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RESPIRATORY SINUS ARRHYTHMIA IN POSTTRAUMATIC GROWTH AND PTSD

A THESIS

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Abstract

Most the human population will experience a traumatic event during their lifespan and will then cope with such trauma in a wide variety of ways. It is well documented that traumatic events are associated with both short and long-term psychological distress and that a small number of those trauma-exposed individuals will even develop a mental health disorder such as PTSD. In more recent empirical studies, some trauma-exposed individuals have been found to experience what has been labeled posttraumatic growth (PTG), or positive personal and psychological changes after experiencing trauma. However, there is significant controversy regarding PTG and its relationship with posttraumatic stress. This project attempts to provide clarity to one area of research about posttraumatic growth: how the physiological response of cardiac vagal regulation differs between those with PTSD and those with reported PTG. It was expected that individuals with PTG will not statistically differ from healthy controls on mean RSA. The findings confirm the proposed relationship between cardiac vagal tone, measured through RSA, and PTG. This study exemplifies that PTG is not an adaptive response to traumatic experiences, but instead a predictor of cardiac vagal rigidity. In each phase, RSA and heart period are lower in the PTG group, signifying less parasympathetic control over respiration and heart activity. More importantly, absence of cardiac vagal regulation post-aversive image task accompanied by lower RSA in PTG during this period strongly suggests that this construct is characterized by extreme affect dysregulation. A limitation in this study is that the sample was homogenous and only females participated in the laboratory portion of the study, which hinders the generalizability. Future research should investigate cardiac vagal activity with a stronger experimental manipulation, which can be done by using specific trauma centered memories or cues to examine the autonomic activity between those with reported PTG and PTSD.

Dedication

Marlow, I'll make the world safe and sound for you!

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Table of Contents

List of Tables....8
List of Figures....9
Introduction: Posttraumatic Growth versus PTSD....10
Evolutionary and Modern History of PTSD....16
Conventional Theories of PTSD....21
Cognitive & Behavioral Theory....21
Development and Attachment Theories....23
Physiological Perspectives....24
Autonomic Arousal Systems....25
Psychological Threat Detection....30
The Social Engagement System, PTSD, and Posttraumatic Growth....32
Chapter 3: Posttraumatic Growth....35
Theories of Posttraumatic Growth....36
Controversy Surrounding Posttraumatic Growth....41
Chapter 4: Current Study....46
Hypotheses....46
Method....48
Participants and Procedure....48
Physiological Measures.....50
Instrumentation....50
Demographic Information.....50
Posttraumatic Growth....51
PTSD Assessments....51
Trauma History Screen....53
General Mental Health Assessment....53
Generalized Anxiety Assessment....54
Depression Assessment....55
The International Affective Picture System....55
The BIOPAC MP150 Amplifier....55
The Distraction Task....55
Data Preparation & Analyses....56
Chapter 5: Results & Discussion....58
Cardiac Assessments....58

Screening Assessments....64
Discussion....65
Limitations....69
Future Directions....69
References....71
Appendix A....89

List of Tables

Table 1 48

Table 2 62

Table 3 63

List of Figures

Figure 1 57

Figure 2 58

Figure 3 59

Figure 4 60

Figure 5 61

Figure 6 61

Introduction: Posttraumatic Growth versus PTSD

Most of the human population will experience a traumatic event during their lifespan and will then cope with such trauma in a wide variety of ways (Bonanno, 2004). Thankfully, most people manage even the most severe stressors and traumas well. They can maintain daily routines, interact with family and friends, and experience positive emotions despite their exposure to adverse experiences. Others, though, experience problematic distress and even psychopathology that can include depression, anxiety, and posttraumatic stress disorder (PTSD). But rather than being only neutral or negative, could experiencing a trauma be a springboard to something positive for certain people? And if so, how?

It is well documented that traumatic events are associated with both short and long term psychological distress (Galea, Nandi, & Vlahov, 2005) and that a small number of those trauma-exposed individuals will even develop a mental health disorder (American Psychiatric Association, 2013). Conversely, the positive effects of traumatic events have been a prominent theme throughout history and are frequently discussed in literature and philosophy (e.g. Kierkegaard, 1983). In more recent empirical studies, some trauma-exposed individuals have been found to experience what has been labeled posttraumatic growth (PTG), or positive personal and psychological changes after experiencing trauma (Zoellner & Maercker, 2006).

According to proponents of this idea (e.g., Tedeschi & Calhoun, 2004), PTG occurs when individuals become stronger and create a more meaningful life in the wake of trauma. This can include a renewed appreciation of life, acting on new opportunities, improved relationships, and enhanced personal strengths. PTG is thought to occur after both natural and man-made traumatic events, including life-threatening disease, war, abuse, and death of loved ones (Zoellner & Maercker, 2006). In this view, while a significant number of individuals will show intense

depression and anxiety after a traumatic event, they will then grow psychologically and emotionally as a result.

However, there is significant controversy regarding PTG and its relationship with posttraumatic stress (Linley & Joseph, 2004; Zoellner & Maercker, 2006). Different theorists have proposed highly diverse models of PTG. For instance, PTG has been conceptualized as both an outcome from the traumatic event (Shaefer & Moos, 1998; Tedeschi & Calhoun, 2004) and a coping strategy (Affleck & Tennen, 1996). There is even speculation if PTG is genuine positive change or a maladaptive, self-deceptive coping mechanism (Frazier et al., 2009; Hall, Hobfoll, Canetti, Johnson, & Galea, 2009).

Theorists in posttraumatic growth research argue the possibility of positive psychological change occurs because of the struggle with a highly challenging life circumstances (Tedeschi & Calhoun, 2004). However, PTG also depends on people's perceptions that they have experienced growth, and so some argue that posttraumatic growth is a positive illusion rather than an objective improvement in psychological wellbeing (Frazier et al., 2009; Mancini, Littleton, & Grills, 2016). Further, the research on the relationship between PTSD and PTG has produced inconsistent and conflicting results.

Given this controversy, significant questions arise. What is the actual relationship between PTSD and PTG? Are they co-occurring phenomena or opposite ends of the same spectrum? If they are opposites, do different trauma types produce different outcomes? Are PTSD and PTG manifested different physiologically? Will people who score highly on measures of PTG respond to stimuli differently than someone who scores highly on measures of PTSD? How is perceived self-reported PTG related to actual growth? Is PTG an adaptive response to traumatic events or does it prevent real healing from occurring? Given these questions and the uncertainty in the

literature, this project will attempt to provide clarity to one area of research: how the physiological response of cardiac vagal regulation differs between those with PTSD and those with reported PTG. A significant portion of the literature and research surrounding trauma has focused on the negative aspects of post-trauma functioning. To understand the complicated construct of PTG it may be beneficial to examine the extant research on PTSD to gain a better understanding of how the disorder manifests itself and how it should differ, theoretically, from PTG.

Chapter 1: Posttraumatic Stress Disorder

Epidemiological research indicates that most people will experience a traumatic event during their life (Morina, Wicherts, Lobbrecht, & Priebe, 2014). Traumatic events can lead to different forms of psychopathology, with posttraumatic stress disorder (PTSD) being the most documented disorder after trauma. Epidemiological studies reveal that rates of PTSD vary depending on the type of trauma: about 55% after rape, 35% after childhood sexual or physical abuse, about 17% after physical assault, and about 7% after severe accidents (Kessler, Sonnega, Bromet & Nelson, 1995; Maercker, Michael, Fehm, Becker, & Margraf, 2004). Most individuals with PTSD appear to suffer for several years then gradually recover functioning, but still live lives clouded by vestiges of their former symptoms, such as persistent sleep problems, occasional intrusive thoughts, planned avoidance, and interpersonal isolation (Tomb, 1994). Others, though, maintain high levels of PTSD symptoms for many years or are functionally impaired for the duration of their life (Goldfeld, Mollica, & Pesavento, 1988).

The current Diagnostic and Statistical Manual of Mental Disorders (fifth edition; American Psychiatric Association, 2013) contains specific diagnostic criteria for PTSD. Chief among these are symptoms that must develop after exposure to a traumatic event and be present for more than one month. Four distinct symptom clusters characterize the disorder. First, involuntary re-experiencing of the traumatic events in one or more of the following ways: recurrent dreams, flashbacks, intense cue sensitivity, recurrent recollections, or physiological reactivity. Second, persistent avoidance of external or internal cues related with the traumatic experience is demonstrated in one or more of the following ways: avoiding thoughts, inability to recall, avoiding activities, diminished interest, detachment, flat affect, and sense of dim future. Third, negative alterations in mood and/or cognitions in one or more of the following ways:

memory problems associated with the traumatic event, negative thoughts about one's self or others, distorted sense of blame for one's self or other associated with the traumatic event, stuck in extreme emotions associated with the trauma, reduced interest in pre-trauma activities, and feeling detached, isolated, or disconnected from other people. Finally, persistent increased arousal in two or more of the following is the fourth symptom cluster of PTSD: problems sleeping, irritability, hypervigilance, difficulty concentrating, and heightened startle reflex.

Over 80% of PTSD diagnoses are accompanied by at least one comorbid disorder, the most common being depression (Kilpatrick et al., 2003; Araujo et al., 2014). The depressive symptoms associated with PTSD further exacerbate the emotional, physical, social, and psychological quality of life deficits (Araujo et al, 2014). Depression is not the only comorbid disorder typically manifested with PTSD, as 50% of PTSD diagnoses are accompanied by two or more comorbid disorders (Kilpatrick et al., 2003). Some other common comorbidities are alcohol and substance abuse that range from 21.6% to 43% (Feldner et al., 2009), prevalence of comorbid panic attacks 35% (Falsetti & Resnick, 1997), and antisocial personality disorder with 48% comorbidity (Keane & Wolfe, 1990).

PTSD is associated with mental and physical distress and high economic costs (Nemeroff et al., 2006). Individuals with PTSD experience greatly decreased quality of life and functional difficulties (Mendlowicz & Stein, 2000). Epidemiologic studies (Breslau, Davis, Andreski, & Peterson, 1991; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995) found prevalence for PTSD of 7.8% to 9.2%, with women experiencing a diagnosis at two times higher rates than men. In an archival analysis of data, researchers measured the impact PTSD had on functioning and quality of life (Mendolowicz & Stein, 2000). A decreased quality of life outcome was more common in people with PTSD. There are significantly elevated odds of poor functioning in all domains for

female veterans with PTSD (Zatzick et al., 1997). Veterans with PTSD were found to be more likely to report marital, parental, and family adjustment problems. Overall, PTSD is associated with elevated odds of poor functioning in self-reported physical health status, days in bed, and role functioning across genders (Mendlowicz & Stein, 2000).

Persons with partial PTSD – i.e., having fewer than the required number of DSM criterion or symptoms – report significantly more problems in work than trauma exposed individuals without PTSD (Mendolowicz & Stein, 2000). Individuals with full PTSD and partial PTSD report comparable levels of family and social interference. Partial PTSD approaches the same level of functional interference as PTSD in several quality of life domains. This highlights the need for effective intervention and treatment even if someone may not meet "full criteria." But, even with treatment, PTSD often leaves a residual functional effect on people despite the remission of their clinical symptoms (Morina et al., 2014). There have been several explanations for this phenomenon put forward. First, PTSD can result in altered cognitive appraisals of one's self and the environment that can affect one's capacity to function healthily. People with PTSD ruminate on the trauma or exaggerate the perceived threat or likelihood of re-experiencing the trauma (Ehlers & Clark, 2000). This form of habitual thinking can contribute to occupational, social, and psychological impairments. Second, PTSD can result in social isolation, which can perpetuate negative affect. Emotional numbing and detachment predict reduced psychosocial functioning, especially in post-trauma adjustment (Riggs, Byrne, Weathers, & Litz, 1998; Samper, Taft, King, & King, 2004). Individuals with the disorder may withdraw from social relationships because of the active PTSD avoidance symptoms and consequently develop socially avoidant habits and behavior. Even when PTSD symptoms abate, these individuals now have fewer social networks, which can in turn produce reduced quality of life.

Relatedly, the perceived absence of supportive relationships is one of the strongest predictors for the development of PTSD (Brewin, Andrews, & Valentine, 2000). Social support diminishes the sense of threat following traumatic events, which protects against anxiety, depression, and PTSD symptoms (Charuvastra & Cloitre, 2008). Additionally, strong social relationships are predictive of wellbeing aside from buffering from the harmful effects of stress (Argyle, 2001). Social support may not only protect people from pathological reactions to stress but may also promote positive effects on wellbeing by improving social relationships.

Evolutionary and Modern History of PTSD

Although PTSD is a relatively recent diagnostic phenomenon, making its debut in the DSM-III after the end of the Vietnam War, the collection of symptoms it is significantly older. Evidence of PTSD can be found wherever catastrophic experiences have occurred, dating back to our early ancestors who lived in a daunting and relentless world in which they experienced life-threatening stressors regularly. How did the functional impairments of PTSD, which appear intrinsically maladaptive, survive natural selection? Traumatized individuals are impaired at several levels, which should be selected against by evolution (Beahrs, 1990). Individual survival is endangered by heightened vulnerability to minor stressors, overt self-destructive actions, and reckless endangerment. Reproductive success is impaired by difficulty maintaining relationships and sexual dysfunction. The maladaptive features of PTSD are problematic in a rapidly changing environment that requires adaptive flexibility and small interpersonal boundaries.

Hominization occurred in the African Savanna, and mankind spent much of its history in primitive settings prior to the advent of civilization (Eibl-Eibesfeldt, 1989). This environment, stable for extended periods of time, fostered the selection of humans' response to stress and trauma. In this environment, catastrophic events occurred regularly and in comparative forms

(e.g., natural disaster, illness, predation, human conflicts). The features of what we now call PTSD evolved to enable mankind to adapt to specific environments in which threats occurred in a repetitive and stereotypical form.

The savannah provided a stable environment to hunter gather societies and successive ancestors of the species encountered the common sources of danger. Natural selection would have favored the survival of those most flexible to responding to threat (Marks, 1987). Natural selection in a stable environment fostered the refinement of innate fear responses that allowed humans to react automatically to common and stereotypic threats (Silove, 1999). However, as populations grew, migration occurred into varied environments, causing expanding groups to encounter new threats to survival. Because the neocortex developed later, early human ancestors lacked the ability to make cognitive discriminations about risks associated with novel threats; they had yet to develop the capacity for language which would have fostered the communication about new sources of danger to fellow group members (Cantor, 2005). Hominids spread to new environments and terrains, natural selection favored the reproductive success of those who learned most efficiently to identify and avoid new sources of threat. As such, PTSD likely reflects defensive behaviors that were adaptive in our ancestral past and have arisen via natural selection, but that become maladaptive due to the level of the responses one is experiencing (Cantor, 2005).

One of the acknowledged limitations of evolutionary theories is their inability to direct empirical testing and dearth of discovered “psychological fossils”. It is possible that PTSD may be a spandrel and has no relationship to survival and reproductive success (Silove, 1999). The term spandrel denotes a structure in biology that may have developed independently of any

survival advantage to the species (Gould & Lewontin, 1979). Therefore, it is possible that PTSD is a historically modern response to trauma.

Extant historical texts reveal numerous individuals with behavior consistent with what we now call PTSD symptoms, with reported cases of agitation, dissociation, or terrifying nightmares (see Birmes, Hatton, Brunet, & Schmitt, 2003 for a review). Ancient literary sources suggest that most such psychopathology is associated with trauma and war. The earliest evidence of PTSD symptoms is even found in the world's oldest written literature, from the *Epic of Gilgamesh* to *The Iliad* and *The Odyssey* (Birmes et al, 2003). Early historical writers reported cases of agitation, terrifying nightmares, and other unusual behavior in the heroes of their stories. It was because of these stories that early philosophers developed their theories of human nature and emotion.

During the 17th century, Rene Descartes observed that events that caused significant fear can change human behavior even after the event had ended (Birmes et al., 2003). During the 18th century, the main references about psychological reactions to trauma were restricted to philosophical or literary pieces. Eventually, medical doctors became more aware of the impact trauma had on the mind; especially war (Briole, Lebigot, & Lafont, 1998). Several decades later, the American Civil War provided an opportunity for observing cases of posttrauma psychosis (Turnball, 1998).

The American Civil War provided an abundance of historical accounts demonstrating the existence of PTSD (Tomb, 1994; Fellman, Gordon, & Sutherland, 2014). Although over a million Americans were killed in this conflict, this number fails to represent the psychological and emotional scars that were inflicted during this time of war. The Civil War occurred in a time when modern scientific understanding and concern for mental wellbeing did not exist. At the end

of the war, returning soldiers were afflicted with terrible physical wounds, malaria, chronic diarrhea, and often psychologically destroyed (Fellman et al., 2014). The Civil War produced large number of soldiers who complained of weakness, heart palpitations, and chest pain, which were attributed to physical stress and referred to as *soldier's heart, effort syndrome, DaCosta's syndrome,* and *irritable heart* (Tomb, 1994). The previous isolated anecdotes of posttraumatic stress symptoms during the Civil War became a medical phenomenon during and after World War I (WWI).

WWI produced the term *shell shock* to describe PTSD symptomatology, which highlighted the physiological arousal seen in combat veterans (Andreasen, 2010). The shell shock theory proposed the idea of predisposition, weakened adaptive capacities, and a shocked nervous system and mind. Shell shock was the result of a brain injury from artillery warfare. When uninjured soldiers expressed the symptoms, it was recognized as a physical reaction to combat stress (e.g., memory loss, neurosis, fatigue, dizziness). The circumstances of each war can affect the psyche of soldiers in different ways. World War I was fought in the trenches of Europe and was marked with artillery bombardments, which gave rise to terms "shell shock" and "gas hysteria," a fear of a poisonous gas attack.

During the Second World War (WWII), medical and psychological observations focused on the psychophysiological responses and loss of impulse control of those affected by trauma (Andreasen, 2010). Individuals who survived concentration camps showed severe biological, psychological and social consequences (Van de Kolk et al., 1996). Shortly after WWII the first edition of the DSM was published, which included the diagnosis of gross-stress reaction (Andreasen, 2010). This was an ill-defined diagnosis for classifying individuals who had been psychologically affected by exposure to stress. The major problem with gross stress reaction was

that it was considered a temporary diagnosis, which would later become a neurotic reaction if the symptoms persevered. Gross-stress reaction was primarily combat-focused and included current characteristic of trauma induced stress.

Surprisingly, the DSM-II deleted gross stress reaction, leaving clinicians without options for diagnosing individuals who had catastrophic experiences. The deletion of gross-stress reaction may have occurred because of the period of peace between WWII and the Vietnam War (Andreasen, 2010). By the time the third revisions to the DSM were occurring, the psychological repercussions of the Vietnam War were unfolding. In the 1970s, many clinicians recognized the need for a new diagnosis for patients suffering from severe and chronic symptoms preceded by exposure to traumatic events. From Gilgamesh to the First World War, more than 3000 years were necessary for medicine and science to establish a structured nosology of traumatic stress (Birmes et al., 2003). These events paved the way for the inclusion of PTSD in the DSM-III.

The current conceptualization of PTSD first appeared in the DSM-III and encompassed a variety of traumatic stressors in addition to combat, including natural disasters, mass catastrophes, industrial accidents, and death camps (3rd ed.; DSM-III; APA, 1980). The main criteria for PTSD was re-experiencing the trauma, avoidance of things associated with the trauma, and increased arousal. The traumatic event criterion specified that the trauma must be so extreme that it would cause marked distress in anyone. The traumatic event criterion was expanded to include other traumas (APA, 1980). The most recent changes to the event criterion specify that simply witnessing a traumatic event happening to others or learning about a traumatic event happening to love ones is sufficient to cause posttraumatic symptoms (4th ed., text rev.; DSM-IV-TR; APA, 2000). In the current edition, the DSM-5, it was expanded to

include events that are recurrent traumatic experiences (e.g., first responders, emergency room staff).

Recent conceptualizations of PTSD describe it as a psychological disorder that develops following the experience or witnessing of life-threatening, sexually violating, or seriously injurious events (APA, 2000). Learning that traumatic events happening to a loved one can also produce PTSD symptoms (Cantor, 2007). Some of the most common events associated with PTSD included sexual assault, accident or fire, violent death of a family member or friend, and witnessing physical assault. In the general population, sexual assault is the most common traumatic event leading to PTSD symptoms (Kilpatrick et al., 2003). PTSD symptomatology includes involuntarily re-experiencing the event, avoiding event-related situations, negative moods and thought patterns, and hyperarousal (5th ed.; DSM-5; APA, 2013). In addition to these symptoms, some individuals experience dissociation from their environment, such as depersonalization or derealization of current surroundings. However, experiencing these types of trauma does not always lead to psychopathology and development of PTSD (Tedeschi & Calhoun 1995; Zoellner & Maercker, 2006).

Conventional Theories of PTSD

Traditional theories explaining PTSD development include cognitive, behavioral, and developmental perspectives. Recent technological advancements have provided new insight into the physiological underpinnings of trauma.

Cognitive & Behavioral Theory

A significant amount of the behavioral research on PTSD is focused on examining the impact of uncontrollable and unpredictable events on the development of pathology (Cantor, 2005). Laboratory animals exposed to uncontrollable and unpredictable events develop PTSD

symptoms (Foa et al., 1989). For example, animals will display marked distress upon re-exposure to trauma stimuli (Cantor, 2005). Non-primate mammals display an inverse relationship between an animal's control over their environment and fear response (Mowrer & Viek, 1948). Rats exposed to inescapable shock showed deficits in escape-avoidance learning, which suggests that psychopathology is the product of instrumental learning and classical conditioning (Jackson, Alexander, & Maier, 1980).

Exposing dogs to inescapable socks interfered with subsequent escape-avoidance responses in other situations (Overmier & Seligman, 1967; Seligman & Maier, 1967). When the dogs were released from their harness they behaved passively and failed to perform escape behavior, this phenomenon was called learned helplessness (Seligman, 1975). Controllability and predictability is central to learned helplessness (Cantor, 2005), and fundamental to understanding PTSD (Foa et al., 1992). An organism that has control over an event can predict when the event will end (Wortman & Brehm, 1975). While some predictable events cannot be controlled, signals preceding the event may allow the organism to prepare for the aversive event. Control and predictability over the termination of the event greatly reduces conditioning of fear responses (Mineka & Zinbarg, 1991).

Cognitive schemas before, during, and after trauma encompass the basis of early cognitive perspectives of PTSD. Posttraumatic stress disorder is a violation of existing schemas and destroying belief systems. Trauma affects an individual's schemas of self-worth, others' trustworthiness, and worldview (McCann & Pearlman, 1990). Traumatic events that are strong enough to change fundamental belief systems will usually result in PTSD symptoms.

According to Emotional Processing theory (Foa, Skeketee, & Rothbaum, 1989) individuals habituate to unpleasant memories causing emotional reactions to decrease. When

habituation does not occur, this fosters the development of PTSD (Foa et al., 1989). Irrational fears and distorted beliefs about the traumatic event lead to avoidance of reminders, which is consistent with PTSD symptomatology.

According to the behavioral-cognitive inhibition theory, PTSD is the product of dysfunctional memories based upon faulty appraisals of the trauma-centered memories (Paunovic, 2010). These faulty appraisals and memories affect current appraisals, memories, and functionality. Current behavioral responses and appraisals are learned from previous appraisals and responses surrounding traumatic events, which lead to impairments in functionality and quality of life.

Development and Attachment Theories

The earlier the trauma occurs developmentally; the more detrimental subsequent functioning is during childhood and into adulthood. From a developmental perspective, a child exposed to trauma would acquire a perception that the environment is untrustworthy and unsafe and they will expect to be betrayed, victimized, and feel unsafe generally (Cantor, 2005). These perceptions and expectations foster an inability for self-regulation, disorganized behavior and thought patterns, anger, and defiant behaviors (van der Kolk, 2005). This pattern of behavior among maltreated children is often understood in terms of attachment theory (Hornor, 2009).

Attachment patterns formed during childhood provide the framework for which the individuals develop future relationships. Individuals exposed to trauma develop maladaptive attachments that continue into adulthood (Allen, Coyen, & Huntoon, 1998; Pearlman & Courtois, 2005). Caregiving behaviors that are neglectful or violent can cause children to be hypervigilant and acquire an anxious attachment. Insecure and disorganized attachment to a caregiver's fosters

anxious and avoidant relational behavioral responses. Trauma-exposed children display disorganized attachments, inability for self-regulation, and dissociative responses.

Physiological Perspectives

Physiological reactivity to exposure to cues similar to a traumatic event is a characteristic feature of PTSD (Sack, Hopper, & Lamprecht, 2004). Individuals with PTSD report the symptoms are beyond their capacity to regulate and control; the inability to regulate levels of arousal and distress is central to PTSD (Frewen & Lanius, 2006). Individuals exposed to severe trauma (i.e., childhood sexual abuse, recurrent domestic violence) may have their neural capacities for regulating arousal levels compromised. PTSD is a disorder of generalized emotional dysregulation (Sack, Hopper, & Lamprecht, 2004). The psychophysiological symptoms of PTSD include hyperarousal (e.g., excessive startle reflex, hypervigilance) and exaggerated reactions to trauma cues, which indicate a dysfunctional physiological stress system (Hauschildt, Peters, Moritz, & Jelinek, 2011).

PTSD has been linked to impaired hypothalamic-pituitary-adrenal axis functioning (Yehuda, 2003), impaired emotional regulation (Lanius, Bluhm, Lanius, & Pain, 2006), and altered gene expression (Yehuda & Bierer, 2007). These changes in physiological functioning produce effects on stress response, physical health, and the capacity to regulate to ongoing stressors from the environment. It is unknown the impact these dysfunctions have after PTSD symptoms are resolved. The neurobiological effects of severe stress can be long-lasting (Cotella, Mestres Lascano, Franchioni, Levin, & Suarez, 2013). People with PTSD may experience persistent alteration of fundamental physiological process that underpin stress response and this can extend beyond remission of symptoms, which can then affect one’s capacity to function.

There is an association between PTSD and elevations in basal heart rate and diastolic blood pressure (Buckley & Kaloupek, 2001). Elevations of cardiac activity may reflect changes in the cardiovascular system in response from repeated cardiovascular stress (Fredrikson & Matthews, 1990). Individuals with PTSD exhibit exaggerated cardiovascular responses to trauma cues (Blanchard & Buckley, 1999). Cardiovascular responses to trauma cues are mediated by the autonomic nervous system, which produces elevated catecholamine levels after exposure to stressors (Blanchard, Kolb, Prins, Gates, & McCoy, 1991).

PTSD populations have on average heart rate resting values approximately five beats per minute faster than control groups (Buckley & Kaloupek, 2001). Individuals with PTSD are more reactive to a variety of stressors, which could influence their baseline measures. The mechanisms and processes by which the amygdala and other structures of the brain affect the heart have been well studied in humans with PTSD (Tomb, 1994; Buckley & Kaloupek, 2001; Blanchard et al., 1991; Sack et al., 2004). Higher resting heart rate and greater heart rate activity to trauma cues in individuals with PTSD have been explained as over activation of the ANS (Sack et al., 2004).

Autonomic Arousal Systems

Sympathetic nervous system (SNS) activation has been a prominent focus in the realm of psychophysiology (Domshke, Stevens, Pleiderer, & Gerlack, 2009). Physiological correlates of PTSD symptomatology are commonly studied through the ANS (Kemp, Felmingham, Falconer, Liddell, Bryant, & Williams, 2009). The regulatory features of the ANS contribute to the body's ability to maintain homeostasis. Parasympathetic activity regulated by the ANS and the influence of the vagus nerve has been the topic of recent psychophysiological studies (Dale, Carroll, Galen, Hayes, Webb, & Porges, 2009; Hauschildt et al., 2011; Park, Bavel, Vasey, & Thayer,

2012). The vagus nerve is the tenth of twelve cranial nerves that originate in the medulla oblongata, where various autonomic and visceral components of the brain reside. The vagus nerve extends from the pharyngeal and laryngeal branches, lungs, heart, and subthoracic renal and gastric organs. The vagus nerve plays a significant role in regulation of the ANS parasympathetic division (Porges, 2011).

Increased understanding of the vagus influence and parasympathetic activation illuminates our understanding of trauma-related stress (Blechert, Michael, Grossman, Lajtman, & Wilhelm 2007; Lewis, Furman, McCool, & Porges, 2012). The influence the vagus nerve on the ANS is called vagal tone and is influenced by the parasympathetic branch of the ANS. Increased vagal tone is associated with an increased ability to deal with stress (Bornestein & Suess, 2000; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996; Stifter & Fox, 1990). Decreased vagal tone leads to a decreased ability to cope with stress (Porges, 2011). Therefore, vagal tone is a physiological marker of one's ability to respond to environmental stressors and is a biomarker to differentially discriminate between different psychopathologies.

Chapter 2: The Polyvagal Theory

The polyvagal theory - a theory describing differentiated vagal systems within mammals that represent phylogenetic adaptive reactions to challenge - describes how and why autonomic processes correspond to affective experiences (Porges, 2007; 2011). When these regulatory processes are dysfunctional, abnormal behavioral, and affective patterns follow. Impaired difficulties with self-regulation are characteristic of trauma-related psychopathology (Blechert, Michael, Grossman, Lajtman, & Wilhelm 2007; Lewis et al., 2012). Cognitive and behavioral responses reflect the dysregulatory patterns of trauma symptomatology. The polyvagal theory is a comprehensive explanation that fits other perspectives in explaining posttrauma experiences. The polyvagal theory provides methodological approaches and theoretical perspectives to study autonomic functioning and emotional regulation. This theory provides framework for understanding pathological deficits of PTSD and potentially PTG. Individuals with PTSD do not display vagal brake in a resting period after a viewing trauma specific stimuli (Sahar, Shalev, & Porges, 2001). To date, no studies have investigated the self-regulatory mechanisms of PTG and vagal brake response after viewing trauma specific stimuli.

As put forth by Stephen Porges (2001; 2007), the polyvagal theory (PVT) proposes that the evolution of the mammalian ANS provides the neurophysiological mechanisms for the emotional processes of social behavior. This theory links the evolution of the ANS to affective experience, emotional expression, facial expressions and head movements, vocalizations, and social behavior (Porges, 2011). The term *polyvagal* itself indicates that there are multiple vagal pathways, and so an understanding of the ANS, including afferent and efferent pathways, target organs, and the bidirectional communication between the central nervous system (CNS) and the heart, is needed (Porges, 1995).

PVT proposes three distinct phylogenetic subsystems of development of the ANS. The three autonomic subsystems are social communication (i.e., the Social Engagement System), mobilization (e.g., fight-flight), and immobilization (e.g., feigning death, vasovagal response, and behavioral shutdown; Porges, 1995). The social communication system relies on the myelinated vagus, which calms behavioral and physiological states by inhibiting sympathetic influences to the heart and suppressing the HPA axis (Porges, 2011). The mobilization system relies on the functioning of the SNS, which removes the vagal brake and allows for fight-flight behaviors (Porges, 2007). The immobilization system is dependent on the unmyelinated vagus, which regulates heart rate deceleration and freezing behavior. The three subsystems provide adaptive physiological and behavioral responses to various types of environments and stimuli, from safe and relaxed to dangerous, stressful, or life-threatening events.

The PVT rests on the five following premises regarding the anatomy and physiology of the vagus in mammals (Porges, 2011). First, the vagus is family of neural pathways originating in several areas of the brain stem. Second, there are several branches of the vagus. Third, the vagus is not only an efferent pathway; indeed, some 80% of the vagal fibers are afferent (Agostoni, Chinnock, DeBurgh Daly, & Murray, 1957). Fourth, the vagus is lateralized, with nerve trunks originating in the left and right sides of the brainstem. Fifth, the vagus is asymmetrical, with the different sides performing distinct tasks, such as the right vagus being involved in regulation of the heart. The origin in different areas of the brainstem mean that the different vagal members that make up the vagus play unique roles in the regulation of visceral function (Grossman & Kollai, 1993; Porges, 1995a), even in the same target organ (Berthoud & Neuhuber, 2000).

For example, in mammals there are two functionally distinct vagal motor systems: the left pathway originating in the dorsal motor nucleus of the vagus (DMNX) and the right pathway originating in the nucleus ambiguous (NA). Both pathways, in turn, innervate the sinoatrial node (SA), which is the primary cardiac pacemaker. The NA influence on the SA is referred to as vagal tone (Park et al., 2012) and is seen across many types of animals, the NA pathway is unique to mammalian orienting response (Porges, 2011). Vagal tone is removed/decreased to support mobilization (e.g., fight-flight behaviors) and maintained/increased to support social engagement behaviors. Vagal tone is related to behavioral and psychological processes along a continuum from prosocial interactions to fight-flight behaviors.

During mammalian, orienting responses there is an increase in vagal outflow from the DMNX area that produces heart rate deceleration (e.g., causing neurogenic bradycardia) while the nucleus ambiguous suppresses Respiratory Sinus Arrhythmia (RSA; Porges, 1995b). RSA is a naturally occurring variation in heart rate that occurs during inhalation and expiration of the breathing cycle (Porges, 2007). The time between heartbeats is shortened during inhalation and lengthened during expiration. RSA is an accurate measurement of the influence of the myelinated vagal efferent pathways on the sinoatrial node, and as such provides a noninvasive indirect measurement of the vagal tone, which is the influence of the myelinated vagus on the heart (Porges, 1995b).

RSA is an accurate measurement of the influence of the myelinated vagal efferent pathways on the sinoatrial node, and as such provides a measurement of the vagal tone. Reliable suppression of RSA is an index of positive social and emotional regulation, while low levels and unreliable RSA regulation indicates poor social and emotional regulation, and in some cases, has been associated with psychiatric disorders (Porges, 1995a). For example, children with

behavioral problems have lower RSA and suppressed RSA during empirical tests, while children with reliable and stable RSA suppression displayed fewer behavioral problems, decreased negative affectivity, and better social skills (Blair & Peters, 2003). High RSA appears to buffer children from the effects of marital hostility (Gottman, Jacobson, Rushe, & Shortt, 1995) and predicts greater self-control and decreased negative emotional arousal in adults (Fabes & Eisenberg, 1997). Poor RSA regulation in adults has also been associated with greater social anxiety and lower RSA is associated with more defensiveness (Movius & Allen, 2005). Individuals with clinical levels of anxiety exhibit lower and less suppression of RSA (Friedman & Thayer, 1998). Importantly, RSA parallels positive effects of treatment, with increases in RSA following treatment for depression (Rottenberg, Gross, & Gotlib, 2005). Outside of mental health, physical factors including cardiovascular risk (Hayano et al., 1990), diabetes, and obesity are related to low RSA (Quilliot et al., 2001).

Psychological Threat Detection

How does the nervous system know when the environment is safe or dangerous and what neural mechanisms evaluate risk? To efficiently switch from social engagement to defensive strategies the nervous system needs to perform two important tasks: a) assess risk to determine if the environment is safe; and b) to suppress the limbic system that regulates fight-flight or freeze behaviors (Porges, 2007, 2011). The nervous system, through continual processing of information from the environment, evaluates risk. The neural process of evaluating risk does not require conscious awareness and involves subcortical limbic structures (Morris et al., 1998). Such *neuroception* is the capability of distinguishing environmental features that are life threatening, dangerous, or safe (Porges, 2011).

When an organism perceives the environment to be safe, the bodily state is regulated to promote growth and restoration (Porges, 1995; 2011). This process occurs through the influence of the myelinated vagal pathways on the cardiac pacemaker that inhibits fight-flight responses, and suppresses stress response system of the HPA-axis (Porges, 2007). The brainstem nuclei that regulate the myelinated vagus are linked to the nuclei that regulate the muscles of the face and the head. This link produces a bidirectional communication system between bodily states and social engagement behaviors.

Neuroception also involves detectors in the temporal cortex, these structures respond to voice, hand, and face movements and influence limbic reactivity (Porges, 2007). The nervous system evaluates risk and matches physiological states with the actual risk of the environment. When the environment is perceived as safe, the defensive limbic structures are suppressed enabling social engagement and calm states (Porges, 1995). However, some individuals perceive the environment as dangerous when it is safe. This mismatch produces physiological states that support fight-flight or freeze behaviors, rather than social engagement behaviors, in safe environments. The SES allows for social communication only when the defensive circuits are inhibited (Porges, 2011). Neuroception is the neural process that allows for mammals to engage in social behaviors and mediates the expression affect regulation and homeostasis.

The three physiological and behavioral subsystems – immobilization, mobilization, and the Social Engagement System - respond to stress in a determined hierarchy consistent with the Jacksonian principle of dissolution (Porges, 2007). Jackson proposed that newer neural circuits inhibited older neural circuits and when higher circuits are unsuccessful, the older circuits take over (Jackson, 1884). Through the hierarchy of adaptive responses, the newest circuits are used first, and if that circuit fails to provide safety the older circuits are recruited. Within the

polyvagal system, the subsystems from oldest to newest are immobilization, mobilization, and the Social Engagement System (Porges, 2011).

The Social Engagement System, PTSD, and Posttraumatic Growth

The phylogenetic origin of the social engagement system is associated with the ANS (Porges, 2011). As the muscles involved in head movement and the muscles of the face intertwined into social engagement structures, a new component of the ANS regulated by the NA through the myelinated vagus evolved (Porges, 2007). The Social Engagement System (SES) controls upper motor neurons of the cortex, regulates lower motor neurons in the brainstem nuclei that control facial muscle for emotional expression, eyelid opening for looking, middle ear muscles for extracting human voices, muscles for mastication for ingestion, muscles of the larynx and pharynx for intonation, and head turning muscles for social gesture and orientation (Porges, 2011).

The vertebrate ANS follows three stages of development, with each stage having various adaptive functions (Porges, 2011). As a reminder, those stages are immobilization, mobilization, and the Social Engagement System. The neural circuitry in each stage supports different behavior, with the phylogenetically newer myelinated vagus capable of supporting social engagement behavior (Porges, 2011). When the SES is compromised, this causes a change in autonomic regulation that is characterized by reduced influence of the myelinated vagus on the heart. The removal of the vagal brake then allows for the expression of the two phylogenetically older neural systems. These two older systems allow for mobilization behaviors of fight-flight through the sympathetic nervous system, or immobilization behaviors of breezing, death feigning, and behavioral shut down via the unmyelinated vagus (Porges, 2007).

Autonomic functioning as a feature of PTSD has been examined through heart activity, skin conductance, and breathing patterns (Cantor, 2005). Sympathetic ANS activation is evidence in decreased heart-rate variability, increased skin conductance, and attenuated respiration (Porges, 2007). A healthy functioning ANS system regulates physiological arousal through an opponent-process method involved the sympathetic and parasympathetic nervous systems. The sympathetic nervous system arouses the body by increasing heart rate, attenuating respiration, and producing perspiration (Kreibig, 2010). The sympathetic activation is often called the fight-flight response. The parasympathetic nervous system counterbalances the processes of the sympathetic system by suppressing excitatory functions and returning the body to homeostasis. Diagnostic features of PTSD rely on sympathetic activation or hyperarousal. The literature on PTSD has focused specifically on sympathetic activation (Fani et al., 2012; Orr & Roth, 2000; McTeague, Lange, Laplante, Cuthbert, Shumen, & Bradley, 2010).

Under current diagnostic criteria for PTSD, a person must be experiencing hyperarousal after experience a traumatic event (APA, 2013). Irritability, angry outbursts, hypervigilance, exaggerated startle response, and difficulty concentrating characterize hyperarousal. Prolonged sympathetic activation and a lack of parasympathetic activation can translate to autonomic dysregulation, which is a common feature observed in PTSD populations (Tan, Dao, Farmer, Sutherland, & Gevirtz, 2010). The role of cardiac activity in the ANS has been extensively studied in trauma research (Porges, 2007). Abnormalities in heart rate (HR) to trauma related stimuli has been exhibited in a plethora of PTSD samples (Hauschidlt et al., 2011; Sack, Hopper, Lamprecht, 2004; Pole, 1994). Individuals with PTSD exhibit elevated tonic cardiovascular activity (Buckley & Kaloupek, 2001; Pole, 2007) and excessive HR reactivity to trauma reminders (Orr et al., 2004; Pole, 2007). In contrast, some trauma-exposed individuals respond

with a reduced basal HR (hypoarousal) or even dissociation when confronted with trauma cues (Lanius et al., 2006). Individuals with PTSD compared to trauma-exposed individuals without PTSD exhibited amplified heart rate, attenuated respiration, and decreased heart rate variability or RSA (Sack et al., 2004). These differences are exaggerated when individuals are exposed to trauma-specific stimuli. Individuals with PTSD tend to remain physiologically aroused and fail to return to baseline levels (Norte et al., 2012). Therefore, it is evident that individuals with PTSD have a unique physiological response to trauma-specific stimuli; this physiological response may illuminate our understanding of posttraumatic growth and how individuals with PTG respond physiologically to trauma specific stimuli.

Chapter 3: Posttraumatic Growth

Some trauma survivors may experience positive psychological changes after a traumatic event (Tedeschi & Calhoun 1995; Zoellner & Maercker, 2006). Posttraumatic growth (PTG) is defined as the subjective experience of positive psychological change after traumatic experiences. PTG is a meaning-making process where trauma-exposed individuals make sense of the trauma. This process of coping and finding meaning then produces genuine and long-lasting positive change (Tedeschi & Calhoun, 2004). People often report positive outcomes following traumatic or stressful situations, either as a direct result of the event or a product of learning that occurred through their effort to cope with the event (Park, 1998). PTG describes the experience of individuals who not only recover from the trauma by returning to their baseline of functioning, but also because of the trauma develop and benefit from the experience. The beneficial outcomes include increased appreciation of life, improved closeness of intimate relationships, positive spiritual change, setting new priorities, or a sense of increased personal strength (Tedeschi, Park, & Calhoun, 1998). Surprisingly, individuals who have been through two horrific events experience more growth than individuals who have one, and individuals who have three horrific events are stronger than those with only two (Seligman, 2012). This was reflected in a study of imprisoned airmen, where 61% of them reported benefiting psychologically from their imprisonment. The more severe their torture, the greater their reported PTG was (Sledge, Boydstun & Rabe, 1980).

PTG may even be present at a community level, in some interpretations of the research. For example, after mass traumas there is an outpouring of emotional and material support to help survivors cope with disasters (Solnit, 2009). Post-disaster communities are characterized by a high degree of unity, an increase in prosocial behaviors, and a reduction in intergroup conflicts (Solnit, 2009; Mancini et al., 2016). Some survivors of mass trauma experience a reduction in

depression and anxiety post-trauma due to increased perceived social support, gains in social resources, and altruistic behavior of others due to the traumatic event (Vollhardt, 2009). After mass trauma, communities unite, grievances are forgiven, and feelings of unity and common purpose increase, all of which promote wellbeing (Mancini, Littleton, & Grills, 2016). The pain of mass trauma can disrupt preexisting distress by providing a sense of relief from current stressors.

Theories of Posttraumatic Growth

There are several different explanations of why and how people experience PTG. One model of PTG proposes that it is the product of attempts to cope with stressful and traumatic experiences. Stressful events are not always negative but can allow for personal development (Aldwin, Levenson, & Spiro, 1994). Different coping strategies result in different outcomes after a traumatic event. In this model, homeostatic coping leads individuals back to their pretrauma baseline levels of functioning; transformational negative coping leads to lower psychological functioning and wellbeing; and transformational positive coping leads to higher levels of psychological coping. As such, PTG occurs when an individual undergoes transformational positive coping after a traumatic event.

According to Shaefer and Moos' (1992) model of life crises, factors of personal growth, environmental, and personal differences influence the life of the individual post-trauma. The environmental factors include support from family, friends and social environment, personal relationships, and financial resources. The interpersonal factors include differences in resilience, optimism, self-confidence, self-efficacy, motivation, health status, and previous experience with trauma (Zoellner & Maercker, 2006). These factors affect coping responses and cognitive appraisals, which impact the outcome after the trauma. All components of the model are directed

by feedback loops. Shaefer and Moos (1992) propose that for growth to occur an individual will need to use active coping approaches and have the right environmental and personal factors.

Per Tedeschi and Calhoun (2004), PTG occurs after a traumatic event of catastrophic proportions destroys fundamental elements of a person's goals and worldview. The traumatic event causes significant challenges to beliefs, goals, and the ability to regulate and manage emotional distress. The emotional distress causes a pattern of recurrent rumination and attempt to engage in behavior to reduce the emotional distress. The rumination results in actively thinking about the trauma and subsequent issues. Eventually the rumination evolves into more deliberate thinking about the trauma and how it changes one's life. Rumination becomes a constructive process and plays a vital role in the development of growth. It is through this process that PTG occurs and produces changes in one's beliefs, goals, behaviors, and identity.

In summary, these models of PTG (Shaefer & Moos, 1992; Tedeschi & Calhoun, 2004) propose that PTG is predicted by a person's pre-trauma characteristics, schemas, goals and beliefs as well as by factors of rumination, schema changes, and enduring stress. Unfortunately, these proposed models are difficult to test empirically due to the vague definitions of many of the predictors, except for enduring stress.

Some theorists have explained PTG as an individual's attempt to construe meaning from the traumatic experience (Davis, Nolen-Hoeksema, & Larson, 1998). This model is based on the idea that people believe that events in their lives are controllable, comprehensible, and nonrandom. In response to trauma individuals will attempt to engage in the adaptive and important task of making meaning of the event (Davis et al., 1998). PTG is one of the two possible results of making meaning of the event. For PTG to occur individuals must engage in an important construal of meaning to determine if they can find beneficial attributions. If a trauma-

exposed individual can answer the question “What for?” after the trauma and find a subjective perception of personal growth, then PTG will occur.

Park and Folkman (1997) differentiate between situational and global meaning in the context of stress and coping. Global meaning includes a person’s beliefs and goals. Situational meaning is the interaction between a person’s global meaning and their interaction with the environment. A traumatic event damages their global meaning, thus starting the meaning-making process. Through the coping process the individual must integrate situational meaning and their understanding of the trauma with their global meaning. PTG will occur when the individual creates meaning for the event (Zoellner & Maercker, 2006). For example, finding beneficial outcomes from the trauma would fall into the category of assimilation. In contrast, an individual could completely change their philosophy of life because of the trauma, which would cause changes in their global meaning.

Some theorists have attempted to explain the development of PTG through the perspective of differences in explanatory styles. Explanatory style is defined as habitual ways of explaining the causes of events and the pattern of explanations (Peterson & Vaidya, 1995; Ho, Chan, Yau, & Yeung, 2011). An optimistic explanatory style of negative events is the tendency to explain the events, as having external, unstable, and specific causes. A pessimistic explanatory style for negative events is the habit of explaining negative events as internal, stable, and global. The explanatory style an individual espouses affects how one attributes the causes of events, which affects subsequent cognitive processing. Individuals with optimistic explanatory styles will report less posttraumatic stress symptoms and more perceived positive changes after a traumatic event (Ho et al., 2011). Individuals with a pessimistic explanatory style for negative events are more likely to develop depression and anxiety (Mineka, Pury, & Luten, 1995; Robins

& Hayes, 1995). Pessimistic explanatory styles predict the development of PTSD after traumatic events (Joseph, Yule, & Wiliams, 1993). Explanatory style for good events, but not for bad events, is associated with PTG (Ho et al., 2011). Optimistic explanatory styles for good events enables the individual to obtain understanding of self-reaction in a trauma leading to higher self-perceived growth (Gohm & Clore, 2002). Interestingly, explanatory style for bad events does not predict self-perceived PTG, but does predict psychological distress associated with PTSD, depression, and anxiety (Ho et al., 2011). This would suggest that after a traumatic event an individual's explanatory style would not influence their development of PTG, but could for PTSD.

The relationship between PTG and posttrauma adjustment is more evident over time (Shakespeare-Finish & Lurie-Beck, 2014). A study conducted with people from Israel showed that PTG is associated with posttraumatic stress at 6 and 12 months, with higher levels of posttraumatic stress symptoms correlated with higher levels of PTG at both time periods (Hall, Saltzman, Cenetti, & Hobfoll, 2015). These results are consistent with the theory that PTG is the product of the struggle with trauma and distress, per Tedeschi and Calhoun's theory (2004). A recent meta-analysis also revealed an overall positive relationship between PTSD and PTG (Shakespeare-Finsh & Lurie-Beck, 2014). One longitudinal study with prisoners of war found that PTSD predicted PTG over 12 years later (Erbes et al, 2005). There is an interesting interaction effect with time and trauma severity that act as a moderator effect of PTG on psychological adjustment (Zoellner & Maercker, 2006).

PTG as a coping strategy denotes a significant beneficial change in emotional, psychological, and cognitive life that is completely different from PTSD. According to this perspective, PTSD and PTG are distinct and independent constructs representing separate

dimensions, so these constructs are not regarded as two ends of the same continuum of adaptation to trauma (Zoellner & Maercker, 2006). PTG is also not regarded as the same thing as a decrease in distress or an increase in wellbeing (Tedeschi & Calhoun, 2004). In this way, PTG can reportedly coexist with emotional distress for some people.

PTG has also been conceptualized as a positive illusion (Zoellner & Maercker, 2006) with an adaptive function for psychological functioning. The perception of PTG helps individuals cope with the trauma through self-enhancing appraisals (Taylor & Armor, 1996). But, positive relationships between PTG and psychological wellbeing are usually only found in studies with nonstandarized assessments of PTG, thus producing low reliability and validity (Zoellner & Maercker, 2006). The studies that used validated instruments of PTG usually do not find any relationship between PTG and psychological wellbeing.

As can be seen from this brief review, PTG has been difficult to study. There are several reasons why, with the first being that to document growth, researchers must assess exposed persons before the stressor or trauma occurs. Such pre-event assessments are rare in trauma research and most of the research conducted is based on data obtained after the traumatic event has happened. This is problematic for several reasons. In the absence of pre-traumatic assessments, it is impossible to determine the precise impact the trauma has on people's functioning. A second key problem is that a clear majority of previous research has examined the average response to acute stress, and, on average people will experience an increase in distress following a traumatic event (Bonanno et al., 2010). Thus, the possibility of improvement or of other responses will be necessarily be obscured in studies that examine average longitudinal response to trauma. These methodological difficulties have led, in part, to several controversies around PTG.

Controversy Surrounding Posttraumatic Growth

There is significant controversy regarding the significance of PTG for recovery after trauma and the relationship with posttraumatic stress (Linley & Joseph, 2004; Zoellner & Maercker, 2006). Different theorists have proposed diverse models of PTG, with it conceptualized either as an outcome from the traumatic event (Shaefer & Moos, 1992, 1998; Tedeschi & Calhoun, 1995, 2004) or a coping strategy (Affleck & Tennen 1996). Further, there is speculation if PTG is genuine positive change or a maladaptive self-deceptive coping mechanism that prevents healing from occurring (Frazier et al., 2009; Hall, Hobfoll, Canetti, Johnson, & Galea, 2009).

The first controversy concerns whether reported "growth" following trauma represents actual growth (Tedeschi, Park, & Calhoun, 1998), a motivated positive illusion (Taylor, 1983), or a coping process (McMillen & Cook, 2003). Evidence of a significant growth consists of numerous reports of growth from survivors of traumatic events (Tedeschi & Calhoun, 1995). Individuals who report growth after a traumatic event are comparing their current state to the state after the event, which appears to be growth, but could be a return to baseline levels of functioning (McFarland & Alvaro, 2000). Individuals who have recently lost a loved one and are primed to think about their loss report more meaning in life than those who are not primed in such a way (Davis & McKearney, 2003). This would suggest growth following traumatic event is a self-protective strategy for coping with the event. The evidence suggests growth after adversity reflects something other than actual changes in life appreciation, priorities, or relationships with other people.

The second controversy surrounds how the construct of PTG is measured and the validity of the psychometric measures of growth. Posttraumatic growth is measured by asking individuals

after a traumatic event if they perceive they have grown because of the event. Typically, however, self-report questionnaires of personal change are viewed with caution and skepticism for several reasons (Nolen-Hoeksema & Davis, 2004). For one, perceived changes in personal attributes are weak predictors of actual change or growth (Robins, Noftle, Trzesniewski, & Roberts, 2005). Self-perceived changes in personality can be misperceptions, and such misperceptions have been documented over as little as three-month periods of time (Costa & McCrae, 1989; Wilson & Ross, 2000). Studies of relationship growth demonstrate that for couples who report growth in their relationships – just as participants in studies of PTG report growth – prospective ratings reveal no increases and even show declines in relationship strength and quality (Karney & Coombs, 2000; Kirkpatrick & Hazan, 1994).

Another concern is that perceived growth is associated with increased distress, whereas actual growth was associated with decreased distress (Zoellner & Maercker, 2006; Frazier et al., 2009). Perceived PTG as measured by the Posttraumatic Growth Inventory (PTGI) does not appear to measure actual growth from pre- to post-trauma. PTGI scores were unrelated to most measures of actual growth in positive relationships, meaning in life, gratitude, and life satisfaction (Frazier et al., 2009). Perceived growth was associated with positive reinterpretation coping, which suggests that the PGI measures something different from actual growth. This is problematic because the PTGI is the standardized measurement set forth by APA to measure PTG.

Several studies have also found significant *negative* relationships between PTG and psychological wellbeing and adjustment and significant positive relationships between PTG and psychological distress (Zoellner & Maercker, 2006). It appears that PTG could be a maladaptive coping mechanism leading to more difficulty in overcoming traumatic experiences. The positive

correlation between PTG and distress point to maladaptive cognitive processes involved in self-perceived PTG. Posttraumatic growth may be an unfavorable psychological mechanism and prevents real healing from occurring. PTG and PTSD may operate in a loop, providing positive feedback in which they mutually promote each other, delaying the recovery of those exposed to trauma. In support of that idea, one study found no significant relationship between posttraumatic stress at 5 months and PTG 15 months after deployment (Englehard, Lommen, & Sijbrandij, 2014). But, higher levels of self-reported PTG were associated with higher levels of posttraumatic stress 15 months after deployment. This is in line with the idea that PTG is a maladaptive coping mechanism that is negatively associated with mental health (Zoellner, & Maercker, 2006), in direct opposition to the position adopted by people who think it is positive. In other words, early reported PTG was related to later development of posttraumatic stress symptoms and suggest that perceived growth contributes to the development of PTSD symptoms. Initial growth predicted development of PTSD symptoms in deployed soldiers (Engelhard et al, 2010). After the Oslo bombing in 2001, researchers investigated the longitudinal relationship between PTG and PTSD and found conflicting reciprocal effects between PTG and trauma (Blix, Birkeland, Hansen, & Heir, 2016).

The timing at which individuals are asked about their perceived growth after the traumatic events could be fundamental to understanding the bidirectional relationship between PTG and PTSD. Researchers have found that while PTG measured at 10 months was related to subsequent levels of posttraumatic stress, PTG measured at 22 months was not associated with posttraumatic stress (Mancini et al., 2016). This was explained by the fact that PTG measured at 10 months could be a self-deceptive coping mechanism that leads to vulnerability towards

developing PTSD, but the PTG measure at 22 months might reflect a more constructive and genuine growth post-trauma.

Similarly, the longitudinal research about the relationship between PTG and PTSD is conflicted. Some of the research reports a negative longitudinal relationship where early PTG predicted lower levels of PTSD symptoms (Linley, Joseph, & Goodfellow, 2008). Other studies have failed to find a relationship between PTG and PTSD (Phelps, Williams, Raichle, Turner, & Ehde, 2008; Salsman, Segerstrom, Brechting, Carlson, & Andrykowski, 2009). One study examined if posttraumatic stress symptoms predicted subsequent development of PTG, finding that higher levels of posttraumatic stress predicted higher levels of PTG (Lowe, Manove, & Rhodes, 2013). It is apparent there is a need for further research about the course and bidirectional nature of the relationship between PTSD and PTG.

The components and predictive ability of PTG are ambiguous and unclear. The question of what PTG is, if not an adaptive physiological phenomenon, is an interesting one with many implications. One way to examine PTG would be to examine the association between levels of distress, wellbeing, other areas of mental health, and self-regulation. There are many inconsistencies in the literatures and it is unclear what the PTGI measures. There appears to be a large illusory component to PTG and few studies have incorporated methodology that goes beyond self-report. By obtaining a better understanding of how PTSD and PTG are related we can tease apart the unique and dynamic relationship between these two constructs. Through an understanding of the physiological mechanisms behind PTSD the true nature of PTG may become clear.

The Polyvagal Theory may provide answers to some of these elusive questions and how individuals who have been traumatized respond physiologically. Individuals exposed to trauma-

specific stimuli should expect their RSA to parallel shifts in affective states elicited by the stimuli. Elicitation of a negative primary emotion would result in withdrawal of vagal tone along to promote fight-or-flight behaviors. A shift in more pleasant affective states would be associated with an increase in RSA. Theoretically, individuals with PTG should display increased RSA compared to healthy controls and PTSD after withdrawal of vagal tone. However, it is expected that individuals with PTG will not be statistically different than healthy controls on mean RSA.

Chapter 4: Current Study

This study investigated the self-regulatory characteristics of PTG compared to healthy controls and PTSD by examining vagal tone through RSA. This study was the first to examine physiological underpinnings of PTG, as there is no published literature on this topic. Moreover, these physiological indices have direct links to the etiology and developmental course of the construct, in addition to corroborating the symptomology of PTSD and examining if there was physiological evidence for PTG.

Observations of RSA would hypothetically reveal differences between PTG, PTSD, and healthy control groups. RSA indicates the level of cardiac vagal tone from the brainstem NA (i.e., the vagal brake), which was expected to be very low or absent during unpleasant images in all groups. However, during the distraction task following unpleasant images, the control and PTG group's vagal brake should reengage in the post-stress resting period; whereas, vagal brake in the PTSD group was expected to remain unengaged. Furthermore, the PTG group and healthy control should demonstrate greater vagal regulation than the PTSD group across all measurement phases.

Hypotheses

Low RSA was associated with sympathetic activation, which is observed via shorter heart periods (Austin et al., 2007). These effects would be more pronounced in the PTSD group, than in the PTG and control groups.

H_1 = A main effect of group (PTG, PTSD, control) across physiological measures was expected, such that downward trends in RSA and shorter heart periods would be more pronounced in the PTSD group. Furthermore, it was expected that there would be no statistical difference between PTG and control groups.

A high correlation between RSA-change and heart-period-change would hypothetically reveal differences in cardiac vagal oscillations between PTG, PTSD, and control populations. This physiological measurement indicates the level of cardiac vagal tone from the NA (i.e., the vagal brake), which was expected to be absent after viewing aversive images in the PTSD group. During resting period, individuals with PTSD were expected to exhibit more vagal influence than those in the PTG group and the control group. Furthermore, the PTG group was not expected to differ from the control group and that both groups would return to baseline levels of RSA and heart-period.

H_2 = There would be a lower correlation between RSA-change and heart-period-change in the PTSD group verses the PTG and control groups.

There has been a lot of speculation surrounding the Posttraumatic Growth Inventory (PTGI) and if it measures actual growth after traumatic events (Frazier et al., 2009). However, no studies to date have examined if scores on the PTGI are associated with measures of physiological wellbeing. Theoretically, individuals who score highly on the PTGI inventory, indicating growth posttrauma, would also exhibit high RSA and return to baseline after viewing aversive measures. However, it was expected that individuals in the PTG group and the control group would not be statistically different on RSA change and heart period.

H_3 = Individuals in the PTG group would exhibit upward trends in RSA and heart period and will be more pronounced than the PTSD group, but not statistically different than the healthy controls after viewing aversive images.

Method

Participants and Procedure

Students participated in research to earn credit toward their introductory psychology course. Participant's self-reported demographic and diagnostic screening measures via Qualtrics, an online survey system. The PTSD sample was selected to participate in the laboratory protocol if they met or exceeded clinical cut-off scores on the PTSD-Checklist. The PTG sample was selected to participate if they exceeded cut-off scores on the Posttraumatic Growth Inventory (PGTI). The control sample was selected to participate if they were below clinical cut-off scores on all mental health assessments. Research participants were selected from the university subject pool via SONA-Systems, an online research participation recruitment site.

Participants refrained from alcohol, drug use, and caffeinated beverages for four hours before attending the study. Participants who self-reported steroid use, narcotics, and medical illness within three weeks of the study, previous exposure to the photographs used in the study, and frequent exposure to violent computer game or movies were excluded from the study. All participants were 18 years or older and fluent in English.

Table 1
Socio-demographic and clinical characteristics for each group

		Control (n = 8)	PTG (n = 8)	PTSD (n = 7)
Age		*M(SD)*=18.73(.65) Range: 18-28	*M(SD)*=21.4(1.95) Range=18-23	*M(SD)*=26.4(12.1) Range=18-29
Gender				
	Female	n = 8	n = 8	n = 7
	Male	n = 0	n = 0	n = 0
Ethnicity				
White/Caucasian		n = 6	n = 6	n = 6
African American		n = 1	n = 1	n = 1
Asian		n = 0	n = 0	n = 0
Pacific Islander		n = 0	n = 0	n = 0
Native American		n = 1	n = 0	n = 0

Hispanic/Latino	*n* = 0	*n* = 0	*n* = 0
2 or more ethnicities	*n* = 0	*n* = 1	*n* = 0
Education Level			
High school	*n* = 2	*n* = 1	*n* = 2
Some College	*n* = 5	*n* = 5	*n* = 2
Two-Year Degree	*n* = 1	*n* = 2	*n* = 3
Four-Year Degree	*n* = 0	*n* = 0	*n* = 0
Some Graduate Work	*n* = 0	*n* = 0	*n* = 0
Master's Degree	*n* = 0	*n* = 0	*n* = 0
Doctorate	*n* = 0	*n* = 0	*n* = 0
Household Income			
Less than $15k	*n* = 1	*n* = 2	*n* = 3
$15-30k	*n* = 3	*n* = 1	*n* = 1
More than $30k	*n* = 4	*n* = 5	*n* = 3
PTSD Checklist, DSM-IV-TR	*M(SD)*=26.2(5.7)	*M(SD)*=35.75(10.2)	*M(SD)*=62.75(9.1)
Scale Range: 17-85	Range: 17-34	Range: 21-47	Range: 54-76
PTGI	*M(SD)*=51.4(13.2)	*M(SD)*=75.5(4.5)	*M(SD)*=54.2(8.4)
Scale Range: 0-84	(*n* = 8)	(*n* = 8)	(*n* = 7)
Psychotropic Meds			
antidepressant	*n* = 0	*n* = 2	*n* = 5
antianxiety	*n* = 0	*n* = 0	*n* = 2
stimulants	*n* = 0	*n* = 1	*n* = 1
sleepmeds	*n* = 0	*n* = 1	*n* = 3
betablocker	*n* = 0	*n* = 0	*n* = 0
antiseizure	*n* = 0	*n* = 1	*n* = 0
Over-the-counter drowsy	*n* = 3	*n* = 1	*n* = 1
Other Psychotropics	*n* = 0	*n* = 1	*n* = 1
Smoking (< half pack a day)	*n* = 2	*n* = 2	*n* = 1
Marijuana	*n* = 1	*n* = 0	*n* = 1
Alcohol	*n* = 1	*n* = 2	*n* = 4

Physiological Measures. Participants were attached to the ECG amplifier (BIOPAC Systems, Inc., Santa Barbara, CA) using three AG-AgCl electrodes with an 11-mm diameter contact area attached below the clavicles and on the abdomen on the lower left rib. Physiological measurements were recorded throughout the experimental procedure. For the first ten minutes of the study, participants sat quietly while baseline physiological arousal was measured and completed a distraction task. After baseline data was recorded, participants viewed 120 images from the International Affective Pictures System (IAPS; Lang, Bradley, & Cuthbert, 2008) on a desktop computer while ECG data is collected. The photographs were in blocks composed of 60 aversive and 60 pleasant images. Images were selected per the standardized rating set forth by creators of the IAPS (Lang, Bradley, & Cuthbert, 2008). Participants viewed each photograph for 5 seconds. Participants viewed all blocks of images during the experimental procedure in a counterbalanced order. Between each block participants completed a distraction task while physiological data was recorded for five minutes. Following ECG data collection participants were disconnected from the BIOPAC and debriefed on the purpose of the study and thanked for their participation. The experimental protocol lasted approximately an hour. If the participant was still autonomically aroused the researcher conducted deep breathing exercises with them before they left the laboratory.

Instrumentation

Demographic Information. Participants began the screening assessment by answering questions regarding their biological sex, gender affiliation, race, age, socioeconomic status, and previous or current military affiliation. (If participants answered “yes” to military affiliation, they were asked to provide military branch, primary duties, and number of deployments). Questions regarding nicotine consumption, specific substances regularly used, and prescribed medications

(being used currently) were addressed. All participants were 18 years old or older and spoke English.

Posttraumatic Growth. The Posttraumatic Growth Inventory (PTGI), Tedeschi & Calhoun, 1996) is a 21-item scales that assesses the following constructs: new possibilities (α = .84), relating to others (α = .85), personal strength (α = .72), spiritual change (α = .85), and appreciation for life (α = .67). The scale has some utility in determining how successful individuals will be able to cope with the aftermath of trauma by strengthening their perceptions of self and the meaning of events. Cronbach's α coefficients, ranging from .67 to .89, indicate high internal consistency for the five subscales (Tedeschi & Calhoun, 1996). Scores range from 17 to 85, with a clinical cut-off score of 60 and above.

PTSD Assessments. Given the recent change in DSM-5 PTSD criteria, both assessments for DSM-IV-TR and DSM-5 symptom sets were included.

DSM-IV-TR PTSD Checklist. Participants completed the PTSD Checklist for Civilians (PCL-C), a self-report diagnostic screening measure assessing the level to which an individual meets DSM-IV-TR criteria for PTSD (Norris & Hamblen, 2003). Twenty items assess three primary symptoms clusters: Criterion B or re-experiencing the traumatic event (e.g., "Repeated, disturbing memories, thoughts, or images of a stressful experience from the past?"), Criterion C or avoidance of reminders and numbing of responsiveness (e.g., "Avoid activities or situations because they remind you of a stressful experience from the past?" and "Feeling emotionally numb or being unable to have loving feelings for those close to you?") and Criterion D or increased arousal (e.g., "Feeling jumpy or easily startled?"). Participants rate how much each item has bothered them during the last month on a 5-point Likert scale ranging from 1 (not at all) to 5 (extremely). To meet DSM-IV diagnostic criteria, an individual must score a three or higher

on at least one Criterion B item, three Criterion C items, and two Criterion D items (APA, 2000). A total symptom severity score is obtained by summing all 17 items together. Cronbach's α coefficients, ranging from .73 to .85, indicate high internal consistency for the three symptom clusters (Weathers, Litz, Herman, Huska, & Keane, 1994). Scores range from 17 to 85, with a clinical cut-off score of 30 and above, in addition to meeting symptom pattern requirements outlined, to determine diagnostic significance.

DSM-5 PTSD Checklist. This assessment is not differentiated between civilian and military populations, as with the DSM-IV PCL. The new checklist reflects the significant changes made to the diagnosis, except for those in Criterion A (Weathers, Litz, Keane, Palmieri, Marx, & Schnurr, 2013). Overall, wording has changed to reflect the possibility of multiple traumas. The primary change to Criterion B, intrusion symptoms, is the exclusion of the hallucinations and illusions as an intrusion symptom. Criterion C, avoidance symptoms, no longer includes memory loss of the trauma, diminished interest in activities, feelings of detachment, restricted affect, and sense of foreshortened future. However, this symptom set was moved to Criterion D, which also includes symptoms involving negative alterations in cognitions or mood. This criterion is new to the DSM-5, but most of the symptoms come from DSM-IV Criterion C of except for negative beliefs and emotional state and blame. Criterion E, increased arousal symptoms, was previously Criterion D with the addition of reckless or self-destructive behavior. Verbal or physical have been added to specify the typical expression of irritable and angry outburst (APA, 2013). Finally, a dissociative subtype was added. Validity and reliability have not yet been formally examined in published literature. Participants rate how much they have been bothered by each item during the last month on a 5-point Likert scale ranging from 0 (not at all) to 4 (extremely). To meet DSM-5 diagnostic criteria, an individual must score a two

or higher on at least one Criterion B item, one Criterion C item, two Criterion D items, and two Criterion E items (APA, 2013). A total symptom severity score is obtained by summing all 20 items together. Scores range from 0 to 80, with a cut-off score of 38, in addition to meeting symptom pattern requirements, to determine diagnostic significance.

Trauma History Screen. The THS contains a list of traumatic events that fit Criterion A of the current PTSD diagnosis (APA, 2013; Carlson, 2005). Changes in Criterion A from DSM-IV include the broadening of qualifying traumas, such as sexual violence, vicarious traumatic experience, and repeated exposure to trauma. Vicarious trauma can be learning of or watching a loved one experience the threat of death, physical injury, or sexual violence. Because the trauma list has been broadened in the new DSM edition, this will be used to examine Criterion A with both PCL versions. This will be considered in the screening procedures. Participants will indicate whether (Yes or No) they have experienced any of the events. If they respond "Yes" on any events, they will be asked to provide details about each event, including age of occurrence and a description of what happened in their own words. They will also respond to specific questions about the event, such as "When this happened, did anyone get hurt or killed?" and "After this happened, how long were you bothered by it?" In addition to its diagnostic utility, this measure also illuminates how types of trauma affect autonomic regulation and which types are associated with PTG versus PTSD.

General Mental Health Assessment. Participants completed the Behavioral Health Screening Measure (BHSM), a self-report measure designed to detect emotional problems in young adults (Zygowicz, & Saunders, 2003). This was used to screen out potential participants who have emotional problems that would disqualify them from the healthy control group. The BHSM is a 22-item measure that detects a variety of DSM-IV defined symptoms, such a

depression (e.g., “I feel unhappy, sad, or depressed”), anxiety (e.g., “I feel fearful, nervous, or anxious without knowing why”), and substance use problems (e.g., “I feel unhappy or guilty about my drinking or drug use”). Respondents indicate how much each item has distressed or bothered her or him in the past two weeks on a 5-point Likert scale (0 = None of the Time; 4 = All the Time). Scores, ranging from 0-88, are added together to indicate the level of emotional problem. Scores of 16 or above indicate potential clinically significant problems, and was thus used as the clinical cut-off score. The BHSM has a very high internal consistency of .93 (Zygowicz & Saunders, 2003), and is sensitive to specific mental health difficulties.

Generalized Anxiety Assessment. The Penn State Worry Questionnaire (PSWQ) assesses the trait of worry as it relates to DSM-III-R criteria for Generalized Anxiety Disorder (GAD; Meyer, Miller, Metzger, & Borkovec, 1990). This measure was used to screen out potential participants who have generalized anxiety that would disqualify them from the healthy control group. The PSWQ contains 16 items, with 11 items measuring characteristic (e.g., “I am always worrying about something”) and five items measuring non-characteristic (e.g., “I do not tend to worry about things”) traits. Participants respond with a 5-point Likert scale (1 = not typical at all of me; 5 = very typical of me). The five non-characteristic items are reverse scored and the sum of scores indicate an individuals’ level of worry, ranging from 16 to 80; score of 45 or above were considered clinically significant. Research indicates that the PSWQ measures the construct of worry as separate from other depressive and anxiety symptoms. Specifically, individuals meeting criteria for GAD have reliably higher PSWQ scores than individuals meeting criteria for PTSD. Additionally, internal consistency (.91) and test-retest reliability (.92) are shown to be very high for the PSWQ (Meyer et al., 1990).

Depression Assessment. Participants also completed the Zung Depression Scale (ZDS), a 20-item scale assessing severity of depression (Zung, 1965). This measure was used to screen out potential participants who have depression that would disqualify them from the healthy control group. Participants rate the level to which each item was characteristic of them over the past week on a 4-point Likert scale (0 = none or a little of the time; 3 = most of the time). Depression severity is obtained when scores are summed. Scores between zero and 50 are normal, between 50 and 59 are minimal to mild, between 60 and 69 are moderate to severe, 70 or above is in the severe range; 65 was the clinical cut-off for this study. High split-half reliability of .73 and high internal consistency of .79 has been found (Knight, Waal-Manning, & Spears, 1983)

The International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2005). The IAPS is a well-established standardized pictorial stimulus to study the autonomic psychophysiology of defensive behaviors in humans (Hermans, Henckens, Roelofs, & Fernandez, 2013). A set of 120 photographs were selected from IAPS and were composed of 60 aversive, 60 neutral photographs, and 60 pleasant photographs. Images were assigned to block depending on their standardized arousal ratings (Lang, Bradley, & Cuthbert, 2005).

The BIOPAC MP150 Amplifier (BIOPAC Systems, Inc., Santa Barbara, CA) records electrocardiogram data using three AG-AgCl electrodes with an 11mm diameter contact area with two electrodes attached below each clavicle and the third electrode attached to the lower left rib. The BIOPAC records the electrocardiogram signal at 500 Hz. An automatic R-wave detector identified interbeat interval in milliseconds to calculate RSA.

The Distraction Task is a symmetrical abstract pattern-coloring page that participants will color in with coloring pencils that will be provided by the researcher (see appendix A). The

purpose of this task is to get an accurate baseline measurement. Theoretically, the coloring pages should distract the participant from being in a new environmental setting, which could be autonomically arousing.

Data Preparation & Analyses

Heart period and RSA was extracted from raw ECG data. Using CardioEdit and CardioBatch, RSA amplitude was derived from raw ECG wave. First, HRV from ECG data was edited using CardioEdit software. The editing procedure followed procedures outlined by Heilman et al (2013), who also examined cardiopulmonary oscillations from a polyvagal perspective. Editing consisted of integer arithmetic or manual insertion/deletion of missing/spurious detection based on the ECG recording. Heart period was derived from the time intervals between successive R-waves in milliseconds. Using CardioBatch software, RSA was derived from a high-frequency band of HRV waves and will reflect spontaneous breathing patterns. Age and sex differences in spontaneous breathing frequency are accounted for by using age-specific parameters for calculating RSA amplitude.

Cardiac vagal influence was calculated by correlating the change scores (baseline to target point) for both heart period and RSA (i.e., when RSA-change and heart period change are highly correlated, this indicates increased cardiac vagal regulation). A total of eight change scores will be calculated, four for RSA and four for heart period, indicating change from baseline to image to baseline to distraction tasks.

A repeated-measures within-participant's multivariate analysis of variance was used to examine RSA and heart period trajectory differences in PTG, PTSD, and healthy control groups. The last 5 minutes of the physiological data collection periods was used for analysis.

Furthermore, a repeated measures ANOVA to examine individual differences between different experimental conditions was used.

Chapter 5: Results & Discussion

Cardiac Assessments

Group comparisons using a repeated measures ANOVA demonstrated a main effect of group RSA across all measurement phases (baseline, images blocks, and distraction tasks; see Figure 1), F (2, 20) = 3.905, p = .037, $\eta^2_{partial}$ = .28, obs. power = .636, such that average RSA is lowest in the PTG group (M = 5.23, SD = 1.49), followed by the PTSD group (M = 5.68, SD = 1.39), and the control group had the highest RSA across all phases (M = 6.25, SD = 0.92). Although mean RSA between groups was not in the order expected, with PTG being the lowest, univariate post hoc comparisons revealed significant differences between PTG and control groups.

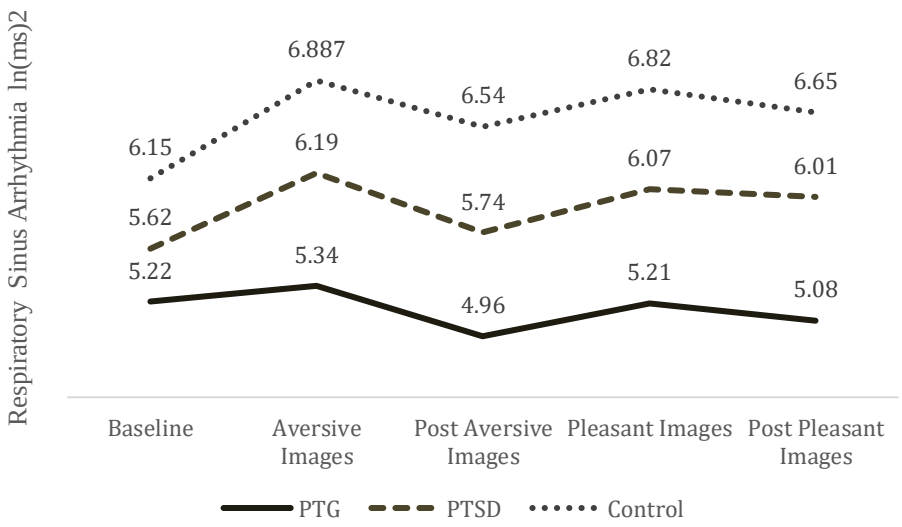

Figure 1. RSA distributions, natural logarithm of ms^2, by group across baseline, blocks of images, and distraction phases.

Next, a group comparisons for heart period was examined using a repeated measures ANOVA, which demonstrated no main effect of group on heart period across all measurement phases (baseline, image blocks, and distraction tasks; see Figure 2), F (2, 20) = .451, p = ns,

$\eta^2_{partial}$ = .043, obs. power = .11. The average heart period is lowest in the PTG group (*M*= 729.79, *SD*= 126.25), followed by the PTSD (*M*= 734.76, *SD*= 77.91) group, and the control group had the highest heart period across most phases (*M*=767.62, *SD*= 130.89). Although mean heart period between groups was not in the order expected, with PTG being the lowest, data analysis confirms expected hypothesis that PTG is not associated with physiological measurements of adaptive functioning measured through RSA and heart period.

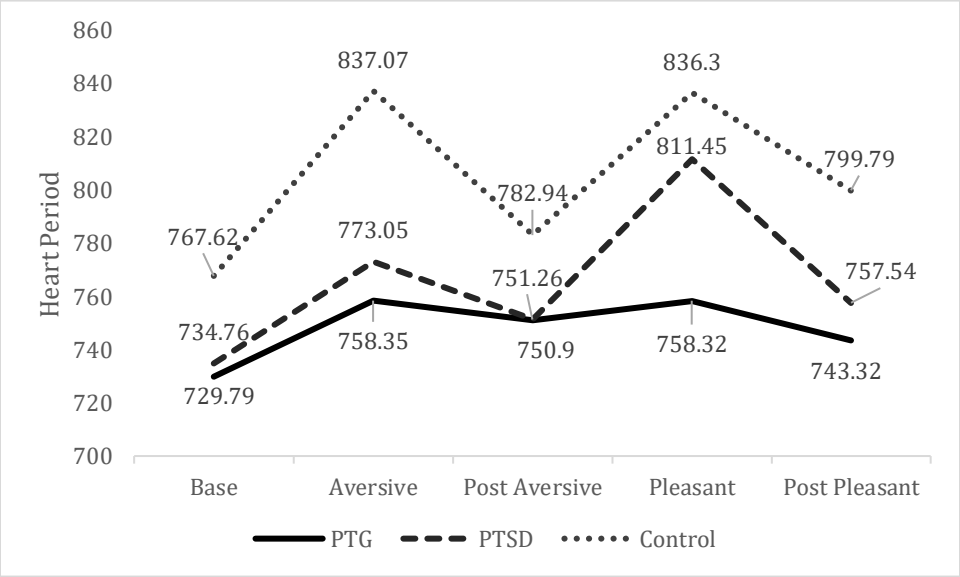

Figure 2. Heart Period distributions in ms by group across baseline, image blocks, and distraction phases.

Trajectories of RSA and HP across phases slightly differ by group from hypothesized expectations, in that post-image distraction task period measures are higher than baseline in control and PTSD groups, but not the PTG group (Figures 1 and 2). Based on previous research, we anticipated that baseline measures would have the highest indications of parasympathetic activity through vagal brake and would be manifested with the highest levels of RSA and heart period. However, RSA and heart period values across phases did not respond physiologically as expected to experimental manipulations. During aversive images, all groups exhibited increases

in heart period and RSA from baseline. Participants exhibited the most physiological arousal during initial baseline recording. Heart period is highest in the control group, in most phases, indicating longer interbeat intervals between R-waves (see Figure 2). Sometimes, this can be an indication of less sympathetic arousal. Furthermore, RSA in the control group was consistently higher than the PTSD and PTG groups, demonstrating increased vagal brake activity (Denver et al., 2007).

Data revealed the vagal brake was removed during the aversive images in the control (r = .24, *ns*), PTG (r = -.16, *ns*), and PTSD groups (r = .54, *ns*) through correlations in change scores of heart period and RSA (see Figure 3). While the PTSD group demonstrated vagal brake reengagement (r = .86, p = .01) in the post-aversive image distraction period (see Figure 4), both the PTG group and the control group did not exhibit vagal brake engagement during the post-aversive image distraction task. This indicates that heart period was not regulated by the vagus, but by other neuroanatomical structures. The correlation between RSA and HP in the post-image distraction period was significantly higher in the PTSD group than the PTG group. Specifically, both RSA and HP change are lower in the PTG group during the post aversive phase, indicating that cardiac output is being influenced by other ventral vagal pathways (see Table 2). These results are different than hypothesized, in that individuals in the PTG group performed physiologically lower than the PTSD group.

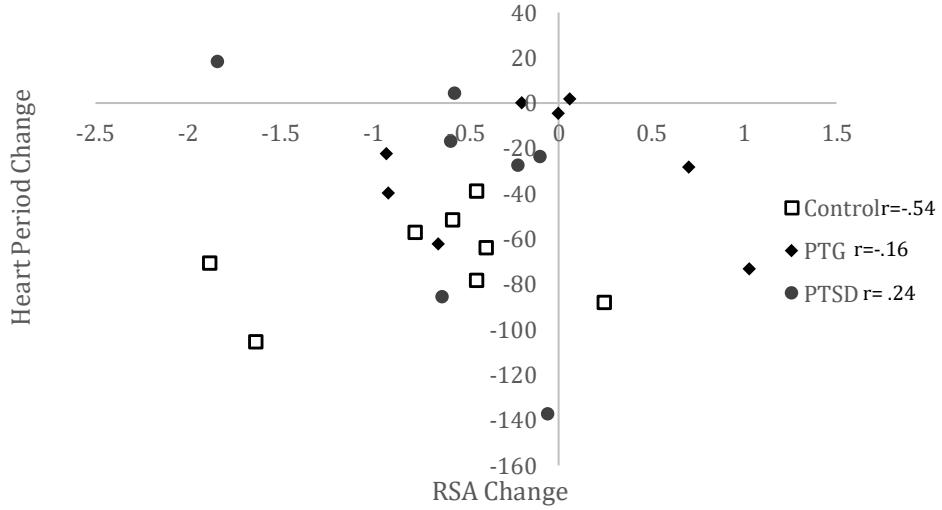

Figure 3. RSA and Heart Period Change Scores (differences between baseline and viewing aversive images).

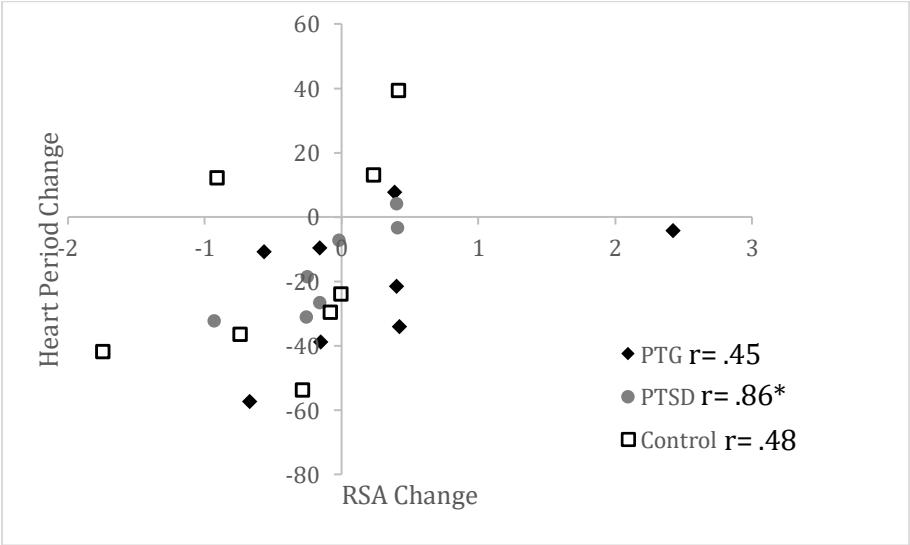

Figure 4. RSA and Heart Period Change scores (differences between baseline and post aversive images while completing distraction task).

Data revealed that the vagal brake was removed during positive images in the control (r=.61, *ns*) and PTG groups (r=.60, *ns*), but not the PTSD groups (r=.78, *p*= .037) in correlations between change scores on RSA and heart period (see Figure 5). The control group demonstrated vagal brake reengagement (r= .86, *p*= .013) in the post image distraction task, while the PTG group did not exhibit vagal brake reengagement (See Figure 6). The PTSD group was already

demonstrating vagal brake activity while viewing pleasant images and did not have vagal brake activation during the post-pleasant image distraction task. The correlation between RSA and HP in the post-pleasant-image-distraction-task was lower in the PTG group compared to both PTSD and control groups, indicating the heart periods are mediated by other vagal pathways.

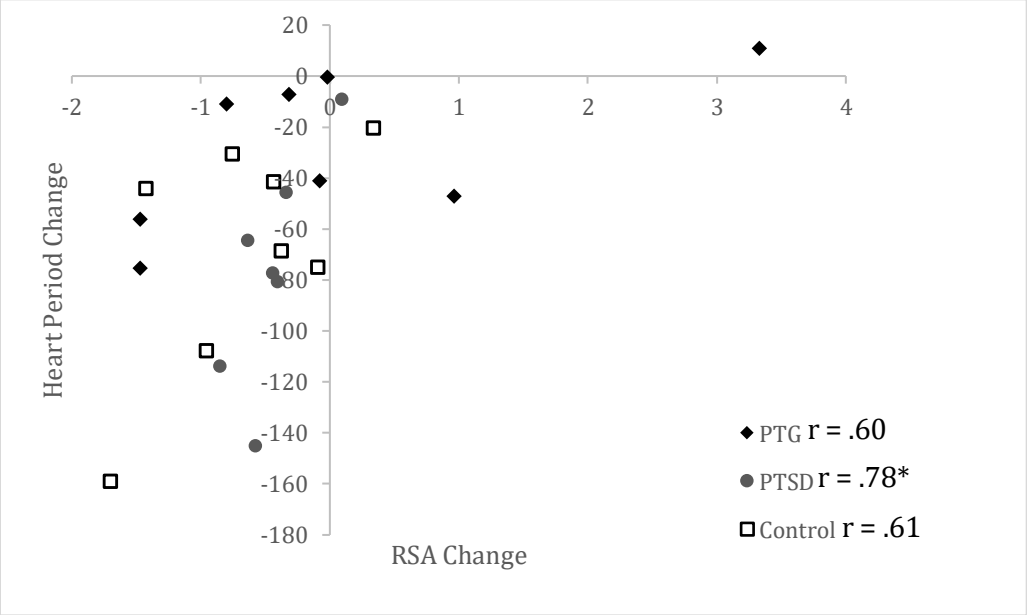

Figure 5. RSA and Heart Period change scores, which are the differences between baseline and viewing pleasant images.

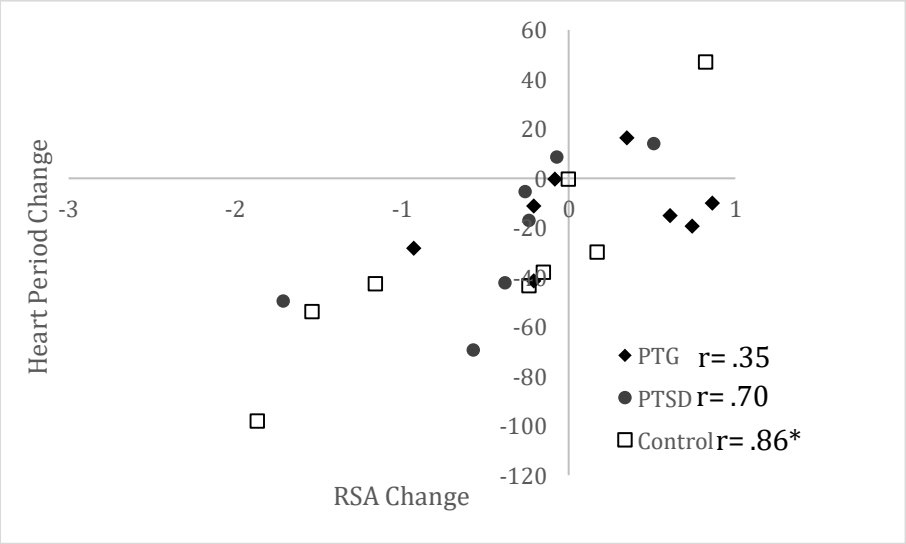

Figure 6. RSA and Heart Period change scores, which are the differences between baseline and post pleasant images while completing distraction task.

Table 2.
Descriptive statistics of physiological variables after viewing unpleasant images for each group

	RSA (SD)	HP (SD)	HR (SD)
Initial baseline			
Control	6.15(0.99)	767.89(111.24)	81.00(15.99)
PTG	5.22(1.03)	730.24(124.69)	84.72(14.33)
PTSD	5.62(1.28)	734.18(77.06)	82.92(7.32)
Aversive Images			
Control	6.88(1.27)	837.07(142.02)	74.39(14.61)
PTG	5.34(1.00)	757.33(121.47)	81.46(14.33)
PTSD	6.19(1.28)	773.97(129.47)	79.83(11.02)
Post Aversive			
Control	6.54(0.93)	782.79(123.31)	78.77(14.97)
PTG	4.96(1.25)	751.92(139.30)	82.53(16.24)
PTSD	5.74(1.05)	750.09(72.97)	80.31(6.57)
Pleasant Images			
Control	6.82(1.35)	801.61(134.20)	74.06(14.52)
PTG	5.21(1.49)	758.32(138.23)	81.44(15.67)
PTSD	6.07(1.12)	811.45(112.84)	75.83(6.96)
Post Pleasant			
Control	6.65(1.06)	799.79(136.55)	70.06(28.52)
PTG	5.08(.919)	743.32(136.34)	83.44(15.67)
PTSD	6.01(.876)	760.74(71.69)	79.83(6.964)

Note. RSA= Respiratory Sinus Arrhythmia. HP = Heart Period. HR = Heart Rate. PTG= Posttraumatic Growth. PTSD= Posttraumatic Stress Disorder.

Bivariate correlations examined the relationship between psychometric scores of pathology and RSA (see Table 3). There was a strong negative correlation between PTG and base RSA, r (23) = -.40, $p < .05$, PTG and RSA during aversive images, r (23) = -.55, $p < .05$, PTG and RSA during post-aversive image distraction task, r (23) = -.56, $p < .05$, PTG and RSA during pleasant images, r (23) = -.53, $p < .05$, and PTG and RSA during post-pleasant images during distraction task, r (23) = -.60, $p < .05$. The strong negative relationship between PTG and all phases of RSA is surprising because it indicates that scores on the PTGI are better at

predicting *less* heart rate variability and *less* flexibility in adapting to stressors. Furthermore, PTG was unrelated to PTSD and other psychometric variables of psychopathology. Individuals in the PTSD group did not have a significant relationship with any of the phases of RSA measurement. There was a strong positive relationship between severity of PTSD and the Behavioral Health Screening Measure, r (23) = .86, p <.05, between severity of PTSD and the number of traumas, r (23) = .50, p < .05, and between severity of PTSD and Zung Depression scale, r (23) = .51, p< .05.

Table 3.

Correlations between Posttraumatic Growth, PTSD, RSA, and other psychometric variables of psychopathology.

	PTG	PTSD	Base	UI	PUI	PI	PPI	BHSM	Zung	Trauma	PSWQ
PTG											
PTSD	-.16										
Base	-.40*	.01									
UI	-.55*	.10	.84*								
PUI	-.56*	.11	.80*	.86*							
PI	-.53*	.16	.72*	.79*	.93*						
PPI	-.60*	.18	.78*	.90*	.92*	.92*					
BHSM	-.22	.86*	.17	.32	.32	.40	.28				
Zung	.09	.51*	.13	-.01	-.01	.05	.05	.60*			
Trauma	.04	.50*	-.03	-.14	-.14	-.12	-.18	.28	.11		
PSWQ	-.26	.36	-.21	-.13	.00	.07	-.07	.24	.06	.42	

Note. PTG= Posttraumatic Growth. PTSD= Posttraumatic Stress Disorder. UI= Unpleasant Images. PUI= Post Unpleasant Images. PI= Pleasant Images. PPI= Post-Pleasant Images. BHSM= Behavioral Health Screening Measure. PSWQ= Penn State Worry Questionnaire.

Screening Assessments

It was hypothesized that different trauma histories produced different outcomes, specifically if a certain type of trauma history would predict PTG or PTSD. For example, was

there higher incidence of childhood maltreatment in the PTSD group. To do this a multiple regression analyzed data from the screening questionnaire completed by over 1,000 participants. Multiple regression analyses were calculated to predict scores on the PTGI from different trauma histories. A multiple regression indicated natural disasters (β =.07), transportation accidents (β = .07), wartime violence (β = -.08), sudden death of a loved one (β = .12), and witnessing a death (β = .09) significantly predicted scores on the PTGI (R^2 = .06, p < 001).

Moreover, another multiple regression analysis was calculated to predict scores on the PCL-5 from different trauma histories. A multiple regression indicated accidents at home (β =.12), transportation accidents (β = .10), physical abuse as a child (β = .08), sexual abuse as a child (β = .09), sexual abuse as an adult (β = .11), witnessing a death (β = .08), and other emotionally distressing events (β = .12) significantly predicted scores on the PCL-5 (R^2 = .15, p < 001). As can be seen, there is some overlap in the different types of traumatic experiences and the outcome of either PTSD or PTG, but not total agreement. There is a statistically significant but weak positive correlation between scores on the PTGI and scores on the PCL-5, r (969) = .17, p < .001. These results indicate that PTG and PTSD are not mutually exclusive outcomes from traumatic experiences.

Discussion

A main effect of group across physiological measures was expected, such that downward trends in RSA and shorter heart periods would be more pronounced in the PTSD group. Furthermore, it was expected that there would be no statistical difference between PTG and control groups. These findings somewhat confirm the proposed relationship between cardiac vagal tone, measured through RSA, and PTG. This study supports that PTG is not necessarily an adaptive response to traumatic experiences, but instead a predictor of cardiac vagal rigidity. In

each phase, RSA and heart period are lower in the PTG group, signifying less parasympathetic control over respiration and heart activity. More importantly, absence of cardiac vagal regulation after viewing aversive images accompanied by lower RSA in PTG during this period strongly suggests that the PTG construct is characterized by affect dysregulation, which would predispose individuals to the development of various pathologies related to emotional regulation. The results of this study may explain the complicated relationship between PTG and PTSD. Individuals with PTG have less cardiac vagal flexibility, which leads to an inability to respond to environmental stressors, and low RSA is predictive of various forms of psychopathology. A tremendous amount of the controversy surrounding PTG is based on its bidirectional relationship between PTSD. Individuals with PTG, potentially due to low RSA, are at risk of developing psychopathology because of their inability to flexibly respond to stressors, which would explain the relationship between PTSD. The present study does not support the proposition that the PTGI measures growth after traumatic events, but instead shows that measure is more predictive of pathology and cardiac vagal rigidity. These results support the position that PTG is a maladaptive post-trauma response and that the cardiac rigidity found in this study may be an explanation for the complex nature of PTG and the bidirectional relationship it has with PTSD.

Somewhat unexpectedly, the greatest differences in RSA between groups were observed during the baseline period. It was hypothesized that the prospect of coming into the laboratory to participate in the study is in and of itself an autonomic arousing event. Therefore, this study implemented the use of the distraction task during baseline and post-image tasks to capture an accurate baseline of activity. However, RSA scores did not follow expected trajectory, even with the incorporation of a distraction task. It was expected that the RSA would be lower than baseline during the aversive image phase, but RSA was higher than baseline in this phase. Based

on previous research, it was expected that exposure to aversive images would be associated with a decrease in RSA (Lang, Bradley, & Cuthbert, 2008). These unexpected results could be due to weak experimental manipulation, as the images used in this study did not induce the expected physiological activity of decreased RSA and shorter heart periods.

Hypothetically, individuals who score highly on the PTGI inventory, indicating growth posttrauma, would also exhibit high RSA and return to baseline after viewing aversive measures. Therefore, it was expected that individuals in the PTG group and the control group would not be statistically different on RSA change and heart period. Surprisingly, the PTG group had lower RSA than the PTSD group throughout the study and significantly lower RSA than the control group. Furthermore, there were no significant differences between PTSD and control during any of the phases of the study. Research examining PTSD and RSA are inconsistent in terms of baseline differences. Several studies have found that RSA differed significantly between PTSD and RSA during baseline (Blechert et al., 2007; Cohen et al., 2007), but others (Austin et al, 2007; Sahar et al, 2001), did not observe differences in RSA during baseline or stressor phases. Austin et al (2007) did find differences in the post-resting phase, while Sahar et al (2001) did not between the PTSD and control groups.

It was expected that there would be a lower correlation between RSA-change and heart-period-change in the PTSD group verses the PTG and control groups. Current findings indicate that people who report PTG are characterized by a consistent autonomic state absence of vagal regulation, supported by the lack of significant correlations between heart period and RSA change. Accordingly, repeated-measures comparisons with the PTG group reveal that RSA did not reliably differ by phase. Similar comparisons with the PTSD group indicate that baseline RSA does reliably differ from image RSA and post image distraction tasks, which demonstrates

an increase in RSA during the image blocks and a decrease during the distraction tasks. RSA changes in the control group also differ reliably across the phases, however the trajectory of change did not follow anticipated results. Individuals in the control and PTSD group showed an increase in RSA during the image tasks and a decrease during the post-image distraction tasks. This would suggest that more cardiac vagal activity was occurring while the participants were looking at the images and less activity during the post-image distraction task (refer to Figure 1).

Lower heart period in the control group than clinical groups may be explained by extraneous variables, such as current medications for both groups. For example, 35% of participants in the PTG group were currently taking antidepressants, antiseizure medication, and drowsy medication, all of which can have a profound effect on heart rhythms (Julien, 2007). In the PTSD group, 45% of participants were currently taking antidepressants, antianxiety medication, and drowsy medication. These types of medications can have a profound effect on heart rhythms, but it is difficult to control for the effects of these necessary medications.

Post-trauma pathology has been shaped by the present social and political climate and has largely been a war-related disorder. Investigation into post-trauma psychopathology continues to indicate the variety of traumatic experiences extending beyond combat exposure (Herman, 1992). The APA appears to have the right mindset, in that it has attempted to broaden the scope of post-trauma functioning, by standardizing the PTGI as a measurement of growth post-trauma, but these goals do not appear to be manifesting (Kilpatrick et al., 2013). The results from this study would suggest that the PTGI does not measure actual growth after trauma, but is indicative of something maladaptive that prevents individuals from returning to pre-trauma levels of functioning.

Limitations

Sample size was likely the strongest threat to study validity; Field suggests that multivariate group comparisons should have at least 20 cases per group to achieve adequate power (2010). However, Levene's test of normality indicated that all RSA measures had normal distributions and Box's M test for equality of covariance across measures proved to be true for the RSA measures. Both tests bolster the main effects and power of the MANOVA.

Another limitation is that the sample was homogenous and only females participated in the laboratory portion of the study, which hinders the generalizability. The inclusion of only females in the second portion of the study was not intentional but the result of a combination of factors. For instance, females are more likely to experience a trauma and develop a disorder as a result (Mendlowicz & Stein, 2000). Secondarily, participants were recruited from a university population where females are the majority population and are more likely to participate in the study. In the future, efforts to recruit males specifically will help to determine how generalizable these findings are.

Future Directions

This study contributes significantly to the current literature, as it is the first study to examine autonomic differences between individuals who have self-reported PTSD symptoms and those who report PTG. The potential therapeutic implications of this study are important, as this provides objective evidence that self-reported PTG is a maladaptive response and may even be preventing treatment efficacy. Additional investigation into PTG will continue to increase our understanding of the phenomenological differences between it and PTSD and help with understanding the role PTG plays in the development of pathology. Future research should investigate cardiac vagal activity with a stronger experimental manipulation, which can be done

by using specific trauma centered memories or cues, to examine the autonomic activity between those with reported PTG and PTSD.

Furthermore, future research should investigate the influence of the dorsal motor nucleus of the vagus (DMNX) to dissociative symptoms commonly observed in fear responses to trauma and learned helplessness (van der Hart, Nijenhuis, & Steele, 2005). Research has identified freezing in response to threat as tonic immobility manifested through rapid bradycardia or heart rate deceleration in trauma exposed individuals (Volchan et al., 2011). Currently, there is no literature on the relationship between trauma-related bradycardia and DMNX vagal influence on the heart and the differential diagnosis between PTG and PTSD. A study of this nature could further elucidate potential differences between these two maladaptive post-trauma outcomes.

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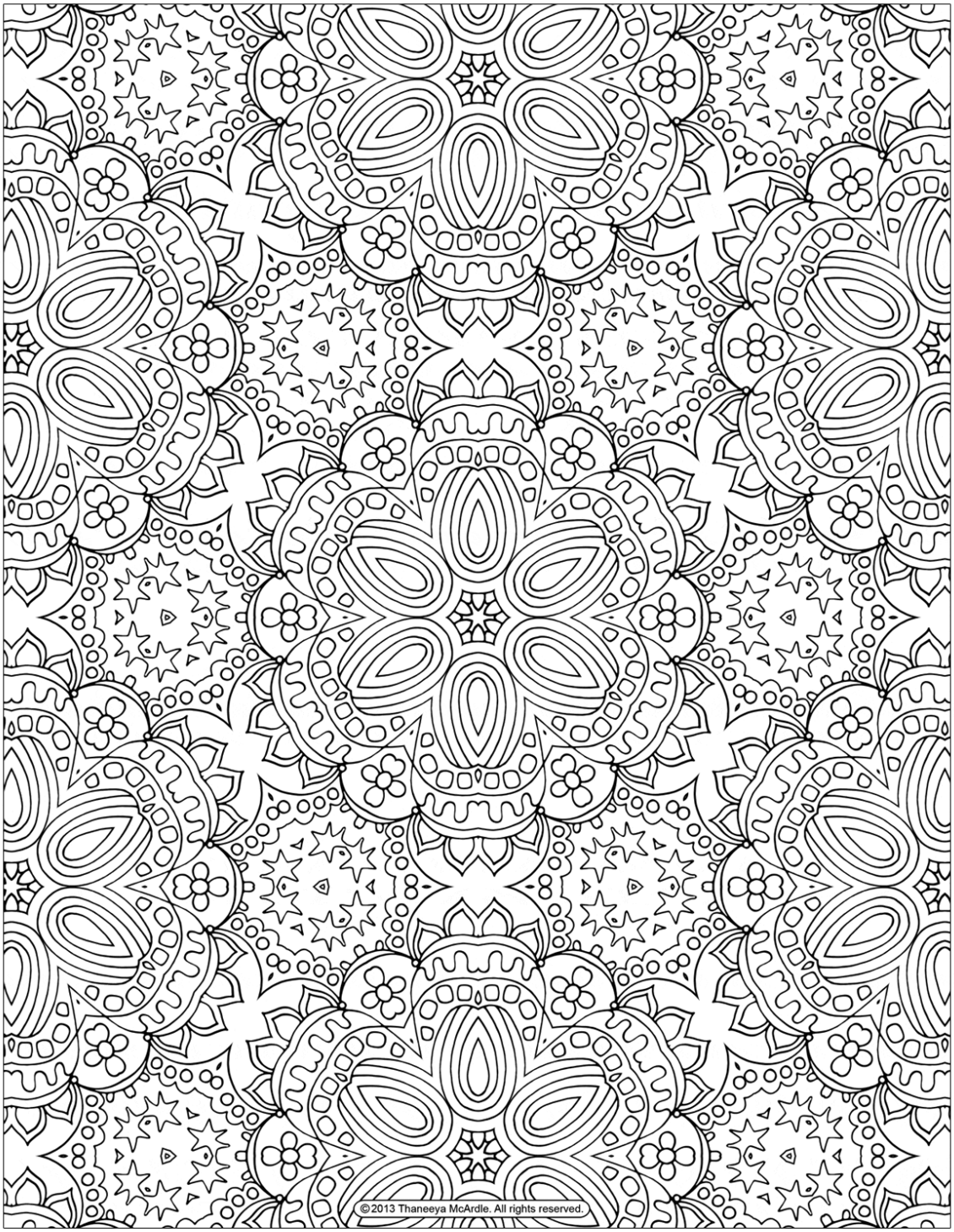
Appendix A



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