closest to the end of the baseline period that contained valid IBI data.

The hand-corrected, time-reduced IBI series was then inputted into John J.B. Allen’s publicly available program, CMetX Cardiac Metric Software (Allen et al., 2007) for Microsoft Windows, which converted the IBI data to HRV data. Specifically, IBIs were converted to a time series sampled at 10 Hz, and the series was then filtered through a 241-point optimal finite impulse response digital filter with half-amplitude frequencies of 0.12-0.4 Hz (this is the high

frequency band of HRV). After the filter was applied, CMetX took the natural log of the variance of the filtered waveform as the estimate of respiratory sinus arrhythmia (RSA) (Allen et al., 2007). Log RSA has been used as an index of emotional regulation in other studies of trauma writing and HRV (O'Connor et al., 2005; Sloan & Epstein, 2005). Though Log RSA data were collected at sessions one and three, for the purposes of this study, only the session one baseline period Log RSA data were used. After obtaining the final Log RSA data, the data were screened again and it was determined that two participants had invalid data. The Log RSA data from these two participants were excluded from all future analyses, leaving 224 participants with valid Log RSA data.

Skin Conductance (SC): Off-line, the SC data were hand-screened in order to detect and correct any artifacts that were present. Preliminary examinations of the data indicated that the SC data were positively skewed, as is often the case, so a log 10 transformation was applied to the session one baseline and writing SC data, resulting in Log SC for these variables. Though Log SC data were collected at both sessions one and three, for the purposes of this study only session one Log SC at baseline and during the writing period was used in analyses. The most stable measure of session one baseline Log SC was the mean of the last five minutes of the baseline period of session one, for which 239 participants had valid Log SC data. The most stable measure of Log SC during the session one writing period was determined to be the mean of minutes 3-8 of the session one writing period. These are the measures that will be reported throughout this study, for which 239 participants had valid Log SC data.

Data Analysis Plan

Dependent variables included: 1) physical illness symptoms at one-month follow-up (PILL); 2) depression symptoms at one-month follow-up (CES-D); 3) PTSD symptom severity at

one-month follow-up (DTS-Long form); 4) PTSD symptom severity at post-writing session three (DTS-Short form); 5) HR during the session one writing period (mean of minutes 3-8 of writing period); and 6) Log SC during the session one writing period (mean of minutes 3-8 of writing period).

Independent measures included: 1) Log RSA (mean of the last two minutes of the session one baseline period containing valid IBI data, mean-centered for the full sample [hypotheses 1-4] and for trauma writers only [hypotheses 5-6]); 2) baseline symptoms of physical illness, depression, and PTSD (PILL, CES-D, and DTS-Long form, respectively); 3) symptoms of PTSD at post-writing session one (DTS-Short form); 4) resting HR and Log SC (mean of last five minutes of session one baseline); 5) Gender (coded as 0 = male and 1 = female); 6) Trauma Condition (coded as 0 = neutral and 1 = trauma); 7) Trauma Condition X Log RSA; 8) Response Condition (dummy coded as 1 = response, 0 = stimulus and no training); 9) Stimulus Condition (dummy coded as 1 = stimulus, 0 = response and no training); 10) Response Condition X Log RSA; and 11) Stimulus Condition X Log RSA.

For hypothesis 1, the relationships between Log RSA and baseline symptoms of physical illness, depression, and PTSD were investigated using Pearson product-moment correlations.

For hypothesis 2, Pearson product-moment correlations were used to investigate the relationship between Log RSA and resting HR and Log SC.

To test hypothesis 3, hierarchical multiple regressions were performed for each outcome measure (one-month follow-up PILL, CES-D, and DTS-Long form; post-writing session three DTS-Short form). Variables were entered into the model in three steps. Variables entered in the first step consisted of the questionnaire data at baseline (baseline PILL, CES-D, and DTS-Long form; session one post-writing DTS-Short form) and Gender. These variables were entered first

in order to remove the effects of baseline symptom levels and gender from later models, as baseline and follow-up measures were expected to be correlated, and research suggests that gender is related to RSA (De Meersman & Stein, 2007; Wang et al., 2005). Step two variables included Log RSA and Trauma Condition. Finally, the third step consisted of the Trauma Condition X Log RSA interaction term.

To test hypothesis 4, hierarchical multiple regressions were performed for the outcome measures HR and Log SC during writing. Variables were entered into the model in three steps. Variables entered in the first step consisted of resting HR and Log SC and Gender. Step two variables included Log RSA and Trauma Condition. The third step consisted of the Trauma Condition X Log RSA interaction term.

Hypothesis 5 analyses were restricted to only trauma writers. Hierarchical multiple regressions were performed for each outcome measure (one-month follow-up PILL, CES-D, and DTS-Long form; post-writing session three DTS-Short form). Variables were entered into the model in three steps. Variables entered in the first step consisted of the questionnaire data at baseline (baseline PILL, CES-D, and DTS-Long form; session one post-writing DTS-Short form) and Gender. Step two variables included Log RSA, Response Condition, and Stimulus Condition. Finally, the third step consisted of the Response Condition X Log RSA interaction term and the Stimulus Condition X Log RSA interaction term.

Hypothesis 6 analyses were restricted to only trauma writers. Hierarchical multiple regressions were performed for the outcome measures HR and Log SC during writing. Variables were entered into the model in three steps. Variables entered in the first step consisted of resting HR and Log SC and Gender. Step two variables included Log RSA, Response Condition, and

Stimulus Condition. The third step consisted of the Response Condition X Log RSA interaction term and the Stimulus Condition X Log RSA interaction term.

Results

Demographics

Table 1 summarizes demographic characteristics for the entire sample and separately for trauma writers, neutral writers, and for each Trauma Condition X Training Condition group. Overall, participants were mostly female (72.0%); in their early twenties (mean 21.5 years, S.D. = 5.5); were college freshmen or sophomores (57.7%); and identified English as their first language (85.8%). The sample was ethnically diverse, as 48.0% identified their race as White; 28.0% as Black; 11.0% as Asian; 2.0% as Hispanic; 1.2% as Native Hawaiian or other Pacific Islander; and 9.8% as Other.

Comparisons for participants randomized to each trauma condition and each training condition were made using independent sample t-tests and one-way ANOVAs for continuous measures (age) and chi-square analyses for categorical measures (gender, race, year in school and native language). One significant group difference was found. There was a significant association between response condition and native language, with the no training group containing a greater percentage of non-native English speakers ($n = 19$; 22.9% of no training group) than the response training ($n = 9$; 11.4% of response training group) or the stimulus training groups ($n = 7$; 8.3% of stimulus training group); $\chi^2(2, n = 246) = 8.02, p = .02$, Cramer's $V = 0.18$.

Attrition

As seen in Figure 1, attrition rates were high in this study, with 97.6% of participants participating in writing sessions one and two ($n = 240$), and 95.1% of participants participating in

Table 1.

Demographic Information

Variable	Trauma	Response Trained Trauma	Stimulus Trained Trauma	No Training Trauma	Neutral	Response Trained Neutral	Stimulus Trained Neutral	No Training Neutral	Total
Age	21.9 (6.5) <i>(Range: 18-53)</i>	20.9 (4.2) <i>(Range: 18-37)</i>	20.9 (2.8) <i>(Range: 18-28)</i>	23.9 (9.8) <i>(Range: 18-53)</i>	21.1 (4.3) <i>(Range: 18-44)</i>	20.8 (4.3) <i>(Range: 18-44)</i>	21.1 (4.7) <i>(Range: 18-43)</i>	21.4 (4.0) <i>(Range: 18-35)</i>	21.5 (5.5) <i>(Range: 18-53)</i>
Gender									
Male	28 (24.8%)	9 (22.5%)	9 (25.7%)	10 (26.3%)	41 (30.8%)	10 (25.6%)	10 (20.4%)	21 (46.7%)	69 (28.0%)
Female	85 (75.2%)	31 (77.5%)	26 (74.3%)	28 (73.7%)	92 (69.2%)	29 (74.4%)	39 (79.6%)	24 (53.3%)	177 (72.0%)
Race									
White	58 (51.3%)	23 (57.5%)	16 (45.7%)	19 (50.0%)	60 (45.1%)	23 (59.0%)	19 (38.8%)	18 (40.0%)	118 (48.0%)
African American	27 (23.9%)	8 (20.0%)	11 (31.4%)	8 (21.1%)	42 (31.6%)	7 (17.9%)	19 (38.8%)	16 (35.6%)	69 (28.0%)
Asian	10 (8.8%)	4 (10.0%)	4 (11.4%)	2 (5.3%)	17 (12.8%)	6 (15.4%)	5 (10.2%)	6 (13.3%)	27 (11.0%)
Hispanic	3 (2.7%)	1 (2.5%)	1 (2.9%)	1 (2.6%)	2 (1.5%)	1 (2.6%)	0 (0%)	1 (2.2%)	5 (2.0%)
Pacific Islander	2 (1.8%)	1 (2.5%)	0 (0%)	1 (2.6%)	1 (0.8%)	0 (0%)	0 (0%)	1 (2.2%)	3 (1.2%)
Other	13 (11.5%)	3 (7.5%)	3 (8.6%)	7 (18.4%)	11 (8.3%)	2 (5.1%)	6 (12.2%)	3 (6.7%)	24 (9.8%)
Year in School									
Freshman	35 (31.0%)	14 (35.0%)	9 (25.7%)	12 (31.6%)	57 (42.9%)	15 (38.5%)	19 (38.8%)	23 (51.1%)	92 (37.4%)
Sophomore	28 (24.8%)	13 (32.5%)	7 (20.0%)	8 (21.1%)	22 (16.5%)	9 (23.1%)	6 (12.2%)	7 (15.6%)	50 (20.3%)
Junior	20 (17.7%)	8 (20.0%)	7 (20.0%)	5 (13.2%)	21 (15.8%)	5 (12.8%)	9 (18.4%)	7 (15.6%)	41 (16.7%)
Senior	30 (26.5%)	5 (12.5%)	12 (34.3%)	13 (34.2%)	33 (24.8%)	10 (25.6%)	15 (30.6%)	8 (17.8%)	63 (25.6%)
Native Language									
English	94 (83.2%)	35 (87.5%)	32 (91.4%)	27 (71.1%)	117 (88.0%)	35 (89.7%)	45 (91.8%)	37 (82.2%)	211 (85.8%)
Other	19 (16.8%)	5 (12.5%)	3 (8.6%)	11 (28.9%)	16 (12.0%)	4 (10.3%)	4 (8.2%)	8 (17.8%)	35 (14.2%)

M (SD) or N (%)

all three writing sessions (n = 234). Additionally, 79.3% of all participants participated in all three writing sessions and sent back one-month follow-up questionnaire data (n = 195).

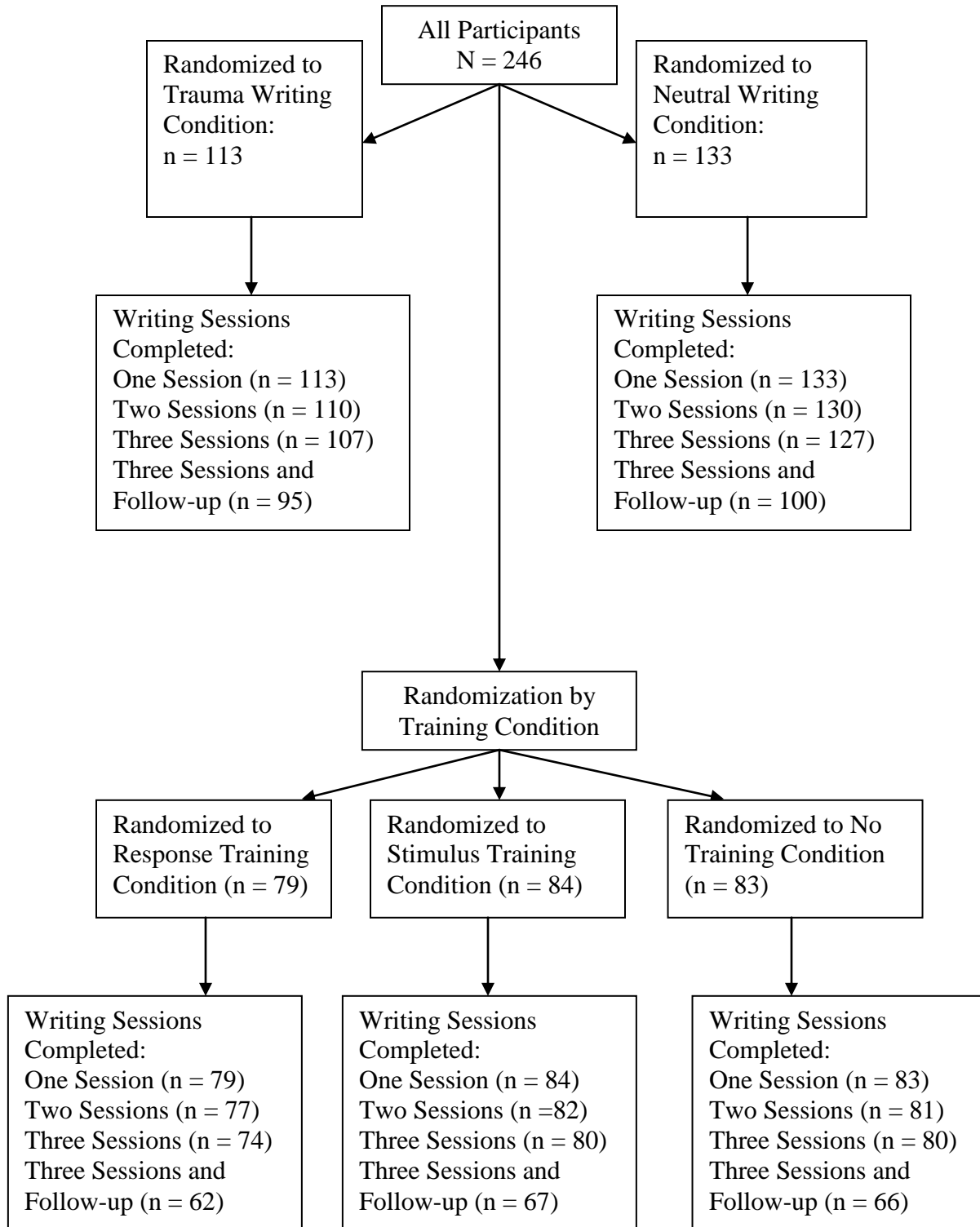


Figure 1. Attrition Rates

Preliminary Data Screening

For physiological and questionnaire data, univariate outliers, skewness and kurtosis were examined using IBM SPSS Statistics Version 21. Descriptives, frequencies, and histograms were obtained for HR, Log SC, Log RSA, PILL, CES-D, DTS-Long form and DTS-Short form data.

Univariate outliers for the physiological data were defined as any values that were less than or greater than three standard deviations from the mean. For HR, kurtosis was within acceptable limits given the sample size for both baseline and writing periods (Tabachnick & Fidell, 2007). One outlier was detected for baseline HR and was removed from the sample. No outliers were detected for HR during the writing period. Using the transformed Log SC data, kurtosis was within acceptable limits given the sample size for both baseline and writing periods (Tabachnick & Fidell, 2007). One outlier was detected for Log SC during the writing period and was removed from the sample. No outliers were found for Log SC during the baseline period. For Log RSA, kurtosis was within acceptable limits, given the sample size, and one outlier was detected, which was excluded from the final sample.

For all questionnaire data at baseline and follow-up, as well as for PTSD symptoms at post-writing sessions one and three, kurtosis was within acceptable limits considering the sample size (Tabachnick & Fidell, 2007). The questionnaire data were hand-screened for any outlying variables indicating invalid data (i.e. data entry or recording errors). No invalid data were found for any of the questionnaire data.

All regressions (Hypotheses 3-6) were screened for multivariate outliers using IBM SPSS Statistics Version 21. Residuals scatterplots were generated for each regression, and Mahalanobis distance and Cook's distance were obtained for each regression. Though a limited number of cases exceeded the recommended Mahalanobis distance, it was determined that they

represented the true tail of the data and not erroneous outlying cases, so they were left in the dataset (Tabachnik and Fidel, 2007). None of the cases exceeded a Cook's distance of 1, as recommended by Tabachnik and Fidel (2007). Thus, no multivariate outliers were excluded from the final dataset.

In summary, once the data were screened and all outliers were excluded, the final analyses contained 232 cases for baseline HR, 230 cases for HR during writing, 239 cases for baseline Log SC, 238 cases for Log SC during writing, 223 cases for Log RSA, 236 cases for baseline PILL, 190 cases for follow-up PILL, 242 cases for baseline CES-D, 193 cases for follow-up CES-D, 241 cases for baseline DTS-Long, 184 cases for follow-up DTS-Long, 242 cases for post-writing session one DTS-Short, and 222 cases for post-writing session three DTS-Short.

Baseline Data

Table 2 displays means and standard deviations for baseline HR and Log SC as well as HR and Log SC during the writing period, with outliers removed. Baseline comparisons for participants randomized to each trauma condition and each training condition were made using independent sample t-tests and one-way ANOVAs. Only one significant group difference was found. There was a statistically significant difference at the $p < .05$ level in baseline Log SC for the three training conditions: $F(2, 236) = 5.84, p = .003$. Post-hoc comparisons using the Tukey HSD test indicated that baseline Log SC for stimulus-trained participants was significantly lower than that of response-trained or no-training participants.

Table 2.

Heart Rate and Skin Conductance Means (and Standard Deviations) at Baseline and During Writing Period

	Baseline			Writing		
	Trauma	Neutral	Total	Trauma	Neutral	Total
Heart Rate (BPM)						
Response	81.20 (11.20)	78.04 (11.77)	79.75 (11.50)	86.60 (12.53)	82.18 (13.75)	84.51 (13.22)
Stimulus	75.29 (10.80)	78.62 (12.79)	77.24 (12.05)	79.11 (10.43)	82.49 (12.86)	81.11 (11.97)
No Training	75.40 (9.98)	78.12 (13.38)	76.79 (11.85)	79.23 (10.18)	81.70 (13.53)	80.51 (12.03)
Total	77.46 (10.95)	78.29 (12.61)	77.89 (11.83)	81.78 (11.60)	82.15 (13.22)	81.98 (12.46)
Log Skin Conductance (μmho)						
Response	0.293 (0.51)	0.367 (0.46)	0.329 (0.49)	0.577 (0.45)	0.536 (0.44)	0.557 (0.44)
Stimulus	0.061 (0.47)	0.145 (0.52)	0.110 (0.50)	0.318 (0.50)	0.314 (0.53)	0.315 (0.52)
No Training	0.305 (0.49)	0.365 (0.43)	0.337 (0.46)	0.506 (0.44)	0.568 (0.38)	0.540 (0.41)
Total	0.225 (0.50)	0.282 (0.48)	0.256 (0.49)	0.473 (0.47)	0.462 (0.47)	0.467 (0.47)

* Values in parentheses indicate Standard Deviations

Table 3 displays means and standard deviations for Log RSA, with outliers removed. Baseline comparisons for participants randomized to each trauma condition and each training condition were made using independent sample t-tests and one-way ANOVAs. Two marginally significant group differences were found. Neutral writers had marginally greater Log RSA than trauma writers, $t(221) = 1.79, p = .075$, and there was a marginally significant difference in Log RSA for the three response conditions: $F(2, 220) = 2.78, p = .06$. Post-hoc comparisons using the Tukey HSD test indicated that response trained participants had marginally lower Log RSA than no training participants.

Table 3.

Baseline Log RSA Means (and Standard Deviations)

Heart Rate Variability (Log RSA non-centered)	Trauma	Neutral	Total
Response	5.81 (1.18)	6.46 (1.13)	6.12 (1.19)
Stimulus	6.34 (1.11)	6.46 (1.11)	6.41 (1.10)
No Training	6.54 (0.95)	6.57 (1.21)	6.56 (1.08)
Total	6.22 (1.12)	6.49 (1.14)	6.36 (1.14)

* Values in parentheses indicate Standard Deviations

Table 4 displays means and standard deviations for all questionnaire measures at baseline and one-month follow-up. Baseline comparisons for participants randomized to each trauma condition and each training condition were made using independent sample t-tests and one-way ANOVAs. Only one significant group difference was found. Neutral writers had significantly higher depression symptoms at baseline, as measured by the CES-D, compared to trauma writers, $t(240) = 2.78, p = .006$.

Table 4.

Questionnaire Data at Baseline and at One-Month Follow-Up

	Baseline			One-Month Follow Up		
	Trauma	Neutral	Total	Trauma	Neutral	Total
PILL						
Response	57.08 (25.50)	63.68 (25.20)	60.34 (25.40)	54.71 (27.09)	58.23 (25.57)	56.47 (26.19)
Stimulus	54.75 (22.25)	59.19 (26.41)	57.41 (24.78)	45.07 (21.28)	51.64 (24.76)	48.77 (23.35)
No Training	55.72 (25.39)	51.19 (21.68)	53.25 (23.40)	50.00 (25.34)	43.88 (20.92)	46.94 (23.26)
Total	55.93 (24.32)	57.85 (24.89)	56.97 (24.60)	50.09 (24.84)	51.19 (24.30)	50.66 (24.50)
CES-D						
Response	14.36 (9.33)	16.68 (11.08)	15.51 (10.23)	13.97 (7.78)	16.40 (11.97)	15.16 (10.05)
Stimulus	11.32 (6.88)	17.88 (10.70)	15.19 (9.83)	11.00 (9.80)	16.24 (11.11)	13.90 (10.79)
No Training	13.03 (6.39)	14.02 (7.90)	13.57 (7.23)	15.09 (7.53)	12.84 (8.59)	14.02 (8.07)
Total	12.97 (7.73)	16.22 (10.02)	14.74 (9.18)	13.43 (8.48)	15.21 (10.68)	14.34 (9.68)
DTS-Long form, Severity Scale						
Response	20.23 (17.54)	22.79 (14.31)	21.49 (15.98)	12.11 (13.60)	10.30 (13.07)	11.17 (13.24)
Stimulus	14.73 (10.60)	17.00 (13.78)	16.07 (12.56)	9.18 (14.50)	9.36 (11.73)	9.28 (13.15)
No Training	21.32 (14.16)	16.47 (12.88)	18.69 (13.62)	12.24 (11.29)	11.31 (12.46)	11.81 (11.77)
Total	18.95 (14.70)	18.50 (13.81)	18.71 (14.20)	11.24 (13.19)	10.25 (12.28)	10.73 (12.71)

* Values in parentheses indicate Standard Deviations

Table 5 displays means and standard deviations for the DTS-Short form at post-writing session one and session three. No baseline comparisons were performed for this measure because this measure was not given at baseline.

Table 5.

PTSD Symptom Severity at Post-Writing Session One and Session Three

	Post-Writing Session One			Post-Writing Session Three		
	Trauma	Neutral	Total	Trauma	Neutral	Total
DTS-Short form, Severity Scale						
Response	12.69 (11.29)	3.39 (5.77)	8.10 (10.09)	7.94 (10.39)	5.06 (6.34)	6.52 (8.69)
Stimulus	12.65 (10.55)	3.10 (4.97)	7.01 (9.03)	7.06 (9.66)	3.47 (7.05)	4.93 (8.35)
No Training	15.61 (11.27)	4.93 (7.52)	9.88 (10.80)	9.88 (11.48)	2.90 (5.12)	5.97 (9.14)
Total	13.68 (11.05)	3.80 (6.16)	8.33 (10.03)	8.31 (10.51)	3.73 (6.25)	5.79 (8.72)

* Values in parentheses indicate Standard Deviations

Hypothesis 1

In hypothesis 1, it was predicted that participants with greater Log RSA would have lower baseline levels of physical illness, PTSD, and depression symptoms. These relationships were investigated using Pearson product-moment correlation coefficients, with predicted negative correlations indicating that higher Log RSA was related to fewer symptoms. No significant relationships were found. For baseline symptoms of PTSD and depression, the correlations with Log RSA were in the predicted direction, but were small and not significant. The correlation between baseline physical symptoms and Log RSA was near zero and nonsignificant (see Table 6).

Hypothesis 2

Hypothesis 2 predicted that participants with greater Log RSA would have lower resting heart rate and skin conductance. These relationships were investigated using Pearson product-moment correlation coefficients, with predicted negative correlations indicating that higher

resting Log RSA was related to lower baseline HR and Log SC. As predicted, a significant negative correlation was found between resting Log RSA and baseline HR ($r = -0.58, p < .0005$), and a marginally significant negative correlation was found between resting Log RSA and baseline Log SC ($r = -0.12, p = .08$) (see Table 6).

Table 6.

Pearson Product-Moment Correlations Between Baseline Log RSA and Baseline Measures

	<i>r</i>	<i>n</i>	<i>p</i>
DTS-LONG*	-0.10	218	0.14
CES-D	-0.07	219	0.32
PILL	0.02	213	0.77
HR	-0.58	222	<.0005 [†]
SC*	-0.12	220	0.08 [‡]

[†]Indicates $p < .05$

[‡]Indicates $p < .10$

*DTS LONG = DTS-Long Form Severity Scale; SC = log transformation of the mean skin conductance of the last 5 minutes of Session 1 baseline

Hypothesis 3

Hypothesis 3 predicted that, among trauma writers, Log RSA would moderate the effects of trauma writing at follow up for physical illness symptoms, PTSD symptom severity, and depression symptoms. Among neutral writers, Log RSA was not predicted to have a moderating role for any of the measures. For all of the outcome variables, it was expected that trauma writers with greater Log RSA would have greater symptom improvement at follow-up, compared to neutral writers. To test hypothesis 3, hierarchical multiple regressions were performed including the dependent variable's baseline symptoms in order to remove the effects of baseline symptom levels, gender, Log RSA, trauma condition, and the interaction between trauma condition and Log RSA, with the hypothesized effect being a Trauma Condition X Log RSA interaction indicating that Log RSA was indeed playing a moderating role.

No significant Trauma Condition X Log RSA interactions were found for any of the follow-up measures. Relationships among trauma condition, Log RSA, and each follow-up measure took a number of forms. Overall, trauma writers experienced modest increases in depression symptoms from baseline to one-month follow-up, while neutral writers experienced modest reductions in depressive symptoms from baseline to one-month follow-up (see Table 4). Though not significant, the pattern of modification between Log RSA and trauma writing was in the expected direction for depressive symptoms, as greater Log RSA was related to greater improvements in follow-up depression symptoms among trauma writers but not among neutral writers (Trauma Condition X Log RSA: $\beta = -.78$, $t = -0.81$, $p = .42$). Figure 2 is included to illustrate this expected pattern, although the finding is not reliable.

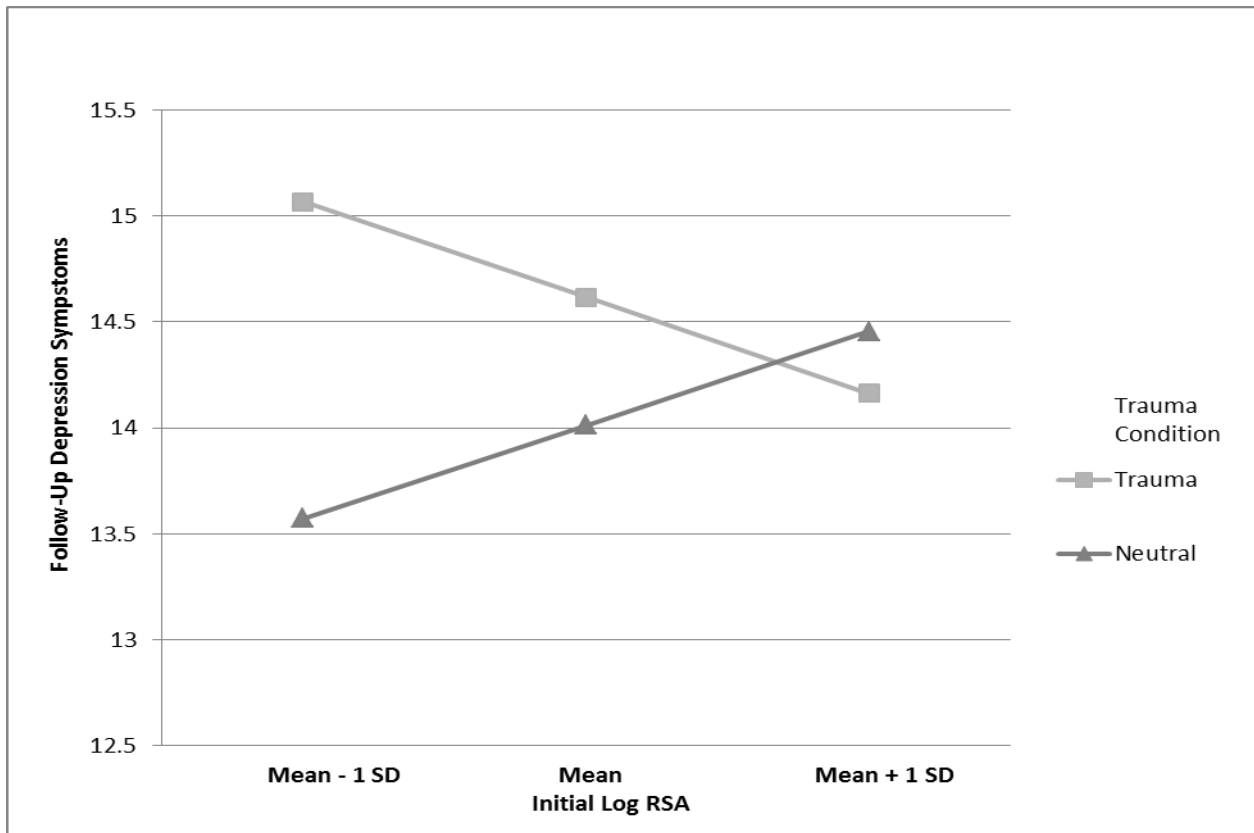


Figure 2. Moderation of the Effect of Trauma Condition on Follow-Up Depression Symptoms by Log RSA

On average, both writing groups experienced improvements in PTSD symptom severity and physical illness symptoms from baseline to one-month follow-up (see Table 4), but no hypotheses were confirmed for PTSD symptom severity (Trauma Condition X Log RSA: $\beta = -0.63$, $t = -0.41$, $p = .68$) or physical illness symptoms (Trauma Condition X Log RSA: $\beta = 0.58$, $t = 0.25$, $p = .80$) at follow-up, as no significant patterns of modification were found between Log RSA and trauma writing for these variables. For all of the follow-up measures, the baseline symptoms (physical illness, PTSD severity, and depression) were significant variables in the full regression, showing that participants' baseline symptoms were related to symptoms at one-month follow-up (see Appendix I for full regression results).

In addition to measuring change in symptoms from baseline to one-month follow-up, this study also asked participants to report on PTSD symptom severity immediately after writing in each session using the DTS-Short form Severity Scale. Hypothesis 3 also predicted that, among trauma writers, Log RSA would moderate the change in post-writing PTSD symptoms from session one to session three. Among neutral writers, Log RSA was not predicted to moderate the change in post-writing PTSD symptoms. It was expected that trauma writers with greater Log RSA would have greater post-writing PTSD symptom improvement at session three, compared to neutral writers. To test this hypothesis, a hierarchical multiple regression was performed including the variables post-writing PTSD symptoms at session one, gender, Log RSA, trauma condition, and the interaction between trauma condition and Log RSA, with the hypothesized effect being a Trauma Condition X Log RSA interaction indicating that Log RSA was indeed playing a moderating role.

A marginally significant interaction between Log RSA and trauma was found, with the pattern of interaction in the expected direction. While both writing groups showed

improvements in post-writing PTSD symptom severity from session one to session three (see Table 5), trauma writers showed greater reductions in post-writing PTSD symptom severity at session three as Log RSA increased, while neutral writers showed somewhat less improvement in post-writing PTSD symptom severity at session three as Log RSA increased (Trauma Condition X Log RSA: $\beta = -1.50$, $t = -1.74$, $p = .08$). Figure 3 illustrates this interaction. Post-writing PTSD symptom severity at session one was a significant variable in the full regression, showing that participants' post-writing PTSD symptoms severity at session one was related to post-writing PTSD symptom severity at session three (see Appendix I for full regression results).

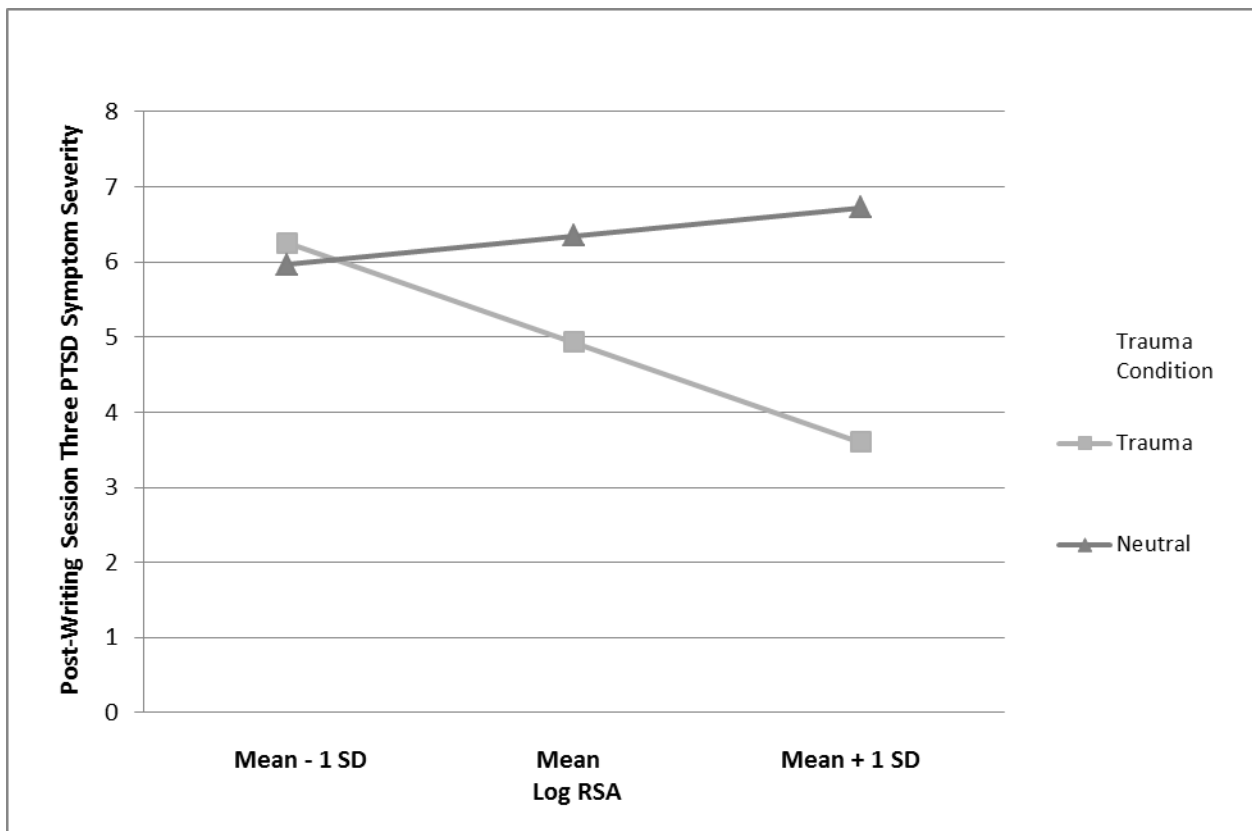


Figure 3. Moderation of the Effect of Trauma Condition on Post-Writing Session Three PTSD Symptom Severity by Log RSA

Hypothesis 4

Hypothesis 4 predicted that, among trauma writers, participants who displayed greater Log RSA would also display appropriately elevated heart rate and skin conductance reactivity in response to the first trauma writing session, while neutral writers would not show this increase in physiological reactivity in response to the first writing session. To test hypothesis 4, hierarchical multiple regressions were performed including the variables baseline HR or Log SC, gender, Log RSA, trauma condition, and the interaction between trauma condition and Log RSA, with the hypothesized effect being a Trauma Condition X Log RSA interaction indicating that Log RSA was indeed playing a moderating role.

Though, on average, both writing groups experienced enhanced HR and Log SC reactivity during the writing period compared to the baseline period (see Table 2), no significant Trauma Condition X Log RSA interactions were found for HR reactivity ($\beta = -.23, t = -.37, p = .71$) or Log SC reactivity ($\beta = .009, t = .31, p = .76$). Baseline HR and Log SC were significant variables in the full regressions, indicating that baseline HR or Log SC levels were related to HR or Log SC during the session one writing task (see Appendix J for full regression results).

Hypothesis 5

Hypothesis 5 analyses were restricted to only trauma writers. It was hypothesized that response-trained trauma writers with greater Log RSA would show the greatest improvements in physical illness, PTSD severity, and depression symptoms at one-month follow-up compared to trauma writers in all other training groups (stimulus and no training). To test hypothesis 5, hierarchical multiple regressions were performed including the variables baseline symptoms, gender, Log RSA, response training (dummy-coded), stimulus training (dummy coded), the interaction between response training and Log RSA, and the interaction between stimulus

training and Log RSA. Only trauma writers were included in these analyses, with the hypothesized effect being a Response X Log RSA interaction.

No significant Response X Log RSA interactions were found for any of the measures. For physical symptoms (PILL), effects followed the predicted pattern but did not meet statistical significance. On average, all trauma writing groups experienced reductions in physical illness symptoms from baseline to one-month follow-up (see Table 4). Response-trained trauma writers showed modest reductions in physical illness symptoms at follow up as Log RSA increased, while stimulus and no training trauma writers showed less improvement in physical illness symptoms at follow up as Log RSA increased (Response X Log RSA: $\beta = -6.55$, $t = -1.52$, $p = .13$). Figure 4 is included to illustrate this expected pattern, although the finding is not reliable. Baseline physical symptoms were a significant variable in the full regression model, indicating that there was a relationship between physical illness symptoms at baseline and at follow-up (see Appendix K for full regression results).

The response-trained and stimulus-trained trauma writing groups showed modest reductions in depressive symptoms (CES-D) from baseline to one-month follow-up, while the no training trauma writing group showed increased depressive symptoms from baseline to one-month follow-up (see Table 4). No significant Response X Log RSA interaction was present ($\beta = -1.50$, $t = -.92$, $p = .36$). Baseline depression symptoms were a significant variable in the full regression model, indicating that baseline depression symptoms were related to depression symptoms at follow-up (see Appendix K for full regression results).

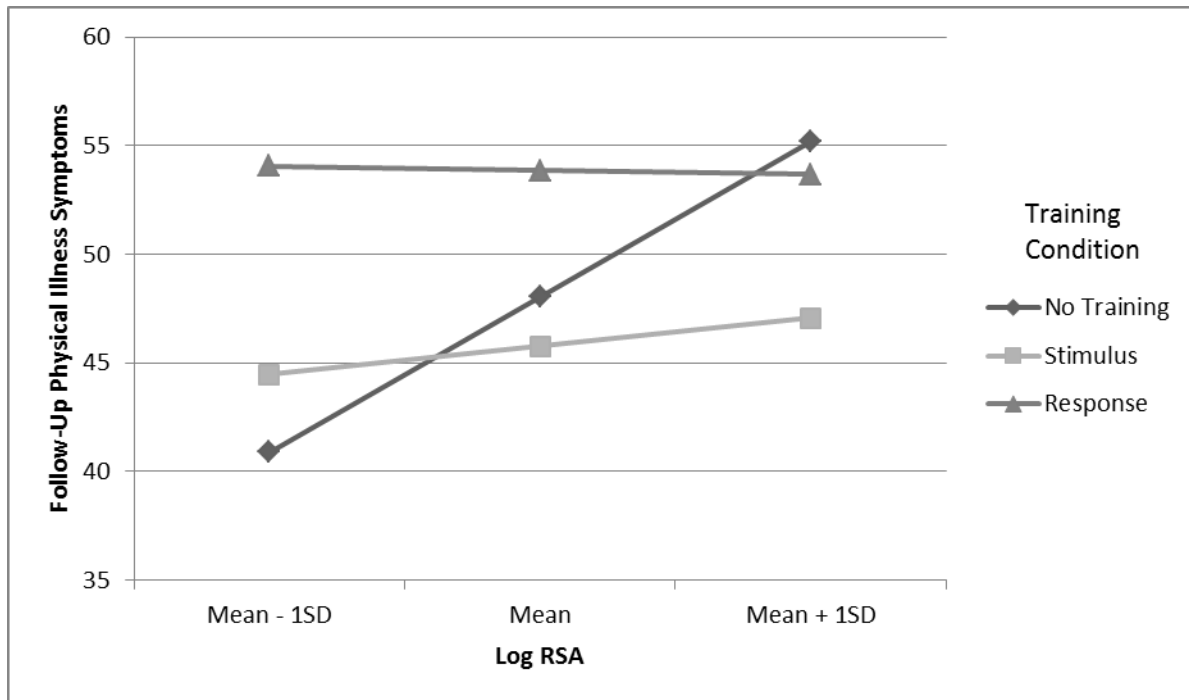


Figure 4. Moderation of the Effect of Response Condition on Follow-Up Physical Illness Symptoms by Log RSA for Trauma Writers Only

On average, all trauma writing groups showed reductions in PTSD symptoms (DTS Long) from baseline to one-month follow up (see Table 4), but no significant Response X Log RSA interaction was present ($\beta = -1.61, t = -.57, p = .57$). The only significant variable in the full regression was baseline PTSD symptoms, indicating a relationship between PTSD symptoms at baseline and at one-month follow-up (see Appendix K for full regression results).

In addition to measuring change in symptoms from baseline to one-month follow-up, participants also reported on PTSD symptom severity immediately after writing in each session using the DTS-Short form Severity Scale. Hypothesis 5 also predicted that response-trained trauma writers with greater Log RSA would report the greatest improvement in post-writing PTSD symptom severity from session one to session three, compared to stimulus-trained or no training trauma writers. To test this hypothesis, a hierarchical multiple regression was performed

including the variables post-writing PTSD symptom severity at session one, gender, Log RSA, response condition, stimulus condition, the interaction between response condition and Log RSA, and the interaction between stimulus condition and Log RSA. Only trauma writers were included in this analysis, with the hypothesized effect being a Response X Log RSA interaction.

On average, all trauma writing groups showed reductions in PTSD symptoms (DTS-Short) from post-writing session one to post-writing session three (see Table 5), but no significant Response X Log RSA interaction was present ($\beta = .90, t = 0.45, p = .66$). Post-writing PTSD symptom severity at session one was the only significant variable in the full regression, indicating that post-writing session one PTSD symptom severity is related to post-writing session three PTSD symptom severity (see Appendix K for full regression results).

Hypothesis 6

Hypothesis 6 analyses were restricted to only trauma writers. It was hypothesized that response-trained trauma writers with greater Log RSA would have the most elevated HR and Log SC reactivity in response to the first trauma writing session, compared to stimulus-trained or no training trauma writers. To test hypothesis 6, hierarchical multiple regressions were performed, including the variables baseline HR or Log SC, gender, Log RSA, response training (dummy-coded), stimulus training (dummy coded), the interaction between response training and Log RSA, and the interaction between stimulus training and Log RSA. Only trauma writers were included in these analyses, with the hypothesized effect being a Response X Log RSA interaction.

Although all trauma writing groups showed enhanced HR and Log SC reactivity from baseline to the writing period (see Table 2), no significant Response X Log RSA interactions were found for HR reactivity ($\beta = 0.24, t = .22, p = .83$) or SC reactivity ($\beta = -0.004, t = -.06, p =$

.96). For HR reactivity, a marginally significant main effect was present for response condition, indicating that response-trained trauma writers had greater HR increases compared to stimulus-trained or no training trauma writers ($\beta = 2.14, t = 1.84, p = .07$). Baseline HR and Log SC were significant variables in the full regressions, indicating that baseline HR or Log SC levels were related to HR or Log SC reactivity during the writing task (see Appendix L for full regression results).

Discussion

The objectives of this study were to: 1) investigate the relationship between resting RSA (an index of emotion regulation) and symptoms of depression, PTSD and physical illness, as well as baseline physiological levels; 2) determine how RSA may moderate the effects of trauma writing and training protocol (response, stimulus, or none) on symptoms of physical illness, depression and PTSD; and 3) explore the expectation that higher resting levels of emotion regulation will be associated with elevated heart rate and skin conductance reactivity in response to trauma writing during the first writing session.

In this study, no relationship was found between emotion regulation and pre-writing symptoms of depression, PTSD, and physical illness. The anticipated relationship was found between a greater degree of emotion regulation and lower baseline levels of HR and SC. Emotion regulation moderated the effects of trauma writing on change in PTSD symptoms immediately after writing. However, emotion regulation did not moderate the effects of trauma writing or the training protocol on symptoms of physical illness, depression, or PTSD at the one-month follow-up, or on HR and SC during the first writing period. Discussion of the results will be organized around the specific hypotheses of this study.

Hypothesis 1

Though the hypothesized relationships between resting RSA and baseline symptoms of PTSD, depression, and physical illness were not statistically significant, relationships between RSA and depression and RSA and PTSD were in the expected direction, with greater resting RSA, an index for emotional regulation, being associated with lower baseline PTSD and depression symptoms. Four previous studies have found that participants with a clinical diagnosis of PTSD had lower resting heart rate variability than healthy controls (Blechert et al., 2007; Cohen et al., 1997; Cohen et al., 1998; Cohen et al., 2000). The current study employed a sample of nonclinical college students and it may be that there was not enough of a range of PTSD symptoms in the sample to find a relationship with RSA. In normative studies, the mean score on the DTS-Long form (which was used to assess PTSD symptoms in this study) for people with PTSD was 62 ($SD = 38.0$) compared to a mean score of 15.5 ($SD = 13.8$) for people without PTSD (Davidson et al., 1997). In the current study, the mean DTS-Long form score at baseline was 36.9 ($SD = 26.4$), with 44 participants out of 241 obtaining a total score of 62 or above. Thus, the range of PTSD symptoms among participants in the current sample may not have been sufficient to provide robust findings, though the relationship was in the expected direction.

Traditionally, it has been thought that RSA and depression symptoms are inversely related. This line of thinking has largely grown out of meta-analyses by Kemp and colleagues (2010) and Rottenberg (2007), which looked at studies comparing the heart rate variability of healthy control groups to groups with major depressive disorder and found that the depressed groups had lower heart rate variability than healthy control groups. The current study, however, consisted of a nonclinical sample of college students, and as was the case with PTSD symptoms, may not have had a great enough range of depressive symptoms to find a relationship with RSA.

Three other studies were found that examined the relationship between RSA and symptoms of depression and included at least some nonclinical participants (Fagundes et al., 2012; Hopp et al, 2013; Yaroslavsky et al., 2013). Fagundes and colleagues' (2012) sample consisted of healthy 14-year-old children, and resting RSA was not significantly related to scores on the CES-D ($r = .02$; $p > .05$). Yaroslavsky and colleagues' (2013) all-female sample consisted of both healthy controls and adults who had childhood-onset depressive disorder. No differences in resting RSA were found between the healthy controls and the participants with childhood-onset depression, and the entire sample's resting RSA was not significantly related to scores on the Beck Depression Inventory ($r = .09$; $p > .05$). Hopp and colleagues (2013) studied a community sample of adults who reported a stressful event within the past eight weeks. These adults were somewhat older (mean age = 40.1 years) than the college-aged students in the current study. Hopp and colleagues found that greater baseline RSA corresponded with lower baseline depression levels, as measured with the Beck Depression Inventory ($r = -.20$, $p < .01$).

The current study's correlation of $r = -.07$ between depressive symptoms and RSA sits just about at the midpoint among the findings of these three other studies. Thus, it would appear that the current study's findings are very comparable to other studies in which the population is not strictly clinical (i.e. did not have major depressive disorder), and that the relationship between resting RSA and depression may only hold when clinically-depressed samples are examined.

In the current study, higher resting RSA did not have any relationship with physical illness symptoms. This is somewhat surprising, as past reviews have found that lower heart rate variability is associated with poorer health outcomes (Haensel et al., 2008; Thayer & Lane, 2007). For example, a review by Thayer and Lane (2007) found that, independent of other risk

factors, decreased vagal function is associated with all-cause mortality. In the same review, decreased vagal function was also associated with cardiovascular disease and inflammation. A closer examination of the review, however, reveals that not all studies used heart rate variability as a measurement of vagal function, and many of the included studies were composed of samples that had been selected for their history of cardiovascular risk factors, such as history of myocardial infarction, obesity or hypertension (Thayer and Lane, 2007).

The review by Haensel and colleagues (2008) investigated the relationship between heart rate variability and inflammation, conducting separate analyses for healthy samples and samples with various health conditions. Within Haensel's review, four studies were composed of healthy samples (Kon et al., 2006; Owen & Steptoe, 2003; Sajadieh et al., 2004; and Sloan, McCreath, et al., 2007), thus more closely mirroring the current study's sample. Participants in these four studies were older than the participants in the current study, and findings were mixed. Two studies found that lower heart rate variability was related to more inflammation markers (Sajadieh et al., 2004; Sloan, McCreath, et al., 2007), one study found no relationship between heart rate variability and inflammation (Owen and Steptoe, 2003), and the other study found a relationship between heart rate variability and inflammation only for participants whose heart rate variability was within the lowest quartile of the sample (Kon et al., 2006). In their review of vagal changes across the lifespan, De Meersman and Stein (2007) found that increased age is associated with decreased heart rate variability, and older people also tend to have higher levels of physical symptoms than younger people. Given that the current study, composed of younger individuals unselected for physical disorders, found no relationship between RSA and physical illness symptoms, more research needs to occur to disentangle the relationships among physical illness, RSA, and age.

Hypothesis 2

As predicted, greater resting levels of RSA corresponded with lower skin conductance and heart rate levels at baseline. The finding that RSA corresponds with lower HR is rather robust. In designing and testing the CMetX software, Allen and colleagues (2007) found that greater resting RSA was significantly correlated with lower resting HR ($r = -.61, p < .05$), a finding replicated by Hibbert and colleagues (2012) ($r = -.47, p < .05$) and consistent with the correlation of $r = -.50$ found in this study. However, far less research exists that investigates the relationship between HRV and SC, and no studies could be found that directly correlated resting HRV and resting SC for adults. One study investigated the relationship between baseline RSA and baseline SC for a nonclinical sample of 14-year-olds, and found no relationship between them ($r = .13; p > .05$) (Diamond et al., 2012). SC is generally thought to be an index of sympathetic nervous system activity (Boucsein, 1992), which is why an inverse relationship between resting HRV (which is strongly related to parasympathetic activity) and SC was anticipated in the current study.

Hypothesis 3

This hypothesis predicted that increased emotion regulation would be associated with greater reduction in PTSD, depression, and physical illness symptoms after trauma writing. As predicted, greater emotion regulation, as assessed by RSA, was associated with a greater reduction in posttraumatic symptoms at the end of session 3 compared to the end of session 1. Trauma writers were asked to write about their most upsetting event with as much emotion and feeling as possible (Pennebaker, 1997), and it was thought that trauma writers with higher resting RSA, and by inference a greater degree of emotional flexibility, would have more enhanced, appropriate reactivity to the task. “Appropriate” reactivity at the first trauma writing session

would likely entail an enhancement of negative emotions, given the writing content. By the third writing session, however, the “appropriate” response to trauma writing was expected to be habituation of negative emotional symptomatology – in this case, a reduction in posttraumatic symptoms.

In the current study, participants with higher RSA (better emotion regulation) appeared to make more effective use of the emotions elicited in the context of the written disclosure and experienced reductions in PTSD symptoms by the final writing session, whereas participants with lower RSA (poorer emotion regulation) did not benefit to the same extent from trauma writing. Unlike trauma writing, neutral writing did not elicit traumatic memories, so the ability to regulate emotion was not expected to affect PTSD symptoms, and in fact there was no relationship between RSA and reduction in posttraumatic symptoms for neutral writers.

The results described above are based on the DTS-Short form, which was administered immediately following each writing session. However, emotion regulation did not moderate the effect of trauma writing on symptoms of physical illness, depression, or PTSD at the one-month follow-up assessment. Only two other studies have investigated the moderating role of RSA on psychological and physical health outcomes following trauma and neutral writing. The first study, by Sloan and Epstein (2005), employed a nonclinical college student sample, much like the current study, and found that RSA moderated the effect of the trauma writing intervention on symptoms of physical health and depression collected one month later, but not one-month follow-up symptoms of anxiety or stress. However, Sloan and Epstein investigated the moderating effect of RSA collected while participants were writing about their trauma during the first session, not baseline RSA as in the current study. Sloan and Epstein’s use of RSA involves

a response to trauma writing, rather than a resting baseline, and so the two studies are not comparable.

The second study, by O'Connor and colleagues (2005), was comprised of mostly female participants experiencing bereavement and only had one outcome variable – depression symptoms. They found that trauma writers with greater emotion regulation experienced greater improvements on follow-up symptoms of depression than did trauma writers with less emotion regulation. Neutral writers did not differ on follow-up depressive symptoms based on their ability to regulate emotion – not surprising given the fact that the neutral writing condition was not expected to elicit a great deal of emotion. Given the methodological differences in the Sloan and Epstein study (2005) and the differences in sample composition in the O'Connor study (2005), further research with nonclinical samples using tonic RSA as a moderator is needed to see if the current study's findings are reliable.

Recent research has suggested that both resting RSA and RSA reactivity be investigated within the same study to obtain an optimal index of emotion regulation. Yaroslavsky and colleagues (2013) found that, among adults with childhood-onset depressive disorder histories, the combination of high resting RSA and RSA withdrawal while watching a sad film was predictive of depression levels, but neither resting RSA nor RSA reactivity was predictive of depression levels on its own. Yaroslavsky and colleagues (2013) defined the “optimal” RSA pattern, predictive of low depression, as consisting of both high resting RSA and RSA withdrawal in the face of negative mood triggers. “Suboptimal” RSA patterns include low resting RSA coupled with RSA withdrawal to negative mood triggers, as well as high resting RSA coupled with RSA augmentation in response to negative mood triggers. Thus future studies

should investigate RSA reactivity as well as resting RSA in order to best characterize the emotional regulation capacities of their participants.

Hypothesis 4

Emotion regulation was not associated with HR or SC during writing for either trauma or neutral writers. To date, no studies have investigated the potential moderating role of emotion regulation on physiological response during trauma writing. Past research has shown that people with higher resting RSA demonstrate greater physiological responses to emotional stimuli. For example, women with higher resting heart rate variability showed greater startle responses to unpleasant pictures and greater startle inhibition to pleasant pictures than women with lower resting heart rate variability (Ruiz-Padial, Sollers, Vila, & Thayer, 2003). Thus, it was hypothesized that participants in the current study with higher RSA (better emotion regulation) would experience greater HR and SC in response to trauma writing than participants with lower RSA (poorer emotion regulation). However, no evidence was found for this hypothesis.

Hypothesis 5

Emotion regulation did not influence the effect of response-training on one-month follow-up symptoms of physical illness, depression, or PTSD, or the effects of response-training on change in post-writing PTSD symptoms from the first to the third session. No prior studies have investigated the potential moderating effect of emotion regulation on the psychological and physical health outcomes of response training.

Past research has shown that people with greater physiological reactivity when confronted with personally-relevant fear or trauma cues experience beneficial physical and mental health outcomes (Beckham et al., 1990; Epstein et al., 2005; Foa & Kozak, 1986; Lang et al., 1970; Sloan et al., 2005). The response training protocol was specifically designed by Lang

and colleagues to enhance physiological reactivity in response to imagery exposure. The neurovisceral integration model, as explained by Thayer and colleagues (2012), views higher resting RSA as indicative of better overall flexibility and regulation in a variety of domains, including emotion and physiology. Thus it was anticipated that, among trauma writers, higher RSA coupled with response training would greatly enhance physiological reactivity, resulting in the greatest reductions in symptoms of physical illness, depression, and PTSD at one-month follow-up.

Given these initial expectations, it is interesting that the only outcome for which the relationship even weakly followed the expected pattern was for physical illness symptoms. Response training is focused on eliciting appropriate physiological reactivity by facilitating access to motor programs. Physical health symptoms may be more directly connected to such physiological reactivity than symptoms of depression or PTSD, which, despite having a physiological component, are more psychological in nature.

It is also important to consider that this study was comprised of a nonclinical sample of college students. In this study, trauma writers were asked to identify the most upsetting event of their lives and write about it. As this was a nonclinical sample, individuals with better emotion regulation skills may have already more effectively processed their traumatic events than their counterparts with less effective emotion regulation abilities, thus limiting the potential benefit they could glean from trauma writing or response training. To disentangle these effects, future studies could pre-screen samples to include only participants with stressors that are currently bothersome to them, or use clinical samples.

Hypothesis 6

Because high levels of resting RSA are indicative of better overall flexibility, including emotional and physiological flexibility, it was originally hypothesized that trauma writers with greater baseline RSA would be able to most effectively use the response training, thus experiencing a greater increase in HR and SC compared to the stimulus or no training groups. However, differences in emotion regulation had no influence on the effects of response training on HR or SC reactivity during writing. This is the first study to investigate the potential moderating effect of emotion regulation on physiological reactivity during trauma writing, so explanations are largely speculative at this point. As was the case with Hypothesis 5, participants with better emotion regulation may have already processed their most upsetting events in healthier ways than participants with reduced emotion regulation, thus limiting the extent to which participants with the highest levels of HRV can actually benefit from response training.

Limitations and Future Directions

There were some limitations to the current study. The current sample was composed of nonselected, nonclinical college students. Though previous research indicates that 84% of college students have experienced a significant traumatic event (Vrana & Lauterbach, 1994), the range and intensity of symptoms in an unselected college sample are likely restricted compared to samples that included participants with more severe levels of trauma-related symptoms. As a result, the current sample's self-reported scores on measures of PTSD tended to be low. Though Tabachnik and Fidell (2007) advise that having a large sample lessens the impact of skewness, they caution that running parametric tests on skewed data can lessen the chances of finding significant effects. Use of a nonselected college student sample also limits generalizability of findings to clinical samples.

There is also some question regarding the effect of the diaphragmatic breathing training on RSA. If participants in a study are asked to consciously try to control breathing, the mental effort involved may actually end up reducing HRV (Berntson et al., 1997). In this study, all participants were briefly trained in diaphragmatic breathing prior to the baseline period and were encouraged to use diaphragmatic breathing during the baseline period, but were told they could breathe normally if they preferred. The researchers in this study did not follow up with participants to see if they had indeed used the diaphragmatic breathing technique or had breathed normally, which could have affected baseline RSA. Additionally, there is controversy in the RSA literature regarding whether or not respiration should be measured in addition to RSA, with some researchers in favor of measuring both processes concurrently (Berntson et al., 1997) and others asserting that concurrent measurement of respiration is not required (Denver et al., 2007). In this study, respiration was neither controlled nor measured.

While a large body of research supports the idea that trauma writing has a beneficial effect on physical and psychological symptoms (see review by Frattaroli, 2006), not everyone benefits from trauma writing (Batten et al., 2002; Kloss & Lisman, 2002; Stroebe et al., 2002) and some people even experience adverse effects (Gidron et al., 1996). There has been speculation that RSA (an index for emotion regulation) moderates trauma writing outcomes (O'Connor et al., 2005; Sloan & Epstein, 2005). The current study found that resting RSA did moderate the effects of trauma writing on PTSD symptoms at post-writing session three. The study also investigated the relationships between resting RSA and baseline physical, physiological, and psychological symptoms in a healthy population, finding that better emotion regulation was related to lower levels of resting HR and SC. Finally, this study is the first of its kind to investigate resting RSA as a potential moderator of response training outcomes. Because

trauma writing is increasingly being used as a treatment for PTSD, future research is recommended that investigates whether RSA plays a moderating role on trauma writing outcomes in clinical samples.

List of References

List of References

- Allen, J.J.B., Chambers, A.S., & Towers, D.N. (2007). The many metrics of cardiac chronotropy: A pragmatic primer and a brief comparison of metrics. *Biological Psychology*, *74*, 243-262. doi: 10.1016/j.biopsycho.2006.08.005
- Batten, S.V., Follette, V.M., Hall, M.L.R., & Palm K.M. (2002). Physical and psychological effects of written disclosure among sexual abuse survivors. *Behavior Therapy*, *33*, 107-122. doi: 10.1016/S0005-7894(02)80008-9
- Beckham, J.C., Vrana, S.R., May, J.G., Gustafson, D.J., & Smith, G.R. (1990). Emotional processing and fear measurement synchrony as indicators of treatment outcome in fear of flying. *Journal of Behavior Therapy and Experimental Psychiatry*, *21*, 153-162. doi: 10.1016/0005-7916(90)90002-3
- Berntson, G.G., Bigger, J.T., Eckberg, D.L., Grossman, P., Kaufmann, P.G., Malik, M.,... Van der Molen, M.W. (1997). Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiology*, *34*, 623-648. doi: 10.1111/j.1469-8986.1997.tb02140.x
- Berntson, G.G., & Stowell, J.R. (1998). ECG artifacts and heart period variability: Don't miss a beat! *Psychophysiology*, *35*, 127-132. doi: 10.1111/1469-8986.3510127
- Blechert, J., Michael, T., Grossman, P., Lajtman, M., & Wilhelm, F. H. (2007). Autonomic and respiratory characteristics of posttraumatic stress disorder and panic disorder. *Psychosomatic Medicine*, *69*, 935-943. doi: 10.1097/2FPSY.0b013e31815a8f6b
- Booth, R.J., Petrie, K.J., & Pennebaker, J.W. (1997). Changes in circulating lymphocyte numbers following emotional disclosure: Evidence or buffering? *Stress Medicine*, *13*, 23-29. doi: 10.1002/(SICI)1099-1700(199701)13:1<23::AID-SMI714>3.0.CO;2-E
- Boucsein, Wolfram. (1992). *Electrodermal Activity*. New York: Plenum Press.
- Bradley, M. M., & Lang, P. J. (1994). Measuring emotion: The self-assessment manikin and the semantic differential. *Journal of Behavior Therapy and Experimental Psychology*, *25*, 49-59. doi: 10.1016/0005-7916(94)90063-9
- Bradley, M. M., Greenwald, M. K., Petry, M. C., & Lang, P. J. (1992). Remembering pictures:

- Pleasure and arousal in memory. *American Psychological Association*, 18, 379-390. doi: 10.1037/0278-7393.18.2.379.
- Brown, E.J., & Heimberg, R.G. (2001). Effects of Writing about Rape: Evaluating Pennebaker's Paradigm with a Severe Trauma. *Journal of Traumatic Stress*, 14, 781- 790. doi: 10.1023/A:1013098307063
- Choi, J., Hong, S., Nelesen, R., Bardwell, W.A., Natarajan, L., Schubert, C., & Dimsdale, J.E. (2006). Age and ethnicity differences in short-term heart rate variability. *Psychosomatic Medicine*, 68, 421-426. doi: 10.1097/01.psy.0000221378.09239.6a
- Cohen, H., Benjamin, J., Geva, A.B., Matar, M.A., Kaplan, Z., & Kotler, M. (2000). Autonomic dysregulation in panic disorder and in post-traumatic stress disorder: application of power spectrum analysis of heart rate variability at rest and in response to recollection of trauma or panic attacks. *Psychiatry Research*, 96, 1-13. doi: 10.1016/S0165-1781(00)00195-5
- Cohen, H., Kotler, M., Matar, M.A., Kaplan, Z., Loewenthal, U., Miodownik, H., & Cassuto, Y. (1998). Analysis of heart rate variability in posttraumatic stress disorder patients in response to a trauma-related reminder. *Biological Psychiatry*, 44, 1054-1059. doi: 10.1016/S0006-3223(97)00475-7
- Cohen, H., Kotler, M., Matar, M.A., Kaplan, Z., Miodownik, H., & Cassuto, Y. (1997). Power spectral analysis of heart rate variability in posttraumatic stress disorder patients. *Biological Psychiatry*, 41, 627-629. doi: 10.1016/S0006-3223(96)00525-2
- Cohen, J., Cohen, P., West, S.G., & Aiken, L.S. (2003). *Applied multiple regression/correlation analysis for the Behavioral sciences*. New Jersey: Erlbaum Associates.
- Davidson, J.R.T. (1996). *Davidson Trauma Scale*. Ontario, Canada: Multi-Health Systems.
- Davidson, J.R.T., Book, S.W., Colket, J.T., Tupler, L.A., Roth, S., & David, D., ... Feldman, M.E. (1997). Assessment of a new self-rating scale for post-traumatic stress disorder. *Psychological Medicine*, 27, 153-160. doi: 10.1017/S0033291796004229
- Denver, J.W., Reed, S.F., Porges, S. W. (2007). Methodological issues in the quantification of respiratory sinus arrhythmia. *Biological Psychology*, 74, 286-294. doi:10.1016/j.biopsycho.2005.09.005
- De Meersman, R.E., & Stein, P.K. (2007). Vagal modulation and aging. *Biological Psychiatry*, 74, 165-173. doi:10.1016/j.biopsycho.2006.04.008
- Diamond, L.M., Fagundes, C.P., & Cribbet, M.R. (2012). Individual differences in adolescents' sympathetic and parasympathetic functioning moderate associations between family environment and psychosocial adjustment. *Developmental Psychology*, 48, 918-931. doi: 10.1037/a0026901

- Epstein, E.M., Sloan, D.M., & Marx, B.P. (2005). Getting to the heart of the matter: Written disclosure, gender, and heart rate. *Psychosomatic Medicine*, *67*, 413-419. doi: 10.1097/01.psy.0000160474.82170.7b
- Fagundes, C.P., Diamond, L.M., & Allen, K.P. (2012). Adolescent attachment insecurity and parasympathetic functioning predict future loss adjustment. *Personality and Social Psychology Bulletin*, *38*, 821-832. doi: 10.1177/0146167212437429
- Fairclough, S.H., & Mulder, J.M. (2012). Psychophysiological processes of mental effort investment. In: R.A. Wright, & G.H.E. Gendolla (Eds.) *How Motivation Affects Cardiovascular Response* (pp. 61-76). American Psychological Association: Washington, D.C.
- Foa, E.B., & Kozak, M.J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin*, *99*, 20-35. doi: 10.1037/0033-2909.99.1.20
- Frattaroli, J. (2006). Experimental disclosure and its moderators: A meta-analysis. *Psychological Bulletin*, *6*, 823-865. doi: 10.1037/0033-2909.132.6.823
- Friedman, B.H. (2007). An autonomic-flexibility – neurovisceral integration model of anxiety and cardiac vagal tone. *Biological Psychology*, *74*, 185-199. doi: 10.1016/j.biopsycho.2005.08.009
- Friedman, B.H., & Thayer, J.F. (1998). Autonomic balance revisited: Panic anxiety and heart rate variability. *Journal of Psychosomatic Research*, *44*, 133-151. doi: 10.1016/S0022-3999(97)00202-X
- Gidron, Y., Peri, T., Connolly, J.F., & Shalev, A.Y. (1996). Written disclosure in posttraumatic stress disorder: Is it beneficial for the patient? *Journal of Nervous and Mental Disease*, *184*, 505-507. doi: 10.1097/00005053-199608000-00009
- Greenberg, M.A., Wortman, C.B., & Stone, A.A. (1996). Emotional expression and physical health: Revising traumatic memories or fostering self-regulation? *Journal of Personality and Social Psychology*, *71*, 588-602. doi: 10.1037/0022-3514.71.3.588
- Haensel, A., Mills, P.J., Nelesen, R. A., Ziegler, M.G., Dimsdale, J.E. (2008). The relationship between heart rate variability and inflammatory markers in cardiovascular diseases. *Psychoneuroendocrinology*, *33*, 1305-1312. doi: 10.1016/j.psyneuen.2008.08.007
- Henry, B.L., Minassian, A., Paulus, M.P., Geyer, M.A., & Perry, W. (2010). Heart rate variability in bipolar mania and schizophrenia. *Journal of Psychiatric Research*, *44*, 168-176. doi: 10.1016/j.jpsychires.2009.07.011
- Hibbert, A.S., Weinberg, A., & Klonsky, D.E. (2012). Field validity of heart rate variability metrics provided by QRSTool and CMetX. *Psychological Assessment*, *24*, 777-782. doi: 10.1037/a0027284

- Hopp, H., Shallcross, A., Ford, B.Q., Troy, A.S., Wilhelm, F.H., & Mauss, I.B. (2013). High cardiac vagal control protects against future depressive symptoms under conditions of high social support. *Biological Psychology*, *93*, 143-149. doi: 10.1016/j.biopsycho.2013.01.004
- Kemp, A.H., Quintana, D.S., Gray, M.A., Felmingham, K.L., Brown, K., & Gatt, J.M. (2010). Impact of depression and antidepressant treatment on heart rate variability: A review and meta-analysis. *Biological Psychiatry*, *67*, 1067-1074. doi: 10.1016/j.biopsych.2009.12.012
- Kloss, J.D., & Lisman, S.A. (2002). An exposure-based examination of the effects of written emotional disclosure. *British Journal of Health Psychology*, *7*, 31-46. doi: 10.1348/135910702169349
- Knight, R.G., Williams, S., McGee, R., & Olaman, S. (1997). Psychometric properties of the Center for Epidemiologic Studies Depression Scale (CES-D) in a sample of women in middle life. *Behavior Research & Therapy*, *35*(4), 373-380. doi: 10.1016/S0005-7967(96)00107
- Kon, H., Nagano, M., Tanaka, F., Satoh, K., Segawa, T., Nakamura, M. (2006). Association of decreased variation of R-R interval and elevated serum c-reactive protein level in a general population in Japan. *International Heart Journal*, *47*, 867-876. doi: 10.1536/ihj.47.867
- Lang, P.J. (1977). Imagery in therapy: An information processing analysis of fear. *Behavior Therapy*, *8*, 862-886. doi: 10.1016/S0005-7894(77)80157-3
- Lang, P.J. (1979). A bio-informational theory of emotional imagery. *Psychophysiology*, *16*, 495-512. doi: 10.1111/j.1469-8986.1979.tb01511.x
- Lang, P.J. (1984). Cognition in emotion: concept and action. In: C.E. Izard, J. Kagan, R.B. Zajonc (Eds.). *Emotions, Cognition and Behavior* (pp. 192-226). Cambridge University Press.
- Lang, P.J., Kozak, M.J., Miller, G.A., Levin, D.N., & McLean, Jr., A. (1980). Emotional imagery: Conceptual structure and pattern of somato-visceral response. *Psychophysiology*, *17*, 179-92. doi: 10.1111/j.1469-8986.1980.tb00133.x
- Lang, P.J., Levin, D.N., Miller, G.A., & Kozak, M.J. (1983). Fear behavior, fear imagery and the psychophysiology of emotion: The problem of affective response integration. *Journal of Abnormal Psychology*, *92*, 276-306. doi: 10.1037/0021-843X.92.3.276
- Lang, P.J., Melamed, B.G., & Hart, J. (1970). A psychophysiological analysis of fear modification using an automated desensitization procedure. *Journal of Abnormal Psychology*, *76*, 220-234. doi: 10.1037/h0029875

- Lewis, G.F., Furman, S.A., McCool, M.F., Porges, S.W. (2012). Statistical strategies to quantify respiratory sinus arrhythmia: Are commonly used metrics equivalent? *Biological Psychology*, 89, 349–364. doi: 10.1016/j.biopsycho.2011.11.009
- Li, Z., Snieder, H., Su, S., Ding, X., Thayer, J.F., Treiber, F.A., & Wang, X. (2009). A longitudinal study in youth of heart rate variability at rest and in response to stress. *International Journal of Psychophysiology*, 73, 212-217. doi: 10.1016/j.ijpsycho.2009.03.002
- Miller, G. A., Levin, D.N., Kozak, M.J., Cook III, E.W., Mclean, Jr, A., & Lang, P.J. (1987). Individual differences in imagery and the psychophysiology of emotion. *Cognition and Emotion*, 1, 367-390. doi: 10.1080/02699938708408058
- O'Connor, M.F., Allen, J.B., & Kaszniak, A.W. (2005). Emotional disclosure for whom? A study of vagal tone in bereavement. *Biological Psychology*, 68, 135-146. doi: 10.1016/j.biopsycho.2004.04.003
- Owen, N., Steptoe, A. (2003). Natural killer cell and proinflammatory cytokine responses to mental stress: Associations with heart rate and heart rate variability. *Biological Psychology*, 63, 101-115. doi: 10.1016/S0301-0511(03)00023-1
- Park, C.L., & Blumberg, C.J., (2002). Disclosing trauma through writing: testing the meaning-making hypothesis. *Cognitive Therapy and Research*, 26, 597-616. doi: 10.1023/A:1020353109229
- Pennebaker, J.W. (1982). *The psychology of physical symptoms*. New York: Springer Verlag.
- Pennebaker, J.W. (1997). Writing about emotional experiences as a therapeutic process. *Psychological Science*, 8, 162-166. doi: 10.1111/j.1467-9280.1997.tb00403.x
- Pennebaker, J.W., & Beall, S.K. (1986). Confronting a traumatic event: Toward an understanding of inhibition and disease. *Journal of Abnormal Psychology*, 95, 274-281. doi: 10.1037/0021-843X.95.3.274
- Pennebaker, J.W., Colder, M., & Sharp, L.K. (1990). Accelerating the coping process. *Journal of Personality and Social Psychology*, 58, 528-537. doi: 10.1037/0022-3514.58.3.528
- Pennebaker, J. W., & Francis, M. E. (1996). Cognitive, emotional, and language processes in disclosure. *Cognition and Emotion*, 10, 601-626. doi: 10.1080/026999396380079
- Pennebaker, J.W., Kiecolt-Glaser, J., & Glaser, R. (1988). Disclosure of traumas and immune function: Health Implications for psychotherapy. *Journal of Consulting and Clinical Psychology*, 56, 239- 245. doi: 10.1037/0022-006X.56.2.239
- Pennebaker, J.W., & Seagal, J. (1999). Forming a story: The health benefits of narrative. *Journal*

- of Clinical Psychology*, 55, 1243-1254. doi: 10.1002/(SICI)1097-4679(199910)55:10<1243::AID-JCLP6>3.0.CO;2-N
- Petrie, K. J., Booth, R., Pennebaker, J. W., Davison, K. P., & Thomas, M. (1995). Disclosure of trauma and immune response to Hepatitis B vaccination program. *Journal of Consulting and Clinical Psychology*, 63, 787-792. doi: 10.1037/0022-006X.63.5.787
- Porges, S.W. (1995a). Cardiac vagal tone: A physiological index of stress. *Neuroscience and Biobehavioral Reviews*, 19, 225-233. doi:10.1016/0149-7634(94)00066-A
- Porges, S.W. (1995b). Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A polyvagal theory. *Psychophysiology*, 32, 301-318. doi: 10.1111/j.1469-8986.1995.tb01213.x
- Porges, S. W. (1991). Vagal tone: An autonomic mediator of affect. In: Barber, J., Dodge, K.A. (Eds.), *The Development of Emotion Regulation and Dysregulation*. Cambridge University Press, Cambridge, 111-128.
- Radloff, L.S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, 1, 385-401. doi: 10.1177/014662167700100306
- Resick, P.A., Galovski, T.E., Uhlmansiek, M.O., Scher, C.D., Clum, G.A., & Young-Xu, Y. (2008). Randomized clinical trial to dismantle components of cognitive processing therapy for posttraumatic stress disorder in female victims of interpersonal violence. *Journal of Consulting and Clinical Psychology*, 76, 243-258. doi: 10.1037/0022-006X.76.2.243
- Ritz, T., Alatupa, S., Thons, M., Dahme, B. (2002). Effects of affective picture viewing and imagery on respiratory resistance in nonasthmatic individuals. *Psychophysiology*, 39, 86-94. doi: 10.1017.S0048577201020066
- Roberts, R., Vernon, S.W., & Rhoades, H.M. (1989). Effects of language and ethnic status on reliability and validity of the CES-D with psychiatric patients. *Journal of Nervous and Mental Disease*, 177, 581-592. doi: 10.1097/00005053-198910000-00001
- Rottenberg, J. (2007). Cardiac vagal control in depression: A critical analysis. *Biological Psychiatry*, 74, 200-211. doi: 10.1016/j.biopsycho.2005.08.010
- Ruiz-Padial, E., Sollers, J.J., Vila, J., & Thayer, J.F. (2003). The rhythm of the heart in the blink of an eye: Emotion-modulated startle magnitude covaries with heart rate variability. *Psychophysiology*, 40, 306-313. doi: 10.1111/1469-8986.00032
- Sack, M., Hopper, J.W., & Lamprecht, F. (2004). Low respiratory sinus arrhythmia and

- prolonged psychophysiological arousal in posttraumatic stress disorder: Heart rate dynamics and individual differences in arousal regulation. *Biological Psychiatry*, 55, 284-290. doi: 10.1016/S0006-3223(03)00677-2
- Sahar, T., Shalev, A.Y., & Porges, S.W. (2001). Vagal modulation of responses to mental challenge in posttraumatic stress disorder. *Biological Psychiatry*, 49, 637-643. doi: 10.1016/S0006-3223(00)01045-3
- Sajadieh, A., Nielsen, O.W., Rasmussen, V., Hein, H.O., Abedini, S., Hansen, J.F. (2004). Increased heart rate and reduced heart-rate variability are associated with subclinical inflammation in middle aged and elderly subjects with no apparent heart disease. *European Heart Journal*, 25, 363-370. doi: 10.1016/j.ehj.2003.12.003
- Sloan, D.M., & Epstein, E.M. (2005). Respiratory sinus arrhythmia predicts written disclosure outcome. *Psychophysiology*, 42, 611-615. doi: 10.1111/j.1469-8986.2005.347.x
- Sloan, R.P., Huang, M.H., McCreath, H., Sidney, S., Liu, K., Dale Williams, O., Seeman, T. (2008). Cardiac autonomic control and the effects of age, race, and sex: The CARDIA study. *Autonomic Neuroscience*, 139, 78-85. doi: 10.1016/j.autneu.2008.01.006
- Sloan, D.M., & Marx, B.P. (2004) a. A closer examination of the structured written disclosure procedure. *Journal of Consulting and Clinical Psychology*, 72, 165-175. doi: 10.1037/0022-006X.72.2.165
- Sloan, D.M., & Marx, B.P. (2004)b. Taking pen to hand: Evaluating theories underlying the written disclosure paradigm. *Clinical Psychology: Science and Practice*, 11, 121-137. doi: 10.1093/clipsy.bph062
- Sloan, D.M., Marx, B.P., & Epstein, E.M. (2005). Further examination of the exposure model underlying the efficacy of written emotional disclosure. *Journal of Consulting and Clinical Psychology*, 73, 549-554. doi: 10.1037/0022-006X.73.3.549
- Sloan, D.M., Marx, B.P., Epstein, E.M., & Lexington, J.M. (2007). Does altering the writing instructions influence outcome associated with written disclosure? *Behavior Therapy*, 38, 155-168. doi: 10.1016/j.beth.2006.06.005
- Sloan, R.P., McCreath, H., Tracey, K.J., Sidney, S., Liu, K., & Seeman, T. (2007). RR interval variability is inversely related to inflammatory markers: The CARDIA study. *Molecular Medicine*, 13, 178-184. doi:10.2119/2006-00112.Sloan
- Smyth, J.M. (1998). Written emotional expression: Effect sizes, outcome types, and moderating variables. *Journal of Consulting and Clinical Psychology*, 66, 170-184. doi: 10.1037/0022-006X.66.1.174
- Smyth, J.M., Hockemeyer, J.R., Heron, K.E., Wonderlich, S.A., Pennebaker, J.W. (2008).

- Prevalance, type, disclosure, and severity of adverse life events in college students. *Journal of American College Health*, 57, 69-76. doi:10.3200/JACH.57.1.69-76
- Smyth, J.M., True, N., & Souto, J. (2001). Effects of writing about traumatic experiences: The necessity for narrative structuring. *Journal of Social and Clinical Psychology*, 20, 161-172. doi: 10.1521/jscp.20.2.161.22266
- Stroebe, M., Stroebe, W., Schut, H., Zech, E., & van den Bout, J. (2002). Does disclosure of emotions facilitate recovery from bereavement? Evidence from two prospective studies. *Journal of Consulting and Clinical Psychology*, 70, 169-178. doi: 10.1037/0022-006X.70.1.169
- Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics (5th ed.)* Boston: Allyn and Bacon.
- Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. (1996). Heart rate variability: Standards of measurement, physiological interpretation, and clinical use. *Circulation*, 93, 1043-1065. doi: 10.1161/01.CIR.93.5.1043
- Thayer, J.F., Ahs, F., Fredrikson, M., Sollers, J.J., & Wager, T.D. (2012). A meta-analysis of heart rate variability and neuroimaging studies: Implications for heart rate variability as a marker of stress and health. *Neuroscience and Behavioral Reviews*, 36, 747-756. doi: 10.1016/j.neubiorev.2011.11.009
- Thayer, J.F., & Lane, R.D. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders*, 61, 201-216. doi: 10.1016/S0165-0327(00)00338-4
- Thayer, J.F., & Lane, R.D. (2007). The role of vagal functioning in the risk for cardiovascular disease and mortality. *Biological Psychology*, 74, 224-242. doi: 10.1016/j.biopsycho.2005.11.013
- van Emmerik, A.A.P, Reijntjes, A., & Kamphuis, J.H. (2013). Writing therapy for posttraumatic stress: A meta-analysis. *Psychotherapy and Psychosomatics*, 82, 82-88. doi: 10.1159/00343131
- Vrana, S., & Lauterbach, D. (1994). Prevalence of traumatic events and post-traumatic psychological symptoms in a non-clinical sample of college students. *Journal of Traumatic Stress*, 7, 289-302. doi: 10.1002/jts.2490070209
- Wang, X., Thayer, J.F., Treiber, F., & Snieder, H. (2005). Ethnic differences and heritability of heart rate variability in African- and European American youth. *American Journal of Cardiology*, 96, 1166–1172. doi: 10.1016/j.amjcard.2005.06.050
- Wesseling, K.H., & Settels, J.J. (1985). Baromodulation explains short term blood pressure

variability. In: J.F. Orlebeke, G. Mulder, & L.P.J. van Doornen (Eds.), *The Psychophysiology of Cardiovascular Control* (pp. 69-97). New York: Plenum Press.

Yaroslavsky, I., Bylsma, L.M., Rottenberg, J., & Kovacs, M. (2013). Combinations of resting RSA and RSA reactivity impact maladaptive mood repair and depressive symptoms. *Biological Psychiatry, 94*, 272-281. doi: 10.1016/j.biopsycho.2013.06.008

Yehuda, R., McFarlane, A.C., & Shalev, A.Y. (1998). Predicting the development of posttraumatic stress disorder from the acute response to a traumatic event. *Biological Psychiatry, 44*, 1305-1313. doi: 10.1016/S0006-3223(98)00276-5

Appendix A

Imagery Response Training Protocol

Imagery Response Training Protocol

“Today we’ll begin by teaching you to relax through the use of a breathing technique. This technique, called diaphragmatic breathing, has been found to be effective for reducing feelings of tension. Essentially, there are two ways of breathing-- from the chest, or from the diaphragm. With chest breathing, the chest expands with each inhalation, while the abdomen remains relatively motionless. When breathing from the diaphragm, the stomach or abdomen expands as the diaphragm moves downward to allow air to fill the lungs. We now know that when we breathe predominantly from our chest that this can create bodily tension, and that when we breathe with our diaphragm we can create feelings of relaxation. I will teach you this technique so you can use it later to relax before writing.

"Now I would like for you to practice this breathing technique. First, place one hand on your chest and the other on your abdomen, like this (*demonstrate*). Next, inhale slowly through your nose and try to make the hand on your abdomen rise. Try to push up your hand as much as it feels comfortable. Your chest should move slightly, but not more than your abdomen. After you’ve taken a full breath, pause for a second, and then exhale slowly and fully through your nose or mouth and count to one to yourself as you exhale. As you practice this procedure, imagine that there is a balloon in your stomach, and that each time you inhale, the balloon inflates and each time you exhale, the balloon deflates."
(Demonstrate this breathing technique for 30s).

“Do you have any questions?”

“Now I would like for you to practice this technique for a few minutes with your eyes closed. Again, try to imagine a balloon inflating and deflating in your stomach as you practice this technique. Concentrate on your abdomen moving up and down, the air moving in and out of your lungs, and the feelings of relaxation that deep breathing gives you.”

Have subject practice for 3 minutes. Watch, and provide feedback (minimal) about whether he/she is doing the procedure correctly.

After 3 minutes, if the subject is not doing the procedure correctly, additional practice may be needed. Thus, explain the procedure again reading from the bolded paragraph above. If the

subject is breathing correctly, continue with the next part of the experiment. For every subject, say the following line before continuing to the next part.

How did that feel?

(For some people it is not relaxing. If it is not relaxing for you, you can just close your eyes and breathe when you are instructed to relax.)

"You will be asked to use this breathing technique later in the experiment. Do you have any questions?"

As mentioned earlier, I will ask you to write about an event in your life and we will do this in a little while. When you write you will be calling upon memories of the experience. I want to share with you a technique that I want you to use that will help you recall and visualize the experience. I would like to help you learn to be able to vividly recall the feelings of the actual experience.

Imagery Response Training

"As I mentioned earlier, visualization, or vividly imagining scenes and events, is part of our experimental procedure. We will begin this phase of the experiment now. I'd like you to practice visualizing some commonplace scenes. It is like daydreaming, but I'd like you to bring this more under your control, to imagine specific events, for a given period of time. It will help you to do this if you remain relaxed, as you've learned.

As you are sitting there, deeply relaxed, completely calm, I'd like you to try some scenes. Try to imagine these situations as vividly as you can. Involve yourself fully in the image as an active participant in the imagined scene. For example, the first scene I will ask you to imagine involves reading a book. I want you to try to move your eyes in the image just as if you were actively scanning the words and lines of a real book. The idea of a vivid image is that you get the feeling of a real, actual experience.

As I describe the scenes, create the image in your mind, doing exactly what you would do in the real situation. When I finish the description, keep imagining the scene until I tell you to stop and focus on relaxation. Now I will present the first scene. Please close your eyes and keep them closed until I tell you to open them."

"You are sitting in a chair reading a popular science magazine. Your eyes dart from word to word and from line to line down the page as you make rapid progress through the text. You shift to a full page illustration of the muscles of the arm, and you look up and down all over the page, noting first the hand on the upper right corner of the page, then inspecting the elbow in the center, and finally the upper arm muscles in the lower left part of the page. You turn the page, and your eyes follow the text into the next chapter."

Have subject imagine scene for 20s

"Now open your eyes."

“How were you able to imagine the scene?”

“Did you move your eyes in the image?”

“Did you move your hand in the image?”

“Remember, it is important to scan the book with your eyes in the image just as if you were looking at a real book. A vivid image depends on making the scene like a real, actual experience. You must do in the image what you would do in the real situation.”

“Alright, now that we’ve reviewed the idea of vividness, let’s try another scene. Don’t worry if the first scene wasn’t very vivid. Some people are initially better than others at this, but practice will help everyone to imagine events as if they are really happening. We’re ready to try again.”

“Close your eyes and take a few seconds to get in a comfortable position and relax again. (20s).

“Remember, what we are trying to learn is vivid imagery through your active participation in what you imagine. Just like with the last scene, this means doing just what the image requires. For example, the next image involves muscle tension you feel while you are reading. I want you to actually tense your muscles in imagining this. This will make the image more vivid, that is, more like an actual experience of the scene I present.

Now I will present the scene. Create the image in your mind, doing exactly what you would do in the real situation. When I finish the description, keep imagining the scene until I tell you to stop. Here is the next scene. Please close your eyes and keep them closed until I tell you to open them.”

“You are in the language laboratory listening to an assignment over headphones, and following the conversation with your book. You listen to the words and follow the script from line to line down the page. Your neck and shoulder muscles are tense and stiff from being held so long in the same position. Trying to concentrate, you tense the muscles in your forehead and around your eyes, and you feel a full headache. Taking off the headphones, you breathe deeply and get up from the desk for a break.”

Have subject imagine scene for 20s

“Now open your eyes.”

“How were you able to imagine the scene?”

“Did you tense your muscles in the image?”

“Did you move your eyes in the image?”

“Did you take the deep breath?”

“It is important to do in the scene what you would do in the real situation. This means things like tensing your muscles, moving your eyes, and breathing deeply. Many of us are not used to this type of vivid imagery, and the point of this portion of the experiment is for you to learn to practice this kind of active involvement in your images.”

“Let’s practice another scene again. Sit back, close your eyes, and get relaxed. Try to focus on breathing deeply from your diaphragm.” (20s)

“Now that you are sitting there, deeply relaxed, completely calm, I’d like you to imagine another situation. Try to imagine the scene as vividly as you can.”

“You are standing at the base of an observation tower as some of your friends ascend the stairs. Your eyes follow their hands, gliding upwards on the handrails, as they slowly climb the metal staircase. You tense the muscles on your face, squinting to avoid the sun, which glints through the metalwork of the tower. Craning your neck, you continue to watch closely, following with your eyes their steady upward progress toward the observation deck. They reach the top, and you look up as someone drops a hat. You follow the hat with your eyes while it sails gently down to the ground at your feet.”

Have subject imagine scene for 20s

“Now open your eyes.”

“How were you able to imagine the scene?”

“Did you move your eyes in the image?”

“Did you use the muscle in your face and neck?”

“Recall that I want you to move your eyes in the image just as if you were looking up and down a real tower. Likewise, you are to tense the muscles used in the image. Actually do what you would do in the real situation.”

“Close your eyes and relax. We’ll do another scene. I’ll give you a few seconds to get relaxed, and then we’ll go into the next scene.” (20s)

“You are doing some isometric exercises and you look at the diagram in your exercise book. It is a schematic diagram, showing only the muscles themselves. It shows the face and the neck muscles, and you glance up and down the whole page, while you note the muscles involved in the exercise. You breathe deeply and tense all the muscles in your face and neck. Your heart races and sweat beads up on your forehead with strain.”

Have subject imagine scene for 20s

“Open your eyes.”

“How were you able to imagine the scene?”

“Did your heart beat change any? How about sweating?”

“Did you tense your muscles? Which ones? Did you breathe deeply?”

“This scene was a little different than the other ones we did, in that you were asked to imagine heart rate and perspiration changes. It may not be obvious that you can actually do these things in the images like you can with eye movements, muscle tension, and breathing changes, but don’t let this concern you. The practice here with imagining these responses can help you to increase your skill.”

“Okay, you have practiced a relaxation technique, and a way of achieving vivid imagery by doing in the scene what you would do in the real situation. The next part of this experiment will involve writing and I ask that you use the techniques you were just taught in order to more fully involve yourself in your writing.

How to reinforce the participant:

Reinforce response statements i.e. muscle movement, actions and perceptual movements. Ignore stimulus descriptions.

When response statements are reported you can say you did a good job with that. If the description did not involve response statements ask the participant if they experienced X. If they report that they did say “good, that will make your image more vivid.” If they did not experience X provide corrective feedback i.e. in future images try to imagine that you are actually in the scene doing what is described. For example, if the scene states that your muscles are tense, actually tense your muscles as if you were experiencing the scene.

You can ask prompting questions such as “What did you do when the hat fell down?” If the participant reports several response statements you can say, “It sounds like you had a very vivid image.”

After the participant has described their image, the experimenter should summarize the image and provide reinforcement and corrective feedback when appropriate.

Avoid interrupting the participant because interruption can be a punisher.

Imagery Stimulus Training Protocol

“Today we’ll begin by teaching you to relax through the use of a breathing technique. This technique, called diaphragmatic breathing, has been found to be effective for reducing

feelings of tension. Essentially, there are two ways of breathing-- from the chest, or from the diaphragm. With chest breathing, the chest expands with each inhalation, while the abdomen remains relatively motionless. When breathing from the diaphragm, the stomach or abdomen expands as the diaphragm moves downward to allow air to fill the lungs. We now know that when we breathe predominantly from our chest that this can create bodily tension, and that when we breathe with our diaphragm we can create feelings of relaxation. I will teach you this technique so you can use it later to relax before writing.

"Now I would like for you to practice this breathing technique. First, place one hand on your chest and the other on your abdomen, like this (*demonstrate*). Next, inhale slowly through your nose and try to make the hand on your abdomen rise. Try to push up your hand as much as it feels comfortable. Your chest should move slightly, but not more than your abdomen. After you've taken a full breath, pause for a second, and then exhale slowly and fully through your nose or mouth and count to one to yourself as you exhale. As you practice this procedure, imagine that there is a balloon in your stomach, and that each time you inhale, the balloon inflates and each time you exhale, the balloon deflates."
(Demonstrate this breathing technique for 30s).

"Do you have any questions?"

"Now I would like for you to practice this technique for a few minutes with your eyes closed. Again, try to imagine a balloon inflating and deflating in your stomach as you practice this technique. Concentrate on your abdomen moving up and down, the air moving in and out of your lungs, and the feelings of relaxation that deep breathing gives you."

Have subject practice for 3 minutes. Watch, and provide feedback (minimal) about whether he/she is doing the procedure correctly.

After 3 minutes, if the subject is not doing the procedure correctly, additional practice may be needed. Thus, explain the procedure again reading from the bolded paragraph above. If the subject is breathing correctly, continue with the next part of the experiment. For every subject, say the following line before continuing to the next part.

How did that feel?

(For some people it is not relaxing. If it is not relaxing for you, you can just close your eyes and breathe when you are instructed to relax.)

"You will be asked to use this breathing technique later in the experiment. Do you have any questions?"

As mentioned earlier, I will ask you to write about an event in your life and we will do this in a little while. When you write you will be calling upon memories of the experience. I want to share with you a technique that I want you to use that will help you recall and visualize the experience. I would like to help you learn to be able to vividly recall the feelings of the actual experience.

“As I mentioned earlier, visualization, or vividly imagining scenes and events, is part of our experimental procedure. We’ll begin this phase of the experiment now. I’d like you to imagine some situations. I’ll be reading descriptions of the events to help you imagine them. It is like daydreaming, but I’d like you to bring this more under your control, to imagine specific events, for a given period of time. It will help you to do this if you remain relaxed, as you’ve learned.

As you sit there, relaxed and calm, I’d like you to imagine some events. Try to imagine the situations as vividly as you can. Picture the scene in your mind as clearly as possible. For example, the first scene I will ask you to imagine involves reading a magazine. I want you to visualize the picture of the magazine with as much detail as you can, just as if the book were real. The idea of a vivid image is that you get a realistic picture of the scene in your mind.

Now I’ll set up the image. As I describe the situation, create the image in your mind, getting a detailed picture of what the real situation would be like. When I finish the description, keep imagining the scene until I tell you to stop and focus on relaxation. Now I will present the first scene. Please close your eyes and keep them closed until I tell you to open them.”

“You are sitting in a chair reading a popular science magazine. You see the words in paragraphs in black ink. You shift to a full page illustration of the muscles of the arm, and you notice that different colors are used to illustrate different parts of the arm, noting first the hand, which is yellow, then inspecting the elbow which is green, and finally the upper arm muscles which are shown in orange. You notice the fine detailed lines of the muscles in each part of the arm.

Have subject imagine scene for 20s

“Now open your eyes.”

“How were you able to imagine the scene?”

“Were you able to see the words in paragraphs in black ink?”

“Were you able to see the different muscles of the arm in the different colors?”

“Were you able to see the fine detailed lines of the muscles in each part of the arm?”

“Remember, it’s very important to include in the picture all the details that you can, and to visualize the scene just as if it were really happening. A vivid image depends on your having a realistic picture in your mind. Many of us aren’t used to this way of imagining things vividly, and the point of these group sessions is for you to learn and practice this kind of active involvement with your imagery. A vivid image depends on your making the picture look as real as possible. You must include in the image colors, shapes, sizes, and relationships. This can help you to have more realistic images.

All right, now that we’ve reviewed the ideas of vividness, let’s try another scene. Don’t worry if the first scene wasn’t very vivid. Some people are initially better than others at this, but

practice will help everyone to imagine events as if you were really seeing them. We are ready to try again.”

“Close your eyes and take a few minutes to get in a comfortable position and relaxed again (20s).”

“Remember, what we’re trying to learn is vivid imagery by your including as many details as possible in the picture in your mind. Just like in the last scene this means including colors, textures, and relationships, in the picture. For example, be involved in the next situation by attending carefully to the details of situation just as if they were right in your line of sight. This will make the image more vivid. Now I will present the scene. When I finish the description, keep imagining the scene until I tell you to stop. Here is the next scene. Please close your eyes and keep them closed until I tell you to open them. Here is the next scene.”

“You are in the language laboratory listening to an assignment over headphones and following the conversation with your book. The words flow too fast and the lines of text are a gray blur against the creamy white surface of the page. A color photograph of a farm on the adjoining page distracts you from the text. The texture of the page with the color plate is smooth looking and glossy, while the page with the text is rough and dull.”

Have subject imagine scene for 20s

“Now open your eyes.”

“What did you see in the image?”

“Were you able to see gray blurred lines on the page?”

“Did you see colors in the photograph?”

“Did you see the glossy vs. dull textures?”

“It is important to include lots of details in the image, picturing the situation in your mind as if it were a real situation. Many of us are not used to this type of vivid imagery, and the point of this portion of the experiment is for you to learn to practice including details in your images.

“Let’s practice another scene again. Sit back, close your eyes, and get relaxed. Try to focus on breathing deeply from your diaphragm.” (20s)

“Now that you are sitting there, deeply relaxed, completely calm, I’d like you to imagine another situation. Try to imagine the scene as vividly as you can.”

“You are at the base of an observation tower as some of your friends ascend the stairs. The sun glints through the metal staircase. Slowly they make upward progress toward the

tower's observation deck. They reach the top and wave to you from the platform. One of your friends drops a white hat, which gently sails down to the ground at your feet."

Have subject imagine scene for 20s

"Now open your eyes."

"What did you see in the image?"

"Did you see the gray tower, the sun, the platform?"

"Did you see the white hat falling?"

"It is important to include lots of details in the image, picturing the situation in your mind as if it were a real situation. Many of us are not used to this type of vivid imagery, and the point of this portion of the experiment is for you to learn to practice including details in your images.

"Let's practice another scene. Sit back, close your eyes, and get relaxed. Try to focus on breathing deeply from your diaphragm."(20s)

"Now that you're sitting there, deeply relaxed, completely calm, I'd like you to imagine another situation. Try to imagine the scene as vividly as you can."

"Try to picture in your mind as much detail as you can, as if the situation were real."

Close your eyes and relax again. An interesting thing about this training is that you can apply what you have learned to your images in a variety of settings. For example, the experiences you have when you watch a film or see a play are like the pictures you imagine here. If you are willing to focus on as many details as possible, the action on screen or on stage helps you to believe in the situation and picture it as if it were real. My picturing as many details as possible in your mind, you can experience situations as if they were real."

"Close your eyes and relax. We'll do another scene. I'll give you a few seconds to get relaxed, and then we'll go into the next scene." (20s)

Close your eyes and relax again. (20 seconds) Let's do another image now.

"You are flying a kite on the beach on a bright summer day. Your red kite shows clearly against the cloudless blue sky, and whips quickly up and down in spirals with the wind. The sun glares at you from behind the kite and makes the white sandy beach sparkle with reflection. The long white tail dances from side to side beneath the soaring kite."

Have subject imagine scene for 20s

“Open your eyes.”

“What did you see in the image?”

“What colors did you see?”

“Did you see the texture of the beach?”

“What shape was the kite?”

“I want to remind you again of the purpose of the imagery practice. You let yourself see situations as real by including lots of details about colors, shapes, sizes, etc., in your images. You have practiced a relaxation technique, and a way of achieving vivid imagery by including rich detail in the pictures in your mind. The next part of this experiment will involve writing and I ask that you use the techniques you were just taught in order to more fully involve yourself in your writing.”

How to reinforce the participant:

Reinforce descriptive statements i.e. the sky is blue, or the sun is shining Ignore response statements i.e. muscle movement, actions and perceptual movements.

When stimulus statements are reported you can say you did a good job with that. If the description did not involve stimulus statements ask the participant if they experienced X. If they report that they did say “good, that will make your image more vivid.” If they did not experience X provide corrective feedback i.e. in future images try to let yourself see situations as real by including lots of details about colors, shapes, sizes, etc., in your images.

You can ask prompting questions such as “What did the hat look like?”

If the participant reports several stimulus statements, you can say, “It sounds like you had a very vivid image.”

After the participant has described their image, the experimenter should summarize the image and provide reinforcement and corrective feedback when appropriate.

Avoid interrupting the participant because interruption can be a punisher.

Appendix B

Writing Instructions

Overview of Writing Instructions Given to All Participants

This study is an extremely important project looking at writing. During the next three lab sessions, you will be asked to write about one of several different topics for 20 minutes each day.

The only rule we have about your writing is that you write continuously for the entire time. If you run out of things to say, just repeat what you have already written. In your writing, don't worry about grammar, spelling, or sentence structure. Just write. Different people will be asked to write about different topics. Because of this, I ask that you not talk with anyone about the experiment. Because we are trying to make this a tight experiment, I can't tell you what other people are writing about or anything about the nature or predictions of the study. Once the study is complete, however, we will tell you everything. Another thing is that sometimes people feel a little sad or depressed after writing. If that happens, it is completely normal. Most people say that these feelings go away in an hour or so. If at any time over the course of the experiment you feel upset or distressed, please tell your experimenter or contact Dr. Vrana immediately.

[Note: All participants will receive a sheet with contact information for Dr. Vrana.]

Another thing. Your writing is completely anonymous and confidential. Your writing is coded with an ID number. Please do not include your name in your writing. Some people in the past have felt that they didn't want anyone to read them. That's OK, too. If you don't feel comfortable turning in your writing samples, you may keep/delete them. We would prefer if you turned them in, however, because we are interested in what people write. I promise that none of the experimenters, including me, will link your writing to you. The one exception is that if your writing indicates that you intend to harm yourself or others, we are legally bound to match your ID with your name. Above all, we respect your privacy. Do you have any questions at this point? Do you still wish to participate?

Experimental Condition Instructions

(Do Not state the next sentence to participants in the no training group) I would like you to use the imagination techniques you were just taught in order to more fully involve yourself in recalling and writing about your experiences.

What I would like to have you write about for the next three days is the most traumatic, upsetting experience of your entire life—the same experience that you identified when you filled out a questionnaire earlier about posttraumatic symptoms. In your writing, I want you to really let go and explore your very deepest emotions and thoughts. It is critical that you really delve

into your deepest emotions and thoughts. Ideally, we would like you to write about significant experiences or conflicts that you have not discussed in great detail with others. Remember that you have three days to write. You might tie your personal experiences to other parts of your life. How is it related to your childhood, your parents, people you love, who you are, or who you want to be. Again, in your writing, examine your deepest emotions and thoughts and remember to use the techniques you were just taught in order to more fully involve yourself in your writing.

On the Second Day of Writing

How did yesterday's writing go? Today, I want you to continue writing about the most traumatic experience of your life using the techniques you were taught in the first session in order to more fully involve yourself in your writing. While you are recalling your experience, remember to [actually do in your recollection what you were doing in the actual situation] or [involve yourself fully in the sights, sounds, and smells of the actual situation]. I really want you to explore your very deepest emotions and thoughts...and remember to use the techniques you were taught in the first session in order to more fully involve yourself in your writing.

On the Third Day of Writing

Today is the last writing session. In your writing today, I again want you to explore your deepest thoughts and feelings about the most traumatic experience of your life using the techniques you were taught in the first session in order to more fully involve yourself in your writing. While you are recalling your experience, remember to [actually do in your recollection what you were doing in the actual situation] or [involve yourself fully in the sights, sounds, and smells of the actual situation]. Remember that this is the last day and so you might want to wrap everything up. For example, how is this experience related to your current life and your future? But feel free to go in any direction you feel most comfortable with and delve into your deepest emotions and thoughts...and remember to use the techniques you were taught in the first session in order to more fully involve yourself in your writing.

Control Condition Instructions

(Do Not state the next sentence to participants in the no training group) I would like you to use the imagination techniques you were just taught in order to more fully involve yourself in recalling and writing about your experiences.

What I would like you to write about over the next three days is how you use your time. Each day, I will give you different writing assignments on the way you spend your time. In your writing, I want you to be as objective as possible. I am not interested in your emotions or opinions. Rather I want you to try to be completely objective. Feel free to be as detailed as possible. In today's writing, I want you to describe what you did yesterday from the time you got up until the time you went to bed. For example, you might start when your alarm went off and you got out of bed. You could include the things you ate, where you went, which buildings or objects you passed by as you walked from place to place. The most important thing in your

writing, however, is for you to describe your days as accurately and as objectively as possible and remember to use the techniques you were just taught in order to more fully involve yourself in your writing.

On the Second Day of Writing

How did your writing go yesterday? Today, I would like you to describe what you have done today since you woke up using the techniques you were taught in the first session in order to more fully involve yourself in your writing. While you are recalling your experience, remember to [actually do in your recollection what you were doing in the actual situation] or [involve yourself fully in the sights, sounds, and smells of the actual situation]. Again, I want you to be as objective as possible to describe exactly what you have done up until coming to this experiment... and remember to use the techniques you were taught in the first session in order to more fully involve yourself in your writing.

On the Third Day of Writing

This is the last day of the writing sessions. In your writing today, I would like you to describe what you will be doing over the next week and remember to use the techniques you were taught in the first session in order to more fully involve yourself in your writing. While you are recalling your experience, remember to [actually do in your recollection what you were doing in the actual situation] or [involve yourself fully in the sights, sounds, and smells of the actual situation].

Appendix C

Demographic Questionnaire

ID Number:

Name _____

- 1) Age _____
- 2) Gender _____
- 3) What is your Race? Please check **All that apply**:

American Indian/Alaska Native	<input type="checkbox"/>
Asian	<input type="checkbox"/>
Black or African- American	<input type="checkbox"/>
Hispanic	<input type="checkbox"/>
Native Hawaiian or Other Pacific Islander	<input type="checkbox"/>
White	<input type="checkbox"/>
Other	<input type="checkbox"/>

- 4) What year are you in school? Please check one of the following:

Freshman	<input type="checkbox"/>
Sophomore	<input type="checkbox"/>
Juitor	<input type="checkbox"/>
Senior	<input type="checkbox"/>

- 5) Is English your native language? _____
If not, what is your native language? _____

- 6) Are you currently receiving psychotherapy? _____

- 7) Have you smoked cigarettes in the last 6 hours? _____

- 8) Have you used any other tobacco products in the last 6 hours? _____ - If yes, what kinds? _____

- 9) Do you use any prescription medications?
If yes, please list: _____

- 10) Please include your **e-mail address** to receive your Follow-Up Packet **one month from now**:

Appendix D

Davidson Trauma Scale

Intials: _____
Date/session: _____
Idnum: _____

Please identify the trauma which is most disturbing to you: _____

A. In the past week, how much trouble have you had with the following, keeping in mind the event described above.

Frequency

0= Not at all
1= Once only
2= 2-3 times
3= 4-6 times
4= more than 6 times

Severity

0= Not at all distressing
1= Minimally distressing
2= Moderately distressing
3= Markedly distressing
4= Extremely distressing

- | | | |
|---|-------|-------|
| 1) Have you had painful images, memories or thoughts of the event? | _____ | _____ |
| 2) Have you had distressing dreams of the event? | _____ | _____ |
| 3) Have you felt as though the event was re-occurring? | _____ | _____ |
| 4) Have you been upset by something which reminded you of the event? | _____ | _____ |
| 5) Have you been avoiding any thoughts or feelings about the event? | _____ | _____ |
| 6) Have you been avoiding doing things or going into situations which remind you about the event? | _____ | _____ |
| 7) Have you found yourself unable to recall important parts of the event? | _____ | _____ |
| 8) Have you had difficulty enjoying things? | _____ | _____ |
| 9) Have you felt distant or cut off from other people? | _____ | _____ |
| 10) Have you been unable to have sad or loving feelings? | _____ | _____ |
| 11) Have you found it hard to imagine having a long life span fulfilling your goals? | _____ | _____ |
| 12) Have you had falling asleep or staying asleep? | _____ | _____ |
| 13) Have you been irritable or had outbursts of anger? | _____ | _____ |
| 14) Have you had difficulty concentrating? | _____ | _____ |
| 15) Have you felt on the edge, been easily distracted, or had to stay on guard? | _____ | _____ |
| 16) Have you been jumpy or easily startled? | _____ | _____ |

17) Have you been physically upset by reminders of the event?

Appendix E

Short Version of the Davidson Trauma Scale

Intials: _____
Date/session: _____
Idnum: _____

Please identify the trauma which is most disturbing to you: _____

A. In the past 10 minutes, how much trouble have you had with the following, keeping in mind the event described above.

Frequency	Severity
0= Not at all	0= Not at all distressing
1= Once only	1= Minimally distressing
2= 2-3 times	2= Moderately distressing
3= 4-6 times	3= Markedly distressing
4= more than 6 times	4= Extremely distressing

a. Have you had any painful images, memories or thoughts of the event?

b. Have you felt as though the event was reoccurring? Was it as if you were reliving it?

c. Have you been upset by something which reminded you of the event?

d. Have you been avoiding any thoughts or feelings about the event?

e. Have you found yourself unable to recall important parts of the event?

f. Have you had difficulty enjoying things?

g. Have you felt distant or cut-off from other people?

h. Have you been unable to have sad or loving feelings, or have you generally felt numb?

i. Have you been irritable or had outburst of anger?

j. Have you had difficulty concentrating?

k. Have you felt on edge, been easily distracted, or had to stay on guard?

l. Have you been jumpy or easily startled?

m. Have you been physically upset by reminders of the event? (This includes sweating, trembling, racing heart, shortness of breath, nausea, diarrhea.)

Appendix F

Center for Epidemiological Studies-Depression Scale (CES-D)

CES-D

Below is a list of the ways you might have felt or behaved. Please check the appropriate box to tell how often you have felt this way during the past week.

Rarely or none of the time (less than 1 day)	Some or a little of the time (1-2 days)	Occasionally or a moderate amount of time (3-4 days)	Most or all of the time (5-7 days)
--	---	--	------------------------------------

- | | | | | |
|--|--------------------------|--------------------------|--------------------------|--------------------------|
| 1. I was bothered by things that usually don't bother me. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 2. I did not feel like eating; my appetite was poor. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 3. I felt that I could not shake off the blues even with help from my family or friends. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 4. I felt I was just as good as other people. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 5. I had trouble keeping my mind on what I was doing. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 6. I felt depressed. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 7. I felt that everything I did was an effort. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 8. I felt hopeful about the future. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |

- | | | | | |
|--|--------------------------|--------------------------|--------------------------|--------------------------|
| 9. I thought my life had been a failure. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 10. I felt fearful. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 11. My sleep was restless. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 12. I was happy. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 13. I talked less than usual. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 14. I felt lonely. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 15. People were unfriendly. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 16. I enjoyed life. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 17. I had crying spells. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 18. I felt sad. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 19. I felt that people disliked me. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| 20. I could not get "going". | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |

Appendix G

The Pennebaker Inventory of Limbic Languidness (PILL)

Several common symptoms or bodily sensations are listed below. Most people have experienced most of them at one time or another. We are currently interested in finding out how prevalent each symptom is among various groups of people. On the page below, write how frequently you experience each symptom. For all items, use the following scale:

	1		2		3		4		5
	Have never or almost never experienced the symptom		Less than 3 or 4 times per year		Every month or so		Every week or so		More than once every week
_____ 1	Eyes Water				_____ 28		Swollen joints		
_____ 2	Itchy eyes or skin				_____ 29		Stiff or sore muscles		
_____ 3	Ringing in ears				_____ 30		Back pains		
_____ 4	Temporary deafness or hard of hearing				_____ 31		Sensitive or tender skin		
_____ 5	Lump in throat				_____ 32		Face flushes		
_____ 6	Choking sensations				_____ 33		Tightness in chest		
_____ 7	Sneezing spells				_____ 34		Skin breaks out in rash		
_____ 8	Running nose				_____ 35		Acne or pimples on face		
_____ 9	Congested nose				_____ 36		Acne/pimples other than face		
_____ 10	Bleeding nose				_____ 37		Boils		
_____ 11	Asthma or wheezing				_____ 38		Sweat even in cold weather		
_____ 12	Coughing				_____ 39		Strong reactions to insect bites		
_____ 13	Out of breath				_____ 40		Headaches		
_____ 14	Swollen ankles				_____ 41		Feeling pressure in head		
_____ 15	Chest pains				_____ 42		Hot flashes		
_____ 16	Racing heart				_____ 43		Chills		
_____ 17	Cold hands or feet even in hot weather				_____ 44		Dizziness		
_____ 18	Leg cramps				_____ 45		Feel faint		
_____ 19	Insomnia or difficulty sleeping				_____ 46		Numbness or tingling in any part of body		
_____ 20	Toothaches				_____ 47		Twitching of eyelid		
_____ 21	Upset stomach				_____ 48		Twitching other than eyelid		
_____ 22	Indigestion				_____ 49		Hands tremble or shake		
_____ 23	Heartburn or gas				_____ 50		Stiff joints		
_____ 24	Abdominal pain				_____ 51		Sore muscles		
_____ 25	Diarrhea				_____ 52		Sore throat		
_____ 26	Constipation				_____ 53		Sunburn		
_____ 27	Hemorrhoids				_____ 54		Nausea		

In the last month, how many:

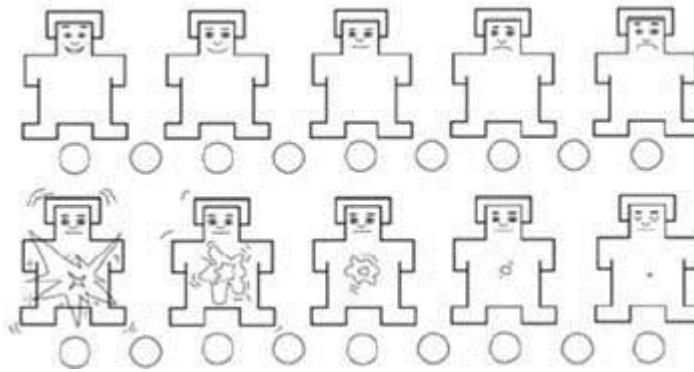
_____ _____ Visits have you made to the student health center or private physician for illness?

_____ _____ Days have you been sick?

_____ Days has your activity has been restricted due to illness?

Appendix H

Self-Assessment Manikin (SAM)



Appendix I

Hypothesis 3 Regression Table

DV	IV	Unstandardized Beta	t value	Standardized Beta	<i>P</i>
PILL (follow-up)	(Constant)	8.826	2.361		0.019 [†]
	PILL (Baseline)	0.754	14.580	0.757	<.0005 [†]
	Gender	-1.935	-0.676	-0.036	0.500
	Log RSA	0.567	0.368	0.026	0.713
	Trauma	0.692	0.273	0.014	0.785
	Trauma X Log RSA	0.578	0.248	0.018	0.804
CES-D (follow-up)	(Constant)	2.420	1.706		0.090 [‡]
	CES-D (Baseline)	0.752	12.890	0.713	<.0005 [†]
	Gender	0.704	0.596	0.033	0.552
	Log RSA	0.386	0.601	0.045	0.549
	Trauma	0.601	0.558	0.031	0.577
	Trauma X Log RSA	-0.783	-0.805	-0.061	0.422
DTS-LONG (follow-up)	(Constant)	2.112	1.079		0.282
	DTS-LONG (Baseline)	0.520	8.930	0.581	<.0005 [†]
	Gender	-2.330	-1.263	-0.083	0.208
	Log RSA	1.251	1.249	0.112	0.213
	Trauma	1.140	0.692	0.045	0.490
	Trauma X Log RSA	-0.629	-0.413	-0.037	0.680
DTS-SHORT (S3)	(Constant)	1.806	1.821		0.070 [‡]
	DTS-SHORT (S1)	0.598	11.234	0.688	<.0005 [†]
	Gender	-0.612	-0.585	-0.032	0.559
	Log RSA	0.332	0.583	0.043	0.560
	Trauma	-1.417	-1.323	-0.081	0.187
	Trauma X Log RSA	-1.497	-1.737	-0.128	0.084 [‡]

[†]Indicates $p < .05$

[‡]Indicates $p < .10$

Appendix J

Hypothesis 4 Regression Table

DV	IV	Unstandardized Beta	t value	Standardized Beta	<i>P</i>
HR (S1 Writing)	(Constant)	4.681	1.685		0.093 [‡]
	HR (S1 Baseline)	0.985	28.338	0.935	<.0005 [†]
	Gender	0.410	0.551	0.015	0.582
	Log RSA	0.381	0.826	0.035	0.410
	Trauma	0.512	0.764	0.021	0.446
	Trauma X Log RSA	-0.225	-0.369	-0.014	0.712
Log SC (S1 Writing)	(Constant)	0.258	6.932		<.0005 [†]
	Log SC (S1 Baseline)	0.811	23.635	0.847	<.0005 [†]
	Gender	-0.030	-0.795	-0.029	0.428
	Log RSA	-0.033	-1.604	-0.079	0.110
	Trauma	0.051	1.541	0.054	0.125
	Trauma X Log RSA	0.009	0.307	0.015	0.759

[†]Indicates $p < .05$

[‡]Indicates $p < .10$

Appendix K

Hypothesis 5 Regression Table

DV	IV	Unstandardized Beta	t value	Standardized Beta	P
PILL (follow-up)	(Constant)	11.630	2.077		0.041 [†]
	Gender	-8.206	-1.954	-0.143	0.054 [‡]
	PILL (Baseline)	0.761	10.037	0.745	<.0005 [†]
	Log RSA	6.383	1.924	0.288	0.058 [‡]
	Response	5.833	1.293	0.113	0.200
	Stimulus	-2.273	-0.502	-0.043	0.617
	Response X Log RSA	-6.551	-1.523	-0.186	0.132
	Stimulus X Log RSA	-5.220	-1.167	-0.128	0.247
CES-D (follow-up)	(Constant)	4.444	2.146		0.035 [†]
	Gender	2.091	1.319	0.107	0.191
	CES-D (Baseline)	0.693	7.616	0.632	<.0005 [†]
	Log RSA	0.552	0.433	0.073	0.666
	Response	-2.457	-1.414	-0.139	0.161
	Stimulus	-2.765	-1.576	-0.151	0.119
	Response X Log RSA	-1.501	-0.916	-0.125	0.362
	Stimulus X Log RSA	-1.492	-0.866	-0.107	0.389
DTS-LONG (follow-up)	(Constant)	-2.288	-0.661		0.511
	Gender	2.088	0.768	0.069	0.445
	DTS-LONG (Baseline)	0.580	6.892	0.646	<.0005 [†]
	Log RSA	2.101	0.947	0.179	0.347
	Response	1.188	0.396	0.043	0.693
	Stimulus	1.245	0.406	0.044	0.686
	Response X Log RSA	-1.611	-0.573	-0.086	0.568
	Stimulus X Log RSA	-1.697	-0.570	-0.078	0.570
DTS-SHORT (S3)					

(Constant)	1.823	0.764		0.447
Gender	-1.532	-0.796	-0.063	0.428
DTS-SHORT (S1)	0.613	7.943	0.645	<.0005 [†]
Log RSA	-1.412	-0.900	-0.151	0.371
Response	-0.698	-0.327	-0.032	0.744
Stimulus	-1.159	-0.541	-0.051	0.590
Response X Log RSA	0.900	0.448	0.061	0.655
Stimulus X Log RSA	-0.404	-0.192	-0.023	0.848

[†]Indicates $p < .05$

[‡]Indicates $p < .10$

Appendix L

Hypothesis 6 Regression Table

DV	IV	Unstandardized Beta	t value	Standardized Beta	<i>p</i>
HR (S1 Writing)	(Constant)	5.856	1.403		0.164
	Gender	0.119	0.110	0.004	0.913
	HR (S1 Baseline)	0.970	17.950	0.916	<.0005 [†]
	Log RSA	0.462	0.498	0.045	0.619
	Response	2.135	1.844	0.088	0.068 [‡]
	Stimulus	0.178	0.153	0.007	0.879
	Response X Log RSA	0.239	0.219	0.015	0.827
	Stimulus X Log RSA	-0.951	-0.821	-0.050	0.414
Log SC (S1 Writing)	(Constant)	0.290	4.339		<.0005 [†]
	Gender	-0.021	-0.344	-0.019	0.732
	Log SC (S1 Baseline)	0.781	14.437	0.827	<.0005 [†]
	Log RSA	-0.019	-0.390	-0.045	0.697
	Response	0.064	0.967	0.065	0.336
	Stimulus	-0.004	-0.061	-0.004	0.951
	Response X Log RSA	-0.004	-0.057	-0.005	0.955
	Stimulus X Log RSA	0.009	0.132	0.011	0.895

[†]Indicates $p < .05$

[‡]Indicates $p < .10$

Vita

Alison Marie Eonta was born in Sewickley, Pennsylvania and is an American citizen. She graduated from Quaker Valley High School in Leetsdale, Pennsylvania in 2001. She received her Bachelor of Arts in Foreign Affairs from the University of Virginia in 2005. In 2010, she received her Master of Science in Clinical Psychology from Virginia Commonwealth University in Richmond, Virginia. She then continued in the Clinical Psychology Doctoral Degree program at Virginia Commonwealth University. She completed a clinical internship at the Veterans Affairs Pittsburgh Healthcare System in Pittsburgh, Pennsylvania in August 2013. Alison will graduate in December 2013 with her doctorate in Clinical Psychology. She will then complete a post-doctoral fellowship in the Combat Stress Recovery Clinic at the Veterans Affairs Pittsburgh Healthcare System.