



VCU

Virginia Commonwealth University
VCU Scholars Compass

Theses and Dissertations

Graduate School

2010

The influence of ethnicity and gender on the relationship between Posttraumatic Stress Disorder status and cardiovascular responding

Alison Eonta
Virginia Commonwealth University

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



Part of the [Psychology Commons](#)

© The Author

Downloaded from

<https://scholarscompass.vcu.edu/etd/2184>

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

THE INFLUENCE OF ETHNICITY AND GENDER ON THE RELATIONSHIP
BETWEEN POSTTRAUMATIC STRESS DISORDER STATUS AND
CARDIOVASCULAR RESPONDING

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

By: ALISON EONTA
Bachelor of Arts, University of Virginia, 2005

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

Virginia Commonwealth University
Richmond, Virginia
May, 2010

Acknowledgements

I would like to thank my advisor, Dr. Scott Vrana, for his help and for his direction with this project. I would also like to thank the entire Department of Psychology at Virginia Commonwealth University for the support and guidance they provided.

Table of Contents

| | Page |
|---|------|
| Acknowledgements | ii |
| List of Tables | v |
| List of Figures | vi |
| Abstract | vii |
| Introduction | 1 |
| Literature Review | 2 |
| Ethnic and gender differences in physiological response | 3 |
| The psychophysiology of PTSD | 6 |
| Gender | 9 |
| All-male studies | 11 |
| All-female studies | 16 |
| Mixed-gender studies | 18 |
| Ethnicity | 22 |
| Statement of the Problem | 23 |
| Method | 24 |
| Participants | 25 |
| Apparatus | 27 |
| Design and Procedure | 27 |
| Data Analysis Plan | 29 |
| Results | 30 |
| Analyses of Variance and Covariates | 30 |
| Baseline HR | 31 |
| Baseline SBP | 33 |
| Baseline DBP | 34 |
| HR Response to Anger | 35 |
| SBP Response to Anger | 36 |
| DBP Response to Anger | 37 |
| Discussion | 37 |
| Baseline Findings | 38 |
| Anger Recall Findings | 52 |
| List of References | 60 |
| Appendix | 69 |

Vita 75

List of Tables

| | Page |
|--|------|
| Table 1. PTSD Studies that Report Baseline Physiological Findings | 14 |
| Table 2. Demographic Information by Group | 26 |
| Table 3. Mean Baseline Heart Rate by PTSD Status, Gender, and Ethnicity | 33 |
| Table 4. Mean Baseline Systolic Blood Pressure by PTSD Status, Gender, and Ethnicity | 34 |
| Table 5. Mean Baseline Diastolic Blood Pressure by PTSD Status, Gender, and Ethnicity | 34 |
| Table 6. Mean Heart Rate Change Score During Anger Recall by PTSD Status, Gender, and Ethnicity | 35 |
| Table 7. Mean Systolic Blood Pressure Change Score During Anger Recall by PTSD Status, Gender, and Ethnicity | 36 |
| Table 8. Mean Diastolic Blood Pressure Change Score During Anger Recall by PTSD Status, Gender, and Ethnicity | 37 |

List of Figures

| | Page |
|---|------|
| Figure 1. Baseline heart rate as a function of Ethnicity, Gender, and PTSD status | 32 |

Abstract

THE INFLUENCE OF ETHNICITY AND GENDER ON THE RELATIONSHIP BETWEEN POSTTRAUMATIC STRESS DISORDER STATUS AND CARDIOVASCULAR RESPONDING

By Alison Eonta, B.A.

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

Virginia Commonwealth University, 2010

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

Past research has found inconsistent effects of posttraumatic stress disorder (PTSD) status on cardiovascular responding. Inconsistencies may be explained by demographic differences in study samples. In this study, the influence of gender and ethnicity on the relationship between PTSD status and cardiovascular responding was explored. Participants' (N = 245) heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) readings were taken throughout baseline and anger recall periods. For all gender by ethnicity groups, baseline HR was higher in participants with PTSD than without PTSD, except for Black men. Whites with PTSD had lower baseline SBP than Whites without PTSD; the opposite was true for Blacks. Men and Blacks with PTSD had larger HR increases during anger imagery than men and Blacks without PTSD, whereas women and Whites showed the opposite pattern. Results suggest demographic variables may account for inconsistent effects of PTSD on cardiovascular responding.

The Influence of Ethnicity and Gender on the Relationship Between Posttraumatic Stress Disorder Status and Cardiovascular Responding

Posttraumatic Stress Disorder (PTSD) is a serious condition that affects 6.8 – 7.8% of the population (National Center for Posttraumatic Stress Disorder, 2007; Resick, Monson, & Rizvi, 2008). To receive a diagnosis of PTSD, a person must have been exposed to a traumatic event and experienced a response that involved fear, helplessness, or horror. The traumatic event then must be reexperienced, and the person must try to avoid cues that are associated with the trauma. General responsiveness is numbed, and increased arousal is present post-trauma (American Psychiatric Association [APA], 2000). PTSD is a relatively new diagnosis, first appearing in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (APA, 1980). Since then, many studies have been conducted that attempt to delve into the causes and effects of this disorder. There is still a long way to go, however, before this disorder is fully understood, particularly its physiological effects in response to both everyday and trauma-related cues.

According to the DSM-IV-TR, one of the criteria for PTSD (Posttraumatic Stress Disorder) is increased physiological arousal (APA, 2000). Such arousal may take the form of higher resting heart rate (HR) and blood pressure (BP), or the arousal may be manifested in the presence of cues reminiscent of the original trauma. Increased cardiovascular arousal, over time, can lead to a number of health issues. Specifically, higher resting HR has been shown to be positively related to early death from cardiovascular disease (Greenland et al., 1999). Both PTSD status and having a higher average heart rate have been significantly associated with an increased number of physical health symptom complaints in male veterans (Beckham et al., 2003).

Overall, the literature shows some consistent findings regarding the physiology of PTSD, like an elevated resting heart rate and increased responsivity to trauma cues in people with PTSD. However, there are also inconsistencies in the literature for both BP and HR findings. It may be that some of these inconsistencies can be accounted for by demographic group differences in PTSD profiles and physiological responses. Men and women, and Blacks and Whites have different rates of exposure to trauma, suffer different types of trauma, and have different rates of PTSD. Additionally, there exist gender and ethnic differences in baseline physiology and physiological response to trauma cues. Unfortunately, it is difficult to pinpoint whether or not the ethnic and gender differences play a role in the inconsistencies because most studies do not examine results separately for different genders or ethnicities. In fact, many studies do not even report the ethnic compositions of their samples. Because PTSD affects people of all backgrounds, it is important to investigate in more depth the physiological symptoms of PTSD in different populations. In order to gain more insight into gender and ethnic differences in a PTSD sample, the current study will measure cardiovascular levels at baseline and in response to an anger elicitation task for males and females and Blacks and Whites with and without PTSD.

The literature review will first describe the basic gender and ethnic differences in physiological response that may contribute to the inconsistencies in PTSD group differences. Then, recent meta-analyses will be compared to explore the basic physiological trends that are present in people with and without PTSD. In order to ascertain possible effects of gender and ethnicity on the physiology of PTSD, the PTSD literature will be examined with particular attention to the gender and ethnic composition of the subject samples studied.

Literature Review

Ethnic and gender differences in physiological response. Because few studies exist that directly investigate differences in physiological responding between men and women or Blacks and Whites in a PTSD sample, basic physiological differences will be reviewed by ethnicity and gender. The literature suggests that there are ethnic differences in resting baseline blood pressure, but not heart rate. A large-scale (Ohira, Roux, Prineas, Kizilbash, Carnethon, & Folsom, 2008) study showed that no significant differences in resting heart rate were found among White, African-American, Hispanic, and Chinese participants. This resting HR finding was replicated in a smaller-scale sample of college students, but resting SBP was found to be higher in Blacks than in Latinos or Whites (Salomon & Jaguszyn, 2008). It is also alarming that Blacks have much higher death rates from high blood pressure (hypertension) than Whites, with Black males having the highest death rates of all from hypertension (American Heart Association, 2008). Most researchers attribute the higher prevalence of hypertension in African Americans to environmental factors (Kaufman & Hall, 2003).

In response to certain stimuli, the physiological responses of Blacks and Whites may differ. African-Americans, in comparison to Caucasians, may experience an enhanced physiological response to anxiety cues, especially cues that involve physical anxiety. Blood pressure responsivity, especially, is believed to be more sensitive in African-American participants than Caucasian participants. In a recent study that included an oral anger recall task, however, Caucasian participants showed greater heart rate reactivity to the task than did African-American participants (Richman, Bennett, & Pek, 2007).

There is also evidence that the racial context of tasks affects cardiovascular responding. It is thought that African Americans, from an early age, are taught to inhibit

their anger expression. On the other hand, White males tend to be conditioned to outwardly express anger, particularly when interacting with someone of equal or lesser perceived social status. Given this conditioning, it has been shown that Blacks experience elevated physiological reactions when asked to express anger outwardly, whereas White males show elevated physiological reactions when asked to inhibit anger. (Dorr, Brosschot, Sollers, & Thayer, 2007).

Given the literature, physiological differences in responsivity between Blacks and Whites are inconsistent and depend upon the nature of the task that participants complete. These inconsistencies occur even in nonclinical populations and highlight the importance of researching ethnic differences in physiological responsivity among clinical populations with PTSD.

Some basic physiological trends emerge when comparing the baseline heart rates of males and females. It is useful to review basic sex differences at baseline and in response to different cues, as these basic sex effects could translate into different responses to PTSD, like differences in reactivity. In a large study of over 6500 adults aged 45-84 who did not suffer from cardiovascular disease, female participants had significantly higher resting heart rates than male participants (Ohira et al., 2008). Even at younger ages, this difference appears to be present. In a recent community study of adolescents, male participants had significantly lower resting heart rate than female participants (Crozier et al., 2008). International studies have found similar results. In a Belgian study of 276 healthy participants, women had baseline higher heart rates than men by about five beats per minute (Ramaekers, Ector, Aubert, Rubens, & Van de Warf, 1998). A Finnish study with 224 participants also found

resting heart rates to be higher for women than for men (Piha, 1993). Overall, there seems to be agreement in the field that women have higher resting heart rates than men.

When participants were confronted with a laboratory task, however, physiological results were more mixed. Men had increased HR reactivity in response to a task where participants, prompted by a video, were asked to imagine themselves as victims (Crozier et al., 2008). In a mixed-ethnicity, mixed-gender sample, men had elevated heart rate responses to pain when asked to immerse their hand in very cold water (a task known as a “cold pressor”) (Myers, Robinson, Riley, & Sheffield, 2001). With other tasks, though, no significant gender differences could be found. In one study, male and female participants were assigned to either a disclosure or a control writing condition, and there were no physiological differences between genders in either condition (Epstein, Sloan, & Marx, 2005). There is some evidence that the “gender neutrality” of instructions delivered prior to a task can actually affect physiological responsivity to a task, whereby the more gender-relevant a task is to someone, the more stressful the task will seem (Lash, Gillespie, Eisler, & Southard, 1991). Also, males and females have been shown to have different physiological responses when expressing anger. White men, for instance, tend to be conditioned to outwardly express anger, while women have been taught that it is more socially acceptable to inhibit outward expressions of anger (Hokanson, Willers, & Koropsak, 1968; Bjorklund & Kipp, 1996). Because of these findings, male and female physiological responses may be somewhat dependent on the type of task being performed.

Men tend to have higher resting SBP and DBP than women, a finding that has been supported by many studies (e.g. Lawler, Wilcox, & Anderson, 1995; Maixner & Humphrey, 1993; Myers et al., 2001). In response to various tasks, men’s BP tends to elevate more than

women's BP. Men display an enhanced blood pressure response when presented with both pleasant and unpleasant arousing stimuli and when under stress, compared to women (Sarlo, Palomba, & Buodo, 2005). In a study that included an oral anger recall task, men also exhibited greater blood pressure reactivity than women (Lawler et al., 1995). Physical discomfort produces similar results. Men show greater blood pressure increases in response to pain than women do (Maixner & Humphrey, 1993; Myers et al., 2001).

Overall, it appears that women have higher resting HR but lower resting BP than men. When participants are confronted with different tasks and stimuli, however, physiological results fall less clearly along gender lines, though men's BP responses tend to be greater than women's.

The Psychophysiology of PTSD. There have been a number of recent meta-analyses and reviews regarding the psychophysiology of PTSD (Bedi & Arora, 2007; Buckley & Kaloupek, 2001; Pole, 2007). Cardiovascular response (especially HR) and electrodermal activity are the most studied physiological measures in PTSD research (Bedi & Arora, 2007). Because this study will examine cardiovascular responding due to the health implications of excess cardiovascular responding, this review will be confined to heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP).

Overall, people with PTSD tend to have higher resting HR than people without PTSD, though it was acknowledged that studies on resting baseline measures have yielded inconsistent results (Bedi & Arora, 2007; Buckley & Kaloupek, 2001; Pole, 2007). The Buckley and Kaloupek (2001) meta-analysis was devoted solely to baseline psychophysiological activity in people with PTSD. It contained thirty-four studies, and most of the 2670 participants were male combat veterans (Buckley & Kaloupek, 2001). Using a

weighted means effect size, HR was significantly elevated in people with PTSD compared both to controls without PTSD who had not experienced trauma ($d = .43$, 95% CI = .24-.62) and to controls without PTSD who had been exposed to trauma ($d = .36$, 95% CI = .27-.45) (Buckley and Kaloupek, 2001).

Pole's (2007) more recent meta-analysis included fifty-eight resting baseline studies but also looked at physiological responses to different tasks. Pole's (2007) meta-analysis included data from 3,424 adults and, as in Buckley and Kaloupek's (2001) article, most of the participants were males who had experienced military trauma. Using conservative weighted means effect sizes, HR was significantly higher in people with PTSD ($r = .18$; 95% CI = .13-.23) (Pole, 2007).

In most of the studies contributing to the meta-analyses described above, baseline cardiovascular measures were recorded prior to a manipulation in which subjects were exposed to some stimulus that served as a reminder of their trauma. One hypothesis for why people with PTSD exhibit elevated baseline cardiovascular activity is the presence of anticipatory anxiety prior to participation in a study. Knowing that they may be exposed to stimuli reminiscent of their trauma, some people with PTSD may have a higher-than-normal heart rate response during study participation, even the baseline phase (Bedi & Arora, 2007; Buckley & Kaloupek, 2001; Pole, 2007). The act of entering a lab or medical center could be considered a trauma cue (Gerardi, Keane, Cahoon, & Klauminzer, 1994).

To combat this potential effect, ambulatory monitoring studies have been conducted, during which people with and without PTSD are hooked up to devices that measure HR and/or BP for hours at a time, with readings being taken unobtrusively at given intervals. During these ambulatory studies, participants can go about their daily routines without the

need to come into the laboratory. Most of the ambulatory monitoring studies reviewed support the finding that people with PTSD have higher resting HR than people without PTSD (Muraoka, Carlson, & Chemtob, 1998; Buckley, Holohan, Greif, Bedard, & Suvak, 2004)), while one ambulatory monitoring study has yielded no baseline cardiovascular differences between PTSD and non-PTSD participants (Beckham et al., 2000).

In addition to having higher resting HR, people with PTSD tend to have higher HR responses to external stimuli that are either associated with the diagnostic criteria or reminiscent of their trauma. Most studies that investigate psychophysiological differences in PTSD share a similar format. In these laboratory studies, a baseline period is typically followed by a task designed to elicit a psychophysiological reaction. The stimuli presented in these tasks include startling sounds, standardized trauma cues, and personalized trauma cues (Bedi & Arora, 2007; Pole, 2007). This increased reactivity is reflected across many different types of trauma, including combat, motor vehicle accidents, and sexual assault (Bedi & Arora, 2007). In Pole's (2007) meta-analysis, people with PTSD had significantly higher HR responses to the presented stimuli than people without PTSD across 25 startle studies ($r = .23$; 95% CI = .12-.32), 17 standardized trauma cue studies ($r = .27$; 95% CI = .14-.39), and 22 personalized trauma cue studies ($r = .22$; 95% CI = .09-.35).

Blood pressure is another oft-studied component of cardiovascular responding. The differences in resting blood pressure between people with and without PTSD are not as strong as the HR differences. Pole's (2007) meta-analysis found that people with PTSD have elevated resting SBP ($r = .12$; 95% CI = .02-.21) and DBP ($r = .23$; 95% CI = .13-.32) when looking at unweighted mean effect sizes. In Buckley and Kaloupek's (2001) meta-analysis devoted solely to baseline psychophysiological responses in PTSD, weighted mean effect

sizes indicated that higher resting SBP ($d = .35$; 95% CI, .07-.63) and DBP ($d = .71$; 95% CI, .34-1.08) were found in people with PTSD compared to people without PTSD who had not experienced a traumatic event. When people with PTSD were compared to people without PTSD who had experienced trauma, however, only resting DBP ($d = .10$; 95% CI, .00-.21) was elevated in people with PTSD (Buckley & Kaloupek, 2001).

In response to traumatic cues like slides, sounds and scripts, greater BP responses were found in PTSD populations (Bedi & Arora, 2007). In the Pole (2007) meta-analysis, though, no blood pressure data was reported for the startle studies, and there were no significant differences in SBP or DBP responses when PTSD and non-PTSD samples were presented with standardized trauma cues. In response to personalized trauma cues, however, unweighted mean effect sizes showed that there were significantly higher DBP ($r = .49$; 95% CI, .02-.81) responses in people with PTSD than in people without PTSD, but there were no SBP differences between PTSD and non-PTSD groups (Pole, 2007).

In summary, the strongest findings supported by the meta-analyses are that people with PTSD have higher resting heart rates than people without PTSD, and that people with PTSD have elevated HR responses to trauma cues, compared to people without PTSD. There is some support for the assertion that resting BP levels are elevated for people with PTSD, but less support for the idea that BP responses in reaction to trauma cues are higher in people with PTSD compared to non-PTSD groups.

Gender.

Despite their common findings, the meta-analyses of both Pole (2007) and Buckley and Kaloupek (2001) both acknowledge that there are still inconsistencies in the literature. These inconsistencies may be due to differences in the gender composition of various studies.

In both meta-analyses, there were more male participants than female participants (Buckley & Kaloupek, 2001; Pole, 2007). Gender is a potentially important variable in the development and experience of PTSD. Traditionally, subjects in studies on PTSD were primarily male combat veterans, since the PTSD diagnosis was formulated in 1980 to reflect symptoms observed in combat veterans, particularly those returning from Vietnam (Kulka et al., 1990). In recent years, however, participants in PTSD-related studies have come from increasingly different backgrounds and have suffered diverse traumas.

An article by Tolin and Foa (2006) suggests that there are a number of important gender differences in PTSD. For example there are gender differences in the rate at which traumatic events are experienced, the type of traumas experienced, and diagnostic rates for PTSD. Specifically, women and girls were more likely than men and boys to meet diagnostic criteria for PTSD (Tolin & Foa, 2006), a finding supported by prevalence data from the National Center for PTSD, which reports that women are over twice as likely as men to suffer from PTSD during their lives (National Center for PTSD, 2007). On the other hand, men were more likely than women to report having suffered a traumatic event, leading to the question of whether men are experiencing less fear than women or if they are more culturally likely to deny feeling fear and anxiety. The answer to this question is not yet known, but it may have implications for the large gender difference in prevalence rates of PTSD (Tolin & Foa, 2006). There are also differences in the types of trauma experienced by men and women, with men tending to experience more trauma due to combat, and women experiencing more instances of sexual assault (Tolin & Foa, 2006).

As noted earlier, gender differences in baseline physiology and physiological responsivity exist even in the absence of PTSD. Women tend to have higher resting heart

rates and lower resting blood pressures than men, while findings on gender differences in cardiovascular responsivity to different stimuli are less consistent. Because of these existing differences, and the fact that all of the reviewed studies reported on the gender composition of their samples, gender serves as a natural way to categorize different studies.

All-male studies.

Consistent with the overall finding in the meta-analyses, most of the recent all-male laboratory studies support the finding that men PTSD have higher resting heart rates than men without PTSD (Keane et al., 1998; Orr, Lasko, Metzger, & Pitman, 1997; Orr, Metzger, Lasko, Macklin, Peri, & Pitman, 2000; Tarabrina, Lazebnaia, Zelenova, Lasko, Orr, & Pitman, 2001). In the all-male studies reviewed below, men came into the lab, were assessed for baseline HR, and completed tasks. These tasks included personalized trauma scripts (Tarabrina et al., 2001), non-startling tones (Orr et al., 1997), and self-selected levels of electrical shock (Orr et al., 2000). Sources of trauma in these studies were also varied, from mixed trauma samples (Orr et al., 2000) to clean-up experiences post-Chernobyl (Tarabrina et al., 2001), to combat (Orr et al., 1997). None of the all-male laboratory studies listed above reported the ethnic composition of their sample, except for one study (Tarabrina et al., 2001) that reported an all-Russian sample. An early Blanchard (1990) article also reviewed all-male laboratory studies and found that male veterans with PTSD displayed higher resting HR than men without PTSD, a result that led to warnings of increased risk of cardiovascular disease in the veteran population.

In one study, resting heart rate was collected during a routine hospital check-up. This was a departure from the laboratory studies in that there was no task to be completed. Male veterans with PTSD had higher heart rates than male veterans without PTSD (Gerardi et al.,

1994). Adding further credence to the baseline findings is a study looking at male monozygotic twin pairs (Orr et al., 2003). In each pair, one twin had served in combat in Vietnam, and the other twin had not been in combat and did not have PTSD. In about half of the twin pairs, the combat veteran twin had been diagnosed with current PTSD. At baseline, combat-exposed twins with PTSD had higher resting heart rates than the non-combat twins without PTSD (Orr et al., 2003). These differences could be due to PTSD status or combat exposure. As was mentioned earlier, some all-male ambulatory monitoring studies also support the finding that men with PTSD have elevated resting HR compared to people without PTSD (Muraoka et al., 1998; Buckley et al., 2004). It should also be kept in mind that most participants in the meta-analyses were male (Buckley & Kaloupek, 2001; Pole, 2007).

Though most of the all-male studies reviewed were consistent with the findings of the meta-analyses, a few all-male laboratory-based studies did indicate that men with PTSD had equivalent or lower resting heart rates than men without PTSD. Most trauma studies involving male veterans have relied on Vietnam veterans. In one such study that reported the ethnic composition of its sample, male Vietnam combat veterans had slightly but not significantly higher baseline HR than their counterparts without PTSD (Litz, Orsillo, Kaloupek, & Weathers, 2000). In another study, males with PTSD had lower resting heart rates than male combat veterans without PTSD, though this difference was not significant (Pitman, Orr, Fogue, de Jong, & Claiborn, 1987). Another study showed that anxious male veterans had insignificantly higher resting HR than male veterans with PTSD (Pitman, Orr, Fogue, Altman, de Jong, & Herz, 1990). In another study, World War II and Korean combat veterans with and without PTSD – an all white, male sample – were found to have nearly

equivalent resting heart rates (Orr, Pitman, Lasko, & Herz, 1993). One all-male ambulatory monitoring study reports no mean cardiovascular differences between men with and without PTSD at baseline (Beckham et al., 2000). It should be noted that the Beckham et al. (2000) study contains some of the same male participants that took part in the current study.

Only a few all-male studies gathered baseline blood pressure data. During the Gerardi et al. (1994) study, male veterans with PTSD who visited a medical center had higher SBP and DBP levels than a comparable group of male veterans without PTSD. This was consistent with the findings of the meta-analyses. According to the literature, it would be expected that both resting HR and BP would be higher for men with PTSD. In a twenty-four hour ambulatory monitoring study, however, SBP was only slightly higher for men with PTSD than for men without PTSD, and DBP levels were nearly identical between groups (Buckley et al, 2004) (see Table 1 for information about PTSD studies that reported baseline cardiovascular findings).

In line with the findings of the meta-analyses, heart rate increased in response to traumatic stimuli like personalized combat scripts (Orr et al., 1993) for white male veterans with PTSD. The distinction between traumatic and non-traumatic stimuli is not always so clear-cut, however. No difference in heart rate response was found between male Vietnam combat veterans with and without PTSD during an individualized script task where men were asked to recall a stressful (but not traumatic) memory (Pitman et al., 1987). Although a traumatic memory certainly has stressful elements, there may be a different physiological reaction to very trauma-specific cues, as opposed to cues that are merely stressful, or that evoke other types of emotional reactions. Overall, the clearest differences in physiological

Table 1.

PTSD Studies That Report Baseline Physiological Findings

| Study Reference | N | Baseline Measures Reported | Gender Reported? | Ethnicity Reported? | Effects of Gender and Ethnicity on PTSD Reported? |
|------------------------|------|----------------------------|-------------------------|--------------------------------------|---|
| Blanchard et al., 1996 | 159 | HR, SBP | 31% Male; 69% Female | 92% White; 8% non-White | No |
| Bremner et al., 2003 | 41 | HR, SBP, DBP | 63% Male; 37% Female | No | Yes; Males and Females Compared |
| Buckley et al., 2004 | 36 | HR, SBP, DBP | 100% Male | 75% White; 25% non-White | No |
| Carson et al., 2000 | 38 | HR, SBP, DBP | 100% Female | No | No |
| Carson et al., 2007 | 128 | HR | 100% Female | No | No |
| Casada & Roache, 2006 | 23 | HR | 57% Male; 43% Female | No | No |
| Cohen et al., 2000 | 50 | HR | 40% Male; 60% Female | No | No |
| Cuthbert et al., 2003 | 130 | HR | 38% Male; 62% Female | “Predominantly Caucasian” | No |
| Elsesser et al., 2004 | 86 | HR | 48% Male; 52% Female | No | No |
| Forneris et al., 2004 | 92 | HR, SBP, DBP | 100% Female | 53% White; 39% Black; 8% Other | No |
| Gerardi et al., 1994 | 58 | HR, SBP, DBP | 100% Male | No | No |
| Griffin, 2008 | 40 | HR | 100% Female | 33% White; 63% Black; 4% Other | No |
| Halligan et al., 2006 | 61 | HR | 54% Male; 46% Female | No | No |
| Keane et al., 1998 | 1328 | HR, SBP, DBP | 100% Male | 9% Hispanic | No |
| Kellner et al., 2003 | 34 | HR, SBP, DBP | 35% Male; 65% Female | No | No |
| Litz et al., 2000 | 61 | HR | 100% Male | 82% White; 11% Black; 5% Hispanic | No |
| Metzger et al., 1999 | 57 | HR | 100% Female | No | No |

Table 1 (continued)

PTSD Studies That Report Baseline Physiological Findings

| Study Reference | N | Baseline Measures Reported | Gender Reported? | Ethnicity Reported? | Effects of Gender and Ethnicity on PTSD Reported? |
|------------------------------|-----|----------------------------|----------------------|---|---|
| Muraoka et al., 1998 | 18 | HR, SBP, DBP | 100% Male | 61% White; 39% non-White | No |
| Newton et al., 2005 | 39 | HR, SBP, DBP | 100% Female | 84% White; 10% Black; 3% Hispanic; 3% Asian | No |
| Orr et al., 1993 | 20 | HR | 100% Male | 100% White | No |
| Orr et al., 1995 | 56 | HR | 100% Male | No | No |
| Orr et al., 1997 | 39 | HR | 100% Male | No | No |
| Orr, Lasko, et al., 1998 | 71 | HR | 100% Female | No | No |
| Orr, Meyerhoff, et al., 1998 | 35 | HR, SBP, DBP | 100% Male | No | No |
| Orr et al., 2000 | 33 | HR | 100% Female | No | No |
| Peri et al., 2000 | 86 | HR | 76% Male; 24% Female | No | No |
| Pitman et al., 1987 | 33 | HR | 100% Male | 100% White | No |
| Pitman et al., 1990 | 14 | HR | 100% Male | 100% White | No |
| Pitman et al., 2001 | 37 | HR | 100% Female | No | No |
| Schmahl et al., 2004 | 40 | HR, SBP, DBP | 100% Female | 76% White; 13% Black; 8% Hispanic; 3% Asian | No |
| Tarabrina et al., 2001 | 39 | HR | 100% Male | 100% Russian | No |
| Veazey et al., 2004 | 132 | HR | 27% Male, 73% Female | 89% White, 11% non-White | No |
| Wessa et al., 2006 | 47 | HR | 77% Male; 23% Female | 100% German | No |

responding between men with and without PTSD occur in the presence of trauma-related cues.

All-female studies.

Most all-female studies have supported the findings of all-male studies (Carson, Paulus, Lasko, Metzger, Wolfe, Orr, & Pitman, 2000; Forneris, Butterfield, & Bosworth, 2004). Samples of all-female veterans (Forneris et al., 2004) and Vietnam-era nurses (Carson et al., 2000) with PTSD had higher resting HR than female veterans or nurses without PTSD. Similar findings were also supported by a number of all-female studies that included three categories: current PTSD, lifetime or past PTSD, and never had PTSD (Metzger, Orr, Berry, Ahern, Lasko, & Pitman, 1999; Orr, Lasko, Metzger, Berry, Ahern, & Pitman, 1998; Pitman et al., 2001).

In a study of women with a self-reported history of child abuse, the resting heart rates of women with current PTSD were higher than the resting HR of women with lifetime but not current PTSD, which were higher than the resting HR of women who had never had a PTSD diagnosis (Metzger et al., 1999). The directionality of the results were consistent with the Pitman et al. (2001) study, in which female breast cancer patients with current PTSD had the highest resting heart rates, and women who had never had PTSD had the lowest resting heart rates. The same directionality was found in the Orr, Lasko, et al. study (1998), where all women had a self-reported history of child abuse. Here, the resting HR of women with PTSD was also higher than the resting HR of women who had never had PTSD (Orr, Lasko, et al., 1998).

In another study with these three categories, however, the results for resting HR did not follow the same pattern as the previous studies. All participants were female Vietnam

nurse veterans, and women with past PTSD actually had higher resting HR levels than women with current PTSD. In line with standard findings, however, female nurses with current PTSD still had higher baseline HR than female nurses who had never had PTSD (Carson et al., 2007).

The severity of PTSD may also play a role in physiology. Using an all-female sample with a history of criminal victimization, one 18-hour ambulatory monitoring study showed a positive relationship between increased PTSD symptomatology and elevated heart rate (Newton, Parker, & Ho, 2005).

A few all-female studies found opposite results, however. In a sample composed entirely of physical and/or sexual abuse survivors, women who had suffered abuse but had not developed PTSD actually had higher resting heart rates than women with PTSD (Schmahl et al., 2004). Likewise, another study consisting of physical assault and rape survivors found the same pattern, with women with PTSD having lower baseline HR than women without PTSD (Griffin, 2008).

Few all-female studies were found that collected baseline systolic and diastolic blood pressure in PTSD and non-PTSD samples. Forneris et al. (2004) found no significant differences in blood pressure between female veterans with and without PTSD, though the female veterans with PTSD had slightly lower SBP and DBP, in contrast to traditional findings. No significant differences in SBP or DBP were found by Schmahl et al. (2004) either, though pre-script baseline DBP was higher for abused control women than for women with PTSD.

More all-female studies reported on ethnicity than did all-male or mixed-gender studies. The ethnic composition of an aforementioned all-female ambulatory monitoring

study was predominantly Caucasian (Newton et al., 2005), as was the Schmahl et al. (2004) study investigating responses to neutral, abandonment, and trauma scripts in women with a history of physical and/or sexual abuse. About half of the female veterans in the Forneris et al. (2004) study were Caucasian, and nearly 40% were African-American. The Griffin (2008) study is unique in that over half of the participants were African American. Interestingly, this is also a study that does not fit the typical baseline heart rate pattern. This suggests that there may be a connection worth investigating between the ethnic composition of a study and the baseline heart rate findings. Even in the studies that did state the ethnic composition of their samples, though, physiological differences between ethnic groups were not reported (see Table 1 for information about PTSD studies that reported baseline cardiovascular findings).

Cardiovascular responsivity findings in all-female studies supported the findings of the meta-analyses. Women with PTSD had enhanced HR responses to traumatic cues like startling tones (Carson et al., 2007), personalized nursing trauma scripts (Carson et al., 2000), and breast cancer trauma scripts (Pitman et al., 2001). Women with PTSD also showed a greater SBP response to traumatic scripts than to neutral or abandonment scripts (Schmahl, 2004).

Mixed-gender studies.

Only one study could be found in the literature that directly compared the psychophysiological data of men and women with PTSD. In a mixed-gender study that examined heart rate and blood pressure differences in PTSD sufferers who had been abused as children, it was found that HR was significantly higher for women during a series of cognitive challenges and subsequent recovery period than for men. There was no significant

interaction involving gender and PTSD status, however. In the same study, there had been no significant PTSD-status differences found in HR or BP before, during, or after the cognitive challenge, and there was also no gender difference in BP responding (Bremner et al., 2003).

Resting heart rate results appeared to be less consistent for studies that included a mixed-gender sample, though most studies are consistent with the findings of the meta-analyses. In samples ranging from victims of motor vehicle accidents (Blanchard, Hickling, Buckley, Taylor, Vollmer, & Loos, 1996) to witnesses of an airshow disaster (Wessa, Jatzko, & Flor, 2006) to mixed trauma samples (Cohen, Benjamin, Geva, Matar, Kaplan, & Kotler, 2000; Cuthbert, Lang, Strauss, Drobles, Patrick, & Bradley, 2003; Peri, Ben-Shakhar, Orr, & Shalev, 2000), most studies upheld the finding that PTSD-status is associated with higher resting heart rates.

As noted earlier, though, a number of mixed-gender studies do not follow the typical baseline heart rate pattern. For a number of studies, baseline heart rate was higher in people who did not have PTSD than people who do have PTSD (Veazey, Blanchard, Hickling, & Buckley, 2004; Elsesser, Sartory, & Tackenberg, 2004; Halligan, Michael, Wilhelm, Clark, & Ehlers, 2006) or nearly equivalent between PTSD and non-PTSD groups (Kellner, Yassouridis, Hubner, Baker, & Wiedemann, 2003). This is a direct contradiction to the findings of the meta-analyses and the all-male studies. Samples targeted in these mixed-gender studies included motor vehicle accident survivors (Veazey et al., 2004), victims of physical and sexual assault (Halligan et al., 2006), and mixed traumatic samples (Elsesser et al., 2004). Most of the studies followed a traditional format, with a baseline period followed by tasks such as self-generated trauma recall (Halligan et al., 2006), listening to a

personalized audiotaped trauma script (Veazey et al., 2004), and completing dot-probe and startle tasks (Elsesser et al., 2004).

Few mixed-gender studies collected blood pressure data. Consistent with the meta-analyses, people who had developed PTSD after a motor vehicle accident had higher resting SBP than people who had been in motor vehicle accidents but had not developed PTSD. No DBP was reported in this study. (Blanchard et al., 1996). In another study, though, experimenters administered corticotropin-releasing hormone to a relatively small mixed-gender sample of people with and without PTSD while measuring baseline HR, DBP, and SBP. The only significant finding was that people with PTSD had higher resting SBP levels (Kellner et al., 2003).

As in the all-male studies, few mixed-gender studies provided information about the ethnic makeup of the sample. The only two mixed-gender studies to report the ethnic composition of their samples investigated psychophysiological responding in motor vehicle accident survivors at baseline (Blanchard et al., 1996; Veazey et al., 2004). Following baseline, participants in both studies completed a mathematics task and listened to two personalized audiotapes related to motor vehicle accidents, while participants in the Blanchard et al. (1996) study also watched a standardized videotape depicting car crashes. In both articles, the samples were predominantly white and female across all study groups. (Blanchard et al., 1996; Veazey et al., 2004). To allow for accurate characterization of samples, it would be helpful if studies began specifying the ethnic composition of their samples (see Table 1 for information about PTSD studies that reported baseline cardiovascular findings).

As mentioned in the meta-analyses, one of the most consistent findings in the literature is that people with PTSD tend to display increased cardiovascular reactions to trauma-related stimuli. In one part of a study by Elsesser et al. (2004), participants were shown trauma-related pictures while heart rate was recorded. People with PTSD and participants who had recently experienced a trauma had accelerated heart rate responses while healthy controls showed deceleration in their heart rates (Elsesser et al., 2004). Another recent study included a task in which motor vehicle accident survivors listened to personalized trauma scripts, during which time the group with PTSD had significantly more reactive heart rates than the group without PTSD or the subsyndromal group (Veazey et al., 2004).

This finding is not uniform, however. In one mixed-gender study, assault victims were asked to vividly imagine their trauma while giving a verbal narrative about their trauma. The group without PTSD actually displayed higher heart rates than the group with PTSD (Halligan et al., 2006). The authors attributed this finding to possible differences between cue-driven trauma tasks and intentional, self-generated trauma recall tasks and proposed a number of other factors that might limit the physiological responses of people with PTSD. People with PTSD may have more difficulty recalling traumatic memories in an intentional, organized way than non-PTSD controls. PTSD sufferers who exhibit dissociation may also show decreased physiological responsivity. Finally, excessive rumination over past events may also inhibit physiological responses (Halligan et al., 2006).

When confronted with non-trauma stimuli, people with PTSD and control groups generally tend to have similar responses (Casada & Roache, 2006). When asked to complete twenty minutes of cognitively challenging tasks, for example, participants with PTSD and a

control group showed no significant differences in HR or BP before, during, or after the cognitive stressor (Bremner et al., 2003).

Ethnicity.

There may be ethnic differences in the development, maintenance, and experience of PTSD. Ford (2008) suggests that the racism ethnic minorities face may lead to an increased risk for PTSD, although the exact mechanism of this risk is unclear. For example, racism may act as a risk factor for exposure to trauma, it may be a moderator variable that can make the effects of psychological trauma worse, thus increasing the risk for PTSD, or it could be a direct form of psychological trauma (Ford, 2008). These ideas are supported in Pole's (2007) meta-analysis, which suggests that African Americans may have higher rates of exposure to traumatic stress than Caucasians, and that perceived racial discrimination may be related to more severe PTSD symptoms.

In the Buckley et al. (2004) ambulatory monitoring study, it was shown that minority status in people with PTSD was associated with significantly higher SBP and DBP, and with lower HR than minority status in people without PTSD. Unfortunately, the label "minority status" does not specify exactly which minority groups were represented in the study. Additionally, physiological differences related to minority status were examined using the entire sample, and not in a manner that compared people of minority status with PTSD to people of minority status without PTSD (Buckley et al., 2004). In the Beckham et al. (2003) ambulatory monitoring study, ethnicity was not significantly related to mean HR and BP levels.

As noted in the Introduction, some basic ethnic differences in physiological responding exist, especially as BP is concerned. Though no studies exist that specifically

compare the physiological baselines and responses of African-Americans with PTSD to Caucasians with PTSD, it is logical to think that some of the aforementioned basic ethnic differences in physiology may apply to people with PTSD as well. This could affect results without researchers being aware of it if ethnic data is not reported.

Statement of the Problem

Recent meta-analyses and studies have shown some consistency in their physiological findings. People with PTSD tend to have higher resting heart rates than people without PTSD. This finding seems more pronounced in all-male and all-female studies than in mixed-gender studies. Research on basic gender effects has shown that women tend to have higher HR and lower BP than men, a finding that could be influencing physiological findings in PTSD populations as well. Elevated responses to traumatic cues are also present in people with PTSD, though there do not seem to be clear-cut response differences between people with and without PTSD in response to non-traumatic cues. Though results are more mixed than with baseline HR, resting BP also appears to be elevated in people with PTSD. Comparisons of PTSD to non-PTSD groups' physiological responses to traumatic cues have yielded inconsistent results for SBP and DBP.

Overall, though, very little research exists on gender or ethnic differences in physiological responsivity or baseline differences in populations with PTSD. Of the studies reviewed, only one directly compared the psychophysiological data of men and women with PTSD (Bremner et al., 2003). No studies were found that directly compared the cardiovascular data of Blacks and Whites with PTSD. In order to more fully understand the physiology of PTSD, research must examine the response of different groups. PTSD in and of itself is a debilitating disease, but its potentially adverse effects on blood pressure and

heart rate responses are also worrisome. Because PTSD is such a widespread, serious problem for a diverse range of people, it will be important to conduct focused research regarding the gender and ethnic differences that may be present in cardiovascular responding in people with PTSD. In the current study, differences in cardiovascular responding will be examined at baseline and in response to an anger elicitation task for male and female participants, and for Blacks and Whites, with and without a PTSD diagnosis. This will allow for direct comparisons of physiological results between genders and between ethnic groups within the same study and using the same research methodology. This type of direct comparison has not been done before in the PTSD literature, making this study an important advance in the literature.

Given the existing literature, the researcher expects to find that differences in gender, ethnicity, and PTSD status will elicit differences in heart rate, systolic blood pressure, and diastolic blood pressure levels at baseline and in response to an anger recall task. Specifically, the researcher predicts that participants with PTSD will have higher baseline HR, SBP, and DBP and greater HR response to anger than participants without PTSD. The researcher also expects to find the typical demographic differences in cardiovascular responses: women will have higher baseline HR than men; men will have higher baseline BP and greater BP responses to anger than women; and Blacks will have higher baseline SBP and DBP than Whites. However, the main purpose of the study is to explore how effects of PTSD may differ between gender and ethnic groups (i.e., interactions of PTSD with gender and ethnicity). Due to lack of information and inconsistencies in the literature, interactions of gender, PTSD, and ethnicity will be exploratory in nature.

Method

Participants

Participants were men with (n = 61) and without PTSD (n = 56) and women with (n = 61) and without PTSD (n = 67). Of the total number of participants (N = 245), there were 29 Black men and 31 Black women with PTSD. Non-PTSD participants included 19 Black males and 33 Black females. Participants' ages ranged from 20 to 70 years, while the average age was 44.8 years. Additional participant demographics appear in Table 2. All participants in the current study had been recruited to take part in larger studies on health and hostility. Approval was sought from the appropriate review boards, and all who partook in the study provided informed consent and were compensated for their participation.

Male participants were all Vietnam combat veterans recruited in Durham, North Carolina at the Veterans Affairs Medical Center (VAMC). All male participants reported combat as their main trauma. Female participants were not necessarily veterans, and were recruited through the Durham VAMC, Women's Health Clinic and Women's Mental Health Clinic, and the Duke University Medical Center Anxiety Disorder Clinic. Of women with PTSD, 61% reported that their main trauma was physical or sexual assault; the death of a loved one was the main trauma for 23% of women with PTSD; and 16% of women with PTSD named other types of trauma as their main trauma. In the non-PTSD group, 85% of women reported that they had experienced a traumatic event. Of these women without PTSD who had experienced a trauma, 42% reported that their main trauma was the death of a loved one; 39% reported that their main trauma was physical or sexual assault; and 19% reported other types of trauma as their main trauma. All participants filled out questionnaires to provide information about gender, ethnicity, behaviors like smoking and substance abuse,

Table 2.

Demographic Information by Group

| Demographics | PTSD | | | | Non-PTSD | | | |
|---|-------|-------|--------|-------|----------|-------|--------|-------|
| | Male | | Female | | Male | | Female | |
| | Black | White | Black | White | Black | White | Black | White |
| N | 29 | 32 | 31 | 30 | 19 | 37 | 33 | 34 |
| Age | 49.66 | 48.91 | 39.39 | 44.1 | 49.53 | 50.7 | 41.27 | 36.88 |
| Education | 13.79 | 13.94 | 13.52 | 15.3 | 14.42 | 15.76 | 14.21 | 16.94 |
| SES | 51.45 | 51.31 | 49.13 | 44.30 | 42.68 | 31.38 | 44.94 | 30.00 |
| % Married | 75.9% | 71.9% | 29.0% | 40% | 78.9% | 89.2% | 30.3% | 41.2% |
| % Employed | 58.6% | 37.5% | 58.1% | 60% | 84.2% | 83.8% | 78.8% | 85.3% |
| % Veteran | 100% | 100% | 22.6% | 30% | 100% | 100% | 9.1% | 11.8% |
| % Current smoker | 41.4% | 56.3% | 38.7% | 33.3% | 26.3% | 18.9% | 21.2% | 11.8% |
| % with History of Substance/Alcohol Abuse | 69.0% | 53.1% | 35.5% | 50% | 31.6% | 29.7% | 12.1% | 23.5% |
| % with Current Major Depression | 10.3% | 9.4% | 51.6% | 56.7% | 5.3% | 0% | 30.3% | 26.5% |
| % with Current Anxiety | 24.1% | 15.6% | 32.3% | 53.3% | 5.3% | 0% | 27.3% | 17.6% |
| % on Hypertensive Medication | 37.9% | 6.3% | 22.6% | 16.7% | 42.1% | 16.2% | 18.2% | 2.9% |
| % on Beta Blockers | 10.3% | 15.6% | 16.1% | 10.0% | 5.3% | 8.1% | 12.1% | 0% |
| % on Alpha Andrenergics | 27.6% | 25% | 12.9% | 6.7% | 0.0% | 2.7% | 0% | 0% |
| % on Anticholinergics | 6.9% | 9.4% | 3.2% | 0.0% | 0.0% | 2.7% | 0% | 0% |

current and past mental health diagnoses, current medications, and socioeconomic status, among other topics (Beckham et al., 2000).

Male participants filled out the Combat Exposure Scale (CES; Keane, Fairbank, Caddell, Zimering, Taylor, & Mora et al., 1989) and the Mississippi PTSD Scale (Keane, Caddell, & Taylor, 1988). All male participants completed the Structured Clinical Interview for DSM-III-R criteria (SCID; Spitzer, Williams, Gibbons, & First; 1989) for Axis I disorders. Female participants, whose data were collected at a later time period, completed the Clinician Administered PTSD Scale (CAPS; Blake, Weathers, Nagy, Kaloupek, Fusman, & Charney, 1995) and the SCID for DSM-IV-TR criteria (First, Spitzer, Gibbon & Williams, 1997). All PTSD diagnoses were based on the SCID.

Apparatus

In this study, participants' heart rate and blood pressure data were collected using an Ohmeda Finapres Blood Pressure Monitor (Model 2300). This device simultaneously measured blood pressure and heart rate (Vrana, Hughes, Dennis, Calhoun, & Beckham, 2009).

Design and Procedure

Participants were seen individually in a Veterans Affairs Medical Center laboratory over the course of three sessions. In the first session, participants provided consent, completed the appropriate SCID, and filled out a number of demographic, hostility, and health-related questionnaires.

In the second session, participants were asked to sit in a comfortable chair and the middle finger of the non-dominant hand was hooked up to the Finapres finger cuff. Heart rate and blood pressure were monitored continuously on a beat-by-beat basis for all

participants throughout the study (Beckham, Vrana, Barefoot, Feldman, Fairbank, & Moore, 2002). First, participants sat quietly for ten minutes to obtain baseline readings. After the baseline period, all participants read a ninety-second neutral passage aloud to provide a baseline condition to which the upcoming anger verbalization stage could be compared. The reading was followed by two minutes of recovery data.

Participants were then asked to identify and recall a personal anger memory. The recalled situation was to be a time when they felt “really angry, frustrated, or upset with another person, and were not happy with the outcome” (Beckham et al., 2002). Once participants identified a relevant anger memory, they were prompted by the experimenter to give more details about it. In all, the anger memory was discussed for at least five minutes. After retrieving this personal anger memory, participants rated both their current anger and their anger at the time of the situation using a zero to ten scale. Afterwards, five minutes of post-anger recovery data were collected.

In the third session, participants sat in the comfortable chair again, and the Finapres was attached. Ten minutes of baseline psychophysiological measures were collected. After this ten-minute baseline period, the stimulus videotape began providing instructions for the rest of the session. Participants were told to relax and clear their minds while an “X” was displayed on the TV screen and the two-minute pre-anger baseline measures were taken. Next, participants were asked to imagine a time when they felt so angry that they wanted to explode. This memory had been pre-selected during the second session, so the participant was, presumably, easily able to recall the memory. During this anger recall task, participants were asked to raise a finger when they began to actually feel anger as they were recalling this memory. Cardiovascular responses to this anger recall task were recorded during a twenty-

second period seconds after the participants indicated feeling angry by raising a finger. At this point, a ninety-second clip of ocean waves came on the screen. The ocean waves served as a cue for participants to stop reliving their anger. After the ocean waves clip, participants looked at a blank screen for five minutes while post-anger data was collected (Beckham et al., 2002; Runnals, 2006; Vrana et al., 2009).

Data Analysis Plan

The current study examined the group differences in cardiovascular responses at baseline and during an anger recall task. Heart rate, SBP, and DBP were analyzed in a series of separate 2 PTSD status (PTSD vs. non-PTSD) X 2 Gender (Male vs. Female) X 2 Ethnicity (Black vs. White) between-subjects Analyses of Variance (ANOVAs) or Analyses of Covariance (ANCOVAs). Pairwise comparisons were conducted to determine the directionality of all significant two-way and three-way interaction findings. An overall alpha level of .05 was used for all statistical tests. Post-hoc analyses used the Simes (1986) modification of the Bonferroni correction.

For HR, SBP, and DBP, “baseline” was defined as the initial ten-minute baseline period of data collection in the third session. The “change score during anger recall” refers to the difference between the two-minute pre-anger baseline period and the period of twenty seconds directly after participants raised a finger to indicate that they felt angry. The six dependent variables being investigated are baseline heart rate, the heart rate change score during the anger recall task, baseline systolic blood pressure, the systolic blood pressure change score during the anger recall task, baseline diastolic blood pressure, and the diastolic blood pressure change score during the anger recall task.

In investigating the impact of PTSD status, gender, and ethnicity on cardiovascular responding, it is important to take additional variables into account to see if they are accounting for the observed effects. In order to do this, independent-samples t-tests were conducted to see if there were significant differences in cardiovascular responding for the following categorical variables: smoking status, marriage status, veteran status, employment status, current major depressive disorder status, current anxiety disorder status, history of substance or alcohol abuse status, and current use status of hypertensives, beta blockers, alpha adrenergics, and anticholinergics. Similarly, correlation analyses were used to determine the strength of the relationship between each of the cardiovascular response variables and each of the following continuous variables: age, education level, and socioeconomic level.

For any t-tests that were found to be significant ($p < .05$) or any correlations of medium to large strength ($r > .30$), the variables were added as covariates to the PTSD X Gender X Ethnicity analyses of variance (ANOVAs) that were run. This strategy was in line with the recommendations of experts in the field of PTSD research (Keane & Kaloupek, 1997).

Results

Analyses of Variance and Covariates

In order to test hypotheses and explore potential interactions, a series of ANOVAs and ANCOVAs were run (see Tables A1 through A6). Independent-samples t-tests and correlational analyses were conducted to assess potential covariates for analyses of cardiovascular responding. The results indicated that current major depressive disorder status and the use of beta blockers were significantly related to baseline HR and HR response

to anger; marriage status, history of substance or alcohol abuse, and veteran status all significantly affected baseline DBP; and veteran status significantly impacted SBP response to anger. These variables were used as covariates in the analyses reported below.

Baseline HR

Despite initial predictions, there were no significant differences in baseline HR between people with and without PTSD, $F(1, 235) = .21, p = .65$; between men and women, $F(1, 235) = .01, p = .93$; or between Blacks and Whites, $F(1, 235) = .14, p = .71$.

There was a significant PTSD status X Gender effect, $F(1, 235) = 6.24, p = .01$: women with PTSD had slightly but not significantly higher baseline HR than women without PTSD, $F(1, 235) = 2.14, p = .15$. Men actually showed the opposite pattern, as men with PTSD had lower HR than men without PTSD, $F(1, 235) = 4.10, p = .04$.

Baseline heart rate as a function of PTSD status was affected by both gender and ethnicity, PTSD x Gender x Ethnicity $F(1, 235) = 7.16, p = .01$. These data are presented in Figure 1. To follow up on this three-way interaction, baseline HR was compared for participants with and without PTSD for each Ethnicity x Gender group. Data were in the expected pattern of higher baseline HR in participants with PTSD compared to those without PTSD for Black women, $F(1, 235) = 2.90, p = .09$; White women, $F(1, 235) = 0.16, p = .67$; and White men, $F(1, 235) = 0.36, p = .55$. However, Black men with PTSD actually had significantly *lower* baseline HR than Black men without PTSD, $F(1, 235) = 9.72, p < .01$ (see Table 3 for group means and Table A1 for ANCOVA table).

Figure

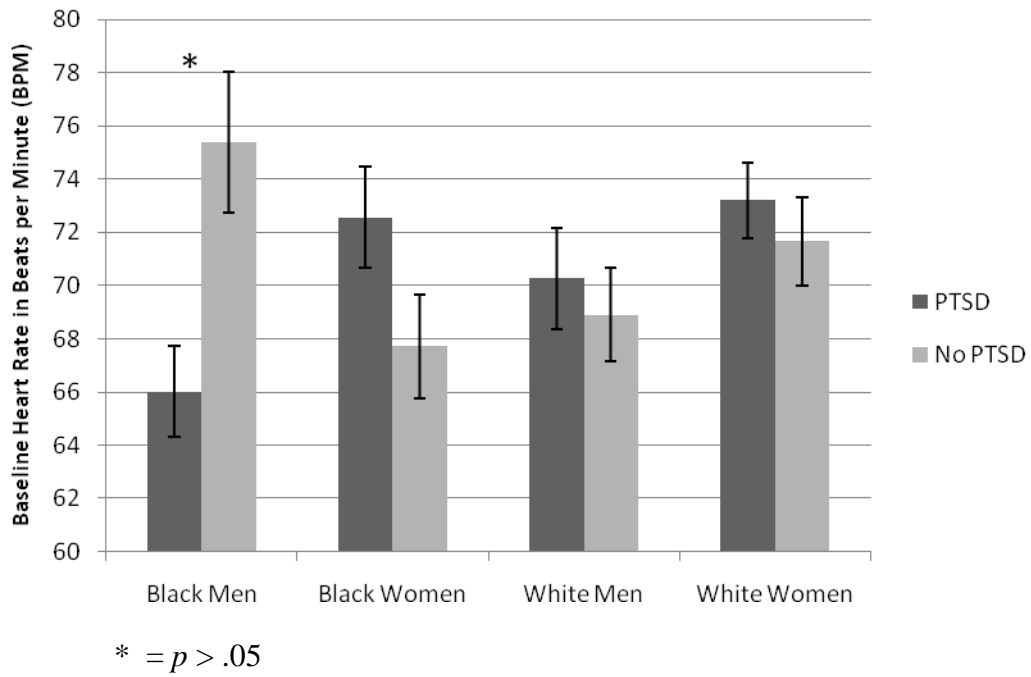


Figure 1. Baseline heart rate as a function of Ethnicity, Gender, and PTSD status.

Table 3.

Mean Baseline Heart Rate by PTSD Status, Gender, and Ethnicity

| | PTSD | | | No PTSD | | |
|-------|-------------|-------------|-------------|-------------|-------------|-------------|
| | Male | Female | Total | Male | Female | Total |
| Black | 66.0 (9.1) | 72.6 (10.6) | 69.4 (10.4) | 75.4 (11.5) | 67.7 (11.1) | 70.5 (11.7) |
| White | 70.3 (10.8) | 73.2 (7.8) | 71.7 (9.5) | 68.9 (10.7) | 71.7 (9.7) | 70.2 (10.3) |
| Total | 68.2 (10.2) | 72.9 (9.3) | 70.6 (10.0) | 71.1 (11.3) | 69.7 (10.5) | 70.3 (10.9) |

Note. Values enclosed in parentheses indicate Standard Deviations.

Baseline SBP

No significant differences in baseline SBP were found between people with and without PTSD, $F(1, 237) = 0.14, p = .71$, contrary to the original prediction that people with PTSD would have higher baseline SBP. Contrary to the original hypothesis, women had slightly but not significantly higher baseline SBP than men, $F(1, 237) = 3.53, p = .06$. In line with the original hypothesis, Blacks displayed higher SBP compared to White participants' baseline SBP, $F(1, 237) = 8.06, p = .01$.

PTSD status was related to baseline systolic blood pressure differently for Blacks and Whites, PTSD X Ethnicity $F(1, 237) = 5.84, p = .02$. Blacks showed the anticipated pattern in that participants with PTSD had slightly but not significantly higher baseline SBP than participants without PTSD, $F(1, 237) = 1.90, p = .17$. Whites with PTSD actually had significantly *lower* baseline SBP than Whites without PTSD, $F(1, 237) = 4.34, p = .04$ (see Table 4 for group means and Table A2 for ANOVA table).

Table 4.

Mean Baseline Systolic Blood Pressure by PTSD Status, Gender, and Ethnicity

| | PTSD | | | No PTSD | | |
|-------|--------------|--------------|--------------|--------------|--------------|--------------|
| | Male | Female | Total | Male | Female | Total |
| Black | 131.8 (15.5) | 140.1 (25.0) | 136.1 (21.1) | 129.1 (16.2) | 132.7 (18.8) | 131.4 (17.9) |
| White | 120.9 (15.4) | 125.1 (18.0) | 122.9 (16.7) | 128.6 (20.2) | 131.1 (19.8) | 129.8 (19.9) |
| Total | 126.1 (16.1) | 132.7 (23.0) | 129.4 (20.0) | 128.8 (18.8) | 131.9 (19.2) | 130.5 (19.0) |

Note. Values enclosed in parentheses indicate Standard Deviations.

Baseline DBP

Contrary to the original hypothesis, there were no significant differences between participants with and without PTSD for baseline DBP, $F(1, 234) = .35, p = .55$. Contrary to expectations, the baseline DBP of women and men did not differ, $F(1, 234) = 0.36, p = .55$. As expected, Black participants had higher baseline DBP than White participants, $F(1, 234) = 9.22, p < .01$ (see Table 5 for group means and Table A3 for ANCOVA table).

Table 5.

Mean Baseline Diastolic Blood Pressure by PTSD Status, Gender, and Ethnicity

| | PTSD | | | No PTSD | | |
|-------|-------------|-------------|-------------|-------------|-------------|-------------|
| | Male | Female | Total | Male | Female | Total |
| Black | 78.0 (12.5) | 75.1 (21.2) | 76.5 (17.4) | 74.4 (10.6) | 71.8 (12.8) | 72.7 (12.0) |
| White | 71.2 (10.7) | 65.2 (11.5) | 68.3 (11.4) | 73.6 (10.8) | 70.0 (14.8) | 71.9 (12.9) |
| Total | 74.4 (12.0) | 70.2 (17.7) | 72.3 (15.2) | 73.9 (10.6) | 70.9 (13.8) | 72.2 (12.5) |

Note. Values enclosed in parentheses indicate Standard Deviations.

HR Response to Anger

There was no main effect indicating a significant difference between people with and without PTSD $F(1, 237) = 1.04, p = .31$.

PTSD status affected HR response to anger differently for Blacks and Whites, PTSD x Ethnicity $F(1, 235) = 5.01, p = .03$. Blacks demonstrated the expected effect of participants with PTSD having a greater positive HR change during anger recall than participants without PTSD, $F(1, 235) = 6.51, p = .01$. The HR response to anger for Whites, however, did not differ based on PTSD status, $F(1, 235) = 0.23, p = .63$.

PTSD status also had a different effect on HR response to anger for men and women, PTSD x Gender $F(1, 235) = 3.86, p = .05$. Men demonstrated the expected effect; men with PTSD had greater positive HR change scores during an anger recall task than men without PTSD, $F(1, 235) = 5.85, p = .02$. Women's HR response to anger, however, did not significantly differ by PTSD status, $F(1, 235) = 0.08, p = .78$ (see Table 6 for group means and Table A4 for ANCOVA table).

Table 6.

Mean Heart Rate Change Score During Anger Recall by PTSD Status, Gender, and Ethnicity

| | PTSD | | | No PTSD | | |
|-------|-----------|-----------|-----------|-----------|-----------|-----------|
| | Male | Female | Total | Male | Female | Total |
| Black | 4.5 (5.6) | 3.0 (5.5) | 3.8 (5.5) | 1.0 (4.0) | 2.6 (3.7) | 2.0 (3.9) |
| White | 4.2 (3.6) | 1.7 (3.7) | 3.0 (3.9) | 4.0 (4.5) | 3.4 (4.7) | 3.7 (4.6) |
| Total | 4.4 (4.6) | 2.4 (4.7) | 3.4 (4.8) | 3.0 (4.5) | 3.0 (4.2) | 3.0 (4.4) |

Note. Values enclosed in parentheses indicate Standard Deviations.

SBP Response to Anger

No differences in SBP response to an anger recall task for participants with and without PTSD were found, $F(1, 236) = 0.33, p = .56$. As expected, men's SBP response to the anger task was greater than women's, $F(1, 236) = 4.69, p = .03$. Whites had higher positive SBP change scores in response to the anger recall task than Blacks, $F(1, 236) = 20.41, p < .01$.

Ethnicity had a different effect on SBP response to anger for men and women, Ethnicity x Gender $F(1, 236) = 4.07, p = .05$. Black men had greater positive SBP responses to an anger recall task than Black women, whose SBP responses actually *decreased* during the anger recall task, $F(1, 236) = 7.63, p = .01$. However, White men's and White women's SBP responses to the anger recall task did not differ, $F(1, 236) = 0.89, p = .35$ (see Table 7 for group means and Table A5 for ANCOVA table).

Table 7.

Mean Systolic Blood Pressure Change Score During Anger Recall by PTSD Status, Gender, and Ethnicity

| | PTSD | | | No PTSD | | |
|-------|------------|-------------|-------------|-----------|------------|-----------|
| | Male | Female | Total | Male | Female | Total |
| Black | 1.3 (10.6) | -3.2 (10.1) | -1.0 (10.5) | 4.5 (7.9) | -2.4 (8.2) | 0.2 (8.7) |
| White | 5.6 (8.8) | 5.0 (7.2) | 5.3 (8.0) | 5.7 (7.2) | 3.6 (6.2) | 4.7 (6.8) |
| Total | 3.5 (9.8) | 0.8 (9.7) | 2.2 (9.8) | 5.3 (7.4) | 0.7 (7.8) | 2.8 (7.9) |

Note. Values enclosed in parentheses indicate Standard Deviations.

DBP Response to Anger

No differences in DBP response to a relived anger task were predicted or found for participants with and without PTSD, $F(1, 237) = 0.07, p = .80$. As predicted, male DBP change scores during the anger recall task were significantly greater than female scores, $F(1, 237) = 6.23, p = .01$. Whites had greater DBP increases over baseline in response to the anger recall task than Blacks, $F(1, 237) = 10.04, p < .01$ (see Table 8 for group means and Table A6 for ANOVA table).

Table 8.

Mean Diastolic Blood Pressure Change Score During Anger Recall by PTSD Status, Gender, and Ethnicity

| | PTSD | | | No PTSD | | |
|-------|-----------|------------|------------|-----------|------------|------------|
| | Male | Female | Total | Male | Female | Total |
| Black | 0.8 (5.3) | -0.9 (8.0) | -0.1 (6.8) | 1.7 (3.5) | -1.1 (3.6) | -0.1 (3.8) |
| White | 2.9 (3.9) | 2.0 (4.2) | 2.5 (4.0) | 2.2 (2.8) | 1.4 (5.0) | 1.8 (4.0) |
| Total | 1.9 (4.7) | 0.5 (6.5) | 1.2 (5.7) | 2.0 (3.1) | 0.2 (4.5) | 1.0 (4.0) |

Note. Values enclosed in parentheses indicate Standard Deviations.

Discussion

The purpose of the current study was to investigate the influence of ethnicity, gender, and PTSD status on cardiovascular responding. As expected, baseline physiological effects of PTSD status varied depending on ethnicity and gender. White men, White women, and Black women with PTSD all had higher baseline HR than their counterparts without PTSD, while Black men with PTSD had lower baseline HR than Black men without PTSD. Following from this data pattern, women with PTSD had slightly higher baseline HR than women without PTSD, while men with PTSD had lower baseline HR than men without

PTSD. Whites with PTSD had lower baseline SBP than Whites without PTSD, whereas Blacks had the opposite pattern. As expected given basic physiological trends, Blacks had higher baseline SBP and DBP than Whites.

The physiological responses to anger also differed depending on PTSD status, gender, and ethnicity. Blacks and men with PTSD had greater HR responses than Blacks and men without PTSD, respectively. The HR responses to anger for Whites and women did not differ by PTSD status, however. Also, no differences in SBP or DBP responses to anger were found between participants with and without PTSD. Black men had greater positive SBP responses to anger than Black women, whose SBP responses decreased during the anger recall task, but the SBP responses of Whites did not differ based on gender. Whites had greater positive SBP and DBP responses to anger Blacks, and men had greater positive SBP and DBP responses to anger than women. The baseline findings and then the responses to anger will be discussed in turn.

Baseline Findings

One of the more interesting results of the current study was the finding that baseline heart rate was slightly higher in people with PTSD than in people without PTSD for all Gender X Ethnicity groups except for Black men. Thus among women and White men the expected pattern, that people with PTSD would have higher baseline HR than people without PTSD, was found. Black men with PTSD, however, actually had lower baseline HR than Black men without PTSD.

This finding for Black males was unexpected. This study was the first to compare the physiological responses of Blacks with PTSD to Blacks without PTSD as well as Whites. Within the literature, no PTSD studies had all-Black samples or directly compared the

physiological responses of Black participants with PTSD to Blacks without PTSD or to other ethnic groups, so the generalizability of this finding is not known.

All of the Black men in the current sample were Vietnam combat veterans, but only three studies with all-male Vietnam combat veteran samples reported that their samples contained non-White participants (Keane et al., 1998; Litz et al., 2000; Muraoka et al., 1998). The Litz et al. (2000) study was the only study with an all-male Vietnam-era veteran sample that specifically reported how many Black participants were in the study (11%) (see Table 1 for information about PTSD studies that reported baseline cardiovascular findings).

Having more information on ethnic differences in samples could help explain discrepant findings. For example, had the ethnic composition of the current study not been reported, and had the baseline HR of Black and White men with and without PTSD not been directly compared to each other in the current study, the data would have only shown that men in the study with PTSD had lower HR than the men without PTSD, when in reality this was only true for the Black males. The lack of similar findings in the literature reflects the fact that this unusual finding needs replication.

Within this PTSD by Ethnicity by Gender interaction, White men with PTSD ($M = 70.3$ bpm) had slightly higher baseline HR than White men without PTSD ($M = 68.9$ bpm). Though the direction of this finding is consistent with other all-male studies of Vietnam-era veterans, the magnitude of the mean difference seems to be less than the magnitude of the mean HR differences in other studies (Gerardi et al., 1994; Keane et al., 1998; Muraoka et al., 1998; Orr, Lasko, Shalev, & Pitman, 1995; Orr et al., 1997; Pitman et al., 1987).

Seven studies with all-male Vietnam combat veteran samples found that men with PTSD had slightly to significantly greater baseline HR than men without PTSD (Gerardi et

al., 1994; Keane et al., 1998; Litz et al., 2000; Muraoka et al., 1998; Orr et al., 1995; Orr et al., 1997; Pitman et al., 1987). Four of these studies reported the ethnic composition of their samples (Keane et al., 1998; Litz et al., 2000; Muraoka et al., 1998; Pitman et al., 1987).

A large-scale lab-based study of over 1300 male Vietnam combat veterans found that men with PTSD ($M = 74.0$ bpm) had significantly higher HR than men who had never had PTSD ($M = 70.5$ bpm) (Keane et al., 1998). The study reported how many Hispanic participants were in the study, but did not provide information on other ethnicities (Keane et al., 1998). In a laboratory-based study consisting entirely of White male Vietnam combat veterans, men with PTSD ($M = 75.3$ bpm) had significantly higher resting HR than men without PTSD ($M = 66.3$ bpm) (Pitman et al., 1987). In two lab-based Orr et al. (1995, 1997) studies made up entirely of male Vietnam combat veterans of unreported ethnicity, the results were quite similar to the current study. Men with PTSD in both studies (1995 $M = 73.0$ bpm; 1997 $M = 76$ bpm) had marginally higher baseline HR than men without PTSD (1995 $M = 68.2$ bpm; 1997 $M = 68.6$ bpm) (Orr et al., 1995; Orr et al., 1997).

In a 24-hour ambulatory monitoring study composed entirely of male Vietnam combat veterans, men with PTSD ($M = 80.8$ bpm) had significantly higher overall HR than men without PTSD ($M = 71.9$ bpm) (Muraoka et al., 1998). The Muraoka et al. (1998) study broke down its sample into White (61%) vs. non-White (39%), and did not investigate any PTSD X Ethnicity differences in baseline HR.

In the Gerardi et al. (1994) study that took place in the waiting area of a hospital, all men with PTSD were Vietnam combat veterans, while the men without PTSD were Vietnam-era medical veterans who had *not* served in Vietnam. Ethnicity was not reported for this study (Gerardi et al., 1994). Male veterans with PTSD ($M = 89$ bpm) had significantly

higher resting HR than male veterans without PTSD ($M = 78$ bpm). The more robust difference between PTSD and non-PTSD groups in the Gerardi et al. (1994) study than in the current study may be due to the fact that the non-PTSD sample, though Vietnam-era veterans, did not have any combat experience. In the current study, all male participants were Vietnam combat veterans. Mean resting HRs for both PTSD and non-PTSD samples were higher in the Gerardi et al. (1994) study than in the current study, as was the magnitude of difference in the resting HR between the PTSD and non-PTSD groups.

A Litz et al. (2000) lab-based study found that, by a magnitude comparable to that of the White men in the current study, male Vietnam combat veterans with PTSD ($M = 68.9$ bpm) had slightly higher baseline HR than veterans without PTSD ($M = 67.6$ bpm). The study's ethnic composition was 82% White and 11% Black. Overall, these studies with all-male Vietnam combat veteran samples found, as did the current study, that men with PTSD had slightly to significantly higher baseline HR than men without PTSD, though the magnitudes of HR difference were larger in the studies detailed above (save for Litz et al., 2000) than in the current study.

Not all studies composed of male Vietnam combat veterans found that men with PTSD had higher resting HR than men without PTSD, however. A study composed entirely of male, White veterans found that baseline HR in men with PTSD ($M = 70.8$ bpm) was slightly lower than the male comparison group without PTSD ($M = 73.6$ bpm) (Pitman et al., 1990). However, in the Pitman et al. (1990) study, all men in the non-PTSD group suffered from anxiety disorders, which was not the case in the current study (see Table 2). In an in-home study by Orr, Meyerhoff, Edwards, & Pitman (1998) containing an all-male Vietnam combat veteran sample, men with PTSD had slightly lower resting HR ($M = 66$ bpm) than

men without PTSD ($M = 68.5$ bpm). The ethnic composition of the sample was not reported (Orr, Meyerhoff, et al., 1998).

Overall, seven all-male studies showed baseline HR differences in the expected direction, with male Vietnam-era veterans with PTSD having higher baseline HR than their counterparts without PTSD (Gerardi et al., 1994; Keane et al., 1998; Litz et al., 2000; Muraoka et al., 1998; Orr et al., 1995; Orr et al., 1997; Pitman et al., 1987). Of these studies, the HR differences between male samples with and without PTSD were significant in four studies (Gerardi et al., 1994; Keane et al., 1998; Muraoka et al., 1998; Pitman et al., 1987), marginal in two studies (Orr et al., 1995; Orr et al., 1997), and almost negligible in one study (Litz et al., 2000). Two studies showed the opposite pattern in which male Vietnam-era veterans with PTSD had slightly *lower* baseline HR than male Vietnam-era veterans without PTSD (Orr, Meyerhoff, et al., 1998; Pitman et al., 1990), though these differences did not approach significance (see Table 1 for additional information about PTSD studies that reported baseline cardiovascular findings).

Upon close examination, no clear differences in sample demographics or study design seem to distinguish studies in which men with PTSD have greater baseline HR than men without PTSD from studies that show the opposite pattern. All of the aforementioned studies were made up of all-male Vietnam-era veteran samples, so no gender differences were observed. Ethnic differences were not related consistently to findings; men with PTSD in one all-White sample (Pitman et al., 1987) had higher resting HR than men without PTSD, while in another all-White sample (Pitman et al., 1990) the opposite pattern occurred. All three studies that explicitly included non-White participants showed that men with PTSD had higher resting HR than men without PTSD (Keane et al., 1998; Litz et al., 2000; Muraoka et

al., 1998). However, three additional studies in which men with PTSD had elevated HR compared to non-PTSD men (Gerardi et al., 1994; Orr et al., 1995; Orr et al., 1997), and one study that showed the opposite pattern (Orr, Meyerhoff, et al., 1998) did not include data on male participants' ethnicities, making it difficult to generalize the effects of ethnicity and PTSD status on resting HR across studies.

Women in the current study with PTSD ($M = 72.9$ bpm) had slightly higher baseline HR than women without PTSD ($M = 69.7$ bpm). This finding is consistent with six all-female PTSD studies (Carson et al., 2000; Carson et al., 2007; Forneris et al., 2004; Metzger et al., 1999; Orr, Lasko, et al., 1998; Pitman et al., 2001) that found that women with PTSD had slightly to significantly higher resting HR than women without PTSD. Of these studies, the HR differences between female samples with and without PTSD were significant in four studies (Carson et al., 2000; Forneris et al., 2004; Metzger et al., 1999; Orr, Lasko, et al., 1998), and almost negligible in one study (Carson et al., 2007; Pitman et al., 2001). Two studies showed the opposite pattern in which women with PTSD had slightly *lower* baseline HR than women without PTSD (Griffin et al., 2008; Schmahl et al., 2004), though these differences did not approach significance.

Within the all-female literature, trends emerge. Overall, women with PTSD have higher resting HR than women without PTSD. Some of the most robust findings occur in all-veteran samples (Carson et al., 2000; Forneris et al., 2004). Findings for women who have experienced sexual or physical abuse are more mixed; two studies supported the overall trend of elevated HR in women without PTSD compared to women without PTSD (Metzger et al., 1999; Orr, Lasko, et al., 1998), while two studies showed the opposite pattern (Griffin et al., 2008; Schmahl et al., 2004).

While resting HR was higher among women with PTSD than without PTSD in the current study, the difference was not significant. This less-robust finding may have been due, in part, to the makeup of the current female sample compared to the composition of other female samples in the literature. Women in the current study had a wide range of traumas. Some had been victims of physical or sexual assault, others had been in car accidents, and still others had been in combat. Women in the current study who did *not* have PTSD are likely more representative of a general population without PTSD, compared to the narrower sample of women in the aforementioned studies, who were either all veterans (Carson et al., 2000; Carson et al., 2007; Forneris et al., 2004), childhood sexual abuse victims (Metzger et al., 1999; Orr, Lasko, et al., 1998), or breast cancer patients (Pitman et al., 2001). The women with PTSD in the current study were recruited through multiple sites (the Durham VAMC, the Women's Health Clinic and Women's Mental Health Clinic, and the Duke University Medical Center Anxiety Disorder Clinic). As such, the current sample of women identified a wider range of traumas. Across a variety of samples, however, women with PTSD tend to have higher resting HR than women without PTSD, particularly in samples made up entirely of veterans.

Overall, the literature on baseline HR differences between people with and without PTSD shows that people with PTSD tend to have higher baseline HR than people without PTSD (Buckley & Kaloupek, 2001; Pole, 2007). In all-male Vietnam combat veteran samples men with PTSD have marginally to significantly higher HR than men without PTSD (Gerardi et al., 1994; Muraoka et al., 1998; Orr et al., 1995; Orr et al., 1997; Pitman et al., 1987). The baseline HR of White men in the current sample was consistent with these findings. However, the Black men in the current sample displayed the opposite pattern.

Since most of these studies in the literature do not include the ethnic breakdowns of their samples, it is unclear whether the findings from the current study are consistent with the literature, and replication of these findings is needed. When women are investigated separately from men, it seems that more robust baseline HR elevations in female PTSD versus non-PTSD samples (particularly samples that have *never* had PTSD) occur when the sample is more homogenized, such as an all-veteran sample (Carson et al., 2000; Forneris et al., 2004).

In the current study, no significant differences in baseline SBP or DBP were found between people with (SBP $M = 129.4$ mm Hg; DBP $M = 72.3$ mm Hg) and without PTSD (SBP $M = 130.5$ mm Hg; DBP $M = 72.2$ mm Hg), despite the initial predictions. Pole's (2007) recent meta-analysis found that resting SBP and DBP are generally higher for people with PTSD than without PTSD. Another meta-analysis, however, drew distinctions when comparing baseline SBP of people with PTSD to people without PTSD who have experienced a trauma versus people with PTSD who have *not* experienced a trauma (Buckley & Kaloupek, 2001). Elevated baseline SBP was found in people with PTSD compared to people without PTSD who had not experienced a traumatic event. When people with PTSD were compared to people without PTSD who had experienced a traumatic event, however, there were no baseline SBP differences (Buckley & Kaloupek, 2001). In the same Buckley & Kaloupek (2001) meta-analysis, baseline DBP was significantly elevated for people with PTSD compared to all people without PTSD (Buckley & Kaloupek, 2001). Though the effect sizes in the meta-analysis were significant, the actual DBP differences were quite small for groups with PTSD compared to groups without current PTSD (only 5 mm Hg) (Buckley

& Kaloupek, 2001). The PTSD vs. non-PTSD difference in DBP was even smaller in the current study, only 0.1 mm Hg.

The participants in the current study had, by and large, experienced trauma; all men had been in combat in Vietnam, and 85% of women without PTSD had experienced some sort of trauma. This suggests that the finding that baseline SBP is greater in people with PTSD than without PTSD may not be as robust when the comparison sample without PTSD has also suffered traumas. All-male studies that consist entirely of combat veterans do not have a need for the “non-trauma-exposed, non-PTSD” category; many of the all-female and mixed-gender studies, however, contain more varied samples in which participants may not have all experienced a traumatic event. In reporting on future PTSD studies, it would be worthwhile to indicate if a comparison sample without PTSD has not suffered from trauma, as this may be associated with different baseline SBP than if the comparison group without PTSD had suffered trauma. Unreported trauma vs. no-trauma distinctions among participants without PTSD could account for some inconsistencies in the literature. Because 96% of participants in the current study experienced a traumatic event, it is possible that the PTSD vs. no PTSD comparisons of the current study are not as robust as in studies in the literature that compare samples with PTSD to samples that have never experienced a traumatic event.

Blacks in the current study with PTSD ($M = 136.1$ mm Hg) had slightly but not significantly higher baseline SBP than Black participants without PTSD ($M = 131.4$ mm Hg), in line with the overall findings of the Buckley & Kaloupek (2001) and Pole (2007) meta-analyses described above. White participants with PTSD ($M = 122.9$ mm Hg) had significantly *lower* baseline SBP than Whites without PTSD ($M = 129.8$ mm Hg), contrary to expectations.

Two studies, both with all-male Vietnam combat veteran samples found that men with PTSD had higher baseline SBP and DBP than men without PTSD (Gerardi et al., 1994; Muraoka et al., 1998). In an ambulatory monitoring study, men with PTSD (SBP $M = 123.0$ mm Hg; DBP $M = 80.1$ mm Hg) had significantly higher DBP and slightly higher SBP than men without PTSD (SBP $M = 119.3$ mm Hg; DBP $M = 71.5$ mm Hg) (Muraoka et al., 1998). In the Muraoka et al. (1998) study, 61% of the participants were White, while 39% were “non-White.” Participant ethnicity was not reported in a study that found that men with PTSD (SBP $M = 133.0$ mm Hg; DBP $M = 88$ mm Hg) had significantly higher resting SBP and DBP than men without PTSD (SBP $M = 124.0$ mm Hg; DBP $M = 79$ mm Hg) while awaiting a physical exam at a Veterans Affairs Medical Center (Gerardi et al., 1994).

Three studies found no significant resting SBP or DBP differences between people with and without PTSD (Blanchard et al., 1996; Keane et al., 1998; Schmahl et al., 2004). In the large-scale, lab-based Keane et al. (1998) study, no SBP or DBP differences were found between male combat veterans with current PTSD (SBP $M = 120.2$ mm Hg; DBP $M = 71.7$ mm Hg) and their counterparts who had never had PTSD (SBP $M = 120.0$ mm Hg; DBP $M = 71.1$ mm Hg). The Keane et al. (1998) study reported how many Hispanic participants were in the study, but did not give any information about other ethnic groups. The participants in the PTSD sample in the Blanchard et al. (1996) study had all been in motor vehicle accidents, while the non-PTSD sample’s involvement in motor vehicle accidents varied. The sample was mostly White (92%, compared to 8% “non-White”) and mostly female (69%, compared to 31% male). DBP was not measured in the study, and no differences in resting SBP were found between people with ($M = 120.4$ mm Hg) and without PTSD ($M = 116.7$ mm Hg) (Blanchard et al., 1996). The Schmahl et al. (2004) study had an entirely female sample with

a history of sexual and physical abuse, but did not report the ethnicity of its participants. No significant baseline SBP or DBP differences were found between women with PTSD (SBP $M = 113.38$ mm Hg; DBP $M = 68.62$ mm Hg) and women without PTSD (SBP $M = 117.36$ mm Hg; DBP $M = 66.43$ mm Hg) (Schmahl et al., 2004).

Two studies, while not significant, found that people with PTSD actually had slightly lower resting SBP and DBP than people without PTSD (Forneris et al., 2004; Orr, Meyerhoff, et al., 1998). An all-male Vietnam combat veteran sample in an ambulatory monitoring study had no significant SBP or DBP differences by PTSD status; if anything, men with PTSD (SBP $M = 119$ mm Hg; DBP $M = 69.5$ mm Hg) had slightly lower resting SBP and DBP than men without PTSD (SBP $M = 123$ mm Hg; DBP $M = 70.5$ mm Hg) (Orr, Meyerhoff, et al., 1998). Ethnic composition of this sample was not reported (Orr, Meyerhoff, et al., 1998). All participants in the Forneris et al. (2004) study were female nurses in Vietnam, and the sample was 53% White, 39% Black, and 8% “other”. Though the differences were not significant, women with PTSD (SBP $M = 126.9$ mm Hg; DBP $M = 74.6$ mm Hg) had slightly lower resting SBP and DBP than women without PTSD (SBP $M = 129.3$ mm Hg; DBP $M = 76.0$ mm Hg) (Forneris et al., 2004).

Overall, two studies found that people with PTSD had higher resting SBP and DBP than people without PTSD (Gerardi et al., 1994; Muraoka et al., 1998). Three studies found no PTSD status differences in baseline SBP (Blanchard et al., 1996; Keane et al., 1998; Schmahl et al., 2004), and two studies found no differences in baseline DBP by PTSD status (Keane et al., 1998; Schmahl et al., 2004). In two studies, people with PTSD had slightly but not significantly lower resting SBP and DBP than people without PTSD (Forneris et al., 2004; Orr, Meyerhoff, et al., 1998).

The current study's finding that Whites with PTSD had significantly lower resting SBP than Whites without PTSD is unusual. No studies in the literature were significant in this direction (with non-PTSD resting SBP being greater than PTSD resting SBP). Two studies did find that people without PTSD had *slightly* higher SBP than people with PTSD, but neither of these studies had an all-White sample; the Forneris et al. (1994) study was 53% White, and the Orr, Meyerhoff, et al. (1998) study did not report on ethnicity. In fact, no studies that measured SBP *and* reported on the ethnic composition of their samples had all-White samples. Also, no studies directly compared the physiological responses of White participants with PTSD to Whites without PTSD or to other ethnic groups (see Table 1 for further information about PTSD studies that reported baseline cardiovascular findings). Further research is needed to replicate this significant finding that Whites with PTSD had lower baseline SBP than Whites without PTSD.

Within the mixed results for baseline, some trends emerge. The finding that people with PTSD have higher resting SBP and DBP than people without PTSD seems most robust in ambulatory monitoring studies (Muraoka et al., 1998) or naturalistic studies (i.e., in a hospital waiting room) (Gerardi et al., 1994). All three traditional lab-based studies showed no differences in baseline SBP for people with and without PTSD (Blanchard et al., 1996; Keane et al., 1998; Schmahl et al., 2004). Studies with all-male Vietnam combat veteran samples also seem to yield more robust findings (Gerardi et al., 1994; Muraoka et al., 1998). None of the all-female (Forneris et al., 2004; Schmahl et al., 2004) or mixed-gender (Blanchard et al., 1996) studies that measured resting SBP or DBP found significant differences based on PTSD status. Overall, non-laboratory studies with all-male Vietnam

combat veteran samples appear to best support the finding that people with PTSD have higher resting SBP than people without PTSD.

In line with the original hypotheses, Blacks had higher baseline SBP and DBP than Whites. This is a fairly robust finding throughout the literature (Salomon & Jaguszyn, 2008). Elevated baseline BP in Blacks compared to Whites has been attributed to different factors. In the past, some scientists attributed this discrepancy to genetic racial differences between Blacks and Whites worldwide (Halberstein, 1999, as cited in Kaufman & Hall, 2003), but a study by Kaufman and Barkey found that Blacks in Africa did not, in fact, have elevated blood pressure, and that Black blood pressure elevation and the high attendant rates of hypertension were confined to the West (as cited in Kaufman & Hall, 2003).

Researchers cite a number of environmental and social factors contributing to higher blood pressure in African-Americans compared to European-Americans (Cooper, Rotimi, et al., 1997; Malan, Van der Merwe, Huisman, & Kruger, 1992; Thorpe, Brandon, & LaVeist, 2008). Blacks in the United States tend to be of lower socioeconomic status than Whites, which is a risk factor for hypertension across industrialized societies. Other disparities exist between Blacks and Whites in American society, as Blacks tend to have higher obesity rates; more limited access to fresh, healthy food; higher intake of fast and processed foods high in sodium; and decreased access to medical information and care. Finally, racial discrimination in American society has also been seen as a major stressor that, over time, can lead to increased blood pressure (Cooper, Rotimi, et al., 1997, Malan et al., 1992; Thorpe et al., 2008).

Contrary to the original hypotheses, women had slightly higher baseline SBP than men, and no gender differences were found for baseline DBP. Generally, studies find that

men have higher baseline blood pressure than women (Lawler et al., 1995; Maixner & Humphrey, 1993; Myers et al., 2001). However, these studies did not look at ethnic differences in baseline BP in tandem with gender differences. When these two demographic variables are combined, a different picture emerges. A recent large-scale study suggests that relative baseline SBP and DBP equality exists for Black men and women, but White men have higher baseline SBP and DBP than White women (Cooper, Wolf-Maier, et al., 2005).

Baseline DBP findings in the current study followed the pattern observed by Cooper, Wolf-Maier, et al., (2005). Black men and women had no DBP differences, $t(110) = -1.104$, $p = .27$, while White men had significantly higher DBP than White women, $t(131) = -2.25$, $p = .03$. Like with resting DBP, the Cooper, Wolf-Maier, et al. (2005) study found that resting SBP and DBP are roughly equivalent for Black men and women, but White men have higher greater resting SBP than White women. In the present study, the data of Black participants follows the pattern of the Cooper, Wolf-Maier, et al. (2005) study, in that Black women have slightly but not significantly higher SBP than Black men, $t(110) = 1.49$, $p = .14$. White women's baseline SBP is slightly greater than that of White men, $t(131) = 0.99$, $p = .32$, however, which is the opposite of the Cooper, Wolf-Maier et al. (2005) pattern.

Another source of deviation from traditional studies on baseline SBP could be the sample composition of the current study. Most studies of baseline differences have been conducted on random samples of large numbers of individuals. The current study is investigating a very specific sample for the purpose of looking at physiological differences in people with and without PTSD. As such, all men in the current study are Vietnam combat veterans, and the women were also recruited through the VA system. Thus it may not be

entirely realistic to expect that all physiological differences found in large-scale, relatively healthy studies will also be observed in the current study.

Similarly, there are clear differences between the male and female participants within the current study. Again, all men in this sample were Vietnam combat veterans recruited in the mid-1990's, while female participants were recruited in the early 2000's. The female participants experienced a greater variety of traumas than the men did, and less time had passed since the women's trauma compared to the men's. The differences in recruitment of men and women could be contributing to the differences in baseline SBP in unknown ways.

Anger Recall Findings

There were no main effects of PTSD indicating that HR, SBP, and DBP increases in response to the anger recall task were significantly different for participants with and without PTSD in the current study. Instead, anger response based on PTSD status differed depending on gender and ethnicity. Blacks with PTSD ($M = 3.8$ bpm) and men with PTSD ($M = 4.4$ bpm) had greater mean increases in HR from baseline levels during the anger recall task than Blacks without PTSD ($M = 2.0$ bpm) and men without PTSD ($M = 3.0$ bpm), respectively. These findings reflect main effects of PTSD status on HR response. The HR responses to anger for Whites and women, however, did not differ based on PTSD status.

Anger is certainly connected to PTSD symptomatology; the DSM-IV-TR (APA, 2000) includes irritability and anger outbursts as hyperarousal symptoms of the disorder. A recent meta-analysis found that anger and hostility are strongly associated with PTSD in adults who have experienced a trauma, especially among people whose trauma was combat-related (Orth & Wieland, 2006). In a study that asked about male Vietnam combat veterans' emotional responses to imagery of individualized trauma scripts, self-reported anger was the

emotion that was most different between PTSD and non-PTSD groups, with veterans with PTSD reporting higher levels of anger than veterans without PTSD (Pitman et al., 1987). An ambulatory monitoring study by Beckham, Flood, Dennis, & Calhoun (2009) that used some of the same female participants as the current study found that increased hostility was connected to greater HR increases in women with PTSD compared to women without PTSD. As this was an ambulatory monitoring study, however, there were no anger-specific tasks involved.

To date, the current study is the only study that has investigated cardiovascular responses to an anger recall task for PTSD versus non-PTSD groups. Thus, it is difficult to generalize the current study's findings that uncovered no overall differences in HR, SBP, and DBP responses between samples with and without PTSD. More research is needed to establish what a normative HR response to anger would be like in a sample with PTSD, and to replicate the current study's findings in other samples.

It is unclear why Blacks with PTSD had higher HR responses to anger than Blacks without PTSD, while Whites with and without PTSD did not have different HR responses. None of the PTSD studies reviewed in the literature had exclusively Black samples or reported specifically on the physiological responses of Blacks with and without PTSD. Both studies that reported all-White samples found that people with PTSD had significantly greater HR responses to traumatic scripts than people without PTSD (Pitman et al., 1987; Pitman et al., 1990); however, there were no studies reporting on anger stimuli.

Overall, in Pole's (2007) meta-analysis, people with PTSD had significantly higher HR responses to various stimuli than people without PTSD, and this finding was particularly robust when the stimuli were trauma-related. The current study, however, included anger-

related rather than trauma-related stimuli. There is a lack of literature on physiological responses to anger in samples with PTSD, let alone studies that directly compare different genders and ethnicities, making it difficult to ascertain what would constitute a “typical” PTSD x Ethnicity pattern of HR response to an anger task.

Black men ($M = 2.6$ mm Hg) showed significantly greater increases in SBP over baseline in response to the anger task compared to Black women ($M = -2.8$ mm Hg), whose SBP levels actually decreased from baseline. Also, SBP and DBP increases in response to the relived anger task were significantly greater for men (SBP $M = 4.4$ mm Hg; DBP $M = 2.0$ mm Hg) and Whites (SBP $M = 5.0$ mm Hg; DBP $M = 2.1$ mm Hg) than for women (SBP $M = 0.7$ mm Hg; DBP $M = 0.3$ mm Hg) and Blacks (SBP $M = -0.5$ mm Hg; DBP $M = -.07$ mm Hg).

The literature on ethnic differences in HR responses to anger does not explain the finding that SBP levels for Black women actually decreased from baseline during the anger recall task, while Black men’s SBP levels increased from baseline. Heart rate and blood pressure responses to anger differ among ethnicities, and there seem to be gender differences in how Blacks respond to anger. A sample of healthy adults (Suarez, Saab, Llabre, Kuhn, & Zimmerman, 2004) participated in an anger recall interview. All Ethnicity x Gender groups showed similarly increased levels of SBP and DBP over baseline in response to the anger recall task, but there were differences in HR response to anger by Ethnicity x Gender group. Black women and Whites showed higher HR increases over baseline in response to the anger task compared to Black men, whose HR increases were significantly lower than the other Ethnicity x Gender groups. Additionally, Black women and White women had significantly greater HR responses to the anger task than did Black men and White men. This study fits in

with literature suggesting that increased SBP and DBP responses in Black men are accompanied by blunted HR responses, while for Black women and Whites, greater SBP and DBP responses tend to be associated with greater HR responses as well (Light, Turner, Hinderliter, & Sherwood, 1993, as cited in Suarez et al., 2004; Saab et al., 1997).

Given the relative agreement in the literature that both Black men and women tend to have increased blood pressure reactivity to anger cues and challenging tasks (Suarez et al., 2004; Saab et al., 1997), both the finding that Black women's SBP levels decreased from baseline during the anger recall task (compared to Black men's SBP levels increasing from baseline), and the finding that Blacks, overall, had lower SBP responses to anger than Whites are unexpected. The lack of literature makes it difficult to generalize these findings, and further research in samples with PTSD will be necessary to get a picture of how normative these findings truly are.

The effect of PTSD status on HR in men is consistent with Pole's (2007) meta-analysis, which found increasingly elevated HR responses to various stimuli for samples with PTSD compared to samples without PTSD. Most of the studies Pole analyzed used male combat veterans as samples, but in the meta-analysis, Pole did not make distinctions by gender or ethnicity. It is important to note that the current study is different from most of the studies in Pole's meta-analysis in that it did not involve standardized cues, personalized trauma cues (though a personalized *anger* cue was used), or startling stimuli. The current study is the only study, to date, that directly compares the physiological responses to anger between people with and without PTSD.

Five all-female studies found that women with PTSD had significantly greater HR elevation over baseline in response to a variety of stimuli (personalized scripts, startling

tones) than women without PTSD (Carson et al., 2000; Carson et al., 2007; Griffin et al., 2008; Metzger et al., 1999; Pitman et al., 2001), and one additional study showed nonsignificant findings in the expected direction (Orr, Lasko, et al., 1998).

The fact that the females in the current study were a less homogenized sample than in the all-female studies, and the stimuli they were responding to was not a trauma-relevant task may help to explain why PTSD versus non-PTSD differences in HR response to a relived anger task did not reach significance. Research also suggests that women have a greater tendency than men to inhibit their physiological responses to anger, which could have affected their reaction to the anger stimuli in the current study.

SBP and DBP increases over baseline in response to anger were greater for men than for women. This is consistent with the literature (Lawler et al., 1995; Maixner & Humphrey, 1993; Myers et al., 2001). In one study, men exhibited significantly greater SBP responses and slightly higher DBP responses to an anger recall interview than women. Men with PTSD actually had slightly lower HR responses to anger than women, however (Lawler et al., 1995). Additionally, evolutionary psychology and social psychology research have suggested that women are more adept at inhibiting their physiological responses to anger than men (Bjorklund & Kipp, 1996). The current data fit the trend laid out by other anger recall studies, in which men tend to have greater SBP and DBP increases in response to an anger task than women.

The results of the current study raise several questions that future research could address. Although this study is an advance over previous studies in that the same methodology was used to look at cardiovascular baseline and response to anger for a large sample of men and women of different ethnicities with and without PTSD, different

recruitment strategies used for the male and female participants make it hard to completely rule out other explanations for the data. In the present study, all male participants were Vietnam combat veterans, but only a small minority of the female participants had veteran status. Different methods were used to recruit male and female participants, which introduced other sample variations into the results based on gender lines, such as age, time since trauma, cardiovascular health, and medication usage. The male and female participants in the current study were also recruited during different time periods, which affected which version of the SCID was administered. Certain gender differences are difficult to control; for example, men and women tend to experience different types of trauma (Tolin & Foa, 2006). Again, the current study was a significant advance in that male and female samples used the same procedures, experimenters, instructions, and settings. However, a more uniform sample of men and women could decrease some of the unwanted variation in other variables, making it easier to home in on physiological differences. It will be important for future research studies to include uniform research methodology when comparing the cardiovascular baseline and response levels of people with and without PTSD.

Because this study is the first of its kind to research the gender and ethnic differences that are present in cardiovascular baseline levels and responses to anger in people with and without PTSD, replication is needed to establish normative findings. Some of the findings of the current study were particularly unexpected, such as the baseline HR finding in which Black men with PTSD had lower HR than Black men without PTSD, or the finding that Black women's SBP actually decreased from baseline levels in response to the anger task. In future studies, one might include other variables that could potentially explain these findings. For example, measures of rumination and dissociation could be administered to participants,

as these types of behaviors have been shown to inhibit physiological responses (Halligan et al., 2006).

Finally, basic PTSD vs. non-PTSD differences in physiological response to anger are not well-established in the literature. The current study is the only study that has investigated these differences. Anger is an important diagnostic criterion and symptom of PTSD and deserves more attention. More research is needed to replicate the current study's findings and establish what normative HR, SBP, and DBP responses to anger would look like in samples with PTSD.

The current study is an important first step toward establishing normative differences in cardiovascular baseline levels and responses to anger among different ethnic and gender samples based on PTSD status. Heightened awareness of these ethnic and gender differences could have important implications for cardiovascular health in people with PTSD, who are already at increased risk for cardiovascular disease and heart disease mortality (Boscarino, 2008).

As has been stated in this manuscript, many studies of PTSD do not report the ethnicity of their samples. Even when studies with PTSD samples do report the ethnic and gender composition of their samples, the physiological responses of different ethnicities and genders are rarely directly compared. Only one study directly compared the cardiovascular data of men and women with PTSD (Bremner et al., 2003), and no studies directly compared the cardiovascular data of Blacks and Whites with PTSD. The current study is a first attempt to investigate the gender and ethnic differences that are present in cardiovascular baseline levels and responses to anger in people with PTSD.

The current study found that demographic variables like gender and ethnicity, when combined with PTSD status, affect cardiovascular responding in manners inconsistent with what would be expected from looking simply at the accepted main effects of PTSD status, gender, and ethnicity. As a result, it is important for researchers to include demographic information when reporting results, especially given the adverse health implications of chronically elevated heart rate and blood pressure.

List of References

List of References

- American Heart Association. (2008). *High blood pressure statistics*. Retrieved October 30, 2008, from <http://www.americanheart.org/presenter.jhtml?identifier=4621>.
- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders: Text revision* (4th ed.). Washington, DC: Author.
- Beckham, J.C., Feldman, M.E., Barefoot, J.C., Fairbank, J.A., Helms, M.J., Haney, T.L., et al. (2000). Ambulatory cardiovascular activity in Vietnam combat veterans with and without Posttraumatic Stress Disorder. *Journal of Consulting and Clinical Psychology, 68*, 269-276.
- Beckham, J.C., Flood, A.M., Dennis, M.F., & Calhoun, P.S. (2009). Ambulatory cardiovascular activity and hostility ratings in women with chronic posttraumatic stress disorder. *Biological Psychiatry, 65*, 268-272.
- Beckham, J.C., Taft, C.T., Vrana, S.R., Feldman, M.E., Barefoot, J.C., Moore, S.D., et al. (2003). Ambulatory monitoring and physical health report in Vietnam veterans with and without chronic Posttraumatic Stress Disorder. *Journal of Traumatic Stress, 16*, 329-335.
- Beckham, J.C., Vrana, S.R., Barefoot, J.C., Feldman, M.E., Fairbank, J., & Moore, S.D. (2002). Magnitude and duration of cardiovascular responses to anger in Vietnam veterans with and without Posttraumatic Stress Disorder. *Journal of Consulting and Clinical Psychology, 70*, 228-234.
- Bedi, U.S., & Arora, R. (2007). Cardiovascular manifestations of Posttraumatic Stress Disorder. *Journal of the National Medical Association, 99*, 642-649.
- Bjorklund, D.F., & Kipp, K. (1996). Parental investment theory and gender differences in the evolution of inhibition mechanisms. *Psychological Bulletin, 120*, 163-188.
- Blake, D.D., Weathers, F.W., Nagy, L.M., Kaloupek, D.G., Gusman, F.D., & Charney, D.S. (1995). The development of a clinician-administered PTSD scale. *Journal of Traumatic Stress, 8*, 75-90.

- Blanchard, E.B. (1990). Elevated basal levels of cardiovascular responses in Vietnam veterans with PTSD: A health problem in the making? *Journal of Anxiety Disorders*, 4, 233-237.
- Blanchard, E.B., Hickling, E.J., Buckley, T.C., Taylor, A.E., Vollmer, A., & Loos, W.R. (1996). Psychophysiology of Posttraumatic Stress Disorder related to motor vehicle accidents: replication and extension. *Journal of Consulting and Clinical Psychology*, 64, 742-751.
- Boscarino, J.A. (2008). A prospective study of PTSD and early-age heart disease mortality among Vietnam veterans: Implications for surveillance and prevention. *Psychosomatic Medicine*, 70, 668-676.
- Bremner, J.D., Vythilingam, M., Vermetten, E., Adil, J., Khan, S., Nazeer, A., et al. (2003). Cortisol response to a cognitive stress challenge in Posttraumatic Stress Disorder (PTSD) related to childhood abuse. *Psychoneuroendocrinology*, 28, 733-750.
- Buckley, T.C., Holohan, D., Greif, J.L., Bedard, M., & Suvak, M. (2004). Twenty-four hour ambulatory assessment of heart rate and blood pressure in chronic PTSD and non-PTSD veterans. *Journal of Traumatic Stress*, 17, 163-171.
- Buckley, T.C., & Kaloupek, D.G. (2001). A meta-analytic examination of basal cardiovascular activity in Posttraumatic Stress Disorder. *Psychosomatic Medicine*, 63, 585-594.
- Carson, M.A., Paulus, L.A., Lasko, N.B., Metzger, L.J., Wolfe, J., Orr, S.P., et al. (2000). Psychophysiologic assessment of Posttraumatic Stress Disorder in Vietnam nurse veterans who witnessed injury or death. *Journal of Consulting and Clinical Psychology*, 68, 890-897.
- Carson, M.A., Metzger, L.J., Lasko, N.B., Paulus, L.A., Morse, A.E., Pitman, R.K., et al. (2007). Physiologic reactivity to startling tones in female Vietnam nurse veterans with PTSD. *Journal of Traumatic Stress*, 20, 657-666.
- Casada, J.H., & Roache, J.D. (2006). Dissociation of physiology and behavior in PTSD. *International Journal of Psychophysiology*, 62, 243-248.
- Cohen, H., Benjamin, J., Geva, A.B., Matar, M.A., Kaplan, Z., & Kotler, M. (2000). Autonomic dysregulation in panic disorder and in post-traumatic stress disorder: application of power spectrum analysis of heart rate variability at rest and in response to recollection of trauma or panic attacks. *Psychiatry Research*, 96, 1-13.
- Cooper, R., Rotimi, C., Ataman, S., McGee, D., Osotimehin, B., Kadiri, S., et al. (1997). Hypertension prevalence in seven populations of African origin. *American Journal of Public Health*, 87, 160-168.

- Cooper, R., Wolf-Maier, K., Luke, A., Adeyemo, A., Banegas, J.R., Forrester, T., et al., (2005). An international comparative study of blood pressure in populations of European versus African descent. *BMC Medicine*, 3:2.
- Crozier, J.C., Dodge, K.A., Fontaine, R.G., Lansford, J.E., Bates, J.E., Pettit, G.S., et al. (2008). Social information processing and cardiac predictors of adolescent antisocial behavior. *Journal of Abnormal Psychology*, 117, 253-267.
- Cuthbert, B.N., Lang, P.J., Strauss, C., Drobles, D., Patrick, C.J., & Bradley, M.M. (2003). The psychophysiology of anxiety disorder: fear memory imagery. *Psychophysiology*, 40, 407-422.
- Dorr, N., Brosschot, J.F., Sollers III, J.J., & Thayer, J.F. (2007). Damned if you do, damned if you don't: The differential effect of expression and inhibition of anger on the cardiovascular recovery in Black and White males. *International Journal of Psychophysiology*, 66, 125-134.
- Elsesser, K., Sartory, G., & Taskenberg, A. (2004). Attention, heart rate, and startle response during exposure to trauma-relevant pictures: A comparison of recent trauma victims and patients with Posttraumatic Stress Disorder. *Journal of Abnormal Psychology*, 113, 289-301.
- Epstein, E.M., Sloan, D.M., & Marx, B.P. (2005). Getting to the heart of the matter: Written disclosure, gender, and heart rate. *Psychosomatic Medicine*, 67, 413-419.
- First, M.B., Spitzer, R.L., Gibbon, M., & Williams, J.B.W. (1997). *Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I), Clinician Version*. Washington, DC: American Psychiatric Publishing.
- Ford, J.D. (2008). Trauma, Posttraumatic Stress Disorder, and ethnoracial minorities: Toward diversity and cultural competence in principles and practices. *Clinical Psychology: Science and Practice*, 15, 62-67.
- Fornieris, C.A., Butterfield, M.I., & Bosworth, H.B. (2004). Physiological arousal among women veterans with and without Posttraumatic Stress Disorder. *Military Medicine*, 169, 307-312.
- Gerardi, R.J., Keane, T.M., Cahoon, B.J., & Klauminzer, G.W. (1994) An in vivo assessment of physiological arousal in Posttraumatic Stress Disorder. *Journal of Abnormal Psychology*, 103, 825-827.
- Greenland, P., Daviglius, M.L., Dyer, A.R., Liu, K., Huang, C., Goldberger, J., et al. (1999). Resting heart rate is a risk factor for cardiovascular and noncardiovascular mortality: The Chicago heart association detection project in industry. *American Journal of Epidemiology*, 149, 853-862.

- Griffin, M.G. (2008) A prospective assessment of auditory startle alterations in rape and physical assault survivors. *Journal of Traumatic Stress, 21*, 91-99.
- Halligan, S.L., Michael, T., Wilhelm, F.H., Clark, D.M., & Ehlers, A. (2006). Reduced heart rate responding to trauma reliving in trauma survivors with PTSD: Correlates and consequences. *Journal of Traumatic Stress, 19*, 721-734.
- Kaufman, J.S., & Hall, S.A. (2003). The slavery hypertension hypothesis: Dissemination and appeal of a modern race theory. *Epidemiology, 14*, 111-118.
- Keane, T.M., Caddell, J.M., & Taylor, K.L. (1988). Mississippi Scale for Combat-Related Posttraumatic Stress Disorder: Three studies in reliability and validity. *Journal of Consulting and Clinical Psychology, 56*, 85-90.
- Keane, T.M., Fairbank, J.A., Caddell, J.M., Zimering, R.T., Taylor, K.L., & Mora, C. (1989). Clinical evaluation of a measure to assess combat exposure. *Psychological Assessment: Journal of Consulting Clinical Psychology, 1*, 53-55.
- Keane, T.M., & Kaloupek, D.G. (1997). Comorbid psychiatric disorders in PTSD: Implications for research. *Annals of the New York Academy of Sciences, 821*, 24-34.
- Keane, T.M., Kolb, L.C., Kaloupek, D.G., Orr, S.P., Blanchard, E.B., Thomas, R.G., et al. (1998). Utility of psychophysiological measurement in the diagnosis of posttraumatic stress disorder: Results from a department of veterans affairs cooperative study. *Journal of Consulting and Clinical Psychology, 66*, 914-923.
- Kellner, M., Yassouridis, A., Hubner, R., Baker, D.G., & Wiedemann, K. (2003). Endocrine and cardiovascular responses to corticotrophin-releasing hormone in patients with Posttraumatic Stress Disorder: A role for natriuretic peptide? *Neuropsychobiology, 47*, 102-108.
- Kulka, R.A., Schlenger, W.E., Fairbank, J.A., Hough, R.L., Jordan, B.K., Marmar, C.R., et al. (1990). Trauma and the Vietnam war generation: Report of findings from the National Vietnam Veterans Readjustment Study. New York: Brunner/Mazel.
- Lash, S.J., Gillespie, B.L., Eisler, R.M., & Southard, D.R. (1991). Sex differences in cardiovascular reactivity: Effects of the gender relevance of the stressor. *Health Psychology, 10*, 392-398.
- Lawler, K.A., Wilcox, Z.C., & Anderson, S.F. (1995). Gender differences in patterns of dynamic cardiovascular regulation. *Psychosomatic Medicine, 57*, 357-365.
- Litz, B.T., Orsillo, S.M., Kaloupek, D., & Weathers, F. (2000). Emotional processing in posttraumatic stress disorder. *Journal of Abnormal Psychology, 109*, 26-39.

- Maixner, W., & Humphrey, C. (1993). Gender differences in pain and cardiovascular responses to forearm ischemia. *Clinical Journal of Pain, 2*, 16-25.
- Malan, N.T., Van der Merwe, J.S., Huisman, H.W., & Kruger, A. (1992). A comparison of cardiovascular reactivity of rural Blacks, urban Blacks and Whites. *Stress Medicine, 8*, 241-246.
- Metzger, L.J., Orr, S.P., Berry, N.J., Ahern, C.E., Lasko, N.B., & Pitman, R.K. (1999). Physiologic reactivity to startling tones in women with Posttraumatic Stress Disorder. *Journal of Abnormal Psychology, 108*, 347-352.
- Muraoka, M.Y., Carlson, J.G., & Chemtob, C.M. (1998). Twenty-four-hour ambulatory blood pressure and heart rate monitoring in combat-related Posttraumatic Stress Disorder. *Journal of Traumatic Stress, 11*, 473-484.
- Myers, C.D., Robinson, M.E., Riley, J.L., & Sheffield, D. (2001). Sex, gender, and blood pressure: Contributions to experimental pain report. *Psychosomatic Medicine, 63*, 545-550.
- National Center for Posttraumatic Stress Disorder (2007). *Epidemiological fact sheet*. Retrieved October 30, 2008, from http://www.ncptsd.va.gov/ncmain/ncdocs/fact_shts/fs_epidemiological.html.
- Newton, T.L., Parker, B.C., & Ho, I.K. (2005). Ambulatory cardiovascular functioning in healthy postmenopausal women with victimization histories. *Biological Psychology, 70*, 121-130.
- Ohira, T., Roux, A.V.D., Prineas, R.J., Kizilbash, M.A., Carnethon, M.R., & Folsom, A.R. (2008). Associations of psychosocial factors with heart rate and its short-term variability: Multi-ethnic study of atherosclerosis. *Psychosomatic Medicine, 70*, 141-146.
- Orr, S.P., Lasko, N.B., Metzger, L.J., Berry, N.J., Ahern, C.E., & Pitman, R.K. (1998). Psychophysiological assessment of women with Posttraumatic Stress Disorder resulting from childhood sexual abuse. *Journal of Consulting and Clinical Psychology, 66*, 906-913.
- Orr, S.P., Lasko, N.B., Metzger, L.J., & Pitman, R.K. (1997). Physiologic responses to non-startling tones in Vietnam veterans with Posttraumatic Stress Disorder. *Psychiatry Research, 73*, 103-107.
- Orr, S.P., Lasko, N.B., Shalev, A.Y., & Pitman, R.K. (1995). Physiologic responses to loud tones in Vietnam veterans with posttraumatic stress disorder. *Journal of Abnormal Psychology, 104*, 75-82.

- Orr, S.P., Metzger, L.J., Lasko, N.B., Macklin, M.L., Hu, F.B., Shalev, A.Y., et al. (2003). Physiologic responses to sudden, loud tones in monozygotic twins discordant for combat exposure. *Archives of General Psychiatry*, *60*, 283-288.
- Orr, S.P., Metzger, L.J., Lasko, N.B., Macklin, M.L., Peri, T., & Pitman, R.K. (2000). De novo conditioning in trauma-exposed individuals with and without Posttraumatic Stress Disorder. *Journal of Abnormal Psychology*, *109*, 290-298.
- Orr, S.P., Meyerhoff, J.L., Edwards, J.V., & Pitman, R.K. (1998). Heart rate and blood pressure resting levels and responses to generic stressors in Vietnam veterans with posttraumatic stress disorder. *Journal of Traumatic Stress*, *11*, 155-164.
- Orr, S.P., Pitman, R.K., Lasko, N.B., & Herz, L.R. (1993). Psychophysiological assessment of Posttraumatic Stress Disorder imagery in World War II and Korean combat veterans. *Journal of Abnormal Psychology*, *102*, 152-159.
- Orth, U., & Wieland, E. (2006). Anger, hostility, and posttraumatic stress disorder in trauma-exposed adults: A meta-analysis. *Journal of Consulting and Clinical Psychology*, *74*, 698-706.
- Peri, T., Ben-Shakhar, G., Orr, S.P., & Shalev, A.Y. (2000). Psychophysiological assessment of aversive conditioning in Posttraumatic Stress Disorder. *Biological Psychiatry*, *47*, 512-519.
- Piha, S.J. (1993). Cardiovascular responses to various autonomic tests in males and females. *Clinical Autonomic Research*, *3*, 15-20.
- Pitman, R.K., Lanes, D.M., Williston, S.K., Guillaume, J.L., Metzger, L.J., Gehr, G.M., et al. (2001). Psychophysiological assessment of Posttraumatic Stress Disorder in breast cancer patients. *Psychosomatics*, *42*, 133-140.
- Pitman, R.K., Orr, S.P., Fogue, D.F., Altman, B., de Jong, J.B., & Herz, L.R. (1990). Psychophysiological responses to combat imagery of Vietnam veterans with Posttraumatic Stress Disorder versus other anxiety disorders. *Journal of Abnormal Psychology*, *99*, 49-54.
- Pitman, R.K., Orr, S.P., Fogue, D.F., de Jong, J.B., & Claiborn, J.M. (1987). Psychophysiological assessment of Posttraumatic Stress Disorder imagery in Vietnam combat veterans. *Archives of General Psychiatry*, *44*, 970-975.
- Pole, N. (2007). The psychophysiology of Posttraumatic Stress Disorder: A meta-analysis. *Psychological Bulletin*, *133*, 725-746.
- Ramaekers, D., Ector, H., Aubert, A.E., Rubens, A., & Van de Werf, F. (1998). Heart rate variability and heart rate in healthy volunteers: Is the female autonomic nervous system cardioprotective? *European Heart Journal*, *19*, 1334-1341.

- Resick, P.A., Monson, C.M., & Rizvi, S.L. (2008). Posttraumatic Stress Disorder. In D. Barlow (Ed.), *Clinical Handbook of Psychological Disorders*. New York: Guilford Press.
- Richman, L.S., Bennett, G.G., & Pek, J. (2007). Discrimination, dispositions, and cardiovascular responses to stress. *Health Psychology, 26*, 675-683.
- Runnals, J.J. (2006). Exploring the cardiovascular response to anger imagery and speech in Vietnam veterans with and without Posttraumatic Stress Disorder. Unpublished master's thesis, Virginia Commonwealth University, Richmond.
- Saab, P.G., Llabre, M.M., Schneiderman, N., Hurwitz, B.E., McDonald, P.G., Evans, J., et al. (1997). Influences of ethnicity and gender on cardiovascular responses to active coping and inhibitory-passive coping challenges. *Psychosomatic Medicine, 59*, 434-446.
- Salomon, K., & Jagustyn, N.E. (2008). Resting cardiovascular levels and reactivity to interpersonal incivility among Black, Latino/a, and White individuals: The moderating role of ethnic discrimination. *Health Psychology, 27*, 473-481.
- Sarlo, M., Palomba, D., & Buodo, G. (2005). Blood pressure changes highlight gender differences in emotional reactivity to arousing pictures. *Biological Psychology, 70*, 188-196.
- Schmahl, C.G., Elzinga, B.M., Ebner, U.W., Simms, T., Sanislow, C., Vermetten, E., et al. (2004). Psychophysiological reactivity to traumatic and abandonment scripts in borderline personality and Posttraumatic Stress Disorders: a preliminary report. *Psychiatry Research, 126*, 33-42.
- Simes, R. J. (1986). An improved Bonferroni procedure for multiple tests of significance. *Biometrika, 73*, 751-754.
- Spitzer, R.L., Williams, J.B., Gibbon, M., & First, M.B. (1989). *Structured Clinical Interview for DSM-III-R (SCID)*. New York: Biometrics Research Department, New York State Psychiatric Institute.
- Suarez, E.C., Saab, P.G., Llabre, M.M., Kuhn, C.M., & Zimmerman, E. (2004). Ethnicity, gender, and age effects on adrenoceptors and physiological responses to emotional stress. *Psychophysiology, 41*, 450-460.
- Tarabrina, N.V., Lazebnaia, E.O., Zelenova, M.E., Lasko, N.B., Orr, S.F., & Pitman, R.K. (2001). The psychophysiological reactivity of workers dealing with the aftermath of the accident at the atomic power station at Chernobyl. *Journal of Russian and East European Psychology, 39*, 43-68.

- Thorpe, R.J., Brandon, D.T., & LaVeist, T.A. (2008). Social context as an explanation for race disparities in hypertension: Findings from the Exploring Health Disparities in Integrated Communities (EHDIC) study. *Social Science and Medicine*, 67, 1604-1611.
- Tolin, D.F., & Foa, E.B. (2006). Sex differences in trauma and Posttraumatic Stress Disorder: A quantitative review of 25 years of research. *Psychological Bulletin*, 132, 959-992.
- Veazey, C.H., Blanchard, E.B., Hickling, E.J., & Buckley, T.C. (2004). Physiological responsiveness of motor vehicle accident survivors with chronic Posttraumatic Stress Disorder. *Applied Psychophysiology and Biofeedback*, 29, 51-62.
- Vrana, S.R., Hughes, J.W., Dennis, M.F., Calhoun, P.S., & Beckham, J.C. (2009). Effects of posttraumatic stress disorder status and covert hostility on cardiovascular responses to relived anger in women with and without PTSD. *Biological Psychology*, 82, 274-280.
- Wessa, M., Jatzko, A., & Flor, H. (2006). Retrieval and emotional processing of traumatic memories in Posttraumatic Stress Disorder: Peripheral and central correlates. *Neuropsychologia*, 44, 1683-1696.

Appendix 1

Analysis of Variance and Covariance Tables

Table A1.

Analysis of Covariance for Baseline Heart Rate

| Source | <i>df</i> | <i>F</i> | <i>p</i> |
|---------------------------|-----------|-----------|----------|
| Major Depressive Disorder | 1 | 4.41* | .04 |
| Beta Blockers | 1 | 6.82* | .01 |
| Ethnicity (E) | 1 | 0.14 | .71 |
| Gender (G) | 1 | 0.01 | .93 |
| PTSD Status (P) | 1 | 0.21 | .65 |
| E X G | 1 | 0.92 | .34 |
| E X P | 1 | 2.02 | .16 |
| G X P | 1 | 6.24* | .01 |
| E X G X P | 1 | 7.16** | .01 |
| Error | 235 | (100.692) | |

Note. Values enclosed in parentheses represent mean square errors.

* $p < .05$. ** $p < .01$.

Table A2.

Analysis of Variance for Systolic Blood Pressure

| Source | <i>df</i> | <i>F</i> | <i>p</i> |
|-----------------|-----------|----------|----------|
| Ethnicity (E) | 1 | 8.06** | .01 |
| Gender (G) | 1 | 3.53 | .06 |
| PTSD Status (P) | 1 | 0.14 | .71 |
| E X G | 1 | 0.29 | .59 |
| E X P | 1 | 5.84* | .02 |
| G X P | 1 | 0.42 | .52 |
| E X G X P | 1 | 0.09 | .77 |
| Error | 237 | (360.82) | |

Note. Values enclosed in parentheses represent mean square errors.

* $p < .05$. ** $p < .01$.

Table A3.

Analysis of Covariance for Baseline Diastolic Blood Pressure

| Source | <i>df</i> | <i>F</i> | <i>p</i> |
|---------------------------------------|-----------|----------|----------|
| Marriage Status | 1 | 5.48* | .02 |
| History of Substance or Alcohol Abuse | 1 | 3.47 | .06 |
| Veteran Status | 1 | 1.42 | .24 |
| Ethnicity (E) | 1 | 9.22** | < .01 |
| Gender (G) | 1 | 0.36 | .55 |
| PTSD Status (P) | 1 | 0.35 | .55 |
| E X G | 1 | 0.91 | .34 |
| E X P | 1 | 3.61 | .06 |
| G X P | 1 | 0.38 | .54 |
| E X G X P | 1 | 0.27 | .60 |
| Error | 234 | (178.11) | |

Note. Values enclosed in parentheses represent mean square errors.

* $p < .05$. ** $p < .01$.

Table A4.

Analysis of Covariance for Heart Rate Change Scores During Anger Recall Period

| Source | <i>df</i> | <i>F</i> | <i>p</i> |
|---------------------------|-----------|----------|----------|
| Major Depressive Disorder | 1 | 2.35 | .13 |
| Beta Blockers | 1 | 3.71 | .06 |
| Ethnicity (E) | 1 | 0.62 | .43 |
| Gender (G) | 1 | 0.32 | .57 |
| PTSD Status (P) | 1 | 2.38 | .13 |
| E X G | 1 | 2.26 | .14 |
| E X P | 1 | 5.01* | .03 |
| G X P | 1 | 3.86* | .05 |
| E X G X P | 1 | 0.28 | .60 |
| Error | 235 | (19.66) | |

Note. Values enclosed in parentheses represent mean square errors.

* $p < .05$. ** $p < .01$.

Table A5.

Analysis of Covariance for Systolic Blood Pressure Change Scores During Anger Recall Period

| Source | <i>df</i> | <i>F</i> | <i>p</i> |
|-----------------|-----------|----------|----------|
| Veteran Status | 1 | 0.18 | .68 |
| Ethnicity (E) | 1 | 20.41** | < .01 |
| Gender (G) | 1 | 4.67* | .03 |
| PTSD Status (P) | 1 | 0.33 | .56 |
| E X G | 1 | 4.07* | .05 |
| E X P | 1 | 1.51 | .22 |
| G X P | 1 | 0.95 | .33 |
| E X G X P | 1 | 0.04 | .85 |
| Error | 236 | (69.877) | |

Note. Values enclosed in parentheses represent mean square errors.

p* < .05. *p* < .01.

Table A6.

Analysis of Variance for Diastolic Blood Pressure Change Scores During Anger Recall Period

| Source | <i>df</i> | <i>F</i> | <i>p</i> |
|-----------------|-----------|----------|----------|
| Ethnicity (E) | 1 | 10.04** | < .01 |
| Gender (G) | 1 | 6.23** | .01 |
| PTSD Status (P) | 1 | 0.07 | .80 |
| E X G | 1 | 1.27 | .26 |
| E X P | 1 | 0.62 | .43 |
| G X P | 1 | 0.14 | .71 |
| E X G X P | 1 | 0.21 | .65 |
| Error | 237 | (22.92) | |

Note. Values enclosed in parentheses represent mean square errors.

p* < .05. *p* < .01.

Vita

Alison Marie Eonta was born on May 24, 1983, in Allegheny County, Pennsylvania, and is an American citizen. She graduated from Quaker Valley High School in Leetsdale, Pennsylvania in 2001. She received her Bachelor of Arts in Foreign Affairs from the University of Virginia in Charlottesville, Virginia in 2005.