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The Effect of Pre-Deployment Physiology as a Predictor of Post-Traumatic Stress Disorder Among a Sample of United States Army National Guard and Reserve Soldiers

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THE EFFECT OF PRE-DEPLOYMENT PHYSIOLOGY AS A PREDICTOR OF POST-TRAUMATIC STRESS DISORDER AMONG A SAMPLE OF UNITED STATES ARMY NATIONAL GUARD AND RESERVE SOLDIERS

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University.

by

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Abstract

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Although a great deal of information is known about Post Traumatic Stress Disorder (PTSD), potential physiological risk factors for PTSD development are still unclear. Further, there are few prospective studies conducted with PTSD. One potential risk factor for the development of PTSD is an individual's cardiovascular reactivity and recovery in response to stressor tasks. The current study was conducted with 763 Army National Guard and Army Reserve soldiers. Participants completed a stressful induction along with self-report measures prior to deployment. Post-deployment, self-report measures were completed to assess PTSD symptomatology and experiences related to deployment and combat. Multiple regression was used to determine the ability of blood pressure response to stress induction to predict PTSD symptoms immediately and one-year post-deployment. Results indicated that soldiers who had a less reactive systolic blood pressure response to stressor tasks reported more PTSD symptomatology immediately after and one year after deployment. Furthermore, slower blood pressure recovery immediately after the stressor was also related to PTSD symptoms both

immediately and one year post-deployment. These results suggest the possibility that soldiers who develop PTSD after deployment have less pre- deployment emotion regulation ability.

The Effect of Pre-Deployment Physiology as a Predictor of Post-Traumatic Stress Disorder Among a Sample of United States Army National Guard and Reserve Soldiers

Within the United States, Post-Traumatic Stress Disorder (PTSD) continues to be a prominent public health issue**.** According to Kessler et al. (2005) the lifetime prevalence rate for PTSD is 6.8% within a community population. Further, 3.5% of individuals report PTSD in the past year (Kessler et al., 2005). Among soldiers, recent estimates indicate a 13.8% prevalence rate of PTSD among OIF/OEF/OND veterans (Tanielian & Jaycox, 2008). More specifically, twelve months after deployment, 16.6% to 30.5% met symptom level criteria for PTSD (Thomas et al., 2010). Thus, PTSD is an especially important public health issue among Veterans.

Because PTSD is associated with a distinct, potentially traumatic event, a unique opportunity exists for prevention. Unlike many other disorders in the DSM, a diagnosis of PTSD requires a significant and life threatening event. This creates a set of discrete periods where prevention can occur, both prior to, and after the occurrence of, a potentially traumatic event. With this in mind, prevention can be separated into three categories as proposed by Caplan & Grunebaum (1967): primary prevention (before the development of a disorder), secondary prevention (treatment once a disorder exists), and tertiary prevention (increasing functioning once a disorder has remitted). Within PTSD, the primary focus has centered on secondary and tertiary prevention efforts. Secondary prevention efforts have focused on treatments that promote symptom remission in PTSD (Foa, Rothbaum, Riggs, Murdock, 1991; Resick & Schnicke, 1992; Monson, Schnurr, Resick, Friedman, Young-Xu, Stevens, 2006; Tuerk et al., 2011). Recent literature has found that treatment of symptoms is only effective for a proportion of the population (Bradley, Greene, Russ, Dutra, & Westen, 2005; Imel, Laska, Jakupcak, & Simpson 2013). Despite a reduction of symptoms, increased vocational productivity, maintenance of employment, and increased quality of life may not co-occur (Schnurr et al. 2006; Steenkamp &

Litz, 2013; Adler et al. 2015). Although widespread secondary and tertiary prevention efforts have occurred (Foa, Rothbaum, Riggs, Murdock, 1991; Resick & Schnicke, 1992; Goldberg & Resnick, 2010; Karlin & Cross, 2013), many individuals still suffer from symptoms of PTSD and reduced quality of life (Steenkamp & Litz. 2013). These treatment efforts and continued tertiary consequences have a substantial impact on society. According to Tanelian & Jaycox (2008) PTSD among soldiers has an estimated societal cost (e.g., lost production, missed work days) of 1.2 billion dollars each year. Further, when compared to other anxiety disorders in the general population, PTSD is associated with the greatest number of lost work days (Kessler & Greenberg, 2002). Based on lack of efficacy associated with treatment, number of lost work days, and continued suffering of individuals with PTSD, there is a need for more primary prevention efforts.

In this paper, I will first review the relatively limited existing literature on primary prevention in PTSD. Due to the limits of the secondary data analysis that will be completed here, I will focus the literature review specifically on potential personality and psychophysiological pre-trauma risk factors for PTSD. These were selected as they were a focus of the larger study from which this study was derived. Based on the literature on potential personality and psychophysiological risk factors for PTSD, I will propose several hypotheses for the current study. Next, I provide an overview of a large prospective cohort study designed to assess potential pre-deployment factors that were hypothesized to be associated with negative post-deployment mental and physical health outcomes. This study focused on a group of high-risk individuals, Army National Guard and Reserve soldiers, whom previous research has found to be at an increased risk of developing PTSD when compared to their active duty counterparts (Baker et al. 2009; Smith et al. 2009; Thomas, Wilk, Riviere, McGurk, Castro, & Hoge, 2010;

Vasterling et al. 2010). We will then describe the methods of the current secondary data analysis, focusing on blood pressure reactivity and recovery as predictors of PTSD symptoms and I will discuss results based on these secondary analyses. Finally I will discuss the theoretical and clinical implications of these results, and frame the current findings within the larger literature on PTSD prevention.

Literature Review

If we hope to decrease the prevalence of PTSD, steps must be taken to prevent the disorder before the occurrence of a potentially traumatic event via primary prevention. Few studies have employed a prospective design to examine factors associated with the development of PTSD diagnosis and symptoms prior to a traumatic stressor. In a recent review, DiGangi, Gomez, Mendoza, Jason, Keys, & Koenen (2013) determined that specific pre-trauma risk factors, including cognitive abilities, poor coping, negative personality types, previous psychopathology, physiological arousal, and social and ecological factors are associated with the diagnosis and development of PTSD symptoms (DiGangi et al., 2013). This review highlights that specific personality and biological factors are potential targets for primary prevention work. Of the factors reviewed in DiGangi et al. (2013) these factors were selected for use in this work because they were assessed as part of the larger parent study.

Several pre-trauma personality factors have been suggested as important in the prediction of PTSD symptoms. Studies measuring personality factors prior to trauma have focused on negative affect (Bramsen, Dirkzwager, & van der Ploeg, 2000), neuroticism (van den Hout & Englehard, 2004; Knezevic, Opacic, Savic, & Priebe, 2005; Parslow, Jorm, & Christensen, 2006; Breslau & Schultz, 2013; Nielsen, Andersen, & Hogh, 2015), and trait anxiety/coping (McNally et al. 2011). Other studies have shown a relationship between pre-trauma personality characteristics including self-efficacy (Heinrichs et al. 2005), hostility (Ogle, Rubin, & Siegler) and trait dissociation (Hodgins, Creamer, & Bell, 2001). Of these factors, neuroticism has been especially important in predicting the development of PTSD symptoms and diagnosis across multiple samples including soldiers, the elderly, pregnant women, and the general population. Although certain personality factors predict an increased risk for PTSD symptoms and diagnosis,

personality is relatively stable (Costa & McCrae, 1986) and therefore, difficult to modify. Therefore, examination of biological factors, which are considered more modifiable (DiGangi et al. 2013) may be more useful in primary prevention efforts.

Biological factors provide a unique opportunity for primary prevention as they have shown an ability to predict the development of PTSD**.** Among the important biological factors studied thus far are alterations in activity of the autonomic, endocrine, and immune systems. The autonomic nervous system is a major division of the peripheral nervous system that mediates the "fight or flight" response via the sympathetic nervous system and supports resting or basal functions via the parasympathetic nervous system (Robertson, Biaggioni, Burnstock, Low, & Paton, 2012). The endocrine system produces hormones responsible for a range of functions including arousal, sexual behavior, growth, and stress (Neal, 2016). The immune system is comprised of mechanisms that protect the body from external and internal threats (Parham, 2015). Interventions that alter autonomic functioning have shown greater promise than those impacting immune and endocrine functioning. There is a growing literature highlighting the ability to alter autonomic functioning through treatment efforts, including biofeedback (Del Pozo, Gevirtz, Scher, & Guarneri, 2004; Nolan et al. 2005; Ginsberg & Fogo, 2014) and pharmacologic agents (Kotler, Matar, & Kaplan, 2000; Vaiva et al. 2003). Further, autonomic changes soon after a potentially traumatic event have been shown to predict the later occurrence of a PTSD diagnosis. For example, research has found that increased autonomic arousal at rest, in reaction to trauma-oriented cues, and slower recovery from trauma cues occur in those with a diagnosis of PTSD (Blanchard, 1990; Buckley & Kaloupek, 2001; Pole, 2007). Studies focusing on the acute stress period have found that alterations in heart rate (HR) and blood pressure (BP), when measured at the scene of accident, hospital admission, and during hospital, are predictive

of PTSD at follow up (Shalev et al. 1998; Bryant, Harvey, Guthrie, and Moulds 2000; Bryant, et al., 2003; Bryant et al., 2008; Coronas, Gallardo, Moreno, Suarez, Garcia-Pares, and Menchon 2011). These studies demonstrate that HR and BP reactivity soon after a potentially traumatic event confer an increased risk of developing PTSD and can be targets of intervention, and discriminative factors in diagnosis. Therefore, further exploration of HR and BP in the pre-trauma phase can provide potential targets for primary prevention.

Other studies have examined immune and endocrine functioning as other potentially important pre-trauma biological factors. In a series of studies measuring endocrine functioning, glucocorticoids have shown promise as a pre-trauma risk factor indicating who is more likely to develop PTSD among Dutch soldiers both immediately after and six months after deployment (van Zuiden, et al., 2009; van Zuiden, et al., 2011; van Zuiden et al., 2012a; van Zuiden et al., 2012b). When measured prior to deployment among a group of U.S. military personnel, markers of immune functioning were predictive of an increased risk for PTSD diagnosis after deployment (Glatt et al., 2013; Eraly et al., 2014). Thus, studies of immune and endocrine functioning provide additional evidence of biological factors that may serve as biological precursors to PTSD.

Measures of change in the autonomic nervous system and facial muscles have provided some of the most extensive evidence for potential prospective psychophysiological risk factors associated with the development of PTSD. In the first prospective study of its kind, Guthrie and Bryant (2006) examined firefighters during cadet training. Their primary autonomic measure was skin conductance (SC), which reflects localized sympathetic activity to the sweat glands in the skin (Dawson, Schell, & Filion, 2007). In a conditioning experiment Guthrie & Bryant (2006) found that slowed extinction of corrugator EMG response (a measure of corrugator

muscle region activity, which is involved in furrowing of the brow, see Larsen, Norris, & Cacioppo, 2003) was predictive of PTSD symptoms twenty-four months after beginning firefighting duties. They did not find that SC during a conditioning procedure prior to a potentially traumatic event was predictive of later PTSD symptomatology. These findings indicate that PTSD symptoms after exposure to a potentially traumatic event were not predicted by autonomic reactions to conditioning. In another study of pre-trauma autonomic functioning, Pole, Neylan, Otte, Henn-Hasse, Metzler, and Marmar (2009) examined the role of multiple autonomic variables as potential predictors of PTSD symptoms. The overall best baseline (pre vocational training) predictors of increased PTSD symptoms after serving as a police officer for twelve months were increased SC responses to loud tones in a high threat of shock condition (where shock was administered) and a return to baseline of SC after loud tones. Contrary to Guthrie & Bryant et al., (2006) Pole et al. (2009) demonstrated that increased SC reactivity and slowed recovery from conditioning prior to a trauma can be predictive of PTSD symptoms after experiencing a potentially traumatic event. In another prospective study, Orr, Lasko, Macklin, Pineles, Chang, and Pitman (2012) measured police and firefighter trainees both before a traumatic event and again after exposure to a potentially traumatic event. Orr et al. (2012) found that increased SC responses to loud tones and greater HR reactivity during pre-trauma conditioning predicted greater post-trauma reactivity on the same measures when reading a script describing the potentially traumatic event. In addition, Orr et al. (2012) found that the only pre-trauma biological factor associated with having more self-reported PTSD symptoms after exposure to a traumatic event was Corrugator EMG during a conditioning procedure prior to beginning vocational training. The findings of Orr et al. (2012) show that increased autonomic functioning in the pre-trauma period is predictive of a significant increase in HR, SC, and EMG

to a traumatic script after a potentially traumatic event but not self-reported PTSD symptoms. Finally, in a prospective study, Minassian et al. (2015) examined autonomic functioning within a sample of United States Marines. Minassian et al. (2015) found that lower resting heart rate variability (HRV) was associated with PTSD diagnosis 6-months after return from deployment. Although prior studies found autonomic predictors related to PTSD symptoms, Minassian et al. (2015) is the first to find an autonomic measure as predictive of a diagnosis of PTSD. Moreover, their finding was within a group of active duty marines, providing the first prospective study of active duty personnel and autonomic functioning.

When considered together, these studies offer a murky picture of pre-trauma autonomic physiology as a predictor of PTSD symptoms and diagnosis. The most frequently studied measure, SC, reveals a mixed picture as only Pole et al. (2009) found that SC reactivity and slowed recovery was associated with PTSD symptoms, while Guthrie & Bryant (2006) and Orr et al. (2012) reported non-significant results related to PTSD symptoms. In addition, Orr et al. (2012) found that pre-trauma SC was predictive of acute post-trauma HR, SC, and EMG but not self-reported PTSD symptoms. These findings though are difficult to interpret, as they only show a change in autonomic and skeletal muscle reactivity but are not associated with increased PTSD symptoms. Furthermore, the prospective studies discussed here suggest that HR was not associated with self-reported PTSD symptoms (Pole et al. 2009) but was predictive of acute post-trauma HR, SC, and EMG when reading a traumatic script (Orr et al. 2012). In contrast to other autonomic measures, HRV was associated with later PTSD diagnosis when measured at rest (Minassian et al., 2015). This finding, unlike those with SC and HR is the first to demonstrate that a cardiovascular variable can prospectively predict a diagnosis of PTSD. Importantly, most of these studies (Guthrie & Bryant, 2006; Pole et al. 2009; Orr et al. 2012) reported relatively

low levels of PTSD, with only one individual meeting criteria for possible PTSD. Therefore, these findings may not provide strong evidence of which psychophysiological factors are most likely to predict later occurrence of PTSD or distressing PTSD symptoms. Therefore, continued research is needed to help further delineate the potential utility of autonomic reactivity as a potential predictive factor for later PTSD symptoms or diagnosis.

The current study will examine the link between BP reactivity and recovery to stressors during the pre-trauma period as a potential susceptibility factor for developing PTSD symptoms after a traumatic experience. I had intended to include personality variables in the current model as well but was unable to because of limitations of data access during the writing of this thesis. The current study will examine a group of Army National Guard and Reserve soldiers (NGR), who are an 'at risk' population for developing PTSD (Vasterling et al. 2010). During the pre-trauma period, NGR soldiers participated in a series of heterogeneous stress tasks that were designed to create changes in autonomic functioning. The stressful tasks included a confrontation speech task, a planning task for the confrontation task, and a subtraction task during which cardiovascular functioning was measured. Participants' cardiovascular functioning was also assessed while completing questionnaires and during a resting baseline. The design of the overall study uniquely positions the current secondary data analysis to assess the ability of blood pressure reactivity and recovery to predict later PTSD symptoms. Therefore, based on the related literature highlighted above, we hypothesize the following results in the current study.

Hypotheses

Hypothesis 1: Those National Guard and Reserve Soldiers who react to stressful tasks with a larger increase in blood pressure (BP), will be more likely to develop PTSD symptoms both immediately after deployment and one-year after return from deployment.

Hypothesis 2: Those National Guard and Reserve Soldiers who have a smaller decrease in BP following stressful tasks will be more likely to develop PTSD symptoms immediately post deployment and one-year post deployment

Hypothesis 3: The effects of BP reactivity and recovery as predictors of PTSD symptoms will hold true over and above the effects of pre-deployment life events, combat exposure, and deployment exposure.

Methods

Participants

The data used in the current study are from a larger study designed to assess soldiers' pre-deployment psychosocial and physiological predictors of physical symptoms, self-reported physical and mental health function, and health care utilization rates (McAndrew, D'Andrea, Lu, Abbi, Yan, Engel, & Quigley, 2013; McAndrew, Helmer, Phillips, Chandler, Ray, & Quigley, 2016; Quigley et al., 2012; Yan, et al. 2012). Collection of data occurred between November 2005 and January 2011. Data were collected at four phases: pre-deployment, immediately post deployment, three months after return from deployment, and one-year post deployment.

The current study used a prospective longitudinal cohort design. Inclusion criteria for the study were a pre-deployment age of 18 to 60 years, a resting BP below 140/90 at pre-deployment, and being within the final states of preparation before deployment. Exclusion criteria were assessed before deployment and included: current officer status; self-reported depression, schizophrenia, or bipolar disorder, and current pregnancy. Additionally, individuals were excluded from the study if they reported taking medications for heart or respiratory conditions, benzodiazepines, anti-depressants (at higher doses than used for depression), stimulants, anticonvulsants, and narcotics.

A total of 805 Army National Guard and Reserve soldiers were recruited prior to deployment from two bases, Fort Dix, New Jersey or Camp Shelby, Mississippi. Of those who were recruited, 795 initially consented to participation. From these 795 soldiers, 32 were officers, killed in action, or did not mobilize to a combat zone and therefore were excluded from analyses. In addition, four were excluded from analyses as they were hypertensive pre-deployment. Thus, 763 soldiers were included in the final pre-deployment analyses. To assess for differences between those who volunteered for the study and those who did not, individuals who declined to participate (n = 410) anonymously reported their health status. A significant difference was found between the two groups on health status (72.1% of the participant sample vs. 78.8% of the non-respondent sample reported excellent/very good health; $X^2 = 8.25$, $p < 0.01$; McAndrew et al. 2016). The number of participants at each phase included: pre-deployment (Phase 1; N=763), immediately post deployment (Phase 2; N=422), three months post-deployment (Phase 3; N=286), and one-year post deployment (Phase 4; N=336). Deployment to warzones typically lasted 12-13 months for soldiers included in the study. Immediate post-deployment data were, in most cases, collected when participants returned to their bases. However, some soldiers returned to bases different than their original deployment base (Fort Dix or Camp Shelby). For soldiers who returned to different bases, questionnaires were mailed to their home address, however, we had no way to verify receipt of the questionnaires. As a result, we 303 soldiers to follow-up at Phase 2, while another 23 soldiers explicitly declined to participate at this phase. Three-month and one-year post deployment data were collected through mailed questionnaire packets. At three months after return from deployment, an additional 45 participants declined to participate, and at one-year after return, another 50 participants declined to do so. The remainder of those who did not complete the questionnaires were lost to follow-up.

Prior to deployment and immediately after deployment participants were not permitted by Department of Defense policy to receive compensation because of their active duty status. Once no longer on active duty, soldiers who participated at three months and one-year after return from post deployment were compensated $30 and $45, respectively.

Immediately post deployment, participants who returned to Fort Dix or Camp Shelby completed questionnaires while on site. As indicated above, since many individuals returned to different bases, they were mailed questionnaires. Three-months and one-year post deployment, all soldiers who had not officially withdrawn consent to participate were mailed questionnaires. No physiological measurement was conducted at any time point after deployment.

Procedures

Soldiers were approached by study staff and asked to volunteer while waiting for, or after completion of, their pre-deployment medical processing. Groups of soldiers were given a verbal briefing about the study. Among those interested, a second in-person verbal briefing occurred in the testing space, at which time those interested signed an informed consent document from the Department of Defense (approved by the Walter Reed Department of Clinical Investigation) and for the Department of Veterans Affairs (approved by the VA New Jersey Healthcare System and the G.V Montgomery VA Medical Center). Soldiers were provided with the appropriate referral services if they endorsed questions that indicated severe anxiety or depression.

See Figure 1 for a detailed outline of timing for each task and flow of tasks for the pre-deployment phase. During the pre-deployment assessment, participants first completed a set of questionnaires on a computer for 20-30 minutes. Next, participants were asked to complete the stressor tasks while psychophysiological measures were recorded. This assessment included a pre-task resting baseline (five minutes), stressor tasks (14 minutes), an initial post-task baseline recorded while the participant completed questionnaires (5 minutes), and then a final baseline

after questionnaires were completed (5 minutes). All baselines were completed while the participant sat still in a quiet room. After the pre-task baseline, task instructions began. The first task induction included four minutes of planning out what they would say to another soldier in their unit whom they were to pretend someone had stolen $500 from them, and that they had to confront the person about the theft. Participants were then asked to deliver their confrontation by speaking into a microphone in front of a computer monitor as if speaking to the guilty person for four minutes. They were also given several points to cover as part of what they said. After completion of the confrontation task, the soldier was asked to count backwards from a random four-digit number by sevens for four minutes. During this task, they were informed when they were incorrect, and if so, were asked to begin again with the last correct answer given. Finally, for up to two minutes, participants were asked to complete a hand cold pressor task in which they placed their hand in icy cold water. Physiological measurements were taken throughout the protocol with no pauses or breaks between tasks. Blood pressure and heart rate was measured using an automated monitor (GE DASH 2000) electronic arterial blood pressure cuff which compressed each minute.

After completion of these tasks the participant started a recovery period in which s/he completed questionnaires. At the completion of this period, participants began the post-task resting baseline period. These two periods comprise the physiological recovery portion of measurement in the current study.

Measures

An appendix contains all items for all self-report measures used for the study.

Blood Pressure and Heart Rate: Both BP and HR were collected using the DASH 2000 meter by General Electric (Jupiter, FL). This is an automated oscillometric device, which was set to obtain readings at one-minute intervals. These readings were written down by a research

assistant from the monitor display. Additionally, the hand written readings were double-checked against the memory of the device and after the check, results were cleared for each participant. Both systolic and diastolic BP reactivity and recovery measures were obtained as follows. Reactivity variables were computed by subtracting the mean of the baseline BP from the mean of the speech planning, speech delivery, and subtraction tasks. To derive the recovery with questionnaires variable we subtracted the mean BP of the post-task resting questionnaire period's BP from the speech planning, speech delivery, and subtraction tasks. Finally, recovery without questionnaires was computed by subtracting the mean BP from the post task resting baseline from the mean BP of speech planning, speech delivery, and subtraction tasks.

PTSD Symptoms. The 17-item PTSD Checklist (PCL-M; Weathers, Litz, Herman, Huska & Keane, 1993) assesses the presence and frequency of PTSD symptoms over the past month related to the individual's military experience. Participants respond on a 5-point scale from *not at all bothered* to *extremely bothered* (Weathers, et al., 1993). The questions on the PCL-M directly assess the seventeen symptoms of PTSD outlined in the Diagnostic and Statistical Manual – Fourth Edition (DSM-IV). Scores on the PCL-M range from 17-85. Higher summed scores indicate higher levels of PTSD symptomatology. Moreover, among military personnel, a cut off score of 50 has been established as a reasonable score for discriminating those with and without likely PTSD (McDonald & Calhoun, 2010). The PCL-M has shown excellent internal consistency and test-retest reliability (Blanchard, Jones-Alexander, Buckley, & Forneris, 1996). Additionally, scores on this measure correlate with the Clinician Administered PTSD scale (Blanchard et al., 1996), the gold standard of PTSD diagnostic measures. In the current study, the PCL-M was administered at all three post-deployment phases.

Deployment Risk and Resilience Inventory: The Deployment Risk and Resilience Inventory (DRRI: King, King, Vogt, Knight, & Samper, 2006) is a collection of measures designed to assess psychosocial risk and resilience factors associated with military personnel and deployment to war zones or other hazardous environments. The DRRI is composed of into fourteen constructs. Of these, the following were included in the study: pre-deployment life events (prior stressors and childhood family environment) combat experiences, and deployment related factors. On the pre-deployment life events and deployment related experiences questionnaire participants respond to questions with "Yes" or "No" responses to queries about events they may have experienced. On questions related to combat experiences participants were asked to respond on a 5-point scale ranging from *never experienced* to *daily or almost daily*. Higher scores on both measures indicate greater exposure to events that may put individuals at greater risk for multiple negative outcomes (King et al., 2006). Follow-up studies have shown good criterion validity for the DRRI on veterans from Operation Iraqi Freedom and Operation Enduring Freedom (Vogt, Proctor, King, King, Vasterling, 2008). We found good reliability for these measures in the current study (Cronbach's alphas: Pre-deployment life events: 0.77, Combat experiences: 0.90, Aftermath of Battle: 0.86).

Analytic Plan

To evaluate the relationship between blood pressure and the development of PTSD symptoms immediately after deployment and one-year after return from deployment, we used hierarchical and stepwise regression analyses. To test hypothesis one, that individuals who respond to stressor tasks with increased blood pressure would develop a greater number of PTSD symptoms immediately post deployment and at one-year post deployment, two simultaneous multiple regression analyses were used. In the first step of each one, both gender and BMI were

used as covariates as both may impact blood pressure. In the second step, SBP and DBP reactivity scores were added to assess for their unique contribution to predicting PTSD symptoms. Two separate regressions were conducted, one to predict the PCL-M total score immediately after deployment and one to predict the PCL-M at one-year after return from deployment.

To test hypothesis two, that individuals who exhibited a smaller decrease in BP after stressful tasks would show more PTSD symptoms immediately post deployment and at one-year post deployment, four simultaneous multiple regression analyses were used. In the first step, both gender and BMI were used as covariates. In the second step, SBP and DBP recovery scores were added as predictors. Two separate regressions were conducted, one to assess their contributions to the prediction of the PCL-M total score immediately after deployment, and to assess their contributions to the prediction of the PCL-M total score one-year after return from deployment. These two regressions described above were each conducted twice with the first pair of regressions conducted with SBP and DBP recovery post task baseline entered as the IV. In the second pair of analyses, SBP and DBP resting post task baseline were entered as the IV.

To test hypothesis three, that pre-deployment physiological responses will predict post deployment PTSD symptoms over and above the effects of pre-deployment life events, combat exposure, and deployment exposure, we conducted stepwise regressions. Gender and BMI were entered in the first step, pre-task baseline SBP and DBP were entered in the second step to control for pre-task differences in blood pressure, pre-deployment life events, combat exposure, and deployment exposure were entered in the third step, and both SBP and DBP reactivity and recovery) were entered into the fourth step.

For the current study, analyses were only conducted using blood pressure because of limitations due to data access imposed by the East Orange Veterans Affairs Medical Center. Thus, analyses using heart rate data were unable to be conducted. These issues were unrelated to the current data and the proposed analyses. As a result, the current thesis does not propose any analyses using heart rate and instead focuses solely on blood pressure. For information of the demographic make-up of the sample, refer to Table 3.

Results

Correlations

Means and standard deviations of all study variables, as well as bivariate correlations between all study variables, can be found in Table 1 and 2.

Table 1: *Correlation Matrix of Study Variables: Immediate post deployment*

	1.	**2.**	**3.**	**4.**	**5.**	**6.**	**7.**	**8.**	**9.**	**10.**	**11.**	**12.**	***M*(SD)**
1. PTSD Symptoms	--	-.004	-.02	-.003	-.08	.21**	.28**	.34**	-.18*	-.08	-.12*	-.10	30.44 (11.76)
2. Gender		--	-.13*	-.23**	-.01	.01	-.16*	-.14*	-.07	-.05	-.09	.03	.09 (.28)
3. BMI		-.13	--	.26**	.21**	.07	.05	.07	-.19*	-.17*	-.19*	-.21**	27.79 (5.10)
4. Pre Task SBP		-.23**	.26**	--	.65**	.07	.06	.08	-.01	-.12*	.10	.04	113.39 (11.21)
5. Pre Task DBP		-.01	.21**	.65**	--	.14	-.05	-.06	.03	-.21**	-.01	-.12*	61.83 (8.98)
6. Pre-Deployment		.01	.07	.07	.14*	--	.13*	.22**	-.07	.01	-.10	-.14*	5.77 (3.55)
7. Combat Exposure		-.16*	.05	.06	-.05	.13*	--	.66**	-.08	-.002	-.03	-.03	7.11 (3.86)

8. Deployment Exposure	-.14*	.07	.08	-.06	.22**	.66**	--	-.13*	-.04	-.04	-.05	4.15 (3.86)
9. SBP Reactivity	-.07	-.19*	-.08	.03	-.07	-.08	-.11*	--	.69**	.77**	.62**	12.17 (8.21)
10. DBP Reactivity	-.05	.17*	-.12*	-.21**	-.09	-.002	-.04	.69**	--	.53**	.72**	7.43 (5.17)
11. SBP Recovery	-.09	-.19*	.10	-.01	-.10	-.03	-.04	.77**	.53**	--	.67**	7.14 (6.75)
12. DBP Recover	.03	-.21**	-.04	-.12*	-.14*	-.03	-.05	.62**	.72**	.67**	--	6.21 (4.88)

Correlation Matrix of Study Variables: Immediate post deployment

**Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

Note: Gender coded 0 = male, 1 = female

Table 2: *Correlation Matrix of Study Variables: One-year post deployment*

	1.	**2.**	**3.**	**4.**	**5.**	**6.**	**7.**	**8.**	**9.**	**10.**	**11.**	**12.**	***M*(SD)**
1. PCL_M_P4	--	-.02	.09*	.01	.04	.25**	.40**	.41**	-.16*	-.09*	-.12*	-.09*	32.75 (14.37)
2. Gender	-.02	--	-.2*	-.34**	-.15*	-.04	-.19*	-.16*	-.09*	-.09*	-.09*	-.04	.09 (.28)
3. BMI	.09*	-.20**	--	.24**	.21**	.03	.19*	.11*	-.12*	-.08	-.11*	-.08	27.79 (5.10)
4. Pre Task SBP	.01	-.34**	.24**	--	.66*	.01	.06	.06	.05	-.04	.17*	.13*	113.39 (11.21)
5. Pre Task DBP	.04	-.15*	.21**	.66**	--	.12*	-.004	-.001	-.02	-.23**	.03	-.06	61.83 (8.98)
6. Pre-Deployment	.25**	-.04	.03	.01	.12*	--	.21**	.19*	-.04	-.09*	-.08	-.13*	5.77 (3.55)
7. Combat Exposure	.40**	-.19*	.19*	.06	-.004	.21**	--	.71**	-.08	-.06	-.04	-.05	7.11 (3.86)

8. Deployment Exposure	.41**	-.16*	.11*	.06	-.001	.19*	.71**	--	-.12*	-.05	-.04	-.02	4.15 (3.86)
9. SBP React	-.16*	-.09*	-.12*	.05	-.02	-.04	-.08	-.12*	--	.70**	.80**	.65**	12.17 (8.21)
10. DBP React	-.09*	-.09*	-.08	-.04	-.23**	-.09*	-.06	-.05	.70**	--	.55**	.72**	7.43 (5.17)
11. SBP Recover	-.12*	-.09*	-.11*	.17*	.03	-.08	-.04	-.04	.80**	.55**	--	.72**	7.14 (6.75)
12. DBP Recover	-.09*	-.04	-.08	.13*	-.06	-.13*	-.05	-.02	.65**	.72**	.72**	--	6.21 (4.88)

Correlation Matrix of Study Variables: One-year post deployment

**Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

Note: Gender coded 0 = male, 1 = female

*Hypothesis 1***:** Those National Guard and Reserve Soldiers who react to stressful tasks with a larger increase in blood pressure (BP), will be more likely to develop PTSD symptoms both immediately after deployment and one-year after return from deployment.

Mean SBP and DBP for each minute of the study are depicted in Figure 1 and 2. To test Hypothesis 1, two hierarchical regression models were run, one for immediate post deployment and one for one-year post deployment. Gender and body mass index (BMI) were entered into the first step of the regression, and SBP and DBP reactivity were entered into the model in the second step. The overall model significantly predicted PTSD symptoms immediately after deployment, $F(4, 413) = 4.22$, $p < .001$, $R^2 = .041$, and this model predicted PTSD symptoms better than gender and BMI alone, $\Delta R^2 = .02$, $p < .001$ (see Table 4 Appendix B for the complete regression results). Of the individual variables entered into the model, only lower SBP reactivity emerged as a significant predictor of PTSD symptoms immediately post deployment, $t(415) = -3.55$, $\beta = -.24$, $p < .001$.

To test this same hypothesis at one-year after deployment, the same predictors were entered into the model, and this time PTSD symptoms at one-year after deployment was the outcome variable. The overall model significantly predicted PTSD symptoms immediately after deployment $F(4, 315) = 2.68$, $p = .03$, $R^2 = .02$, and was a significantly better model than gender and BMI alone, $\Delta R^2 = .02$, $p = .02$ (see Table 5 in Appendix B for the full regression results). Similar to the previous model, the only significant predictor that emerged was lower SBP reactivity $t(315) = -2.32$, $\beta = -.18$, $p = .02$.

Figure 1: *Mean SBP* for each minute of the study

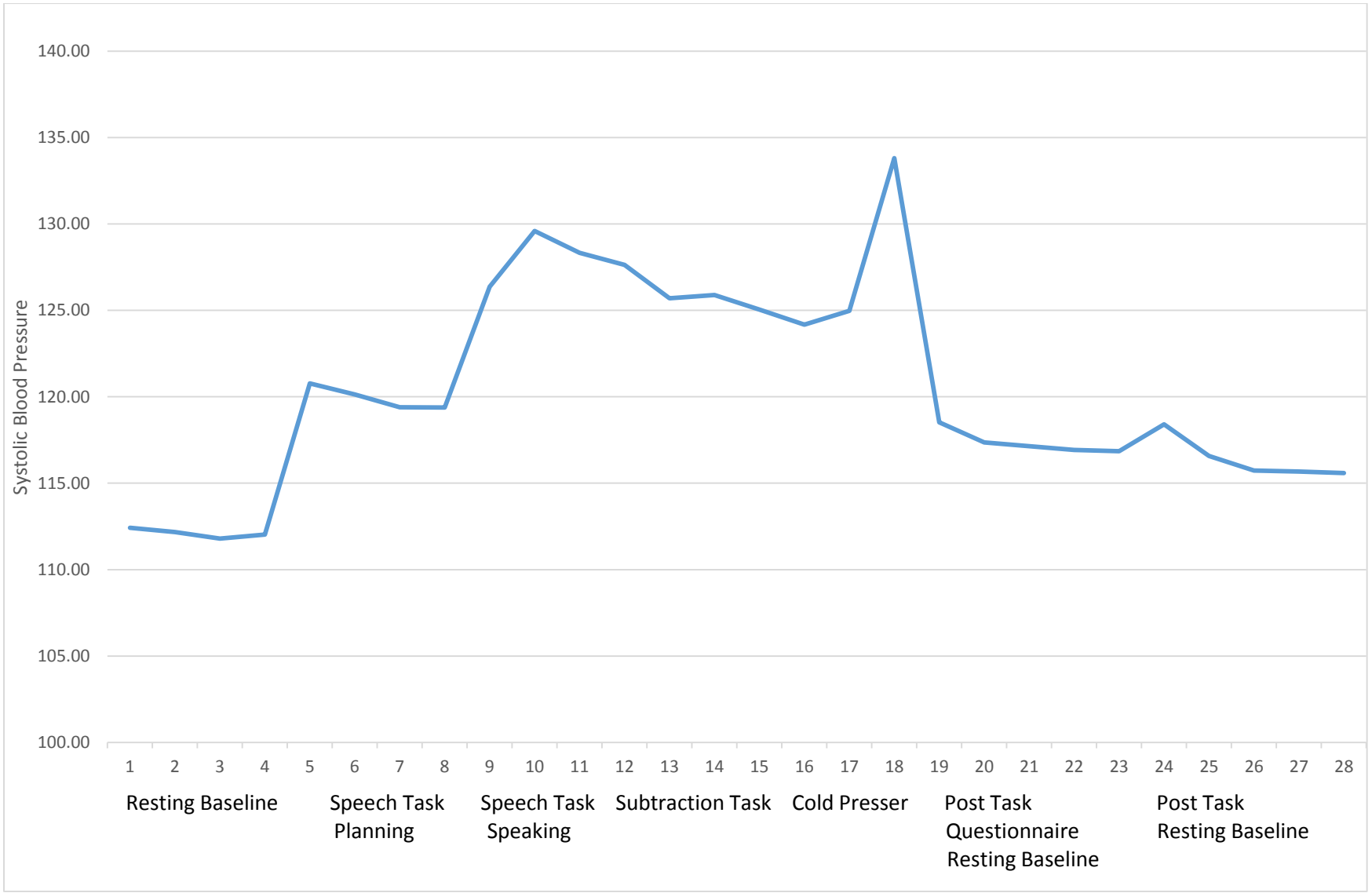
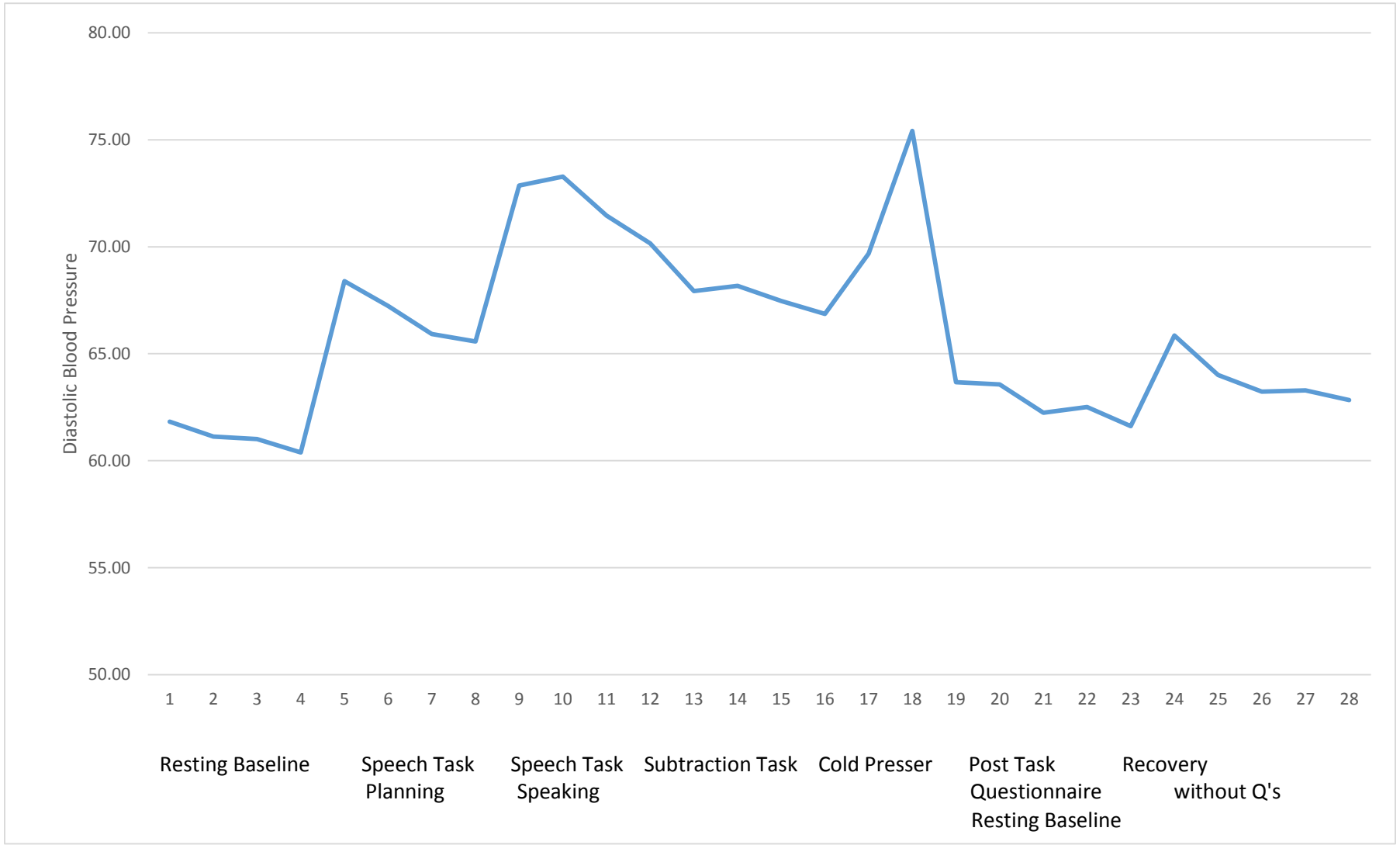


Figure 2: Mean DBP for each minute of the study



*Hypothesis 2***:** Those National Guard and Reserve Soldiers who have a smaller decrease in BP following stressful tasks will be more likely to develop PTSD symptoms immediately post deployment and one-year post deployment

To test the second hypothesis, two pairs of hierarchical regression models were run; one pair predicting immediate post deployment PTSD symptoms and one-year post deployment PTSD symptoms using SBP and DBP recovery as measured during the post-task questionnaire baseline, and the other pair of regression models predicting immediate post deployment PTSD symptoms and one-year post deployment PTSD symptoms using SBP and DBP recovery during the resting baseline that followed the questionnaires. In the first step of each model, gender and BMI were entered. In the second step SBP and DBP recovery were entered into the model.

The first regression used as a predictor the recovery during the post-task questionnaire baseline with the outcome of PTSD symptoms immediately after deployment. The overall model, with all predictor variables entered, significantly predicted PTSD symptoms F (4, 412) = 4.56, p. = 001, , R^2 = .03, and predicted significantly better than did gender and BMI alone, ΔR^2 = .04, p < .001 (see Table 6 in Appendix B for the full regression results). The only significant predictor of PTSD symptoms immediately after deployment was reduced SBP recovery during the post-task questionnaire baseline t (414) = -2.58, β =-.16, p =.01. Another model was used to assess the effect of recovery during the post task resting baseline. This model was significant F (4, 410) = 2.61, p. = .04 and accounted for 1.5% of the variance (see Table 7 in Appendix B for the full regression results). Despite a significant overall model, no individual predictors were significant.

Two additional regression models were run, this time with PTSD symptoms at one-year after deployment as the outcome variable. The overall model with all predictors and using recovery during the post-task questionnaire baseline did not significantly predict PTSD

symptoms one-year post deployment, F (4, 314) = 2.15, p = .08 and was not a significantly better model than BMI and gender alone ΔR^2 = .02, p =.065 (see Table 8 in Appendix B for the full regression results). Furthermore, none of the individual predictors in the model were significant. Of note, DBP recovery while completing questionnaires approached significance as a predictor of PTSD symptoms, t(317) = -1.94, β = -. 14., p =.053, R^2 = .03. The second regression using recovery during the post-task resting baseline was also not significant, F (4, 311) = 1.64, p = .17 (see Table 9 in Appendix B for the full regression results). These results indicate that recovery from tasks only predicted PTSD symptoms immediately after deployment and not at one-year after deployment.

Hypothesis 3: The effects of BP reactivity and recovery as predictors of PTSD symptoms will hold true over and above the effects of pre-deployment life events, combat exposure, and deployment exposure.

Two stepwise regressions were conducted to determine the optimal model for predicting of PTSD symptoms immediately after deployment. In the first step, gender and BMI were entered. In the second step basal SBP and DBP (from prior to beginning the stressor tasks) were entered into the model to control for basal blood pressure. In the third step, the DRRI pre-deployment life events, deployment exposure, and combat exposure were entered into the model. Finally, SBP and DBP reactivity and recovery were entered into the model in the fourth step. All variables were entered using a stepwise procedure. In the first model, SBP recovery during the post task questionnaire baseline was used, while in the second model, recovery during the post task resting baseline was used. This stepwise regression model found that three variables significantly predicted PTSD symptoms immediately after deployment, F (3, 331) = 20.06, p < .001. Specifically, more pre-deployment life events t (334) = 2.57, β = .13, p =.01, more

deployment exposures, t (334) = 5.66, β = .29, p <.001, and less SBP recovery during the post-task questionnaire baseline t (334) = -2.913, β = -.15, p =.004 predicted PTSD symptoms immediately after deployment (see Table 10 in Appendix B). This model predicted 15% of the variance in PTSD symptoms immediately after deployment. In a second analysis, when SBP recovery during the post-task resting baseline was used together with SBP reactivity, lower SBP reactivity t (329) = -2.91, β = -.14, p =.01 emerged as a significant predictor along with pre-deployment life events t (329) = 2.57, β = .14, p =.01 and deployment exposures, t (329) = 5.66, β = -.29, p <.01 whereas SBP recovery was no longer a significant predictor (see Table 11 in Appendix B for the full regression results).

Two additional stepwise regression analyses were conducted to assess predictors of PTSD symptoms at one-year after deployment. The stepwise model significantly predicted PTSD symptoms, F (4, 318) = 22.56, p < .001. Similar to the model immediately after deployment, both pre-deployment life events t (317) = 2.98, β = .15, p =.003 and deployment exposures, t (317) = 3.11, β = .22, p =.002, emerged as significant predictors. In addition, combat exposure emerged as a significant predictor of PTSD symptoms at one-year after deployment, t (317) =2.82, β = .20, p =.01. Finally, reduced SBP reactivity was a significant predictor regardless of which recovery variable was used in the model, t (317) = -2.26, β = -.11, p =.03. The overall model predicted 21.3% of the variance in PTSD symptoms at one-year post deployment (see Table 12 and 13 in Appendix B).

Discussion

The purpose of the current study was to examine the relationship between pre-deployment blood pressure reactivity and recovery to stressful tasks and post- deployment PTSD symptoms. Lower SBP reactivity to a series of stressor tasks emerged as the best predictor of

PTSD symptoms immediately after and one-year after deployment. Less SBP recovery during a post-task questionnaire baseline that immediately followed the stressor tasks was found to be the best predictor of PTSD symptoms immediately after deployment, but blood pressure recovery did not predict PTSD symptoms one-year after deployment. I also used a stepwise regression model to determine the best blood pressure reactivity and recovery predictors of post-deployment PTSD symptoms when controlling for exposure to trauma. Lower SBP recovery during a post-task questionnaire baseline was the best predictor of immediate post-deployment PTSD symptoms, whereas less SBP reactivity better predicted PTSD at one-year after deployment. The final model derived using a stepwise procedure found that pre-deployment life events, deployment exposure, and reduced SBP recovery (while completing questionnaires) accounted for 15% of the variance in PTSD symptoms immediately after deployment. Therefore, the best predictors of PTSD symptoms immediately after deployment were exposure to traumatic events and reduced SBP recovery during a post task questionnaire baseline. Pre-deployment life events, deployment exposures, combat exposures, and reduced SBP reactivity to tasks significantly predicted PTSD symptoms at one-year after deployment. This model accounted for 21.3% of the variance in PTSD symptoms at one year after deployment. Therefore, the best predictors of PTSD symptoms at one year after deployment were exposure to traumatic events and less SBP reactivity to the pre-deployment stressor tasks.

The current study found that soldiers who responded to pre-deployment stressor tasks with lower SBP reactivity to and less SBP recovery from stressors prior to deployment were more likely to report increased PTSD symptoms after deployment. Unlike previous research using autonomic measures of PTSD, here a less reactive profile was predictive of later PTSD. To date, studies have only noted a decrease in autonomic functioning among individuals who were

diagnosed with PTSD and exposed to multiple traumatic events (McTeague, et al. 2010). In this work, individuals who had experienced multiple traumas responded with a blunted cardiovascular response to imagery of their trauma compared to those exposed to a single event that led to PTSD. McTeague et al. (2010) hypothesized that this was due to a decrease in defensive reactivity; that individuals who had experienced multiple traumas no longer saw threats in the same manner as individuals with less trauma exposure. These results are consistent with findings that individuals with more severe symptoms across a spectrum of anxiety disorders exhibit blunted responses when confronted with anxiety imagery (Lang & McTeague, 2009). Although Lang and McTeague (2009) interpret this blunted response as indicating that "normal defensive reactivity may be compromised by an experience of long-term stress" (p. 5), it may be the case that the tendency for blunted responding to stressors predates anxiety disorder symptoms. In fact, the blunted responses associated with more severe anxiety disorders may be associated with a broader range of difficulties, including perhaps poorer emotion regulation abilities that may be, in part, a cause of their later-developing anxiety symptoms. According to Gross (2013), emotion regulation is the capacity of an individual to respond with an appropriate emotion that is of suitable intensity and duration to a situation. Notably, previous research has found alterations in physiological responses among those with decreased emotion regulation (Gross, 2002). In line with the findings of McTeague et al. (2010), I suggest that a blunted physiological response to, and less recovering from, stressors, as found in the current study, may represent a deficit in emotion regulation. Specifically, those people who have trouble regulating emotional responses to stressors also may have greater difficulty regulating their responses to, and recovering from, deployment-related traumatic experiences. Further, deficits in emotion regulation have commonly been found among individuals with PTSD (Ehring & Quack, 2010;

Boden, et al. 2013). What I highlight here is the possibility that emotion regulation deficits pre-date the occurrence of PTSD.

A recent study highlighted the relationship between emotion regulation and blood pressure reactivity to stress (Delgado, Vila, & Reyes del Paso, 2014). Delgado et al. (2014) found, among a sample of high and low trait worriers (top and bottom 20% on a measure of worry), that higher trait worriers reacted to mental and auditory stressors with smaller magnitude BP responses than those lower in trait worry. Based upon these findings, Delgado et al. (2014) posited that increases in BP are a mechanism that reduces emotional distress provoked by the current stressful tasks (McCubbin et al. 2011). In support of the current hypothesis, individuals who are high in worry often exhibit less ability to regulate emotions than those low in trait worry (Salters-Pedneault, Roemer, Tull, Rucker, & Mennin, 2006). Therefore, lower BP reactivity to stressors prior to the experience of a potentially traumatic event could be associated with a decrease in the ability to regulate emotions, similar to those high in trait worry.

Consistent with the findings of the current study, Minassian et al. (2015) found that lower pre-deployment HRV, an autonomic effect suggestive of less resting parasympathetic activity, was predictive of PTSD diagnosis six months later in a sample of marines. Lower HRV also has been associated with a reduced ability to regulate emotions (Thayer & Lane, 2000), and multiple studies have shown that increased emotion regulation is associated with increased basal HRV (Butler et al. 2006; Smith et al. 2011). These findings suggest that soldiers who exhibit lower basal HRV may be more likely to have poor emotion regulation abilities and be more likely to develop PTSD. Further work that directly measures emotion regulation capabilities will be required to directly test this hypothesis.

Clinical Implications

Based on the finding that PTSD is related not only to exposure to traumatic events but to reduced blood pressure reactivity to and recovery from stressors, and the speculation that this is related to reduce emotion regulation capacity, it suggests that one target for primary prevention may be alteration of physiological arousal. One way of altering an individual's physiological reactivity and recovery from anxiety-producing situations is the use of biofeedback. Biofeedback is a method of altering physiological processes through conscious awareness of sensations by monitoring and providing input to the person about the physiological changes underlying those sensations (Schwartz & Andrasik, 2003). Multiple studies have found that biofeedback can be used, e.g., to increase HRV (Del Pozo, Gevirtz, Scher, & Guarneri, 2004; Nolan et al. 2005; Ginsberg & Fogo, 2014). As detailed in Minassian et al. (2015), prior to deployment, decreased HRV was associated with an increased likelihood of developing PTSD. The current results suggest that reduced blood pressure reactivity and recovery to stressors is associated with greater risk of PTSD symptoms. Therefore, an intervention targeted at regulation of BP or HRV or other autonomically-related sensations may buffer the impact of a traumatic event. In support of this claim, Peira, Pourtois, & Fredrickson (2013) found that biofeedback could be used to increase a person's ability to regulate his/her HR. Furthermore, in support of my speculation about the link between blunted blood pressure reactivity and recovery and reduced emotion regulation capacity, Peira et al. (2013) found that an increased ability to regulate HR was associated with an increased ability to regulate emotions to emotionally distressing situations. Participants were not only able to regulate their HR responses but were able to do so when presented with a stimulus they experienced as negative. Moreover, in a follow up study, individuals who had received biofeedback compared those who received sham biofeedback, were better able to regulate their HR responses to negative stimuli (Peira, Fredrickson, & Pourtois, 2014). These studies provide

evidence that biofeedback can help a person to better regulate their physiology when presented with negative stimuli. In addition to HR and HRV, multiple studies have also demonstrated efficacy of biofeedback for the regulation of blood pressure (Lin et al. 2012; Wang et al. 2010) Therefore, biofeedback may aid individuals in regulating multiple different kinds of autonomic functioning.

The use of biofeedback among individuals with PTSD also has empirical support. In an early study of biofeedback among Vietnam veterans with PTSD, Hickling, Sison, & Vanderploeg, (1996) found that EMG biofeedback produced noticeable decreases in PTSD symptoms, specifically decreasing the heightened arousal associated with PTSD, when included as part of a cognitive behavioral therapy treatment regimen. Moreover, recent studies among OEF/OIF veterans have found significant decreases in number of individuals diagnosed with PTSD when HRV biofeedback is included in combination with Prolonged Exposure (PE), Cognitive Processing Therapy (CPT), or Acceptance and Commitment Therapy (ACT; Tan, Wang, & Ginsberg, 2013). According to Tan et al. (2013), decreases in arousal and increases in attention and memory were associated with an overall reduction in PTSD symptoms among patients using biofeedback treatment in conjunction with PE or CPT. A recent pilot study also noted that individuals who underwent trauma-focused CBT achieved symptom remission faster when using HRV biofeedback than those who did not (Polak, Witteveen, Denys, & Olff, 2015). Therefore, there is tentative support for the success of biofeedback as a treatment for individuals who have developed PTSD. If, as suggested by the current study, biofeedback is helpful for people with PTSD because they begin to learn to better regulate their autonomically-mediated physiological responses, it may be useful prior to deployment as a way to assist military

personnel in the regulation of their autonomic functioning, perhaps leading to an increased ability to regulate emotions as a buffer against the effects of traumatic experiences.

Based upon the current findings, screening of autonomic functioning prior to deployment may help identify those vulnerable to developing PTSD. Within the context of the current study, screening individuals in the National Guard/Reserve component of the military is indicated as they have higher rates of PTSD and develop PTSD at a higher rate than their active duty counterparts (Baker et al. 2009; Smith et al. 2009; Thomas, Wilk, Riviere, McGurk, Castro, & Hoge, 2010; Vasterling et al. 2010). Moreover, National Guard/Reserve soldiers who exhibit decreased SBP reactivity and recovery from tasks may then be more closely tracked to assess for signs of PTSD and enrolled in early interventions. Multiple studies have demonstrated efficacy for CBT, brief exposure therapy, beta adrenergic blockade and glucocorticoid administration during the acute stress period following a traumatic event (Kearns, Ressler, Zatzick, & Rothbaum, 2012;Vaiva, et al. 2003). These strategies, implemented prior to PTSD diagnosis, would be considered primary prevention, and could reduce the overall incidence of PTSD. By implementing primary prevention strategies such as biofeedback, enhanced surveillance, and possible pharmacological pre-treatment prior trauma exposure, we may be able to substantially reduce the incidence of PTSD.

Limitations

The current study has a few noteworthy limitations. The findings of the current study are limited to blood pressure and thus do not capture the full scope of physiological reactivity and recovery to tasks. Other variables, including HR, HRV, SC, and corrugator EMG, may provide further clarification of the current findings, because prior research has linked increases in SC responses and corrugator EMG activation to decreases in emotion regulation (Sloan, 2004). By

adding other biological measurements and measures of emotion regulation, a more specific emotion regulation capacity hypothesis can be developed. One variable that should receive significant attention is HR. Heart Rate has shown strong predictive ability in the acute stress phase across multiple studies (Shalev et al., 1998; Bryant et al., 2000; Bryant, Harvey, Guthrie, & Moulds, 2003; Bryant, Salmon, Sinclair, & Davidson, 2007; Bryant, Creamer, O'Donnell, Silove, & McFarlane, 2008; Suendermann, Ehlers, Boellinghaus, Gamer, Glucksman, 2010; Coronas, Gallardo, Moreno, Suarez, Garcia-Pares, & Menchon, 2011). Therefore, HR may be an especially sensitive predictive measure in studies where physiology can be measured prior to a trauma. Another limitation is that the current study's stressor task design does not allow for determination of the unique contribution of different tasks as specific predictors of PTSD symptoms. As there were no rest periods between tasks, it is more difficult to isolate the autonomic responses during each task as a specific and unique predictor. Thus, e.g., one cannot say whether it is the BP response to preparing for the confrontation task or to actually doing the confrontation task that is associated with PTSD symptoms, or whether a more generalized BP reactivity and recovery is most important. In addition, as soldiers completed questionnaires after completing the task period, we cannot disentangle whether the predictive usefulness of the SBP recovery is due to the timing of this period (i.e., it was the first five minutes of recovery) or could also be a function of simultaneously completing questionnaires. Additionally, because recovery was calculated as a reduction from task reactivity, lower recovery is confounded by less reactivity, as this would produce a restriction in the amount of blood pressure reduction needed to return to a resting baseline.

Another limitation of the study is that PTSD symptoms were not assessed prior to deployment, and so one cannot rule out the possibility that participants with blunted

physiological reactivity already had more PTSD symptoms prior to deployment. As detailed above, McTeague et al. (2010) found that lower SBP reactivity to threatening imagery was associated with exposure to multiple traumatic events. Therefore lower SBP reactivity pre-deployment could be a result of a prior trauma history and/or PTSD symptoms prior to deployment, rather than a de novo predictor of PTSD symptoms after deployment. Furthermore, inhibited physiological recovery from stressors is a hallmark of PTSD (Jovanovic & Ressler, 2010) and without pre deployment measurement of PTSD symptoms, there is the possibility that individuals with blunted BP responses already had greater PTSD symptoms at the time of the pre-deployment assessment.

Conclusions

Despite the aforementioned limitations, the current study contributes to the literature by showing that lower SBP reactivity to stressor tasks given prior to deployment is associated with more PTSD symptoms immediately after deployment and one-year after deployment. Furthermore, the current study also demonstrated that lower SBP recovery predicted PTSD symptoms immediately after deployment. These effects were found in a sample of National Guard and Army Reserve soldiers, in whom multiple studies have shown higher rates of PTSD symptoms and diagnosis when compared to their active duty counterparts (Baker et al. 2009; Smith et al. 2009; Thomas, Wilk, Riviere, McGurk, Castro, & Hoge, 2010; Vasterling et al. 2010). Furthermore, the current sample was comprised of individuals who reported substantially higher current PTSD symptoms compared to previous studies examining these issues using a prospective design (e.g. Guthrie & Bryant, 2006; Orr et al. 2012; Pole et al. 2009). Thus, the results of the current study are more representative of a population of individuals who are likely to be diagnosed with PTSD (similar to Minassian et al.; 2015). The current study is the first to show that a less reactive BP response to stressors is a possible risk factor for the development of

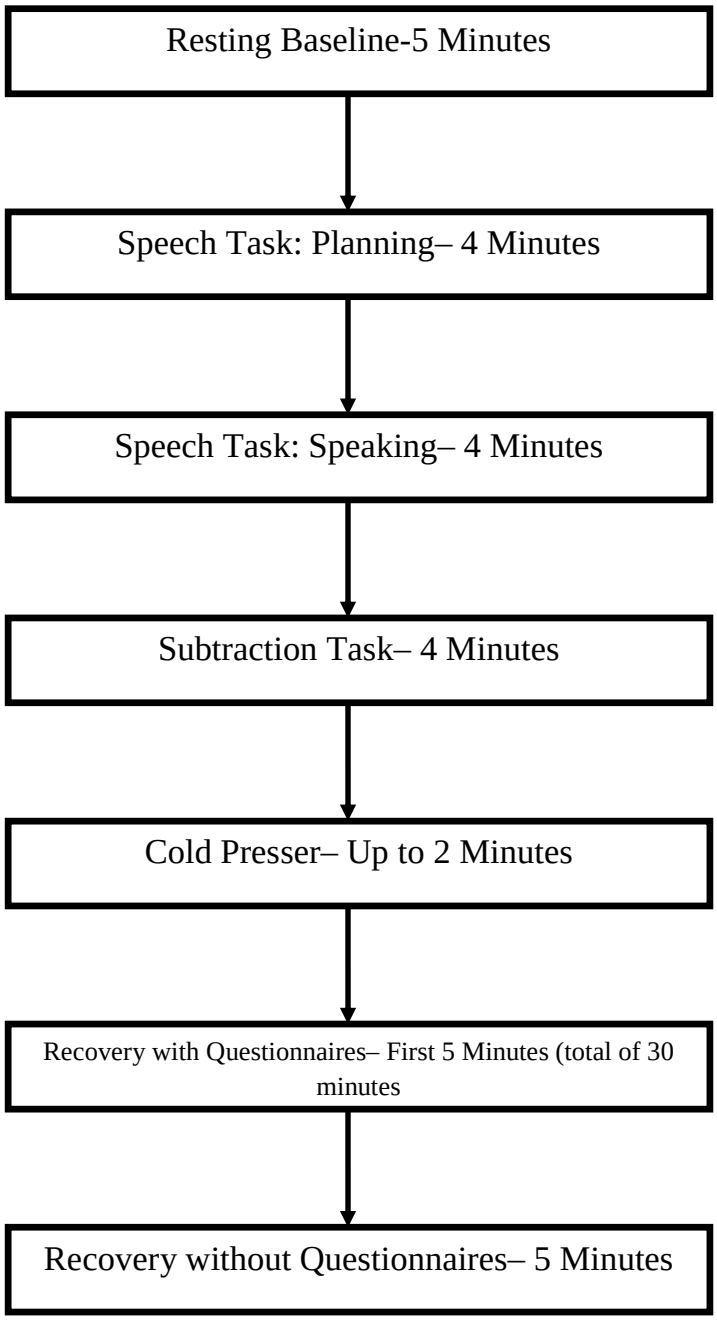
PTSD symptoms, and further substantiates the literature that less BP recovery from stressor tasks is associated with more symptoms of PTSD (Guthrie & Bryant, 2006; Pole et al. 2009; Orr et al. 2012). In doing so, the current study suggests feasible potential targets for primary prevention (i.e., BP biofeedback) prior to deployment for National Guard and Army Reserve soldiers in hopes of reducing the subsequent occurrence of PTSD.

Table 3. Characteristics of Initial Sample.

	Immediately Prior to Deployment Demographics
Gender	
Male	688 (89.7%)
Female	79 (10.3%)
Age – mean years *(SD)*	28.0 *(8.3)*
	Range: 18 - 57
Education	97.4% high school graduate.
	2.0% Bachelors
Military Component	
National Guard	554 (72.2%)
Reserve	202 (26.3%)
Active/Other	11 (1.4%)
Race	
White	592 (77.2%)
Black	69 (9.0%)
American Indian	21 (2.7%)
Asian/Pacific Islander	21 (2.7%)
Mixed race/Other	48 (6.3%)
Ethnicity	
Hispanic*	95 (12.4%)

**Note: Ethnicity was dichotomized as non-white Hispanic vs other.*

Figure 3. Order of physiological measurement



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Appendix

Measures

Figure 4: PTSD Checklist Military

		Frequency:				
No.	Problem or Complaint:	Not at all (1)	A little bit (2)	Moderately (3)	Quite a bit (4)	Extremely (5)
1.	Repeated, disturbing *memories, thoughts,* or *images* of a stressful military experience?					
2.	Repeated, disturbing *dreams* of a stressful military experience?					
3.	Suddenly *acting* or *feeling* as if a stressful military experience *were happening again* (as if you were reliving it)?					
4.	Feeling very upset when something reminded you of a stressful military experience?					
5.	Having *physical reactions* (e.g., heart pounding, trouble breathing, or sweating) when *something reminded* you of a stressful military experience?					
6.	Avoid *thinking about* or *talking* about a stressful military experience or avoid *having feelings* related to it?					
7.	Avoid *activities* or *talking about* a stressful military experience or avoid *having feelings* related to it?					
8.	Trouble *remembering important parts* of a stressful military experience?					
9.	Loss of *interest* in things that you used to enjoy?					
10.	Feeling *distant* or *cut off* from other people?					
11.	Feeling *emotionally numb* or being unable to have loving feelings for those close to you?					
12.	Feeling as if your *future* will somehow be *cut short*?					
13.	Trouble *falling* or *staying* asleep?					
14.	Feeling *irritable* or having *angry outbursts*?					
15.	Having *difficulty* concentrating?					
16.	Being "*super alert*" or watchful on guard?					
17.	Feeling *jumpy* or easily startled?					

Figure 5: DRRI-PDLE

The statements below refer to events you may have experienced. Please circle "yes" or "no" for each item below.

I have experienced…

1. ...a natural disaster (for example, a flood or hurricane), a fire, or an accident in which I was hurt or my property was damaged.	**Yes**	**No**
2. ...exposure to a toxic substance (such as dangerous chemicals, radiation).	**Yes**	**No**
3. ...combat or exposure to a war zone (in the military or as a civilian).	**Yes**	**No**
4. ...the mental illness (for example, clinical depression, anxiety disorder), or life-threatening physical illness (for example, cancer or heart disease) of someone close to me.	**Yes**	**No**
5. ...a parent who had a problem with drugs or alcohol.	**Yes**	**No**
6. ...the death of someone close to me.	**Yes**	**No**

I have ...

7. ...been through a divorce or been left by a partner or significant other.	**Yes**	**No**
8. ...witnessed someone being assaulted or violently killed.	**Yes**	**No**
9. ...been robbed or had my home broken into.	**Yes**	**No**
10. ...lost my job.	**Yes**	**No**
11. ...been emotionally mistreated (for example, shamed, embarrassed, ignored, or repeatedly told I was no good).	**Yes**	**No**
12. ...seen or heard physical fighting between my parents or caregivers.	**Yes**	**No**
13. ...been physically punished by a parent or primary caregiver.	**Yes**	**No**
14. ...been physically injured by another person (for example, hit, kicked, beaten up).	**Yes**	**No**
14a. [IF YES] did this occur (circle all that apply):	**in childhood**	**in adulthood**

15.	...experienced unwanted sexual activity as a result of force, threat of harm, or manipulation.	**Yes**	**No**
15a.	[IF YES] did this occur (circle all that apply):	**in childhood**	**in adulthood**

Figure 6: DRRI-CE

**The response options for the CE subscale were changed from a yes/no format to the following: 0 = never, 1 = a few times over the entire deployment, 2 = a few times each month, 3 = a few times each week, and 4 = daily or almost daily. This was done to more sensitively measure exposure to critical events such as feeling in mortal danger, or anxiety about combat patrols or other missions. The modified CE subscale was on a 0-60 scale.

The statements below are about your combat experiences during deployment. Please circle "yes" if the statement is true or "no" if the statement is false.

While deployed:

1.	I went on combat patrols or missions.	**0**	**1**	**2**	**3**	**4**
2.	I or members of my unit encountered land or water mines and/or booby traps.	**0**	**1**	**2**	**3**	**4**
3.	I or members of my unit received hostile incoming fire from small arms, artillery, rockets, mortars, or bombs.	**0**	**1**	**2**	**3**	**4**
4.	I or members of my unit received "friendly" incoming fire from small arms, artillery, rockets, mortars, or bombs.	**0**	**1**	**2**	**3**	**4**
5.	I was in a vehicle (for example, a truck, tank, APC, helicopter, plane, or boat) that was under fire.	**0**	**1**	**2**	**3**	**4**
6.	I or members of my unit were attacked by terrorists or civilians.	**0**	**1**	**2**	**3**	**4**
7.	I was part of a land or naval artillery unit that fired on the enemy.	**0**	**1**	**2**	**3**	**4**
8.	I was part of an assault on entrenched or fortified positions.	**0**	**1**	**2**	**3**	**4**
9.	I took part in an invasion that involved naval and/or land forces.	**0**	**1**	**2**	**3**	**4**
10.	My unit engaged in battle in which it suffered casualties.	**0**	**1**	**2**	**3**	**4**
11.	I personally witnessed someone from my unit or an ally unit being seriously wounded or killed.	**0**	**1**	**2**	**3**	**4**
12.	I personally witnessed soldiers from enemy troops being seriously wounded or killed.	**0**	**1**	**2**	**3**	**4**

13. I was wounded or injured in combat.	**0**	**1**	**2**	**3**	**4**
14. I fired my weapon at the enemy.	**0**	**1**	**2**	**3**	**4**
15. I killed or think I killed someone in combat.	**0**	**1**	**2**	**3**	**4**

16. How many times were you engaged in a firefight during your deployment? _____

Figure 7: DRRI-DE

1. I observed homes or villages that had been destroyed.	**Yes**	**No**
2. I saw refugees who had lost their homes and belongings as a result of battle.	**Yes**	**No**
3. I saw people begging for food.	**Yes**	**No**
4. I or my unit took prisoners of war.	**Yes**	**No**
5. I interacted with enemy soldiers who were taken as prisoners of war.	**Yes**	**No**
6. I was exposed to the sight, sound, or smell of animals that had been wounded or killed from war-related causes.	**Yes**	**No**
7. I took care of injured or dying people.	**Yes**	**No**
8. I was involved in removing dead bodies after battle.	**Yes**	**No**
9. I was exposed to the sight, sound, or smell of dying men and women.	**Yes**	**No**
10. I saw enemy soldiers after they had been severely wounded or disfigured in combat.	**Yes**	**No**
11. I saw the bodies of dead enemy soldiers.	**Yes**	**No**
12. I saw civilians after they had been severely wounded or disfigured.	**Yes**	**No**
13. I saw the bodies of dead civilians.	**Yes**	**No**
14. I saw Americans or allies after they had been severely wounded or disfigured in combat.	**Yes**	**No**
15. I saw the bodies of dead Americans or allies.	**Yes**	**No**

Appendix B: Summary of Regression Results

Table 4: Summary of Hierarchical Regression Analysis: Hypothesis 1 Immediately Post Deployment

	F	Unstandardized beta	Standardized Beta	*t*	*p*	R^2	ΔR^2
Step 1 Model (Covariates)	.12					.01	
Gender		.03	.001	.01	*.99*		
BMI		-.11	-.05	-.94	*.35*		
Step 2 Model	4.36**					.03	.02
SBP Reactivity		-.34	-.24	-3.55**	<.001		
DBP Reactivity		.12	.05	.81	.42		

DV = PCL-M (PTSD) Immediate Post Deployment

*Significant at the 0.05 level

**Significant at the 0.001 level

Table 5 Summary of Hierarchical Regression Analysis: Hypothesis 1 One year Post Deployment

	F	Unstandardized beta	Standardized Beta	*t*	*p*	R^2	ΔR^2
Step 1 Model (Covariates)	1.38					.002	
Gender		-1.33	-.03	-.53	*.60*		
BMI		.23	.07	1.18	*.24*		
Step 2 Model	2.68*					.02	.02
SBP Reactivity		-.35	-.18*	-2.32	.02		

DBP Reactivity		.04		.11	.47	.64

DV = PCL-M (PTSD) one-year post deployment

Summary of Hierarchical Regression Analysis: Hypothesis 2

Table 6 Summary of Hierarchical Regression Analysis: Hypothesis 2: Immediately Post Deployment

	F	Unstandardized beta	Standardized Beta	*t*	*p*	R^2	ΔR^2
Step 1 Model (Covariates)	.12					.004	
Gender		.72	.02	.35	*.73*		
BMI		-.12	-.05	-1.03	*.30*		
Step 2 Model	4.56**					.03	.04
SBP Reactivity(Distraction)		-.28	-.16	-2.58*	.01		
DBP Reactivity(Distraction)		-.17	.07	-1.09	.28		

DV = PCL-M (PTSD) Immediate Post Deployment

Table 7 Summary of Hierarchical Regression Analysis: Hypothesis 2: One-Year Post Deployment

	F	Unstandardized beta	Standardized Beta	*t*	*p*	R^2	ΔR^2
Step 1 Model (Covariates)	.12					.004	
Gender		.48	.01	.23	*.82*		
BMI		-.09	-.04	-.81	*.42*		

Step 2 Model	2.61					.02	.02*
SBP Recovery (No distraction)		-.12	-.09	-1.34	.18		
DBP Recovery (No distraction)		-.19	-.08	-1.23	.22		

DV = PCL-M (PTSD) Immediate Post Deployment

Table 8

	F	Unstandardized beta	Standardized Beta	*t*	*p*	R^2	ΔR^2
Step 1 Model (Covariates)	1.43				.24	.003	
Gender		-.59	-.01	-.23			
BMI		.22	.07	1.16			
Step 2 Model	2.13				.08		.02*
SBP Recovery(Distraction)		.03	.01	.18	.86		
DBP Recovery(Distraction)		-.45	-.14	-1.94	.053		

DV = PCL-M (PTSD) one-year Post Deployment

Table 9

	F	Unstandardized beta	Standardized Beta	*t*	*p*	R^2	ΔR^2
Step 1 Model (Covariates)	.12					-.004	
Gender		.48	.01	.23	*.82*		

BMI		-.09	-.04	-.81	*.42*		
Step 2 Model	2.61					.02	.02*
SBP Recovery(No distraction)		-.12	-.09	-1.34	.18		
DBP Recovery (No distraction)		-.19	-.08	-1.23	.22		

DV = PCL-M (PTSD) one-year Post Deployment

Summary of Stepwise Regression Analysis: Hypothesis 3 (Recovery during questionnaires)

Table 10

	F	Unstandardized beta	Standardized Beta	*t*	*p*	R^2	ΔR^2
Step 1 Model	16.41**					.04**	
DRRI_PDLE		.72	.22	4.05	<.001**		
Step 2 Model	26.01**						.09**
DRRI PDLE		.49	.15	2.85	.005*		
DRR DE		.93	.31	5.83	<.001**		
Step 3 Model	20.06**						.02*
DRRI PDLE		.44	.13	2.57	.011*		
DRR DE		.90	.29	5.66	<.001**		
SBP *Recovery (Distraction)*		-.26	-.15	-2.91	.004**		

DV = PCL-M (PTSD) Immediate Post Deployment

Summary of Stepwise Regression Analysis: Hypothesis 3 (Recovery without completing questionnaires)

Table 11

	F	Unstandardized beta	Standardized Beta	*t*	*p*	R^2	ΔR^2
Step 1 Model	15.86**					.04**	
DRRI_PDLE		.71	.21	3.98	<.001**		
Step 2 Model	26.01**						.09**
DRRI PDLE		.49	.15	2.81	.005*		
DRR DE		.94	.31	5.88	<.001**		
Step 3 Model	20.06**						.02*
DRRI PDLE		.47	.14	2.57	.007*		
DRRI DE		.90	.29	5.66	<.001**		
SBP Reactivity		-.20	-.14	-2.913	.008*		

DV = PCL-M (PTSD) Immediate Post Deployment

Summary of Stepwise Regression Analysis: Hypothesis 3 (Recovery during questionnaires)

Table 12

	F	Unstandardized beta	Standardized Beta	*t*	*p*	R^2	ΔR^2
Step 1 Model	19.80**					.06**	
DRRI_PDLE		1.03	.24	4.45	<.001**		

Step 2 Model	37.31**						.13**
DRRI PDLE		.72	.17	3.26	.001**		
DRR DE		1.34	.37	7.19	<.001**		
Step 3 Model	28.02**						.02*
DRRI PDLE		.66	.16	3.02	.003*		
DRRI DE		.84	.23	3.28	.001**		
SBP Reactivity		.38	.20	2.80	.005*		
Step 4 Model	22.56**						.01*
DRRI PDLE		.65	.15	2.98	.003*		
DRRI DE		.80	.22	3.11	.002*		
DRRI CE		.38	.20	2.81	.005*		
SBP Reactivity		-.21	-.11	-2.26	.03*		

DV = PCL-M (PTSD) one-year Post Deployment

Summary of Stepwise Regression Analysis: Hypothesis 3 (Recovery without completing questionnaires)

Table 13

	F	Unstandardized beta	Standardized Beta	*t*	*p*	R^2	ΔR^2
Step 1 Model	19.80**					.06**	
DRRI_PDLE		1.05	.25	4.47	<.001**		
Step 2 Model	37.31**						.13**
DRRI PDLE		.74	.17	3.30	.001**		

DRR DE		1.35	.37	7.20	<.001**		
Step 3 Model	28.02**						.02*
DRRI PDLE		.68	.16	3.08	.002*		
DRRI DE		.86	.24	3.34	.001**		
SBP Reactivity		.38	.20	2.72	.005*		
Step 4 Model	22.56**						.01*
DRRI PDLE		.67	.16	3.06	.001**		
DRRI DE		.82	.23	3.18	.002*		
DRRI CE		.38	.20	2.74	.006*		
SBP Reactivity		-.22	-.11	-2.26	.02*		

DV = PCL-M (PTSD) one-year Post Deployment

Vita

David Rothman was born on September 1st, 1991 in New Brunswick, New Jersey. He graduated from Cherry Hill High School East in 2009. He received his Bachelors of Arts in Psychology from The College of New Jersey in December of 2012. He is currently working towards his doctorate in Clinical Psychology, with a focus in behavioral medicine at Virginia Commonwealth University.