POSTTRAUMATIC GROWTH AND BIOMARKERS OF CHRONIC STRESS AMONG COMBAT VETERANS OF OPERATIONS ENDURING AND IRAQI FREEDOM

by

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ABSTRACT

CARA L'ETOILE BLEVINS. Posttraumatic growth and biomarkers of chronic stress among combat veterans of operations Enduring and Iraqi freedom. (Under the direction of DR. RICHARD G. TEDESCHI and DR. JEANETTE M. BENNETT).

Cardiovascular diseases (CVD) are a significant public health problem, representing the leading cause of death in U.S. military combat Veterans. Following deployment, combat Veterans are at an increased risk for CVD due to chronic in-theater exposure to physical and psychosocial stressors. Growing evidence suggests that experiencing posttraumatic growth (PTG), or positive psychological change in the aftermath of trauma, may convey salutary health benefits and increase physical and psychological wellness of combat Veterans. However, scant research has sought to examine if PTG buffers against the adverse effects of combat exposure in combat veterans. Therefore, the present study investigated (1) differences in the diurnal slope of cortisol and morning C-reactive protein (CRP) levels based on trauma perception, and (2) if PTG buffered against adverse effects of stress as indicated by cortisol and CRP in a sample of male combat veterans (N=33) of the wars in Iraq and Afghanistan. Results did not reveal significant differences in either cortisol or CRP based on trauma perception; however, PTG significantly interacted with perceived stress such that the cortisol slope of individuals reporting a greater degree of PTG flattened and became positive as perceived stress levels increased on Day 2. Relatedly, those individuals reporting lower levels of PTG at higher levels of stress did not demonstrate higher levels of cortisol. These findings may reflect the processes by which one seeks to reconstruct their assumptive worldview in the aftermath of trauma, and provide initial physiological support for the

notion that PTG is a not just an outcome, but is also a process requiring bodily support in the form of physiological energy sources (i.e., cortisol). Because this study was cross-sectional and was not powered to detect small effects, larger samples and prospective designs may reveal additional effects of PTG. Future work is warranted before causal assumptions can be made.

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INTRODUCTION

Military Combat and CVD Risk

War and its corresponding sequelae are among the strongest stressors known to mankind. Since October 2001, over 1.64 million men and women have been deployed to Afghanistan and Iraq for Operations Enduring Freedom and Iraqi Freedom (OEF/OIF) (Tanielian, Jaycox, Adamsibm & Metscher, 2008). The psychological cost of these deployments, many involving extended exposure to combat-related stress in multiple rotations, may be disproportionally high in comparison to the physical injuries of combat and has prompted an increased focus on mental health of US Service veterans (Warden, 2006; Tanielian et al., 2008). Stress-responsiveness is related to long-term health outcomes and, accordingly, service veterans exposed to combat, multiple deployments, and other stressful events report higher rates of physical morbidity, mortality and chronic illness when compared to non-traumatized military and civilian counterparts (Heppner, Crawford, Haji, Afari, Hauger, et al., 2009).

Cardiovascular disease (CVD), which encompasses a broad category of cardiac conditions including hypertension, stroke, heart disease, and peripheral artery disease, is the leading cause of mortality among men and women worldwide (American Heart Association (AHA), 2014; Centers for Disease Control (CDC), 2015). CVD and related diseases accounted for over 35% (approximately 611,000) of all United States deaths in 2013 and more deaths each year than any other major cause of death, including cancer, chronic lower respiratory disease, and accidents combined (AHA, 2014). It is estimated that at any one time, over 59,000,000 Americans have one or more types of CVD and over 2,600 Americans (equating to one death every 33 seconds) die each day from CVD

related illnesses (AHA, 2014). It is projected that by 2030, 40.5% of the US population will be diagnosed with CVD or a related illness and associated economic costs are projected to exceed $818 billion (Stults-Holehmanien, 2013). As we move forward into the 21st century, continuing to study and identify CVD-related risk factors and corresponding treatments/interventions will be imperative if we are to effectively combat this growing public health concern.

Although the causes of CVD are vast, currently identified risk factors include age, sex, race, total cholesterol, HDL cholesterol, systolic blood pressure, blood pressure lowering medication use, diabetes status, physical inactivity, smoking status and exposure to chronic or traumatic stress (AHA, 2013). In addition, certain populations have been identified as representing an increased risk for CVD morbidity and mortality including individuals of a lower socioeconomic status (SES), individuals exposed to chronic stressors, and military personnel. A study conducted by Johnson, Pietz, Battleman, & Beyth (2004) found that over half of patients (57.6%) seen at VA medical centers during a three-year period had some form of CVD (hypertension or dyslipidemia). Further, Veteran CVD prevalence rates were found to be nearly double that of average civilian CVD prevalence rates (52.1% Veteran vs 32.8% civilian), suggesting that Veterans tend to have a higher prevalence of CVD and chronic disease and bear a larger burden of disease relative to non-Veteran samples (Johnson et al., 2004).

Behavioral risk factors may be partially responsible for increased Veteran CVD risk. A study by Bray, Pemberton, Hourani, Witt, Olmstead, Brown, and colleagues (2008) found that nearly one-third of active duty military personnel reported smoking

tobacco within the past month and 14% reported smokeless tobacco use (less than 20% of age-matched civilians reported current tobacco use and less than 3% reported using smokeless tobacco). Military service has long been associated with tobacco use; however, recent evidence suggests that OEF/OIF veterans are up to 50% more likely to use tobacco than military peers who did not deploy, placing them at increased risk for hypertension and stroke (Widome, Littman, Laska, & Fu, 2012).

Additionally, while emerging adulthood is generally considered a time of increased risk of excess weight gain, data suggests that OEF/OIF war veterans are more likely to be overweight than same-age civilian samples, leading to an increased risk for hypertension and CVD (Widome et al., 2012). Finally, Cohen, Marmar, Ren, Bertenthal, & Sea, (2009) reported that male and female OEF/OIF veterans with mental health diagnoses had a significantly greater risk of being diagnosed with CVD than those without. Research has shown significant positive associations between combat experiences and post-deployment mental health conditions like posttraumatic stress disorder, anxiety, and depression, and it is estimated that as many as 25% of returning OEF/OIF veterans utilizing VA medical services were given mental health diagnoses, suggesting that the emerging generation of Veterans may be at risk for CVD following their exposure to combat and other deployment experiences (Anderson, Wade, Possemato, & Ouimette, 2010).

Perceived Stress and CVD Risk

Chronic psychological stress is related to CVD emergence (Cohen, Kamarck, & Mermelstein 1983; Melamed, Kushnir, & Shirom, 1992; Belkic, Landsbergis, Schnall, & Baker, 2004; Kashani, Eliasson, & Vernalis, 2011). Psychological stress refers to both

physical and environmental challenges which are judged to threaten one's ability to cope and strain an individual's responses to events (Cohen, Janicki-Deverts, & Miller, 2007). In other words, stress occurs "when an individual perceives that environmental demands tax or exceed his or her adaptive capacity" (Cohen et al., 2007). Stress may be transient and relatively harmless with potentially positive adaptations (i.e., academic examinations or strenuous bursts of exercise) or it may be chronic and uncontrollable producing excessive wear and tear on psychological and physiological systems and resulting in potentially lasting and detrimental impacts to one's physical and mental health (i.e., caregiving for a loved one with a terminal illness or exposure combat exposure) (Cohen et al., 2007; Dimsdale, 2008; Stults-Holehmanien, 2013). Further, psychological stress can be accompanied by a variety of affective responses such as anxiety, depression, anger, fear, and hostility which have been linked to chronic illness and risk for CVD (Kario, McEwen, & Pickering, 2002).

The experience of stress is an evolutionary adaptive process designed to alert us to and protect us from danger or harm (Sapolsky, 2004). However, when negative affective states such as feelings of anxiety, anger, fear, or depression are chronically induced in response to stressors, biological processes may in turn be initiated that can lead to illness and disease and ultimately CVD (Sapolsky, 2004; Cohen et al., 2007; Holman, Silver, Poulin, Andersen, Gil-Rivas, & McIntosh, 2008). Examples of chronic stressors can include stressful events occurring over extended periods of time (i.e., war, caring for a chronically ill family member, etc.) or more acute events which continue to be experienced as stressful even after they have ended (i.e., re-experiencing combat-related events) (Cohen et al., 2007). Behavioral adaptations or attempts to cope with

stressors, such as increases in smoking or decreases in exercise and sleep, provide important pathways to an increased risk of CVD (Cohen et al., 2007). The autonomic nervous system (ANS) represents an additional pathway which impacts the functioning of the cardiovascular system and may lead to increases or decreases in CVD risk (Kario et al., 2002; Sapolsky. 2004; Cohen et al., 2007).

Stress Physiology

The sympathetic nervous system (SNS), one division of the autonomic nervous system (ANS), is specifically activated during stress (Sapolsky, 2004). The SNS is an activation system and responsible for mediating the following functions of behavior: a) fight, b) flight, c) fright, and d) sex (Sapolsky, 2004). In contrast to the activating function of the SNS, the parasympathetic nervous system (PNS) is responsible for inhibiting the SNS and inducing relaxation, calmness, digestion, and reproduction through the secretion of acetylcholine. Working in direct opposition to the SNS, the PNS promotes systemic rehabilitation and energy storage and plays an essential role in regulating the SNS and heart function, causing the variation in time between heart beats (i.e., heart rate variability (HRV)) (Sapolsky, 2004).

The stress response begins when the brain first detects the presence of a potential stressor and activates the SNS. This SNS activation triggers responses in the Sympathetic-Adrenal-Medullary (SAM) axis and the Hypothalamic-Pituitary-Adrenal (HPA) axis. The SAM response is the quick, initial response to stress which releases epinephrine and norepinephrine and is designed to support physical activity (Sapolsky, 2004). Epinephrine and norepinephrine serve to increase and regulate muscular efficiency, release energy stores, and both increase and regulate blood flow to the

arteries, organs, and muscles (Cacioppo & Berntson, 2011). Next, energy and blood flow are shifted to the muscles, blood pressure is increased, and blood flow to peripheral areas is constricted. Thus, the body is mobilized and ready to respond and cope to the stressful situation (Cacioppo & Berntson, 2011).

While the SAM axis response begins the stress signal, the HPA axis response is responsible for actually carrying out the stress response. Stress is perceived by the hypothalamus and triggers a corticotropin releasing hormone (CRH) to the anterior pituitary gland. This stimulates a release of adrenocorticotropinc hormone (ACTH) from the pituitary to the adrenal cortex. Cortisol and glucocorticoids are secreted into the body thus increasing muscular efficiency, brain function (alertness, learning and memory responses), regulating inflammatory responses, energy resources, and the cellular metabolism underlying behavioral adaptation (Cacioppo & Berntson, 2011). In short, the SAM axis response initiates a chain of reactions designed to quickly increase our energy and alertness, slow digestion and reproduction, and effectively prepare us for immediate action to aid in our survival. The HPA axis response then helps to maintain and continue this activation by converting proteins to glucose (energy), increasing blood flow, and decreasing insulin secretion.

The ANS is also responsible for both activating and regulating the immune system, another adaptive component of the “fight-or-flight” response. When the SNS is activated, changes are occurring both to the immune system and the SAM and HPA axes. In essence, SAM axis activation increases the release of epinephrine and norepinephrine, while simultaneously increasing cytokine production. White blood cells are shunted from the bloodstream and sent into visceral and peripheral tissues such as the lymph nodes, GI

tract, skin and bone marrow (Kemeny, 2011). This adaptive function serves to mobilize physiological systems to respond to threat and also mobilize the immune system to prepare to defend against and repair potential wounds or infections which may accompany stressors (Sapolsky, 2004; Kemney, 2011).

The immune system also helps support behavioral recuperation following stress by releasing proinflammatory cytokines. This not only allows the immune system to attack and destroy pathogens and then repair tissue, but also allows the immune system to influence the brain, resulting in behavioral withdrawal to facilitate recuperation (i.e., rest and sleep) and enable the body to devote vital energy to fighting and eliminating infections (Kemeny, 2011). Thus, these proinflammatory cytokines induced with infection are activated by psychological stressors (in addition to physiological stressors) and this may account for the behavioral withdrawal and inhibition in such psychological disorders as depression (Kemney, 2011).

Stress, Allostatic Load, and CVD Risk

Successive or cumulative exposure to stressful or traumatic events such as combat may weaken individual coping abilities and increase activation of the HPA axis resulting in allostatic load and a vulnerability to CVD (Sapolsky, 2004). The human body consistently seeks to maintain a state of relative balance, or homeostasis, across all physiological components (Sapolsky, 2004; Cacioppo & Berntson, 2011, Danese & McEwen, 2011). Homeostatic processes work to regulate, buffer, restore, and protect organisms from the effects of internal and external changes, ultimately striving to keep internal biological processes at ideal levels (Cacioppo & Berntson, 2011). However, the world is not static and the body's homeostatic state is constantly tested and threatened by

changing environmental events or conditions that affect an organism (i.e., it is constantly under stress) (Segerstrom & O'Connor, 2012; Salpolsky, 2004; Danese & McEwen, 2011). To cope with this constant variability, physiological systems must continually evolve and adapt to maintain stability, a process termed allostasis (Sapolsky, 2004; Cacioppo & Berntson, 2011, Danese & McEwen, 2011).

Allostasis, by definition, encompasses broader flexibility than homeostasis and enables humans to respond to physical changes (i.e., wakefulness, sleep, and postural changes) and cope with various stressful situations (danger, hunger, temperature changes, etc.) (Kario et al., 2002). Prolonged activation of allostatic systems may result in allostatic load, or an accrual of wear and tear on physiological regulatory and response systems (Danese & McEwen, 2011; Cacioppo & Berntson, 2011). Frequent or chronic exposure to stressors can result in both failures to adapt as readily to stressors and an inefficiency in terminating allostatic responses once a stressor is removed (Cacioppo & Berntson, 2011); accelerating atherosclerosis, risk of CVD and has been correspondingly linked to earlier mortality (Cacioppo & Berntson, 2011; Sapolsky, 2004; Danese & McEwen, 2011). Chronic stress exposure increases blood pressure, promotes left ventricular hypertrophy and atherosclerosis, reduces cardiac efficiency and vasoconstriction, and increases the risk of myocardial infraction through neurohormonal arousal (Holman et al., 2008; Sapolsky, 2004; Jamieson, Knock, & Mendes, 2012).

Perception of threat and safety appear to be key variables to the stress response (Thayer, Ahs, Fredrikson, Sollers III, & Wager, 2012; Jamieson et al., 2012). When individuals feel equipped with sufficient biological, psychological, and social resources to cope with stressors they experience a challenge response and when the demands of a

stressor exceed perceived resources, individuals experience a threat response (Jamieson et al., 2012). The challenge response activates the SAM axis which is associated with increased cardiac efficiency and vasoconstriction (Jamieson et al., 2012).

The SAM axis is also activated when a threat is perceived, but the outcomes differ in that cardiac efficiency and vasoconstriction are instead *reduced* in such a way to prepare the body for predatory approach and potential damage/defeat (Jamieson et al., 2012). This is evolutionarily advantageous as it protects and warns against approaching potential danger and, if danger is unavoidable, this threat response prepares to defend from harm.

An occasional threat response is unlikely to cause lasting biological damage; however, in the face of chronically perceived and experienced stress, the threat response is associated with impaired decision making, cognitive decline, and CVD (Jamieson et al., 2012). As threat is perceived, blood pressure elevates and strain is put onto the heart and arteries. If individuals experience this strain on a regular basis, the arteries and heart will begin to wear out (Sapolsky, 2004). This begins to damage the ending branch points of arteries resulting in an inflammatory response to boost immunal cells located at damage points (Sapolsky, 2004). Concurrently, the increased blood flow that accompanies sympathetic arousal circulates blood platelets, fatty nutrients, glucose, and cholesterol which also accumulate at damage sites. This can result in atherosclerotic plaque and, ultimately, atherosclerosis. Increased blood flow accompanying the stress response can then loosen plaque and carry it to smaller blood vessels, where vessels can be clogged, potentially resulting heart attack or stroke (Sapolsky, 2004).

Assessment and Measurement of CVD Risk

A growing body of literature has highlighted the role of inflammatory processes in the development of CVD and other chronic diseases, prompting recommendations for the assessment of inflammation to help predict and prevent early onset morbidity and/or mortality (McDade, Hawkley, & Cacioppio, 2006). Chronic stress and trauma exposure contribute to physiologic inflammation and increased risk for CVD (Dockray & Steptoe, 2010; Groer, Kane, & Williams, 2014). Specifically, the experience of chronic stress and trauma have shown positive associations with levels of cortisol and C-reactive protein (CRP) in a variety of samples, suggesting that cortisol and CRP may be important biological pathways through which current levels of stress increase risk for morbidity and mortality (McDade *et al*., 2006).

Cortisol, C-Reactive Protein and Perceived Psychological Stress

Cortisol is a glucocorticoid, or steroid hormone, that is secreted from the adrenal glands during stress (Sapolsky, 2004). Cortisol is involved in emotion, learning, and memory processes, helps to regulate metabolism by influencing glucose storage, and helps to regulate the intensity and duration of inflammatory responses associated with the immune system (Sapolsky, 2004; Miller, Chen, & Shou, 2007). In healthy individuals, cortisol levels typically fluctuate throughout the day, peaking just after awakening and gradually decreasing throughout the course of the day (Bower, Moskowitz, & Epel, 2009; Diaz, Aldridge-Gery, & Spiegel, 2014). This 24-hour cycle is known as the diurnal cortisol rhythm and a steeper decline from morning to evening, or a negative cortisol slope, is typically associated with healthy endocrine function and stress-reactivity (Diaz et al., 2014). Studies have indicated that flatter diurnal cortisol slopes, specifically

indicated by raised late-evening levels of salivary cortisol, are associated with exposure to chronic stressors and are predictive of CVD mortality independent of related covariates including body mass index (BMI), smoking status, age, race, and sex (Kumari, Shipley, Stafford, & Kivamaki, 2011). Diurnal cortisol assessment may therefore serve as a useful indicator of increased risk for CVD following exposure to acute and chronic stressors.

Another physiological biomarker associated with CVD risk is C-reactive protein (CRP), a biomarker indicative of systematic inflammation (e.g. an immunological response to physical damage or pathogenic invasion) (Dockray & Steptoe, 2010; Hansel, Hong, Camara, & von Kanel, 2009; Yamamot, Okazaki, & Ohmori, 2011). Specifically, CRP is an acute phase protein and important component of innate immunity involved in the biological development and progression of disease and illness (McDade et al., 2006). The CDC suggests the following CRP cut-off scores to evaluate risk for CVD and chronic illness: <1 mg/L indicates low risk, 1-2.9mg/L indicates intermediate risk, and >3 mg/L indicates high risk. Highly sensitive CRP (hsCRP) assays have demonstrated that slight elevations of CRP are predictive of CVD and chronic illness independent of traditional risk factors such as age, SES, ethnicity, BMI, and smoking, thereby highlighting the utility of CRP in disease prediction and prevention (McDade et al., 2006; Dockray & Steptoe, 2010).

A large body of research suggests that perceived psychological stress (PPS) – a strained feeling that results from an individual's perception of external demands as too great relative to one's capabilities – contributes to inflammation (McDade et al., 2006; Dockray & Steptoe, 2010). PPS has been linked to increased proinflammatory biomarkers (such as CRP) and a greater propensity to illness and infection in caregivers

reporting higher levels of perceived stress than controls (Kiecolt-Glaser, Preacher, MacCallum, Atkinson, Malarkey, & Glaser, 2003). PPS, cortisol, and levels of CRP have also demonstrated causal links to increased risk for CVD and PPS has been found to be positively associated with increased levels of CRP and a flattened diurnal cortisol slope in several large population-based studies (i.e., the Whitehall II Study and the National Longitudinal Study of Adolescent Health) (McDade et al., 2006; Kumari, Shipley, Stafford, & Kivamaki, 2011). As cortisol and CRP may be important pathways through which current levels of stress increase risk for disease and illness (McDade et al., 2006; Kumari et al., 2011), it is of critical importance to investigate ways in which the impact of stress on biological inflammatory processes may be reduced.

Posttraumatic Growth (PTG) and Health

Exposure to acute and chronic stressors (both in civilian and military populations) may be unavoidable and can have negative outcomes on both physical and psychological health; however, there is growing evidence that stressful events and trauma can also result in positive psychological outcomes and may, in certain circumstances, buffer individuals against the negative physiologic consequences of stress (Bower et al., 2009; Bush, Skoop, McCann, & Luxton, 2011; Tedeschi & McNally, 2011). Specifically, studies have indicated that long-term morbidity and mortality are less likely to occur if benefits are construed following the experience of traumatic circumstances (Affleck, Tennen, Crood, & Levine, 1987). Benefit finding following traumatic circumstances has been associated with the adoption of adaptive health behaviors (i.e., increased exercise and social interaction) in patients recovering from myocardial infraction (Petrie, Buick, Weinman, & Booth, 1999) and a variety of studies have shown that perceiving benefits or

perceiving growth following traumatic experiences may promote better coping, survival, and adaptation (Tedeschi & Calhoun, 1996; Park, Cohen, & Murch, 1996; Petrie et al., 1999; Joseph, Linley, Andrews, Harris, Howle, Woodward, & Shevlin 2005; Bower et al., 2009; Diaz et al., 2014).

Philosophers and scientists have long sought to understand the characteristics associated with those who find benefits, or grow, as a result of their experience with trauma. Tedeschi & Calhoun (1996), coined the term posttraumatic growth (PTG) to refer to the positive psychological changes which may occur following traumatic experience or exposure. The authors suggest that PTG is unique from other similar terms such as adversarial growth (Joseph et al., 2005) or stress-related growth (Park et al., 1996) in that PTG reflects an ongoing, evolutionary, and ultimately transformative process rather than a short-term coping mechanism in response to acute or chronic stressors. Tedeschi & Calhoun (2004) further distinguish the phenomenon of PTG from resiliency, or the ability to resist or bounce back from adversity. According to Tedeschi and Calhoun (2004) PTG represents more than return to pre-trauma levels of functioning, reflecting an ultimate change in character, personality, or identity – a transformation- that occurs within various domains of one's life.

The theory of PTG suggests that when a traumatic experience occurs, one's assumptive world, or system of core beliefs about one's self or the world, is shattered (Tedeschi and Calhoun, 1996; Tedeschi & Calhoun, 2004). It is important to note that simply experiencing a traumatic event is not sufficient to prompt growth, and the shattering of one's belief system does not guarantee an experience of PTG. Rather, the experience of PTG emerges following periods of complex cognitive and emotional

processing during which one actively works to recreate their assumptive world in such a way that it will "withstand future shocks to the system, much as communities rebuild in the aftermath of an earthquake, strengthening the self produces confidence in facing future difficulties, and existential reevaluation can produce a sense of wisdom, life satisfaction, and purpose in life (Tedeschi, 2011, p. 137)." Somewhat paradoxically, the presence of distress is a necessary precursor to an experience of growth. Therefore, it is not uncommon for high correlations to be noted between the presence of PTG and psychological distress (i.e., depression, sadness, anxiety, intrusive thoughts, guilt, etc.) (Tedeschi & Calhoun, 2004). Additionally, studies document that the experience of PTG has the potential to manifest in various domains of an individual's life (Tedeschi & Calhoun, 1996). Specifically, PTG has been noted in the experience of more meaningful and intimate relationships with others, a sense of increased personal strength and ability to face or overcome future struggles, the realization of new possibilities in one's life, the development, recognition, or expression of a greater appreciation for life, and the experience of spiritual change such that one's spirituality is enhanced, redefined, or existentially explored (Tedeschi & Calhoun, 1996).

Substantial literature has documented PTG in a wide variety of populations, and corresponding outcomes have included experiences of increased compassion and empathy for others, changes in religious beliefs and spirituality, a greater sense of social connection and decreased suicidal ideation (Tedeschi & Calhoun, 1996; Bush et al., 2011). Further, the experience of PTG has also been implicated as a potential protective factor for physical health, demonstrating inverse associations with flattened cortisol

slopes and disease progression in samples of breast cancer patients (Dunnigan, Carr, & Steel, 2007; Bower et al., 2009; Bush et al., 2011; Diaz et al, 2014).

Aims of the Present Study

The negative impact that exposure to traumatic or chronic stress may have on one's psychological and physical health is well documented; however, to date there has been relatively little examined on the potential physiological benefits accompanying an experience of PTG following exposure to trauma (Diaz et al., 2014). Further, to the author's knowledge there exists no current studies documenting biological associations of an experience of PTG in military populations. Therefore, the objective of this study was to elucidate salutary benefits of PTG for the physical and mental health of military Service veterans.

Aim 1: To compare a sample of military combat Veterans who perceived their combat experience as traumatic with nontrauma perceiving military combat Veterans on physiological markers of chronic stress as indexed by the diurnal slope of cortisol and C-reactive protein levels.

Research Question 1: Does the perception of combat as traumatic in military combat Veterans predict increases in biomarkers of chronic stress?

Hypothesis 1*:* OEF/OIF Veterans who perceive their combat exposure as traumatic are significantly more likely than non-traumatized combat veterans to have elevations in physiological markers of chronic stress and systemic inflammation, as indicted by the diurnal slope of cortisol and levels of C-Reactive protein (CRP).

Aim 2*:* To assess if the experience of PTG buffers against the negative physiologic effects of stress, protecting combat Veterans against CVD risk.

Research Question 2**:** Will military combat Veterans reporting higher levels of PTG have reduced elevations in physiologic markers of chronic stress associated with CVD?

Hypothesis 2: The experience of PTG in military combat Veterans will moderate the relationship between perceived stress and biomarkers of chronic stress, as indicated by a steeper diurnal cortisol slope and lower levels of CRP.

METHODS

Participants

Participants were recruited from the University of North Carolina at Charlotte (UNCC) and the surrounding community. All participants were male combat Veterans of OEF or OIF; there were no other exclusionary criteria with a goal of 20 participants per combat experience group. At time of recruitment, potential participants were categorized into one of two combat experience groups: (1) Non-trauma: Veterans who do not perceive their combat exposure as traumatic; and (2) Trauma: Veterans who do perceive their combat exposure as traumatic (note that any veteran who indicated their combat was non-traumatic were excluded from Group 2).

Procedure

The proposed study was submitted as a new protocol and approved by the UNCC Institutional Review Board prior to recruitment and data collection. Participants were assigned a unique ID number at the time of consent and all study data was de-identified. All study information was collected and entered using a password protected and encrypted laptop computer. Participants were recruited through UNCC Veteran Student Services, the UNCC Office of Academic Affairs, and through advertisements (print, facebook, and word of mouth) providing contact information for study participation. Prior to meeting with participants in person, research staff communicated with participants either by phone or by email to screen participants for inclusion criteria. Participants had to be male, have served in either OEF or OIF and have been exposed to combat to qualify for participation. Research staff screened participants with a question assessing the perceived traumatization of combat exposure. Participants were asked if

they perceived their combat exposure as traumatic. Responses were recorded on a dichotomous yes/no scale.

Following screening procedures, research staff scheduled an appointment to meet with participants in person. At this time, research staff conducted informed consent and took participant's temperatures. If a participant presented with a temperature at or above 99.6 degrees Fahrenheit, they were asked to postpone beginning the study/saliva collection for two weeks after their temperature dropped to a healthy range (i.e., less than 99 degrees Fahrenheit). All participant temperatures fell within an acceptable range and none were asked to return at a later date.

Following consent, research staff collected biometric information, including height, weight, and blood pressure. Participants then completed the study assessments. This process took approximately 35-75 min to complete. Once participants completed their assessments, research staff provided participants with a saliva collection kit that included detailed instructions regarding how to collect and store saliva samples. Participants were asked to return home and collect saliva samples at three time points- upon waking , at lunch, and before going to bed – across 2 days. Participants placed the synthetic swab from the salivette on their tongue and rolled it around in their mouth for approximately two minutes. The waking sample was to be taken within 30 minutes of waking and the second sample was taken before going to bed. Prior to saliva collection, participants were asked to avoid exercise, eating, or drinking 3 hours before saliva collection, record their waking time, and the saliva collection time. Participants stored salivettes in a refrigerator until returning them to UNCC, at which point research personnel transferred the biospecimens to the Stress*WAVES* BRL for processing, long

term storage, and biomarker assessment. Total participation time, including biospecimen collection, took approximately 60-120 minutes. Upon completing biospecimen and data collection, participants were compensated $25 for participation and an anonymous $5 donation was made to the Wounded Warrior Foundation on behalf of their participation.

Measures

Demographics. Participants reported their age, marital status, current military rank or rank at separation, total number of deployments, and deployment location. For racial and ethnic background, participants reported the group that best represented themselves from preselected options (i.e., White, African American, Hispanic or Latino, etc.). Educational background was assessed by asking respondents the highest level of education they had completed on a six-point scale (1 = less than seven years, 6 = graduate or professional training). Income was assessed by asking participants the range that best described their pre-tax household income in the last year on a seven-point scale (1 = less than $10,000, 7 = more than $100,000).

Average exercise levels were assessed with a single question asking participants how often, on average, they exercised per week (1= *0 days/week*, 5=*6-7 days/week*). Height, weight, and blood pressure were measured and recorded by study staff. BMI was calculated by computing a ratio of participant height to weight as per CDC guidelines.

Smoking status was assessed using questions adapted from the National Social Life, Health, and Aging Project (Drum et al., 2009). Respondents were asked if they currently smoke cigarettes (yes/no) and if so how long they have been smoking and how many cigarettes on average they smoke per day. Participants were coded as 0 if they did not currently smoke cigarettes and 1 if they did.

Perception of Combat Exposure. To screen participants for sample allocation and assess the degree to which their combat exposure is perceived as traumatic or non-traumatic, participants were asked to respond on a dichotomous yes/no scale as to whether they perceived their combat exposure as traumatic. Participants indicating YES to Question 1 were placed into Sample 1 (trauma) and participants indicating NO to question 1 were placed into Sample 2.

Combat Exposure. Combat exposure was assessed with the Combat Experiences Scale (CES, King, King, Vogt, Knight, & Samper, 2006; Vogt, Proctor, King, King, & Vasterling, 2008), a 15-item self-report measure of objective combat experiences. Scale items include "I or members of my unit received hostile incoming fire from small arms, artillery, rockets, mortars, or bombs", "I went on combat patrols of missions", "I was wounded or injured in combat", and "I killed or think I killed someone in combat". Items are noted on a 5-point Likert scale ranging from "Never" to ""Extremely Frequently" and summed. Scores can range from 15-75 and higher total scores indicate greater combat exposure. Cronbach's alpha for this study was 0.92, indicating excellent internal consistency.

Perceived Psychological Stress. Perceived psychological stress (PPS) was assessed using Cohen, Kamarck, & Mermelstein's (1983) fourteen-item Perceived Stress Scale, a well-validated measure designed to assess how overloaded, unpredictable, and uncontrolled respondents felt about their lives in the past 30 days. Scale items include "In the last month how often have you felt unable to control things in your life; how often have your felt confident in your ability to handle personal problems; how often have you felt that things were going your way; and how often have you felt that difficulties were

piling up so high that you could not overcome them?" Items are noted on a 4-point Likert scale ranging from "Never" to ""Very Often" and higher scores indicate higher levels of stress. Cronbach's alpha for this study was 0.83, indicating good internal consistency.

Posttraumatic Stress Disorder. Current levels of PTSD symptomology were assessed with the PTSD Checklist-Version (PCL-5), a widely-used 20-item self- report scale designed to measure distress associated with each PTSD symptom (Weathers *et al*., 2013). Scale items include: In the past month, I have experienced "Avoiding memories, thoughts, or feelings related to the stressful experience", and "Repeated, disturbing, and unwanted memories of the stressful experience". Items are noted on a 5-point Likert scale ranging from "Not at all" to "Extremely" and summed. Scores can range from 0-80, and a cut point of 33 is suggested as indicative of PTSD diagnosis. Cronbach's alpha for this study was 0.95, indicating excellent internal consistency.

Distress. Distress was operationalized as experiences of depression, anxiety, and stress and measured with the Depression, Anxiety, and Stress Scale (DASS; Lovibond & Lovibond, 1995), a 21-item measure assessing symptoms of depression, anxiety, and stress that in the past week. Scale items include "I couldn't seem to experience any positive feeling at all", "I found it hard to wind down", and "I felt I was close to panic". Items are noted on a 4-point Likert scale ranging from "Did not apply to me at all" to "Applied to me very much, or most of the time" and summed. Scores can range from 0-63. Higher scores indicate greater distress. Cronbach's alpha for this study was 0.95, indicating excellent internal consistency.

Posttraumatic Growth. PTG was assessed with the Posttraumatic Growth Inventory (PTGI; Tedeschi & Calhoun, 1996), a 21-item measure assessing positive

psychological changes following exposure to a traumatic experience. Scale items include: As a result of my experience, “I have changed by priorities about what is important in life”, “I can better appreciate each day”, and “I have more compassion for others”. Items are noted on a 5-point Likert scale ranging from “I did not experience this change” to “I experienced this change to a very great degree” and summed. Scores can range from 0-105, and higher scores indicate greater levels of growth. Cronbach’s alpha for this study was 0.91, indicating excellent internal consistency.

Biomarkers**.** Cortisol and CRP were assessed via saliva collected with a synthetic swab salivette (Sarstedt, Newton, NC) and assayed with commercially available enzyme immunoassay kits (Salimetrics, LLC, State College, PA).

Data Analysis

Missing Data

Data was first reviewed for completeness. 41 veterans completed self-report surveys; however, 3 participants did not provide corresponding saliva samples and were therefore excluded from our analyses, bringing our sample to 38. Descriptive statistics were then evaluated to ensure that the data are normal via reasonable standard deviations and means with no signs of outliers or entry errors, but also contained enough variance to be analyzed. We identified 5 outliers with significantly higher levels of CRP then the rest of the sample (i.e., 10.00 or higher). This introduced the possibility that blood could have entered their saliva and skewed both cortisol and CRP data. Therefore, to be conservative, we excluded these 5 participants from our analyses bringing the final sample to 33. Results remained significant with and without the exclusion of the 5 outliers.

Analyses

To test Hypothesis 1 and calculate mean differences in CRP levels and cortisol slope, a one-way analysis of variance (ANOVA) was conducted with trauma perception (0=yes, 1=no) as the grouping variable. Preliminary zero-order Pearson's correlations examined potential confounds. Continuous covariates (*e.g.*, age, BMI, etc.) were transformed to z-scores to aid in interpretation of the first order coefficients and enhance the simple slopes plot (Cohen, Cohen, West, & Aiken, 2003; Hayes & Matthes, 2009). Total daily hours awake were transformed to a 24-hour format. Any bedtime later than 12:00am was added to 24 (i.e., 1am = 25), and minute allotments were divided by 60 to obtain a decimal point. Cortisol and CRP data were natural log transformed before

analysis to achieve a residual distribution that was approximately normal (Bennett, Glaser, Andridge, Peng, Malarkey, & Kiecolt-Glaser, 2014). Cortisol slope was computed by subtracting the cortisol data from the final daily time from the first daily cortisol sample and divided by the total amount of time each participant had been awake.

To test Hypothesis 2 and calculate the moderating effect of PTG on the relationships between perceived stress and both CRP and daily cortisol slopes, an interaction term was calculated and three separate step-wise hierarchical multiple regression analyses were conducted. First, the perceived stress and PTG variables were mean centered then multiplied together to create an interaction term. Centered and control variables were introduced as predictors in step one, and perceived stress (centered) and PTG (centered) as predictors in step two. In step three, the interaction term was added as a predictor to determine if these two variables have an interactive effect above and beyond the variables by themselves (Cohen, Cohen, West, & Aiken, 2003).

Three separate moderated multiple regression analyses were conducted with (1) CRP, (2) Day 1 cortisol slope, and (3) Day 2 cortisol slope as separate criterion. Based on the results of Hypothesis 1 (see below), we did not separate the groups based on trauma perception and ran all 33 participants in each model.

While existing literature (Cohen, Doyle, & Baum, 2006; Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004) associates age, BMI, smoking status, medication use, depression and exercise with the outcome variables, the small nature of our sample made it impossible to include all of these variables as relevant controls and still retain sufficient variability. Therefore, as age was the only control variable

significantly correlated with CRP, we controlled for this variables in each regression model. Analyses were carried out in SPSS (Version 23).

RESULTS

Descriptive Statistics

Detailed information on participant characteristics for the total sample and by trauma group can be found in Table 1. The mean age of participants (100% male) was 30.22 years (*SD* = 6.62; Range: 20-46). The sample was 79% White, 12% Non-European Hispanic or Latino, 3% American Indian/Alaskan Native; 3% Asian, and 3% African American. Military breakdown was as follows: 39% Army, 39% Marine Corps, 6% Navy, 56% Air Force, and 9% National Guard. On average, participants had served for 8.05 years (*SD* 5.50, Range: 1.5-23) and deployed 2.39 times (*SD* 1.65, Range: 1-9). The majority of participants were no longer actively serving (33 % Active Duty; 67% non-active/separated). Regarding educational attainment, 9% held a graduate/professional degree, 6% held a Bachelor's degree, and 85% held a high school diploma and were currently enrolled in college. Finally, income levels for this sample were as follows: 6% < $10,000, 15% $10,000-24,999, 30% $25,000-49,999, 30% $50,000-99,999, 18% > $100,000. The average number of combat experiences was 36.88 (*SD* = 13.83), average PTSD symptom levels were indicative of PTSD (*M*= 49.55, *SD*= 19.81), and participants reported generally high levels of stress (*M*=41.03, *SD*=8.64). The average score on the PTGI was 3.50 (*SD*=.98), and the average DASS score was 40.64 (*SD*=14.55).

Zero-Order Correlations

Zero-order Pearson correlations for all focal variables are presented in Table 2. Combat experiences were positively correlated with BMI ($r(33) = .33$, $p < .05$). DASS scores were positively correlated with both PCL scores ($r(33) = .76$, $p < .05$) and PSS scores ($r(33) = .76$, $p < .001$) and negatively correlated with smoking status ($r(33) = -.36$,

$p < .05$. Day 1 Cortisol slope was positively correlated with Day 2 Cortisol slope ($r(33) = .50$, $p < .01$), and negatively correlated with age ($r(33) = -.51$, $p < .01$). Day 2 Cortisol slope was positively correlated with PTGI scores ($r(33) = .43$, $p < .05$). While CRP was not statistically significant, the correlation with combat experiences was trending towards significance ($r(33) = .24$, $p = .06$. No statistically significant associations were found between PTG and perceived stress, CRP, or the cortisol slope from Day 1.

Hypothesis 1- Group Differences by Trauma Perception

One-way ANOVA Results

A one-way analysis of variance (ANOVA) was used to examine group differences in physiological markers of chronic stress as a function of trauma perception. Results (see Table 3) revealed significant group differences in the following variable means based on perception of combat exposure as traumatic or not: perceived stress ($F(1,33)= 8.64$, $p<.01$; $M_{trauma}= 44.88$, $SE= 1.72$; $M_{nontrauma}=36.94$, $SE=2.10$; $\eta^2= 0.22$); combat experiences ($F(1,33)= 64.02$, $p<.05$; $M_{trauma}= 41.35$, $SE= 3.86$; $M_{nontrauma}=32.13$, $SE=3.45$; $\eta^2= 0.15$); PTSD symptomology ($F(1,33)= 11.77$, $p<.001$; $M_{trauma}= 59.47$, $SE= 3.86$; $M_{nontrauma}=39.00$, $SE=4.59$; $\eta^2= 0.29$); and distress scores ($F(1,33)= 12.64$, $p<.001$; $M_{trauma}= 48.11$, $SE= 2.75$; $M_{nontrauma}=32.69$, $SE=3.39$; $\eta^2= 0.29$). There were no significant differences between mean scores of cortisol slope, CRP, or PTGI scores based on trauma perception.

Conclusion

Hypothesis 1 was not supported. In the present sample, there were not significant differences between mean levels physiological markers of chronic stress and systemic

inflammation, as indicted by diurnal cortisol slope and levels of C-Reactive protein (CRP) based on perception of combat exposure as traumatic or non-traumatic.

Hypothesis 2- The Influence of PTG on PPS and Chronic Stress Biomarkers

Moderated Regression Results

Model 1: Summary of Variables Predicting CRP

In step one, the control variable, age, did not explain a significant amount of variance in CRP (R^2 = -.03., $F(32)$ = .009, p = .93). In step two, the individual predictors did not explain significant amount of variance beyond the first model (R^2 = .06, ΔR^2 = .04, p = .55), and neither perceived stress (β = .01, p = .60) nor PTG (β = .32, p = .33) significantly predicted CRP when holding the control constant. In step three, the interaction term (β = .03, p = .54) did not incrementally predict CRP (R^2 = -.09, ΔR^2 = .01, p = .54) indicating that together, perceived stress and PTG do not have an interactive effect that increases the predictive validity beyond the second model.

Even though the interaction in step three of the hierarchical analysis was not significant, to help aid in data interpretation and to assess data trends given our small sample, the simple slopes were plotted using the Hayes ModProbe macro for SPSS reflecting both centered continuous perceived stress scores and centered continuous PTG scores . The simple slopes (see Figure 1) revealed a disordinal interaction between perceived stress and PTG. Specifically, at low levels of stress PTG did not appear to play a significant role in CRP outcomes. However, at higher levels of stress, higher reports of PTG were associated with an increased CRP values. Thus, PTG was not a buffer and appeared to exacerbate perceived stress' effect on CRP.

Model 2: Summary of Variables Predicting Day 1 Cortisol Slope

As shown in Table 5, in step one, the control variable, age, explained a significant amount of variance in Day 1 cortisol slope (R^2 = .26, F(32) = 10.72, p = .003). In step two, the individual predictors did not explain significant amount of variance beyond the first model (R^2 = .30, ΔR^2 = .03, p = .54) with neither perceived stress (β = .00, p = .38) nor PTG (β = -.00, p = .53) significantly predicting Day 1 cortisol slope, while holding the control constant. In step three, the interaction term (β = .00, p = .24) did not incrementally predict cortisol slope (R^2 = .33, ΔR^2 = .04, p = .24) indicating that together, perceived stress and PTG do not have an interactive effect that increases the predictive validity beyond the second model.

Similar to Model 1, even though the interaction in step three of the hierarchical analysis was not significant, the simple slopes were plotted using the Hayes ModProbe macro for SPSS reflecting both centered continuous perceived stress scores and centered continuous PTG scores. The simple slopes (see Figure 2) revealed a disordinal interaction between perceived stress and PTG. Specifically, at low levels of stress PTG did not appear to play a significant role in Day 1 cortisol slope outcomes. However, at higher levels of stress, higher reports of PTG were associated with a flatter and slightly *positive* cortisol slope across the day. Thus, PTG was not a buffer and appeared to exacerbate perceived stress' effect on Day 1 diurnal cortisol slope.

Model 3: Summary of Variables Predicting Day 2 Cortisol Slope

As shown in Table 6, in step one, control variable, age, did not explain a significant amount of variance in Day 2 cortisol slope (R^2 = .09, F(32) = 2.77, p = .11). In step two, the individual predictors explained a significant amount of variance beyond the

first model (R^2 = .18, ΔR^2 = .18, p = .05) with PTG (β = .01, p = .03) significantly predicting Day 2 cortisol slope, while holding the control constant. Perceived stress (β = .00, p = .30) did not significantly predict cortisol slope in step two. In step three, the interaction term (β = .001, p = .03) incrementally predicted Day 2 cortisol slope (R^2 = .29, ΔR^2 = .12, p = .03) indicating that together, perceived stress and PTG have an interactive effect that increases the predictive validity beyond the second model.

Because step three of the hierarchical analysis revealed a statistically significant interaction between perceived stress and PTG on Day 2 cortisol slope, the simple slopes were plotted using the Hayes ModProbe macro for SPSS reflecting both centered continuous perceived stress scores and centered continuous PTG scores. The simple slopes (see Figure 1) revealed a disordinal interaction between perceived stress and PTG. Specifically, at low levels of stress PTG did not appear to play a significant role in cortisol slope outcomes. However, at higher levels of stress, higher reports of PTG were associated with a flatter and slightly *positive* cortisol slope across the day. Thus, PTG was not a buffer and appeared to exacerbate perceived stress' effect on the flattening of the Day 2 diurnal cortisol slope.

Conclusion

Hypothesis 2 was not supported. PTG did not moderate the relation between perceived stress and CRP or Day 1 cortisol slope. While PTG did moderate the relationship between perceived stress and Day 2 cortisol slope, we hypothesized that PTG would buffer against indicators of chronic stress, resulting in an increasingly negative slope, and the present interaction resulted in a flatter and increasingly positive cortisol slope.

DISCUSSION

The present study sought to examine associations among perceived psychological stress (PPS), posttraumatic growth (PTG), and physiological markers of chronic stress (i.e., CRP and diurnal cortisol slope). Our study had two primary aims: first, to explore if one's perception of military combat as traumatic had a significant influence on physiological markers of chronic stress. Specifically, we predicted that OEF/OIF Veterans who perceived their combat exposure as traumatic would be significantly more likely than non-traumatized combat veterans to have a flatter diurnal cortisol slope and elevated levels of C-Reactive protein (CRP). The second aim of the study sought to assess if experiences of PTG buffered against the negative physiologic effects of stress. Specifically, we predicted that the experience of PTG in military combat Veterans would moderate the relationship between perceived stress and biomarkers of chronic stress, as indicated by a steeper negative diurnal cortisol slope and lower levels of CRP.

Hypothesis 1

Our first hypothesis was not confirmed. There were not statistically significant differences between mean levels of morning or evening cortisol, diurnal cortisol slope, or CRP based on perception of combat exposure as traumatic or non-traumatic. It is possible that the lack of significant differences in chronic stress markers between groups reflects the sustained and enhanced psychoneuroendocrine reactivity that may occur following exposure to combat (Department of Defense, 2015; Van Wingen, Geuze, Vermetten, & Fernandez, 2011). It is also possible that the time since trauma exposure influenced results, a notion that the current study did not control for. Future studies should consider controlling for time since trauma exposure and comparing combat exposed military

personnel to controls never exposed to combat to see if differences in resting levels of cortisol and CRP are noted.

Further, while there were not statistically significant differences between groups on CRP based on trauma perception, the correlation of CRP with combat experiences was trending toward significance (r=.24; p=.06). This is consistent with previous research demonstrating high levels of CRP in combat-exposed service members (Groer, Kane, Williams, & Duffy, 2015), suggesting that combat experiences (i.e., being under enemy fire, witnessing soldiers/civilians seriously injured or killed, participating in firefights, etc.) and inflammatory responses may have an additive effect, increasing the risk for CVD and chronic illness in service members based on degree and number of combat experiences. The present study did not explore this possibility; however, this would be an interesting area of future research.

Hypothesis 2

C-Reactive Protein (CRP)

Our second hypothesis was also not supported. PTG did not moderate the relation between PPS and CRP. Previous research has suggested that frequent exposure to stressors and prolonged states of arousal may take a toll on physiology despite the presence of positive psychological traits and physical resilience (Epel, McEven, & Ickovics, 1998). Therefore, it is possible that the presence of PTG is not sufficient to offset physical toll of serving in theater (i.e., within an active warzone). Alternately, as PTG has been described as both an ongoing process and outcome (Calhoun & Tedeschi, 1998; Cann et al, 2010), it is possible that there had not been sufficient time elapsed since combat exposure for the beneficial impact of PTG to be noted. Future work should

examine the influence of PTG on CRP levels longitudinally to see if study results vary as a function of time.

Diurnal Cortisol Slope

Our buffering hypothesis was not also supported when looking at Day 1 and Day 2 diurnal cortisol slopes; however, PTG significantly interacted with PSS scores to predict a flatter and more positive Day 2 cortisol slope. It is important to note that this interaction was found only with Day 2 cortisol slope. There was not a significant interaction between PTG and PSS scores on Day 1 cortisol slope. It is possible that the differences between Day 1 and Day 2 cortisol could be due to an adjustment effect such that the participants had better adapted to the new procedure of providing saliva samples on Day 2; making the Day 2 cortisol slope a more accurate representation of their average cortisol levels. While the present study was limited to two days due to budgetary constraints, future work should seek to replicate these findings and analyze the cortisol over four or more days in accordance with standard biological procedures.

Finally, it is possible that the difference between days is due to the small sample and overall lack of power. As demonstrated in the non-significant simple slope plots (Figures 2 and 3), the diurnal cortisol slope from both Day 1 and Day 2 follow a similar pattern. Thus, follow-up analyses and future research should seek to assess cortisol slope and CRP in a larger sample to see if increased statistical power may bring congruence to daily cortisol slope significance levels.

The disordinal interaction between PTG and PSS scores on Day 2 cortisol slope revealed that PTG moderated the relationship between PPS and cortisol slope such that those reporting higher PTG at higher levels of stress had a flatter and slightly positive

diurnal cortisol slope in comparison to those reporting lower levels of PTG and higher levels of stress who had a steeper and more negative cortisol slope. This suggests that PTG is a process which may require energy and physiological support (i.e., cortisol) to occur. In addition, the interaction suggests that when current perceptions of stress are higher, one's body may have to work significantly harder to support processes of growth and meaning making compared to when one's perceptions of stress are lower.

While at first glance a positive cortisol slope for those reporting higher PTG may seem counterintuitive, it is important to remember that all participants had been exposed to at least moderate levels of combat for extended periods of time (i.e., 6 months or greater). As indicated by the DASS and PCL scores, average levels of distress and were high and a large number of study participants met criteria for DSM-V PTSD diagnosis. Therefore, it is possible that the positive cortisol slope may reflect a general state of hyperactivity in study participants consistent with prolonged environmental threat (Van Wingen et al, 2011). Future studies should consider comparing combat exposed military personnel to controls not exposed to combat to explore this possibility. Relatedly, the fact that those reporting lower levels of PTG at higher levels of stress did NOT demonstrate higher levels of cortisol may indicate a sign of dysregulation such that their bodies did not respond as sensitively to perceptions of stress. Future research should consider examining this further and employ additional methodology, such as an experimental stress manipulation, to see if these findings reflect overall differences is cortisol reactivity.

Additionally, chronic stress-related alterations in biomarkers are often interpreted as negative and as a manifestation of wear and tear on the body or allostatic load, as

though the system were a rubber band that lost elasticity (McEwen, 2004). However, an alternative interpretation is that these changes may represent not so much a loss of resources, but a reallocation (Aschbacher, O'Donovan, Wolkowitz, Dhabhar, Su, & Epel, 2013). Seen from this light, the positive daily cortisol slope associated with higher levels of PTG may not necessarily be a dysfunction or failure of the system, but a reflection of the processes by which one seeks to reconstruct their assumptive worldview in the aftermath of trauma. Thus, while causal conclusions cannot be derived from the present study, it is possible that the increased cortisol activation demonstrated in those participants reporting higher levels of growth and higher perceived stress supports the notion that PTG is a not just an outcome, but is also a process requiring bodily support in the form of physiological energy sources (i.e., cortisol).

PTG theory posits that PTG emerges following cognitive and emotional struggle that emerges from seismic disruption to the assumptive world (Calhoun, Cann, and Tedeschi, 2010; Calhoun & Tedeschi, 1998; Tedeschi & Calhoun, 2004). This experience is stressful and challenging both emotionally and cognitively and the trauma survivor brings to bear various coping resources in order to calm down the emotional reactions and intrusive ruminations that are set off by the trauma. This experience is also accompanied by ongoing ruminative processes that start with intrusive thoughts and images of trauma and give way to more deliberate, reflective thoughts (Cann et al., 2010). These deliberate ruminations are posited to be effortful processes whereby an individual actively engages with trauma related thoughts, memories, and emotions (Calhoun & Tedeschi, 1998; Tedeschi & Calhoun, 2004). These deliberate ruminations drive the reconstruction of the assumptive world, and their very nature (i.e., deliberate and

effortful) would suggest that they represent a form of stress (albeit good stress, or eustress). Therefore, as PTG is an inherently challenging (i.e., stressful) process, cortisol would be an important source of physiological support as psychological and physical resources are reallocated to incorporate one's new reality.

Finally, the model of PTG suggests that growth is a process which may be dependent on time and degree of resolution. Specifically, as indicated by Zolner and Maecker (2006), PTG may initially be predicted by concurrent intrusion/distress level and openness; however, once meaning has been made and resolution achieved, PTG may be best predicted by openness and initial PTSD severity. The latter finding may point to the fact that those who suffered to a great degree have simultaneously more potential to grow from the experience. From this perspective, that those with higher PTG at higher stress had a positive cortisol slope may be due to the fact that they experienced a higher level of distress overall. Future research must endeavor to replicate these findings and incorporate both a longitudinal design and assessment of resolution before making any causal assumptions.

Limitations and Future Directions

It is important to note that the present cross-sectional study provides only a snapshot (i.e., cross-sectional) view of a group of male OEF/OIF combat veterans with varying histories and attributes. Our sample was small (N=33) and underpowered, and we did not compare participants to a control group. We also did not collect data assessing years since most recent deployment or years since military separation. Thus, this study presents many limitations and should be considered as a first step in understanding the biobehavioral experiences of this unique and growing population. Collection of

longitudinal data and the inclusion of both females and a comparison group of individuals not engaged in military life/combat would be the next logical step in this research program.

CRP is a robust inflammatory marker associated with chronic illness and is widely used by medical and academic professionals to assess systemic inflammation levels. However, it is only a single biomarker of systemic inflammation. Future work should extend these analyses to include other proinflammatory biomarkers associated with chronic illness, such as IL-6, IL-1, and TNFα, to assess the influence of PTG on the relation between PPS and multiple markers of systemic inflammation. Cortisol was assessed at only two time points over the course of two days. Assessing cortisol at three or more time points would allow us to assess the influence of PTG on cortisol from a curvilinear perspective, consistent with past work supporting this type of analysis (Kleim & Ehlers, 2009). Future work should also consider investigating the role of delayed cortisol recovery, which might provide a better assessment of chronic stress reactivity and give support to the wear and tear model. Finally, future research must endeavor to incorporate both a longitudinal design and assessment of traumatic resolution before making any causal assumptions.

Conclusions

Despite these noted limitations, this study provided a beginning view of experiences of PTG in a military population using a perspective provided by physiological stress theory. Results demonstrated that there were not significant differences in physiological markers of chronic stress despite one's perception of military combat as traumatic or not, which may reflect a general physiological hyperactivity

related to prolonged combat exposure. Further, while PTG did not significantly interact with PSS scores to predict a steeper negative cortisol slope or decreased levels of CRP, a significant interaction was found in Day 2 cortisol slope such that those reporting higher PTG at higher levels of stress had a flatter and slightly positive diurnal cortisol slope in comparison to those reporting lower levels of PTG and higher levels of stress who had a steeper and more negative cortisol slope. This flatter and more positive cortisol slope in those reporting higher growth at higher levels of stress may represent a reallocation of physiological resources needed to support the reconstruction of the assumptive worldview following trauma. Alternately, it may reflect that PTG is a coping strategy through which those who are highly reactive to stress attempt to cope with their trauma.

In sum, study findings provide preliminary initial support for biobehavioral associations with experiences of PTG and introduce CRP and cortisol as potential mechanisms through which PTG influences physical health. Caution is directed to future assumptions that PTG has a strictly salutary effect on physical markers like diurnal cortisol slope and CRP, and it is recommended that longitudinal follow-up studies are conducted to investigate more proximal links between PTG and biobehavioral outcomes.

REFERENCES

Affleck, G., Tennen, H., Croog, S., & Levine, S. (1987). Causal attribution, perceived benefits and morbidity after a heart attack: An 8 year study. *Journal of Consulting and Clinical Psychology, 55,* 29–35.

American Heart Association. (2014). Heart disease and strokes statistics – 2014 update. Dallas, TX: *The American Heart Association*.

American Heart Association (2013). Heart disease and strokes statistics – 2013 update. Dallas, TX: *The American Heart Association*.

Anderson, J., Wade, M., Possemato, K., & Ouimette, P. (2010). Association between posttraumatic stress disorder and primary care provider-diagnosed disease among Iraq and Afghanistan veterans. *Psychosomatic Medicine, 72,* 1-7.

Aschbacher, K., O'Donovan, A., Wolkowitz, O. M., Dhabhar, F. S., Su, Y., & Epel, E. (2013). Good stress, bad stress and oxidative stress: insights from anticipatory cortisol reactivity. *Psychoneuroendocrinology*, *38*(9), 1698-1708.

Belkic, K.L., Landsbergis, P.A., Schnall, P.L., & Baker D. (2004). Is job strain a major source of cardiovascular disease risk? *Journal of Work & Environmental Health, 30,* 85–128.

Bennett, J. M., Glaser, R., Andridge, R. R., Peng, J., Malarkey, W. B., & Kiecolt-Glaser, J. K. (2013). Long lasting effects of smoking: Breast cancer survivors' inflammatory responses to acute stress differ by smoking history. *Psychoneuroendocrinology, 38,* 179-187. doi: 10.1016/j.psyneuen.2012.05.012

Binder, E. B. (2009). The role of FKBP5, a co-chaperone of the glucocorticoid receptor in the pathogenesis and therapy of affective and anxiety disorders. *Psychoneuroendocrinology, 34,* S186-S195.

Boscarino, J.A. (2008). Psychobiologic predictors of disease mortality after psychological trauma: Implications for research and clinical surveillance. *Journal of Nervous & Mental Disease,* 196(2), 100-107.

Bower, J.E, Moskowitz, J.T., & Epel, E. (2009). Is benefit finding good for your health? Pathways linking positive life changes after stress and physical health outcomes. *Current Directions in Psychological Science,* 18(6), 337-341.

Bray, R.M., Pemberton, M.R., Hourani, L.L., Witt, M., Olmstead, K.L.R., Brown, J.M., et al. (2008). Department of Defense Survey of Health Related Behaviors Among Active Duty Military Personnel. *RTI International*; 2009.

Bush, N.E., Skopp, N.A., McCann, R., & Luxton, D.D. (2011). Posttraumatic growth as protection against suicidal ideation after deployment and combat exposure. *Military Medicine. 176*(11), 1215-1222.

Buysse, D.J., Reynolds, C.F., Monk, T.H., Berman, S.R., Kupfer, D.J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry, 28,* 193–213.

Cacioppio, J. T., & Berntson, G. G. (2011). The brain, homeostasis, and health: Balancing demands of the internal and external milieu. In H. S. Friedman (Ed.), *The Oxford Handbook of Health Psychology*. New York: Oxford University Press.

Calhoun, L. G., Cann, A., & Tedeschi, R. G. (2010). The posttraumatic growth model: Sociocultural considerations. *Posttraumatic growth and culturally competent practice: Lessons learned from around the globe*, 1-14.

Calhoun, L. G., & Tedeschi, R. G. (1998). Beyond recovery from trauma: Implications for clinical practice and research. *Journal of social Issues*,*54*(2), 357-371.

Cann, A., Calhoun, L.G., Tedeschi, R.G., Kilmer, R.P., Gil-Rivas, V., Vishnevsky, T., & Danhauer, S.C. (2010). The Core Beliefs Inventory: a brief measure of disruption in the assumptive world. *Anxiety, Stress, and Coping*, *23*(1): 19-34.

Centers for Disease Control and Prevention (2015). Web-based. *Health, United States 2015: With Special Feature on Death and Dying.*

Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). Applied multiple regression/correlation analysis for the behavioral sciences, 3rd edition Mahwah, NJ: Erlbaum.

Cohen, S., Doyle, W. J., & Baum, A. (2006). Socioeconomic status is associated with stress hormones. *Psychosomatic Medicine, 68*, 414-420

Cohen, B.E., Marmar, C., Ren, L., Bertenthal, D., & Seal, K.H. (2009). Association of cardiovascular risk factors with mental health diagnoses in Iraq and Afghanistan war veterans using VA health care. *JAMA, 305*(5), 489-492.

Cohen, S., Janicki-Deverts, D., & Miller, G.E. (2007). Psychological stress and disease. *Journal of American Medical Association, 298*, 1685-1687.

Cohen, S., Kamarck, T., Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior*, *24*, 385-396.

Cohen, S., Tyrrell, D. A. J., & Smith, A. P. (1993). Negative life events, psychological stress, negative affect, and susceptibility to the common cold. *Journal of Personality and Social Psychology*, *64*, 131–140.

Danese, A. & McEwen, B. S. (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology & Behavior*, *106*, 29-39.

Diaz, M., Aldridge-Gery, A., & Spiegel. D. (2014). Posttraumatic growth and diurnal cortisol slope among women with metastatic breast cancer. *Psychoneuroendocrinology*, *44*, 83-87.

Dimsdale, J. (2008). Psychological stress and cardiovascular disease. *Journal of the American College of Cardiology, 51*(13), 1237-1246.

Dockray, S., & Steptoe, A. (2010). Positive affect and psychobiological processes. *Neuroscience and Biobehavioral Reviews, 35*(1), 69-75.

Dunnigan, J., Carr, B., & Steel., J. (2007). Posttraumatic growth, immunity and survival in patients with hepatoma. *Digest and Discussions in Science, 52,* 2452-2459.

Epel, E.S., McEwen, B.S., & Ickovics, J.R. (1998). Embodying Psychological Thriving: physical thriving in response to stress. *Journal of Social Issues, 54*(2): 302-322.

Groer, M.W., Kane, B., & Williams, S.N. (2015). Relationship of PTSD symptoms with combat exposure, stress, and inflammation in American soldiers. *Biological Research for Nursing*, *17*(3): 303-310.

Hall, B. J., Hobfoll, S. E., Palmieri, P. A., Canetti-Nisim, D., Shapira, O., Johnson, R. J., & Galea, S. (2008). The psychological impact of impending forced settler disengagement in Gaza: Trauma and posttraumatic growth.*Journal of traumatic stress*, *21*(1), 22-29.

Hänsel, A., Hong, S., Cámara, R. A., & von Känel, R. (2010). Inflammation as a psychophysiological biomarker in chronic psychosocial stress. *Neuroscience and Biobehavioral Reviews, 35*(1), 115-121.

Hayes, A. F., & Matthes, J. (2009). Computational procedures for probing interactions in OLS and logistic regression: SPSS and SAS implementations. *Behavior Research Methods, 41(3),* 924-936**.**

Heppner, P.S., Crawford, E.F., Haji, U.A., Afari, N. Hauger, R.L., et al. *(*2009). The association of posttraumatic stress disorder and metabolic syndrome: A study of increased health risk in veterans. *BMC Medicine, 7*(1), 1-8.

Hobfoll, S. E., Hall, B. J., Canetti-Nisim, D., Galea, S., Johnson, R. J., & Palmieri, P. A. (2007). Refining our understanding of traumatic growth in the face of terrorism: Moving from meaning cognitions to doing what is meaningful. *Applied Psychology*, *56*(3), 345-366.

Holman, E. A., Silver, R. C., Poulin, M., Andersen, J., Gil-Rivas, V., & McIntosh, D. N. (2008). Terrorism, acute stress, and cardiovascular health: A 3- year national study following the September 11th attacks. *Archives of General Psychiatry, 65,* 73-80.

Jamieson, J. P., Knock, M. K., & Mendes, W. B. (2012). Mind over matter: reappraising arousal improves cardiovascular and cognitive responses to stress. *Journal of Experimental Psychology: General, 141*, 417-422.

Johnson, M. L., Pietz, K., Battleman, D.S., & Beyth R.J. (2004). Prevalence of comorbid hypertension and dyslipidemia and associated cardiovascular disease, *American Journal of Managed Care, 8,* 926-932.

Joseph, S., Linley, A., Andrews, L., Harris, G., Howle, B., Woodward, C., Shevlin, M. (2005). Assessing positive and negative changes in the aftermath of adversity: Psychometric evaluation of the changes in outlook questionnaire. *Psychological Assessment, 17*(1), 70-80.

Kario, K., McEven, B.S., & Pickering, T.G. (2002). Disasters and the heart: A review of the effects of earthquake-induced stress on cardiovascular health, *Hypertension Research, 26,* 355-368.

Kashani, M., Eliasson, A., & Vernalis, M. (2011). Perceived stress correlated with disturbed sleep: A link connecting stress and cardiovascular disease, *Stress, 1,* 1-7. DOI: 10.3109/10253890.2011.578266.

Kemeny, M. E. (2011). *Psychoneuroimmunology*. In Oxford Handbook of Health Psychology, H. S. Friedman (pp. 138-161). New York: Oxford University Press.

Kiecolt-Glaser, J. K., Preacher, K. J., MacCallum, R. C., Atkinson, C., Malarkey, W. B., & Glaser, R. (2003). Chronic stress and age-related increases in the proinflammatory cytokine IL-6. *Proceedings of the National Academy of Sciences USA, 100*(15), 9090-9095.

King, L. A., King, D. W., Vogt, D. S., Knight, J., & Samper, R. E. (2006). Deployment risk and resilience inventory: A collection of measures for studying deployment-related experiences of military personnel and veterans. *Military Psychology, 18*, 89–120. doi:10.1207/s15327876mp1802_1

Kleim, B., & Ehlers, A. (2009). Evidence for a curvilinear relationship between posttraumatic growth and posttrauma depression and PTSD in assault survivors. *Journal of traumatic stress*, *22*(1), 45-52.

Kudielka, B. M., Buske-Kirschbaum, A., Hellhammer, D. H., & Kirschbaum, C. (2004). HPA axis responses to laboratory psychosocial stress in healthy elderly adults, younger adults, and children: Impact of age and gender. *Psychoneuroendocrinology, 29*, 83–98.

Kumari, M., Shipley, M., Stafford, M., & Kivamaki, M. (2011). Association of diurnal patterns in salivary cortisol with all-cause and cardiovascular mortality: Findings from the Whitehall II study, *Journal of Clinical Endocrinology and Metabolism,96*(5), 1478-1485.

Lovibond, S.H. & Lovibond, P.f. (1995). *Manual for the Depression anxiety Stress Scales*. (2nd Ed) Sydney: Psychology Foundation

McDade, T. W., Hawkley, L. C., & Cacioppio, J. T. (2006). Psychosocial and behavioral predictors of inflammation in middle-aged and older adults: The Chicago health, aging, and social relations study. *Psychosomatic Medicine, 68*, 376-381.

Melamed, S., Kushnir, T., & Shirom, A. (1992). Burnout and risk factors for cardiovascular diseases. *Journal of Behavioral Medicine, 18*, 53–60.

Miklós, I. H., & Kovács, K. J. (2012). Reorganization of synaptic inputs to the hypothalamic paraventricular nucleus during chronic psychogenic stress in rats. *Biological psychiatry*, *71*(4), 301-308.

Miller, G.E., Chen, E., & Shou, E.S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans, *Psychological Bulletin, 133*(1), 25-45.

Park, C.L., Cohen, L.H., & Murch, R.L. (1996). Assessment and prediction of stress- related growth. *Journal of Personality, 64*, 71-105.

Petrie, K.J., Buick, D.L., Weinman, J., & Booth, R.J. (1999). Positive effects of illness Reported by myocardial infarction and breast cancer patients. *Journal of Psychometric Research, 47*, 537–543.

Pressman, S. D., & Cohen, S. (2005). Does positive affect influence health? *Psychological Bulletin, 131*(6), 925-971. doi:10.1037/0033-2909.131.6.925

Sapolsky, R. M. (2004). Glands, Gooseflesh, and Hormones. In *Why don't zebras get ulcers*, 3rd Edition, New York: Holt paperbacks.Schlotz, W., Yim, I.S., Zoccola, P.M., Jansen, L., & Schulz, P. (2011) The perceived stress reactivity scale: Measurement invariance, stability, and validity in three countries. *Psychological Assessment, 23*(1), 80-94.

Schulz,P., & Schlotz, W. (1999). The Trier Inventory for the Assessment of Chronic Stress (TICS): Scale construction, statistical testing, and validation of the scale work overload. *Diagnostica, 45*:8-19.

Segerstrom, S.C., & O'Connor, D.B. (2012). Stress, health and illness: four challenges for the future. *Psychology & Health, 27,*128-140.

Stults-Holehmanien, M. (2013). The interplay between stress and physical activity in the prevention and treatment of cardiovascular disease. *Frontiers in Physiology, 4*, 1-4. doi: 10.3389/fphys.2013.00346.

Tanielian, T., Jaycox, L. H., Adamson, D. M., & Metscher, K. N. (2008). Introduction. In T. Tanielian & L. H. Jaycox (Eds.), *Invisible wounds of war: psychological and cognitive injuries, consequences, and services to assist recovery* (pp. 3-17). Santa Monica, CA: RAND Corporation.

Taylor, S. E., Kemeny, M. E., Reed, G. M., Bower, J. E., & Gruenewald, T. L. (2000). Psychological resources, positive illusions, and health. *American psychologist*, *55*(1), 99.

Tedeschi, R.G. (2011). Posttraumatic growth in combat veterans. *Journal of Clinical Psychology in Medical Settings, 18*(2), 137-144.

Tedeschi, R.G., & Calhoun, L.G. (1996). The posttraumatic growth inventory: Measuring the positive legacy of trauma. *Journal of Traumatic Stress, 9,* 455-471.

Tedeschi, R.G., & Calhoun, L.G. (2004). Posttraumatic Growth: Conceptual foundation and empirical evidence. *Psychological Inquiry, 15,* 1 – 18.

Tedeschi, R.G., & McNally, R.J. (2011). Can we facilitate posttraumatic growth in combat veterans? *American Psychologist. 66*(1), 19-24.

Tennen, H., & Affleck, G. (2009). Assessing positive life change: In search of meticulous methods.

Thayer, J., Ahs, F., Fredrikson, M., Sollers III, J.J., & Wager, T.D. (2012). A meta-analysis of heart rate variability and neuroimaging studies: Implications for heart rate variability as a marker of stress and health. *Neuroscience and Biobehavioral Reviews, 36,* 747-756.

van Wingen, G. A., Geuze, E., Vermetten, E., & Fernández, G. (2011). Perceived threat predicts the neural sequelae of combat stress. *Molecular psychiatry*, *16*(6), 664-671.

Vogt, D.S., Proctor, S.P., King, D.W., King, L.A., Vasterling, J.J., 2008. Validation of scales from the Deployment Risk and Resilience Inventory in a sample of Operation Iraqi Freedom Veterans. *Assessment, 15,* 391–403.

Warden, D. (2006). Military TBI during the Iraq and Afghanistan wars. *Journal of Head Trauma Rehabilitation, (5)*21, 398-402

Weathers, F.W., Litz, B.T., Keane, T.M., Palmieri, P.A., Marx, B.P., & Schnurr, P.P. (2013).*The PTSD Checklist for DSM-5 (PCL-5).* Scale available from the National Center for PTSD.

Widome, R., Littman, A.J., Laska, M.N.,& Fu, S.S. (2012). Preventing chronic illness in young veterans by promoting healthful behaviors, *Preventing Chronic Disease, 9,* 1-4.

Yamamoto, K, Okazaki, A., & Ohmori, S. (2011). The relationship between psychosocial stress, age, BMI, CRP, lifestyle, and the metabolic syndrome in apparently healthy subjects. *Journal of Physiological Anthropology, 30,* 15-22.

APPENDIX A: TABLES

Table 1

Demographics and descriptive statistics

Variable	Overall Sample (N=33)	Trauma Group (n=17)	Non-Trauma Group (n=16)
Age			
Mean (*SD*)	30.22 (6.62)	30.94(6.98)	29.50(6.38)
Range	20 – 46	24-46	20-36
Race	n (%)	n (%)	n (%)
American Indian/Alaskan Native	1 (3)	0	1 (6)
Asian/Asian American	1 (3)	1 (6)	0
Black/African American	1 (3)	0	1 (6)
Non-European Hispanic/Latino	4 (12)	2 (12)	2 (13)
Caucasian	26 (79)	14 (82)	12 (75)
Education	n (%)	n (%)	n (%)
High School Diploma/Some College	28 (85)	13 (76)	15 (94)
Bachelor's Degree	2 (6)	2 (12)	0
Graduate or Professional Degree	3 (9)	2 (12)	1 (6)
Annual Household Income	n (%)	n (%)	n (%)
Less than $10,000	2 (6)	1 (6)	1 (6)
$10,000-$24,999	5 (15)	3 (18)	2 (13)
$25,000-$49,999	10 (30)	6 (35)	4 (25)
$50,000-$99,999	10 (30)	2 (12)	8 (50)
$100,000+	6 (18)	5 (29)	1 (6)
Military Branch	n (%)	n (%)	n (%)
Air Force	2 (6)	1 (6)	1 (6)
Army	13 (39.4)	8 (47)	5 (31)
Marine Corps	13 (39.4)	5 (29)	8 (50)
Navy	2 (6)	1 (6)	1 (6)

National Guard	3 (9.2)	2 (12)	1 (6)
Service Commitment (years)			
Mean (*SD*)	8.05 (5.50)	9.62 (6.77)	6.59 (3.66)
Range	1.5-23	3-23	1.5-15
Currently on Active Duty	n (%)	n (%)	n (%)
Yes	11 (33)	5(29)	6 (38)
No	22 (67)	12 (71)	10 (62%)
Deployments			
Mean (*SD*)	2.39 (1.65)	2.47 (1.50)	2.8 (2.48)
Range	1-9	1-6	1-9
Current Smoker	n (%)	n (%)	n (%)
Yes	2 (6)	1 (5.9)	1 (6.3)
No	31 (94)	16 (94.1)	15 (93.8)
BMI			
Mean (*SD*)	27.67 (4.51)	27.86 (5.38)	27.48 (3.51)
Range	18.50 – 39.20	18.50 – 39.20	23.40 – 37.50
Combat Experiences			
Mean (*SD*)	36.88 (13.83)	41.35 (12.63)	32.13 (13.82)
Range	15 – 74	24-74	15-59
PCL			
Mean (*SD*)	49.55 (19.81)	59.47 (15.89)	39.00 (18.36)
Range	23 – 90	24-90	23-75
PTGI			
Mean (*SD*)	3.50 (.98)	3.42 (.89)	3.56 (1.08)
Range	1-5	1-5	1-5
DASS			
Mean (*SD*)	40.64 (14.55)	48.11 (11.35)	32.69 (13.54)
Range	21 – 76	25-69	21-76
PSS			
Mean (*SD*)	41.03 (8.64)	44.88 (7.09)	36.94 (8.42)
Range	23 – 59	31-53	23-59
Day 1 Cortisol Slope			
Mean (*SD*)	-0.02 (.02)	-0.03 (.02)	-0.02 (.01)

Range	-0.06 – 0.00	-0.06 - -0.00	-0.04 - -0.00
Day 2 Cortisol Slope			
Mean (*SD*)	-0.02 (.02)	-0.02 (.03)	-0.02 (.02)
Range	-0.11 – 0.01	-0.11 – 0.01	-0.06 - 0.01
CRP			
Mean (*SD*)	6.21 (1.64)	6.30 (1.72)	6.12 (1.61)
Range	3.91 – 9.09	3.91 - 9.09	3.91 – 8.33

Note: *M*= mean. *SD*= standard deviation. PCL= posttraumatic stress disorder check list. PTGI= posttraumatic growth inventory. DASS= depression, anxiety, and stress scales. PSS= perceived stress scale. CRP= C-reactive protein. BMI= body mass index.

Table 2

Zero-order correlations for study variables

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Combat Experiences	--										
2. PCL	.18	--									
3. PTGI	.27	-.06	--								
4. DASS	.05	.83[c]	-.02	--							
5. PSS	.12	.76[c]	-.09	.66[c]	--						
6. Day 1 Cortisol Slope	.16	.03	-.01	.08	.08	--					
7. Day 2 Cortisol Slope	.17	-.16	.43[a]	-.06	-.24	.50[b]	--				
8. CRP	.24	.09	.17	-.17	.09	-.08	.08	--			
9. BMI	.33[a]	.12	.14	.06	.18	.22	.19	.11	--		
10. Age	-.15	.17	-.20	.01	.14	-.51[b]	-.29	.02	.09	--	
11. Smoke	.10	-.28	-.12	-.36[a]	-.13	.03	-.03	.05	.02	.25	--

Note: N=33. [a]p<.05. [b]p<.01. [c]p<.001. M= mean. SD= standard deviation. PCL= posttraumatic stress disorder check list. PTGI= posttraumatic growth inventory. DASS= depression, anxiety, and stress scales. PSS= perceived stress scale. CRP= C-reactive protein. BMI= body mass index.

Table 3.

One-way ANOVA results with trauma perception as the dependent variable

						95% Confidence Interval for Mean				
		N	*M*	*SD*	*SE*	Lower Bound	Upper Bound	*F*	*p*	η^2
Day 1 Cort	yes	17	-.03	.02	.00	-.03	-.02	1.18	.29	.00
Slope	no	16	-.02	.01	.00	-.03	-.01			
	Total	33	-.02	.01	.00	-.03	-.02			
Day 2 Cort	yes	17	-.02	.03	.01	-.04	-.01	.19	.67	.00
Slope	no	16	-.02	.02	.01	-.03	-.01			
	Total	33	-.02	.02	.00	-.03	-.01			
Day 1 Cort	yes	17	-.93	.62	.15	-1.25	-.62	.26	.62	.01
AM (nl)	no	16	-1.04	.64	.16	-1.38	-.71			
	Total	33	-.99	.62	.11	-1.21	-.77			
Day 1 Cort	yes	17	-3.51	1.36	.33	-4.21	-2.82	.02	.90	.01
PM (nl)	no	16	-3.45	1.66	.41	-4.33	-2.56			
	Total	33	-3.48	1.49	.26	-4.01	-2.95			
Day 2 Cort	yes	17	-1.17	.68	.17	-1.52	-.82	.29	.59	.01
AM (nl)	no	16	-1.32	.85	.21	-1.77	-.86			
	Total	33	-1.24	.76	.13	-1.51	-.97			
Day 2 Cort	yes	17	-3.65	1.47	.36	-4.41	-2.89	.05	.82	.04
PM (nl)	no	16	-3.77	1.48	.37	-4.56	-2.97			
	Total	33	-3.71	1.45	.25	-4.22	-3.19			
nlCRP	yes	17	6.30	1.72	.42	5.42	7.18	.10	.75	.01
	no	16	6.11	1.61	.40	5.25	6.97			
	Total	33	6.21	1.64	.29	5.62	6.79			
PSS	yes	17	44.88	7.09	1.72	41.24	48.53	8.64	.006	.22
	no	16	36.94	8.42	2.10	32.45	41.42			
	Total	33	41.03	8.64	1.50	37.97	44.09			
PTGITotal	yes	17	3.42	.89	.22	2.96	3.88	.20	.66	.01
	no	16	3.58	1.08	.27	3.00	4.15			

	Total	33	3.50	.98	.17	3.15	3.84			
CombatTotal	yes	17	41.35	12.63	3.06	34.86	47.85	4.02	.05	.15
	no	16	32.13	13.82	3.45	24.76	39.49			
	Total	33	36.88	13.83	2.41	31.98	41.78			
PCLTotal	yes	17	59.47	15.89	3.86	51.30	67.64	11.77	.002	.29
	no	16	39.00	18.36	4.59	29.22	48.78			
	Total	33	40.64	19.81	3.45	42.52	56.57			
DASS	yes	17	48.11	11.35	2.75	42.28	53.95	12.64	.001	.29
	no	16	32.69	13.54	3.39	25.47	39.90			
	Total	33	40.63	14.55	2.53	35.48	45.80			

Note: Yes= trauma group. No= non-trauma group. *M*= mean. *SD*= standard deviation. *SE*= standard error. Cort= cortisol. nl= natural log. PCL= posttraumatic stress disorder check list. PTGI= posttraumatic growth inventory. DASS= depression, anxiety, and stress scales. PSS= perceived stress scale. CRP= C-reactive protein.

Table 4

Summary of moderated regression analysis for variables predicting CRP

Variable	Step 1		Step 2		Step 3	
	β	*S.E.*	*β*	*S.E.*	*β*	*S.E.*
(Intercept)	6.05***	1.42	5.87***	1.47	5.70**	1.51
Age	.00	.05	.01	.05	.02	.05
PSS			.02	.04	.02	.04
PTGI			.32	.32	.39	.34
PSS*PTGI					.03	.04
R^2		-.03		-.06		-.09
ΔR^2				.04		.01

Note. $n = 32$. $^*p < .05$. $^{**}p < .01$. $^{***}p < .001$. *b* = unstandardized beta weight; *S.E.* = standard error. CRP= C-reactive Protein. PSS = Perceived Stress Scale. PTGI= Posttraumatic Growth Inventory. Dependent variable = CRP.

Table 5

Summary of moderated regression analysis for variables predicting Day 1 cortisol slope

Variable	Step 1		Step 2		Step 3	
	b	*S.E.*	*b*	*S.E.*	*b*	*S.E.*
(Intercept)	.01	.01	.01	.01	.01	.01
Age	-.00**	.00	-.00**	.00	-.00**	.00
PSS			.00	.00	.00	.00
PTGI			-.00	.00	.00	.00
PSS*PTGI					.00	.00
R^2	.26**		.30		.33	
ΔR^2			.03		.04	

Note. $n = 32$. $^*p < .05$. $^{**}p < .01$. *b* = unstandardized beta weight; *S.E.* = standard error. PSS = Perceived Stress Scale. PTGI= Posttraumatic Growth Inventory. Dependent variable = Day 1 Cortisol Slope.

Table 6

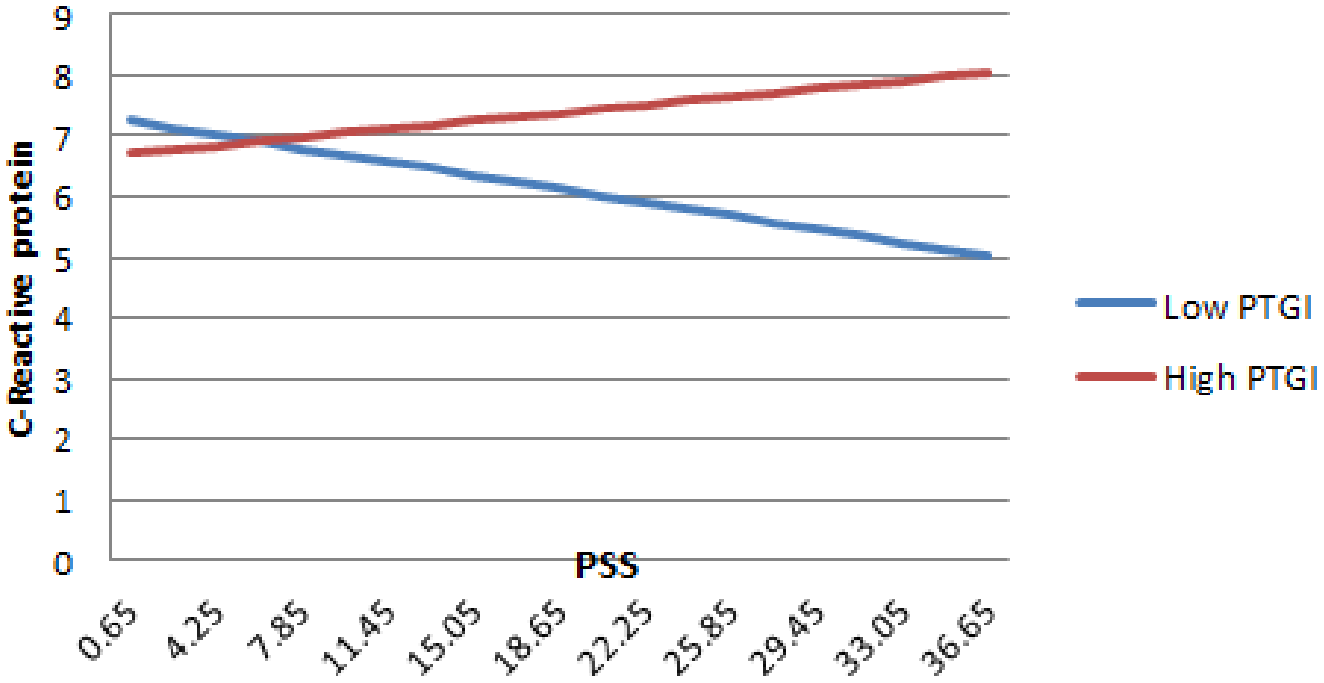
Summary of moderated regression analysis for variables predicting Day 2 cortisol slope

Variable	Step 1		Step 2		Step 3	
	b	*S.E.*	*b*	*S.E.*	*b*	*S.E.*
(Intercept)	.01	.02	.00	.02	-.01	.02
Age	-.00	.00	-.00	.00	.00	.00
PSS			.00	.00	.00	.00
PTGI			.01*	.01	.01**	.01
PTGI*PSS					.001*	.00
R^2	.09		.18*		.29*	
ΔR^2			.18		.12	

Note. $n = 32$. $^*p < .05$. $^{**}p < .01$. *b* = unstandardized beta weight; *S.E.* = standard error. PSS = Perceived Stress Scale. PTGI= Posttraumatic Growth Inventory. Dependent variable = Day 2 Cortisol Slope.

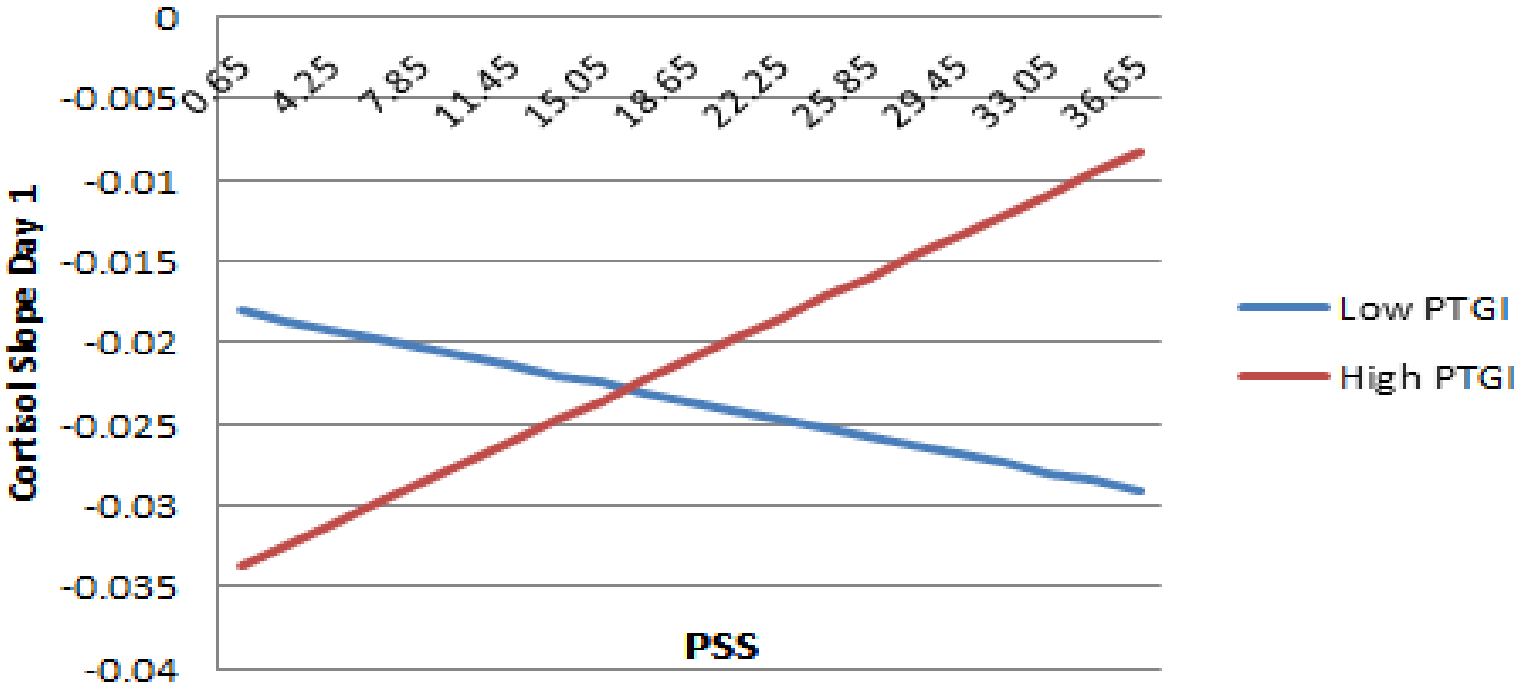
APPENDIX B: FIGURES

Figure 1.
The interactive effect of posttraumatic growth and perceived psychological stress on CRP



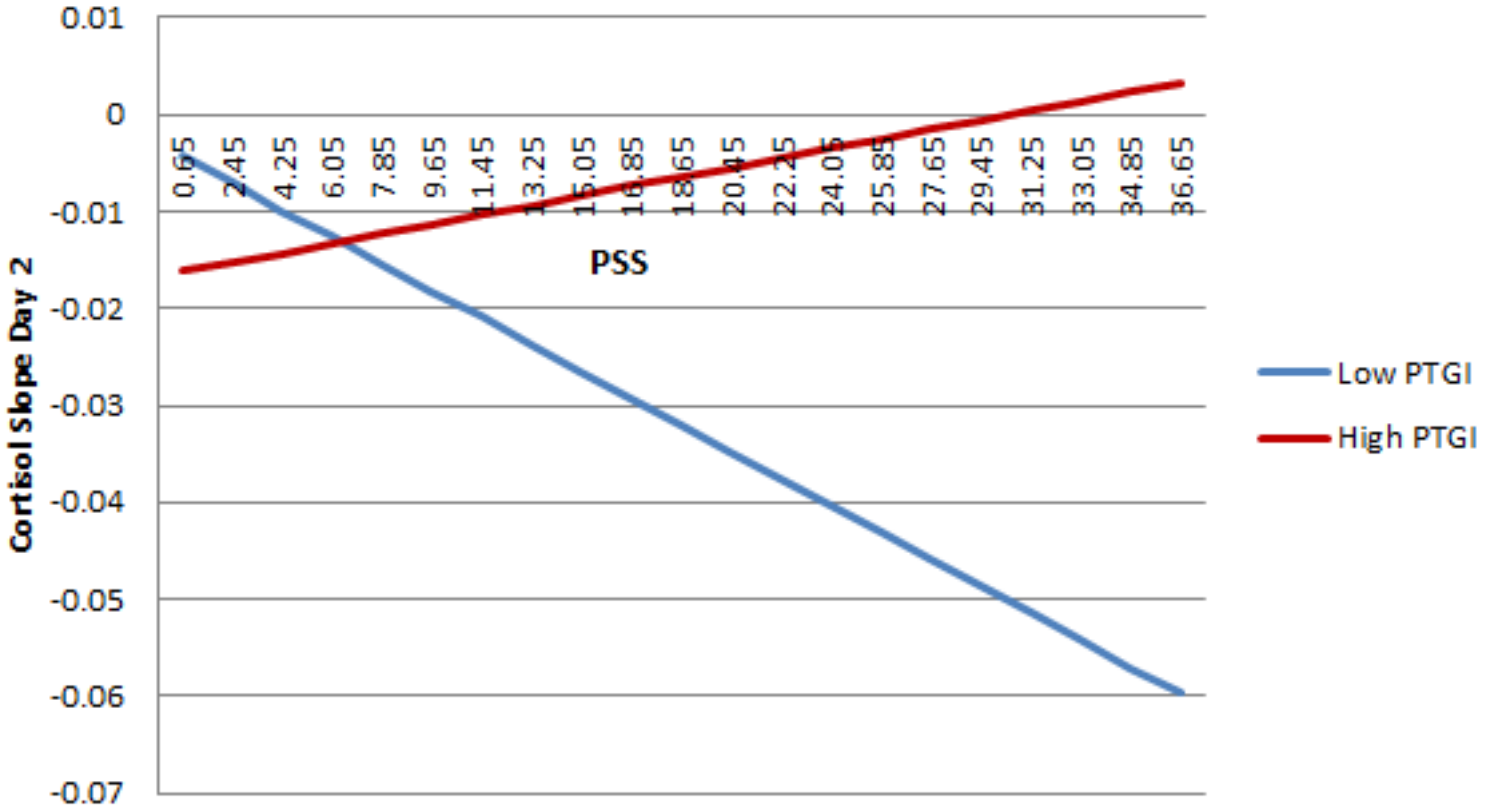
Note: *N*=33. PTGI= Posttraumatic growth inventory. The left axis are natural log-transformed and the right axis are raw values. Cortisol was assessed from saliva samples. Analysis controlled for age. The analyses used the full range of PPS and PTG levels as continuous variables. The interaction was not statistically significant.

Figure 2.
The interactive effect of posttraumatic growth and perceived psychological stress on Day 1 diurnal cortisol slope



Note: *N*=33. PTGI= Posttraumatic growth inventory. The left axis are natural log-transformed and the right axis are raw values. Cortisol was assessed from saliva samples. Analysis controlled for age. The analyses used the full range of PPS and PTG levels as continuous variables. The interaction was not statistically significant.

Figure 3.
The interactive effect of posttraumatic growth and perceived psychological stress on Day 2 diurnal cortisol slope



Note: *N*=33. PTGI= Posttraumatic growth inventory. The left axis are natural log-transformed and the right axis are raw values. Cortisol was assessed from saliva samples. Analysis controlled for age. The analyses used the full range of PPS and PTG levels as continuous variables. The line representing high PTG level does not significantly differ from zero.

APPENDIX C: MEASURES

Thank you for participating in the Veterans Study. This survey will take between 45 to 65 minutes to complete.

We ask that give these questions your full attention as you complete them. If a particular question does not make sense to you, just interpret it as best as you can. You may also skip any questions you do not feel comfortable answering.

Please remember that there is no right or wrong answer to any of these questions. We are sincerely interested in your personal feelings and experiences. Your name will not be directly linked to these data – we strongly encourage you to be as honest as possible.

Also, please do not over-think your responses. We want your initial reactions to each question.

THE FIRST SET OF QUESTIONS ASKS FOR GENERAL INFORMATION ABOUT YOU AND YOUR DEPLOYMENT.

1. What country were you born in?
   a. United States
   b. Other

2. Age:_____

3. What is your race/ethnicity? (check all that apply)
   a. American Indian or Alaskan Native
   b. Asian/Asian-American
   c. Black or African American
   d. Non-European Hispanic or Latino(a)

e. Native Hawaiian or Other Pacific Islander Caucasian/White
f. Other (please specify)
g. Prefer not to answer

4. What is your religious affiliation?
   a. Protestant
   b. Catholic
   c. Judaism
   d. Muslim
   e. Hindu
   f. Buddhist
   g. Mormon
   h. Agnostic
   i. Atheist
   j. None
   k. Other (please specify)

5. How frequently do you attend religious community meetings (e.g., services, prayer meetings)?
   a. One or more times per week
   b. One or more times per month
   c. One or more times per year
   d. Less than once per year
   e. N/A
6. Your Education Level? (Please mark highest level completed)
   a. Less than 9th grade
   b. 9th – 12th grade, No diploma
   c. High School Diploma or Equivalent Some College, No Degree Associate Degree
   d. Bachelor's Degree
   e. Graduate or Professional Degree

7. Annual Household Income?

    a. Less than $10,000
    b. $10,000 - $14,999
    c. $15,000 - $24,999
    d. $25,000 - $34,999
    e. $35,000 - $49,999
    f. $50,000 - $74,999
    g. $75,000 - $99,999
    h. $100,000 - $149,999
    i. $150,000 - $199,999
    j. $200,000 or more

8. Relationship status?
    a. Never Married
    b. Married – 1st marriage
    c. Married – 2nd marriage
    d. Married – 3rd + marriage
    e. Separated
    f. Divorced
    g. Widowed
    h. Cohabitating/Unmarried Partner

9. Employment Status
    a. Employed: Military Occupation- Full time/active duty
    b. Employed: military Occupation- Reserves
    c. Employed: non-military occupation (specify)
    d. Unemployed

10 Do you currently smoke?

e. Yes
f. No

11 If yes, how many cigarettes/day do you smoke?_____
If yes, how many cigarettes/week do you smoke?_____
If yes, how many cigarettes/month do you smoke?_____

12. How often do you exercise, on average?

a. 0 days/week
b. 1 day/week
c. 2-3 days/week
d. 4-5 days/week
e. 6-7 days/week

13. When you exercise, how long do you exercise for? (in minutes)_____

14. What form of exercise do you usually engage in?

15. Have you ever been diagnosed with a mental illness?

a. Yes
i. Specify _____
b. No

16. Are you taking any current medications (including vitamins)?

a. Yes
b. No

Military Service Information

17. Branch of Military Service
    a. Army
        i. Active Duty
        ii. Army Reserves
        iii. Army National Guard
        iv. Army veteran
    b. Air Force
        i. Active Duty
        ii. Air Force Reserves
        iii. Air Force National Guard
        iv. Air Force veteran
    c. Navy
        i. Active Duty
        ii. Naval Reserves
        iii. Naval veteran
    d. Marine Corps
        i. Active Duty
        ii. Marine Corps Reserves
        iii. Marine Corps veteran
    e. Coast Guard
        i. Active Duty
        ii. Coast Guard Reserves
        iii. Coast Guard veteran
    f. National Guard
        i. Active Duty
        ii. National Guard Reserves
        iii. National Guard veteran

18. Number of years military service: Years_____Months_____

19. Current Rank or Rank at Separation_____

20. Did you deploy?
    a. Yes
       Location_____
       Years (MM/YY to MM/YY)
       Length of time in each country_____

21. Do you have a history of combat experience?
    a. Yes
       i. Country_____
       ii. Length of time (Years)_____(Months)_____

Taken by research staff:

1. Height_____
2. Weight_____
3. Waist circumference_____
4. Blood pressure_____
5. Temperature_____
6. Heart Rate_____
7. Carbon Monoxide_____

B. Combat Experiences Scale

King, D. W., King, L. A., & Vogt. D. S (2003). *Manual for the Deployment Risk and Resilience Inventory (DDRI): A Collection of Measures for Studying Deployment-Related Experiences of Military Veterans*. Boston, MA: National Center for PTSD.

The statements below are about your combat experiences during deployment. Please indicate how often you experienced each of the following. **While deployed:**		**Never**				**Extremely Frequently**
1.	I went on combat patrols or missions.	1	2	3	4	5
2.	I or members of my unit encountered land or water mines and/or IEDs.	1	2	3	4	5
3.	I or members of my unit received hostile incoming fire from small arms, artillery, rockets, mortars, or bombs.	1	2	3	4	5
4.	I or members of my unit received “friendly” incoming fire from small arms, artillery, rockers, mortars, or bombs.	1	2	3	4	5
5.	I was in a vehicle (for example, a truck, tank, APC, helicopter, plane, or boat) that was under fire.	1	2	3	4	5
6.	I or members of my unit were attacked by terrorists or civilians.	1	2	3	4	5
7.	I was part of a land or naval artillery unit that fired on the enemy.	1	2	3	4	5
8.	I was part of a mortar team that fired on the enemy.	1	2	3	4	5
9.	I was part of an assault on entrenched or fortified positions.	1	2	3	4	5

10.	I took part in an invasion that involved naval and/or fortified positions.	1	2	3	4	5
11.	My unit engaged in battle in which it suffered casualties.	1	2	3	4	5
12.	I personally witnessed someone from my unit or an ally unit being seriously wounded or killed.	1	2	3	4	5
13.	I personally witnessed soldiers from enemy troops being seriously wounded or killed.	1	2	3	4	5
14.	I was wounded or injured in combat	1	2	3	4	5
15.	I fired my weapon at the enemy.	1	2	3	4	5
16.	I killed or think I killed someone in combat.	1	2	3	4	5

C. Depression, Anxiety, and Stress Scales

Lovibond, S.H. & Lovibond, P.f. (1995). *Manual for the Depression anxiety Stress Scales*. (2nd Ed) Sydney: Psychology Foundation

Please read each statement and circle a number 0, 1, 2 or 3 that indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

1	I found it hard to wind down	0	1	2	3
2	I was aware of dryness of my mouth	0	1	2	3
3	I couldn't seem to experience any positive feeling at all	0	1	2	3
4	I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion)	0	1	2	3
5	I found it difficult to work up the initiative to do things	0	1	2	3
6	I tended to over-react to situations	0	1	2	3
7	I experienced trembling (eg, in the hands)	0	1	2	3
8	I felt that I was using a lot of nervous energy	0	1	2	3

9	I was worried about situations in which I might panic and make a fool of myself	0	1	2	3
10	I felt that I had nothing to look forward to	0	1	2	3
11	I found myself getting agitated	0	1	2	3
12	I found it difficult to relax	0	1	2	3
13	I felt down-hearted and blue	0	1	2	3
14	I was intolerant of anything that kept me from getting on with what I was doing	0	1	2	3
15	I felt I was close to panic	0	1	2	3
16	I was unable to become enthusiastic about anything	0	1	2	3
17	I felt I wasn't worth much as a person	0	1	2	3
18	I felt that I was rather touchy	0	1	2	3
19	I was aware of the action of my heart in the absence of physical exertion (eg, sense of heart rate increase, heart missing a beat)	0	1	2	3
20	I felt scared without any good reason	0	1	2	3
21	I felt that life was meaningless	0	1	2	3

D. The PTSD Checklist for DSM-5

Weathers, F.W., Litz, B.T., Keane, T.M., Palmieri, P.A., Marx, B.P., & Schnurr, P.P. (2013).*The PTSD Checklist for DSM-5 (PCL-5).* Scale available from the National Center for PTSD at www.ptsd.va.gov.

Instructions: Below is a list of problems that people sometimes have in response to a very stressful experience. Please read each problem carefully and then circle one of the numbers to the right to indicate how much you have been bothered by that problem in the past month.

In the past month, how much were you bothered by:	*Not at all*	*A little bit*	*Moderately*	*Quite a bit*	*Extremely*
1. Repeated, disturbing, and unwanted memories of the stressful experience?	0	1	2	3	4
2. Repeated, disturbing dreams of the stressful experience?	0	1	2	3	4
3. Suddenly feeling or acting as if the stressful experience were actually happening again *(as if you were actually back there reliving it)*?	0	1	2	3	4
4. Feeling very upset when something reminded you of the stressful experience?	0	1	2	3	4
5. Having strong physical reactions when something reminded you of the stressful experience *(for example, heart pounding, trouble breathing, sweating)*?	0	1	2	3	4
6. Avoiding memories, thoughts, or feelings related to the stressful experience?	0	1	2	3	4
7. Avoiding external reminders of the stressful experience *(for example, people, places, conversations, activities, objects, or situations)*?	0	1	2	3	4
8. Trouble remembering important parts of the stressful experience?	0	1	2	3	4

9. Having strong negative beliefs about yourself, other people, or the world *(for example, having thoughts such as: I am bad, there is something seriously wrong with me, no one can be trusted, the world is completely dangerous)*?	**0**	**1**	**2**	**3**	**4**
10. Blaming yourself or someone else for the stressful experience or what happened after it?	**0**	**1**	**2**	**3**	**4**
11. Having strong negative feelings such as fear, horror, anger, guilt, or shame?	**0**	**1**	**2**	**3**	**4**
12. Loss of interest in activities that you used to enjoy?	**0**	**1**	**2**	**3**	**4**
13. Feeling distant or cut off from other people?	**0**	**1**	**2**	**3**	**4**
14. Trouble experiencing positive feelings *(for example, being unable to feel happiness or have loving feelings for people close to you)*?	**0**	**1**	**2**	**3**	**4**
15. Irritable behavior, angry outbursts, or acting aggressively?	**0**	**1**	**2**	**3**	**4**
16. Taking too many risks or doing things that could cause you harm?	**0**	**1**	**2**	**3**	**4**
17. Being "superalert" or watchful or on guard?	**0**	**1**	**2**	**3**	**4**
18. Feeling jumpy or easily startled?	**0**	**1**	**2**	**3**	**4**
19. Having difficulty concentrating?	**0**	**1**	**2**	**3**	**4**
20. Trouble falling or staying asleep?	**0**	**1**	**2**	**3**	**4**

E. Posttraumatic Growth

Tedeschi, R.G., & Calhoun, L.G. (1996). The posttraumatic growth inventory: Measuring the positive legacy of trauma. *Journal of Traumatic Stress, 9*, 455-471.

Indicate for each of the statements below the degree to which this change occurred in your life as a result of your experience, using the following scale.		**I did not experience this change**	**I experienced this change to a ___ degree**				
			Very small	**Small**	**Moderate**	**Great**	**Very great**
1.	I changed my priorities about what is important in life.	0	1	2	3	4	5
2.	I have a greater appreciation for the value of my own life.	0	1	2	3	4	5
3.	I developed new interests.	0	1	2	3	4	5
4.	I have a greater feeling of self-reliance.	0	1	2	3	4	5
5.	I have a better understanding of spiritual matters.	0	1	2	3	4	5
6.	I more clearly see that I can count on people in times of trouble	0	1	2	3	4	5
7.	I established a new path for my life.	0	1	2	3	4	5
8.	I have a greater sense of closeness with others.	0	1	2	3	4	5
9.	I am more willing to express my emotions.	0	1	2	3	4	5
10.	I know better that I can handle difficulties.	0	1	2	3	4	5
11.	I am able to do better things with my life.	0	1	2	3	4	5
12.	I am better able to accept the way things work out.	0	1	2	3	4	5

13.	I can better appreciate each day.	0	1	2	3	4	5
14.	New opportunities are available which wouldn't have been otherwise.	0	1	2	3	4	5
15.	I have more compassion for others.	0	1	2	3	4	5
16.	I put more effort into my relationships.	0	1	2	3	4	5
17.	I am more likely to try to change things which need changing.	0	1	2	3	4	5
18.	I have a stronger religious faith.	0	1	2	3	4	5
19.	I discovered that I'm stronger than I thought I was.	0	1	2	3	4	5
20.	I learned a great deal about how wonderful people are.	0	1	2	3	4	5
21.	I better accept needing others.	0	1	2	3	4	5

F. Perceived Stress Scale

Cohen, S., Kamarck, T., Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior*, *24*, 385-396

The questions in the scale ask you about your feelings and thoughts over the past month. In each case, you will be asked to indicate how often you felt or thought a certain way. Although some of the questions are similar, there are differences between them and you should treat each one as a separate question. The best approach is to answer each question fairly quickly. That is, don't try to count up the number of times you felt a particular way, but rather indicate the alternative that seems like a reasonable estimate.

For each question, choose from the following alternatives:
0 = never
1= almost never
2= sometimes
3= fairly often
4= often

1) In the last month, how often have you been upset because of something that happened unexpectedly?
2) In the last month, how often have you felt that you were unable to control important things in your life?
3) In the last month, how often have you felt nervous and "stressed"?
4) *In the last month, how often have you dealt successfully with irritating life hassles?
5) *In the last month, how often have you felt that you were effectively coping with important changes that were occurring in your life?
6) *In the last month, how often have you felt confident about your ability to handle your personal problems?
7) *In the last month, how often have you felt that things were going your way?
8) In the last month, how often have you found that you could not cope with all the things you had to do?
9) *In the last month, how often have you been unable to control irritations in your life?
10) *In the last month, how often have you felt that you were on top of things?
11) In the last month, how often have you been angered because of things that happened that were outside of your control?
12) In the last month, how often have you found yourself thinking about things you have to accomplish?
13) *In the last month, how often have you been able to control the way you spend your time?
14) In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?

*indicates reverse score