2019

Relationship Between Elevated Blood Pressure/ Hypertension in Military Personnel and the Stress of Combat Deployment

Stephen James Pinkerton
Walden University

Walden University

College of Health Sciences

This is to certify that the doctoral dissertation by

Stephen Pinkerton

has been found to be complete and satisfactory in all respects,
and that any and all revisions required by
the review committee have been made.

Review Committee
Dr. Diana Naser, Committee Chairperson, Public Health Faculty
Dr. Suzanne Richins, Committee Member, Public Health Faculty
Dr. Simone Salandy, University Reviewer, Public Health Faculty

Chief Academic Officer
Eric Riedel, Ph.D.

Walden University
2019

Abstract

Relationship Between Elevated Blood Pressure/Hypertension in Military Personnel and the Stress of Combat Deployment

by

Stephen Pinkerton

MPH, University of Oklahoma, 1998

BS, University of Oklahoma, 1992

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Epidemiology

Walden University

May 2019

Abstract

Few studies about elevated blood pressure in the U.S. military exist in which researchers examined exposure to combat and its association with elevated blood pressure. The purpose of this quantitative research was to describe the extent of association between those who were exposed to combat deployment, were 40 or older, and were overweight or obese and had elevated blood pressure for U.S. military personnel who deployed to an area of declared combat between 2012 and 2017. The conceptual basis of this research was best represented by the determinants of health model. Chi-square correlation revealed that being older (equal to or greater than 40 years; $p = .018$) and being overweight/obese (body mass index [BMI] equal to or greater than 25; $p = .000$) both have statistically significant relationships with elevated blood pressure (either systolic blood pressure equal to or greater than 120 mm/hg or diastolic blood pressure equal to or greater than 80 mm/hg) among military personnel, while combat deployment does not (deployment > 30 days; $p =. 487$). However, only being overweight/obese remained significant even when controlling for exposure to combat deployment and being older. Binary logistic regression revealed that elevated blood pressure/hypertension is greater than three times more likely to occur in the presence of the overweight/obese predictor (BMI equal to or greater than 25; $p =. 000$) variable. The findings of this research could be used to proactively enforce medically derived appropriate medical fitness standards such as maintenance of normal BMI during deployments. Social adaptations could be instrumental in improving wellness among deployed military personnel.

Relationship Between Elevated Blood Pressure/Hypertension in Military Personnel and the Stress of Combat Deployment

by

Stephen Pinkerton

MPH, University of Oklahoma, 1998

BS, University of Oklahoma, 1992

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Epidemiology

Walden University

May 2019

Dedication

This work is dedicated to Lorri Anne Pinkerton. Without your love, sacrifice, and support this would not have been possible. I love you.

Acknowledgments

I must also acknowledge Dr. Diana Naser. At the time that we met I was entirely prepared to quit. Your honest advice and sincere help enabled me to reach this point in my academic career. Thank you.

Table of Contents

List of Tables

List of Figures

Chapter 1: Introduction to the Study

Introduction

The military profession is both physically and psychologically taxing, and combat deployment intensifies the stress of this occupation (Granado et al., 2009; Smith et al., 2008; Smoley, Smith, & Runkle, 2008; Wenzell, Pacheco de SouzaI, & Buongermino de Souza, 2009). Recently the Department of Defense (DOD) has increased the length and frequency of deployments to areas of declared combat for U.S. military personnel (Spera, Thomas, Barlas, Szoc, & Cambridge, 2011). Adequate support for active duty military personnel is crucial to promote safe pursuit of combat professions while avoiding health detriments.

This research examined the extent of association between exposure to combat deployment (independent variable), and the incidence of elevated blood pressure/hypertension (either systolic blood pressure equal to or greater than 120 mm/hg or diastolic blood pressure equal to or greater than 80 mm/hg; dependent variable) among military personnel. The purpose of this research was to produce an accurate description of the extent of association between exposure to combat deployment, being overweight/obese, being older, and hypertension among U.S. military personnel who deployed to an area of declared combat between the years 2012 and 2017. This research was needed because of the importance of identifying potentially modifiable sources of elevated blood pressure/hypertension, a high-risk disease that plays a significant role in heart, vascular, and kidney disease and the generation of heart attack, stroke, or aneurysm.

It was my aspiration and aim that the results obtained from the study would encourage positive social change by raising awareness among individual military personnel and within the community of the DOD about how the psychosocial stress of the combat environment affects a soldier's health. This heightened awareness of the increased risk concomitant with military deployment could encourage individual service members to alter personal risk factors that can generate elevated blood pressure/hypertension (Granado et al., 2009). Moreover, it could challenge the U.S. military as an organization and people throughout U.S. society at large who may care about the combat arms profession to champion and campaign for shorter and less frequent combat deployments at the policy level. These adaptations could be instrumental in improving wellness through the reduction of elevated blood pressure/hypertension risk among deployed military personnel.

In the remainder of this chapter, I review the relevant background and state the problem, purpose, and questions of the research. Additionally, the chapter contains the conceptual framework and nature of the study. Finally, there is a list of important definitions, and an exploration of assumptions, scope, delimitations, and limitations.

Background

Instead of stasis, life is perpetually dynamic, always adapting to ease existence in different circumstances (Griffin & Thomson, 1998). This foundational idea upon which the concept of stress is built started with the recognition that circumstances continually cause changes in people and the way in which they live their lives (Griffin & Thomson, 1998). Supplemental and limiting this concept of stress adaptation was Bernard's (as cited in Griffin & Thomson, 1998) postulation that people also resist changing too much in response to an exposure. Cannon (1932) called Bernard’s notion *homeostasis*, meaning a physiological tendency toward equilibrium and additionally, eventually advanced the notion of an automatic fight or flight response to stress. These all contributed to currently held ideas of the interactions between stress and internal responses to stressful stimuli.

Selye is the father of the modern concept of human stress. Selye (1950) described stress as a vague reaction to any adverse circumstance. Today, however, it is believed that stressors in humans are able to stimulate the release of very specific chemicals such as noradrenaline, serotonin, corticotropin-releasing hormone, vasopressin, and cortisol that subsequently can exert a variety of effects from vigilance and alertness to memory or genetic alterations (Joëls & Baram, 2009). The sum and sequence of these chemicals and their actions compose the stress reaction, which allow human adaptation to the environment.

A specific example of a human stress response relevant to this study is the involuntary and automatic changes to blood pressure and heart rate during and after stress. Stress reactions can initially elevate blood pressure and heart rate as adaptive survival mechanisms. However, unimpeded elevations can result in physiologic harm (Charney, 2004; Chiong, 2008; de Kloet et al., 2005). Therefore, these initial blood pressure and heart rate elevations must have caps of time, pressure and rate. These restraints are protective mechanisms in which a secondary physiological response occurs directed at restoring blood pressure and heart rate back to original functional levels (Charney, 2004).

Joëls & Baram (2009) describe the classic stress reaction as immediately creating attentiveness and watchfulness from the release of noradrenaline, serotonin, dopamine and corticotropin-releasing hormone (CRH), followed by sustained endurance or functional adaption produced by corticosteroids. These two components of the reaction occur and are mediated over varying spans of time by means of effects of the different chemicals. This description provides for the possibility of understanding a mechanism for how repetitive exposures of military personnel to the stressors of war could accumulate effects over a 6 to 12-month combat deployment.

Unfortunately, there is a scarcity of studies designed for the examination of the relationship between stress and elevated blood pressure/hypertension among military personnel. Granado et al. (2009) classified a part of the Millennium Cohort, a population-based random sample of 8829 previously deployed US military members as hypertensive. However, this classification was solely based on self-report and the presence of prescribed medications. The relatively few other investigations of the prevalence of elevated blood pressure/hypertension among military personnel were designed so researchers could determine if elevated blood pressure is associated with weight, gender, genetics, tobacco, and activity (see Granado et al., 2009; Shohat et al., 1989; Smoley et al., 2008; Wenzell et al., 2009). Hence, a research deficiency existed in terms of the investigation of the problem regarding elevated blood pressure/hypertension and stress among military personnel. Addressing this gap in knowledge was the reason for this study.

My idea is that elevated blood pressure/hypertension, like any cardiovascular disease state, could occur subject to an exposure, even a stressful combat exposure. Additionally, an aggregation of stressful exposures occurring over a combat deployment could compound blood pressure elevation and ultimately result in new onset of elevated blood pressure/hypertension, or exacerbation of existing and previously controlled elevated blood pressure/hypertension (Foraster et al., 2014; Jorgensen, 2009; Kuchel, 2003). Moreover, in response to these combat stressors, additional harm could occur through the promotion of unhealthy behaviors that could lead to obesity, hyperlipidemia, and nicotine dependence (Lester et al., 2010; Ray, Kulkarni, & Sreenivas, 2011; Smoley et al., 2008).

Holman et al. (2008) found that the elevated stress experienced by people in the United States exposed to the terror attacks of September 11, 2001 resulted in a surge in the incidence of elevated blood pressure/hypertension diagnosed by physicians. Muller et al. (1989) said that thromboses resulting in heart attacks, cerebrovascular accidents, and death could be brought about or triggered by mental or physical stress such as anger, performance of mental arithmetic, cigarette smoking, exercise and exposure to cold. It was clear then that this study could provide additional information in the form of objective data that was currently lacking in the scholarly literature. The results could be important in establishing combat stress as a causal and potentially modifiable source of elevated blood pressure/hypertension in view of the fact that it plays such a significant role in the generation of life-threatening phenomena.

Problem Statement

Human life is fraught with hazards and risks. One of those risks is interference with the perpetuation of internal functions that contribute to the maintenance of life. One potential problem for humans is that blood pressure must be maintained within set parameters. Disruption to the maintenance of blood pressure within those parameters is a general problem that commonly occurs as a link in a sequential chain of events, as is the case, for example, with obesity. Elevated blood pressure/hypertension involves the development of an impairment in excretion of sodium in the urine (Hall, 2003). Furthermore, any precipitant to elevated blood pressure/hypertension is both a real hazard and a major life risk. Elevated blood pressure/hypertension is a central influence in the development of many maladies, including heart, vascular, and kidney disease and represents a principal source of acute events leading to premature death due to occurrences such as myocardial infarctions and cerebrovascular accidents (Ezzati et al., 2002; Kearney et al., 2005; Mensah & Brown, 2007).

Elevated blood pressure/hypertension is associated with chronic kidney dysfunction and obesity (Hall, 2003; Hall et al., 2012). Elevated blood pressure/hypertension has also been associated with obesity and age (Cutler et al., 2008; Fields et al., 2004; Kannel, 2000). Stress such as that produced by exposure to combat could generate elevated blood pressure/hypertension (Granado et al., 2009; Kuchel, 2003; Melby, 1983).

13% of the U.S. military population have hypertension and 62% have elevated blood pressure, suggesting that elevated blood pressure/hypertension is a problem among military personnel (Smoley et al., 2008). Research investigating elevated blood pressure/hypertension among military personnel is scarce. Hence, there was a deficiency of scholarly findings regarding the specific problem of elevated blood pressure/hypertension among military personnel.

Moreover, no research investigating elevated blood pressure/hypertension among military personnel was examining the relationship between stress produced by exposure to combat deployment and the new onset of elevated blood pressure/hypertension in those same military personnel. Furthermore, there was no known research based upon objective and appropriately time-sequenced blood pressure data regarding the relationship between elevated blood pressure/hypertension and the stressful exposure of military personnel to combat deployment. This study offered an opportunity to address that significant dearth in the scholarly literature. This investigation was also an acknowledgement of the lack of objective and temporally relevant data being used in the scholarly literature about this subject. These data issues are fundamental to knowledge that proceeds from observations to the deduction of probable causes.

Purpose of the Study

The purpose of this research was to describe the extent of association between exposure to combat deployment and elevated blood pressure/hypertension (the mean of two recorded blood pressure measurements demonstrating either a systolic blood pressure equal to or greater than 120 mm/hg or diastolic blood pressure equal to or greater than 80 mm/hg) among U.S. military personnel who deployed to an area of declared combat between the years 2012 and 2017. In this investigation, the dependent variable was the deployed soldiers’ blood pressure measured in millimeters of mercury. The dependent variable is influenced by the physical or psychological stress experienced during combat deployment. The experience of combat deployment measured in days of combat deployment was the primary independent variable. Additionally, I designed this study to examine the effects produced by relationships between the dependent variable (blood pressure levels) and other possible predictive or explanatory independent variables such as age and BMI.

Research Questions and Hypotheses

Subsequent to a thorough assessment of the current scientific literature, analysis of the problem, and consideration of the purpose of the study, the primary research questions were developed to explore the extent of association that exposure to combat deployment, being overweight/obese, and being 40 years of age or older have with elevated blood pressure in active duty military personnel. For each research question, I specified null (H_0) and alternative (H_A) hypotheses. H_0 states personnel exposed will have the same or lower prevalence of hypertension as those not exposed. H_A states personnel exposed will have higher prevalence of hypertension than those not exposed.

RQ1: What was the extent of the association between exposure to combat deployment (completing greater than 30 consecutive days of deployment to a geographic area of declared combat) and new onset elevated blood pressure/hypertension (the mean of two recorded blood pressure measurements demonstrating either a systolic blood pressure equal to or greater than 120 mm/hg or a diastolic blood pressure equal to or greater than 80 mm/hg) among active duty military personnel?

H_01: Active duty military personnel exposed to combat deployment have the same or lower prevalence of elevated blood pressure/hypertension as those active duty military personnel not exposed to combat deployment.

H_A1: Active duty military personnel exposed to combat deployment have a higher prevalence of elevated blood pressure/hypertension than active duty military personnel not exposed to combat deployment.

RQ2: What was the extent of the association between being overweight/obese (BMI equal to or greater than 25) and new onset elevated blood pressure/hypertension (the mean of two recorded blood pressure measurements demonstrating either a systolic blood pressure equal to or greater than 120 mm/hg or a diastolic blood pressure equal to or greater than 80 mm/hg) among active duty military personnel?

H_02: Active duty military personnel exposed to being overweight/obese have the same or lower prevalence of elevated blood pressure/hypertension as those active duty military personnel not exposed to being overweight/obese.

H_A2: Active duty military personnel exposed to being overweight/obese have a higher prevalence of elevated blood pressure/hypertension than those active duty military personnel not exposed to being overweight/obese.

RQ3: What was the extent of the association between being 40 years of age or older and new onset elevated blood pressure/hypertension (the mean of two recorded blood pressure measurements demonstrating either a systolic blood pressure equal to or greater than 120 mm/hg or a diastolic blood pressure equal to or greater than 80 mm/hg) among active duty military personnel?

H_03: Active duty military personnel exposed to being 40 years of age or older have the same or lower prevalence of elevated blood pressure/hypertension as those active duty military personnel not exposed to being 40 years of age or older.

H_A3: Active duty military personnel exposed to being 40 years of age or older have a higher prevalence of elevated blood pressure/hypertension than those active duty military personnel not exposed to being 40 years of age or older.

Conceptual Framework

The Health Determinants Model of health is the most appropriate framework for understanding this research. This conceptual model of health describes human health as being influenced through feedback loop relationships (VanLeeuwen, et al., 1999). The idea is that human health is affected by the circumstances of life, including all the events occurring over time and the physical conditions in which they are experienced. More specifically, the feedback loop relationship of recurrent stress from the circumstances of life and the inherent response to it is continuous change that ultimately affects health.

This study was built on Selye's (1950) concept of the general adaption syndrome which described one protective reaction to noxious stress as a peripheral vasoconstriction and elevation of heart rate resulting in a rise in blood pressure. This idea was furthered by Granado's (2009) postulation that psychosocial stress from combat deployment could induce and intensify high blood pressure. Ultimately, what Granado (2009) found was service members exposed to combat were at risk for new onset hypertension.

These findings led to my idea of obtaining actual blood pressure measurements taken on a population of active duty service members before and after deployment to see if elevated blood pressure/hypertension is associated with exposure to combat deployment. Additionally, I planned to obtain age, height, and weight measurements on those same deploying service members to compare the associations of deployment, obesity, and age on elevated blood pressure/hypertension. I postulated that this information could supply evidence supporting Granado's (2009) finding that stressors of combat deployment produce elevated blood pressure/hypertension.

Nature of the Study

This study is a quantitative analysis of the incidence of elevated blood pressure/hypertension occurring over time in a representative group of a larger deployed U.S. military. It measured multiple exposures (independent variables) and one outcome (dependent variable) over time in all the population members. The primary independent variable was combat deployment and the other independent variables were being overweight/obese and being old. The dependent variable was the mean of the first two recorded blood pressure measurements collected at or after the post deployment medical evaluation (Time 2 or T2) in comparison to the mean of the last two recorded blood pressure measurements collected at or before the predeployment medical evaluation (Time 1 or T1). The blood pressure data originated from recordings of objective physical measurements by healthcare personnel during pre and post deployment physical examinations.

Data representing these dependent and independent variables were collected from the Defense Data Manpower Center (DMDC) database, the Military Health System Data Repository (MDR), a Defense Health Agency (DHA) database and the Armed Forces

Health Longitudinal Technology Application (AHLTA) electronic health record, all of which are maintained by the DOD. This particular research was the first study specifically including the measurements of blood pressure, height, and weight, and recording of date of birth performed and recorded at pre- and post-deployment physical examinations by qualified healthcare personnel rather than subjective questionnaire answers. Granado (2009) recommended collection of objective data rather than use of subjective questionnaires for evaluating military deployment and elevated blood pressure/hypertension diagnosis.

Definitions

Deployment: Completing greater than 30 consecutive days of residence in a geographical area of declared combat as defined by the U.S. Army's Central Command personnel deployment qualification document (Office of the Secretary of Defense, 2011).

Elevated blood pressure/hypertension: Persistently elevated blood pressure as measured by the mean of two blood pressure recordings greater than 120mm Hg systolic and/or 80mm Hg diastolic (Chobanian et al., 2003).

Human stress: Phenomena where certain threatening stimuli or stressors are able to be recognized by the human body, which then subsequently is able to create a response in reaction to the presence of those stressors (Bijlsma & Loeschcke, 2005; Dallman, 2003; Griffin & Thomson, 1998; Koolhaas et al., 2011). This reaction can occur secondary to either a single stimulus or a sequence of elements forming a stimulating event. Ordinarily when a person is exposed to a stressor it either immediately or eventually generates a measurable physiological response, such as elevated respiratory rate, heart rate, and blood pressure.

Military personnel: Military personnel are those individuals directly serving on active duty in any of the armed forces in the DOD (Office of the Secretary of Defense, 2011).

Normal weight: Normal weight is defined as a BMI of 24.9 or less. Overweight and obese are defined as a BMI that is equal to or greater than 25 (Pi-Sunyer et al., 1998).

Assumptions

People self-reporting medical diagnoses and health behaviors can be influenced by their circumstances and state of mind. These reports are usually accurate unless there is a reason to believe that a negative consequence would arise from a truthful report or a benefit would accompany a false report, thereby creating a self-reporting bias to answer falsely (Brener, Billy, & Grady, 2003). An assumption is made that the information obtained from any of the DOD databases are both accurate and precise. Another assumption is that the overall size of the active duty U.S. military employee population potentially subject to combat deployment by virtue of the terms of their employment provides a sufficient heterogeneity of subject characteristics, thus increasing the statistical power to identify associations between exposure to combat (independent variable) and elevated blood pressure/hypertension (dependent variable).

Scope and Delimitations

This study included the process of identifying a representative target population, and then collecting, analyzing, and interpreting data from that representative group and finally extrapolating the findings back to the target population as a whole. The target population for this study included all U.S. DOD personnel (military or civilian) who deployed to an area of declared combat operations. The inclusion criteria for the active duty service member sample population required that individuals meet DOD specific deployment qualifications along with recordings of blood pressure measurements (two performed pre and two post deployment), height and weight measurements, and date of birth. These criteria ensure the results of this study are relevant and apply to all deployed U.S. military or civilian men and women.

The underlying theory recognized as the basis for this research was the Health Determinants Model of health. Theories of health offer a coherency to understand how the varied circumstances of life affect human health. Several theories were reviewed but only one other was closely considered: health belief theory. The health belief theory describes how a person's unique perception of their health situation and their environment affects their health (Rimer & Glanz, 2005). It asserts that health occurs because of an individual's confidence in their ability to adapt. Ultimately this theory was not chosen because of my desire to focus on the response to stressful circumstances irrespective of an individual's own confidence in their ability to adapt.

Limitations

The following are design element limitations of this study which could provide evidence of more specific associations with blood pressure changes. One was the dependent variable evaluation based on dual pre and post deployment blood pressure measurements, which provides for recognition of only linear changes. This design prohibits discerning curvilinear trends e.g. initially normal blood pressure that elevates to hypertensive levels secondary to a stressful event, then resolves to normal blood pressure prior to post deployment blood pressure measurement. Another limitation was not parsing combat and noncombat experiences and not differentiating between personally fighting with or killing the enemy, witnessing death, and facing fear of imminent death during a deployment. Additionally, not being able to measure the total number of deployments that an individual service member has completed removes the researcher's ability to discern the impact of cumulative deployments on blood pressure. Finally, only being able to obtain an eligible study population of 308 U.S. active duty service members instead of the G*Power recommended sample size of 395 decreases the statistical power of this research.

Significance

Elevated blood pressure/hypertension is a prominent risk factor for cardiovascular disease, which is one of the greatest causes of death in the world, and yet it is amenable to simple and relatively inexpensive medical treatment (Ezzati et al., 2002; Kearney et al., 2005; Lopez et al., 2006; Mensah & Brown, 2007). Many of elevated blood pressure/hypertension's concomitant risks are a consequence of the absence of symptoms in a great portion of people with the disease. This is the reason elevated blood pressure/hypertension continues to have low rates of diagnosis and the reputation of being a silent killer. However, this deadly factor can remain present even after diagnosis because of insufficient medical therapy (Chiong, 2008).

Deployed military service members are exposed to the unpredictability and peril of combat and must physically and psychologically adjust to these stressful circumstances. These adjustments are made in order to survive and thrive. One automatic human response to experiencing combat can be a rapid elevation of heart rate and constriction of blood vessels (al'Absi & Wittmers, 2003; al'Absi et al., 2002; Bruner & Woll, 2011; Granado et al., 2009; Selye, 1950). Repeated and/or prolonged exposure to stress stimuli generating recurrent and protracted responses may culminate in the development of permanent elevated blood pressure/hypertension.

This study contributes knowledge regarding the extent of the association between stressful combat deployment and new onset of elevated blood pressure/hypertension as well as exacerbation of previously controlled elevated blood pressure/hypertension in military personnel. Elevated blood pressure/hypertension can cause serious harm without experiencing symptoms (Chiong, 2008). Therefore, if this disease is allowed to progress unrecognized and untreated, then disability or death are reasonable and predictable results.

Hence, increasing awareness in the DOD of inherent elevated blood pressure/hypertension risk for military personnel during a combat deployment is both conspicuous and salient in order to decrease hypertension mortality and morbidity. Moreover, this study could increase awareness throughout the DOD about how exposure to a combat deployment can affect soldiers' health, encouraging individual service members to alter risk factors for elevated blood pressure/hypertension, and inspiring military organizations, military communities, and even U.S. society at large to initiate a grassroots political change to demand shorter and less frequent combat deployments. These social adaptations are instrumental in improving wellness among deployed military personnel.

Summary

High blood pressure is an explicit threat to personnel in the United States military (Granado et al., 2009; Smoley et al., 2008). The occurrence of elevated blood pressure/hypertension has multiple sources and is incurable. Moreover, only a few studies about elevated blood pressure/hypertension in the U.S. military population have been done, and even fewer which examine combat stress and its association with elevated blood pressure/hypertension. The problem that generated this study was a deficiency of information in the scholarly literature about whether hypertension has a causal link to exposure to a combat deployment. Chapter 2 is a review of the significant literature relevant to elevated blood pressure/hypertension in military personnel after exposure to combat deployment as well as theories of stress and elevated blood pressure/hypertension. Moreover, this literature review will discuss the relationship between overweight/obesity, tobacco use, age, gender, sleep disordered breathing, mental conditions (stress, PTSD, depression, anxiety), pain syndromes, combat exposure, number of deployments, and alcohol/substance abuse and elevated blood pressure/hypertension.

Chapter 2: Literature Review

Introduction

The literature on the relationship between stress and elevated blood pressure/hypertension is limited and scarce. As a result, many questions about the health effects of personal stress remain unanswered. Additionally, research on stress and its relationship to health in humans has defied many scientific attempts at explaining its causes and effects with precision. This limitation is particularly pertinent for military personnel who are required to experience long and successive deployments. Military service does not come without fears of unknown vulnerabilities to physical or emotional attack or harm.

Military men and women who actively serve in the army are a diverse group. They come out of and typify many demographics in the United States. This makes them a powerful and compelling research population. However, their uniqueness does not occur as a consequence of who they are or what part of the population they represent. It relates much more to the exposures they encounter during the discharge of their duties. These include subjection to physical injury from environmental heat and cold, enemy gunfire and explosives, burn pits, oil fires, viruses, bacteria, and other toxins. They also include vulnerability to psychological trauma from fear, sleep deprivation, and seeing violence, death, and dismemberment (Hakre et al., 2010; Helmer et al., 2007; King et al., 2011; Petruccelli et al., 1999).

The question of how stress influences blood pressure is relevant in today's volatile international relations environment and by those in the military who are required to respond and endure long and successive deployments. Information from this review should be of great interest and help to military commanders and planners who are designing and strategizing ways to more effectively prepare and support military personnel. The studies were representative and include those relevant to the military context.

Literature pertaining to elevated blood pressure/hypertension in military personnel after combat deployment was reviewed in this chapter to provide an appropriate understanding of elevated blood pressure/hypertension and stress as arbiters of ill health. Additional details are furnished regarding the contributions of stress in the genesis and perpetuation of elevated blood pressure/hypertension, and more specifically the role combat deployment plays in the etiology of elevated blood pressure/hypertension in military personnel. It was derived from conveniently available and apposite research. The purpose of this research was to describe the extent of association between exposure to combat deployment, being old, being overweight/obese and elevated blood pressure/hypertension among U.S. military personnel who deployed to an area of declared combat between 2012 and 2017. This research was a quantitative investigation.

Through the referenced literature, stress was defined and explained, accompanied by relevant historical background information. Additionally, Selye’s theory of stress was clarified. An examination of the physiology of stress and its causes and effects was also provided. Finally, a description of the nature of hypertension was stated, incorporating a synopsis of its etiology and pathophysiology, its association with stress and deployment to an area of combat, and epidemiological research concerning hypertension in military populations. All of these were examined as they relate to the primary research questions.

Search Strategy

A reference library of research articles was acquired from a computerized search of health sciences databases (Medline with full Text, CINAHL Plus, PsychINFO, and PubMed at Walden University Library) using the combination of key words—*elevated blood pressure/hypertension*, *stress*, *military*, *deployment*, and *combat* for articles published between 2008 and 2015. That search found an initial and small collection of four full text and peer-reviewed articles per Ulrichsweb Global Serials Directory and one ProQuest dissertation abstract. However, only one of these demonstrated a direct correlation with this research and it was from that article a nascent list of additional articles was initially obtained. This literature search revealed the dearth of research that specifically considers how combat stress is associated with elevated blood pressure/hypertension.

Conceptual Framework

The Health Determinants Model of health describes human health as being influenced through feedback loop relationships (VanLeeuwen, et al., 1999). This conceptual model of health states that human health is affected by the circumstances of life, including all the events occurring over time and the physical conditions in which they are experienced. More specifically, the feedback loop relationship of recurrent stress from the circumstances of life and the inherent response to it is continuous change that ultimately affects health (Walter & Ron, 2011).

Along with determining a functional research model, it is important to remain cognizant of real difficulties in performing medical research. Research is ultimately a search for truth to substantiate knowledge that can then be used to decrease mortality and morbidity. However, discovering isolated truth can create new problems. An example is in the recognition of new risk relationships between specific factors and diseases. This is significant because once a link is established, people desire to reduce their risk by implementing change. The limitation is soon realized when despite change, risk for people still exists. This is because despite having an awareness of many risks, people still may lack a full comprehensive understanding of causation.

Another complication exists with diseased people themselves. Even when they understand that their risk is associated with a specific exposure or behavior, eliminating that exposure or changing themselves and what they have been doing to make their risk go away is an arduous task. There is no shortage of examples of distraught people failing to change their behavior even though they are well acquainted with both the hazard and the risk.

In addition to the determinants of health model which emphasized the recurrent effect of stress on people's lives and an appreciation of the medical research process and its population relevance, the conceptual framework of this research also required a thorough understanding of stress itself. However, studying this state of physical and psychological tension in human life is complex and its causes and effects are difficult to explain with precision. These mystifying circumstances produce many questions that remain objectively unanswered about the health effects of stress. Currently stress is commonly understood as the body's reaction to a stressor, whether it is the prodding of a single stimulus or the impetus of sequential factors comprising an event (Bruner & Woll, 2011; Chrousos, 2009; Cohen, Janicki-Deverts, & Miller, 2007; Dallman, 2003; Day, 2005; de Kloet et al., 2005; Joëls & Baram, 2009; Karatsoreos & McEwen, 2011). Ordinarily, when a person is exposed to a stressor, it either immediately or eventually generates a measurable physiological response, such as elevated respiratory rate, heart rate, and blood pressure.

Selye was the architect of this idea that stress causes a physiological response in humans (Viner, 1999). He started with Cannon's thoughts that animals respond with a constriction of blood vessels and an elevated heart rate when they appear to experience fear or pain (Cannon, 1932). Cannon's thoughts, in turn, originated from Bernard's animal vivisection experiments in physiology (Gross, 1998).

However, historically stress was a concept with a muddled scientific understanding (Bijlsma & Loeschcke, 2005; Mustacchi, 1990). Almost as soon as it was first absconded out of the field of physics—as a quantification of forces that could deform metal—by Selye to use in the biological sciences, its meaning was challenged by others (Bijlsma & Loeschcke, 2005; Griffin & Thomson, 1998; Karatsoreos & McEwen, 2011; Koolhaas et al., 2011; Rosmond, 2005). Selye initially described stress as opposing actions of harm and protection occurring simultaneously on one another (Bijlsma & Loeschcke, 2005; Karatsoreos & McEwen, 2011; Koolhaas et al., 2011; Rosmond, 2005; Selye, 1950; Viner, 1999). Nevertheless, today no known broad scientific agreement exists as to an all-encompassing and specific definition of stress (Bijlsma & Loeschcke, 2005; Karatsoreos & McEwen, 2011; Koolhaas et al., 2011; Rosmond, 2005).

The foundational ideas upon which the concept of stress is built can be argued to have begun with early Greek philosopher scientists (Griffin & Thomson, 1998). These intellectual giants proposed a life that never adapts, never changes—is not normal. A normal life is one that weathers all of life's stressful exposures, sometimes imperceptibly and at other times dramatically, but always adapting in some measure to life's exigencies (1998).

Bernard, because of what he saw while performing live vivisections on animals, proposed his notion that a living body had an as of yet unidentified mixture of internal substances with the ability to stabilize against the adverse effects of stress (Griffin & Thomson, 1998; Gross, 1998; Rosmond, 2005). He described this ability as essential for a life that is exposed to persistent external environmental variability. Building on Bernard's findings, Cannon gave it a name that stuck—"homeostasis" and later advanced another idea and name for a specific stress response—"fight or flight" (Cannon, 1932; Griffin & Thomson, 1998; Koolhaas et al., 2011; Rosmond, 2005).

It was Selye, however, who ultimately came up with a more complete concept of the physiology of stress with his description of a four phased stress reaction (Griffin & Thomson, 1998; Selye, 1950; Viner, 1999). This sequential occurrence proceeds from alarm, to resistance, then adverse effects and finally exhaustion (Griffin & Thomson, 1998; Selye, 1950). However, not everyone has been willing to placidly agree with Selye (Bijlsma & Loeschcke, 2005; Karatsoreos & McEwen, 2011; Koolhaas et al., 2011;

Kuchel, 2003; Mustacchi, 1990). This lack of agreement in the scientific community resulted in a much more simplified popular understanding of stress today as a disparity produced when external demands exceed a person's ability to handle them (Bijlsma & Loeschcke, 2005; Koolhaas et al., 2011; Rosmond, 2005).

At the outset of this debate over stress, it was initially considered a common reaction to any pernicious exposure (Koolhaas et al., 2011). Then the concept was improved by differentiating the stimulus from the response (Bijlsma & Loeschcke, 2005). Later refinement added that when humans sense the possibility of something harmful happening (the stimulus), the brain releases chemicals which then act on specialized nerve cells (the response). The collaboration of these separate actions composes the stress reaction, which provides the means to adapt to change (Joëls & Baram, 2009).

Opposing that adaptation ability is the stable equilibrium which all strive to preserve. This was classically referred to as homeostasis—a concept initially conceived and developed by Cannon (Charney, 2004; Koolhaas et al., 2011; Neylan, 1998). Individuals all have internal processes that though variable, are inherently designed to maintain a stable equilibrium (Charney, 2004; de Kloet et al., 2005; Mustacchi, 1990). Some prominent examples are the involuntary and automatic maintenance of blood pressure and blood glucose at functional levels. Cannon contended that these processes have individual preset limits, beyond which a physiological response occurs directed at restoring the functional level (Karatsoreos & McEwen, 2011; de Kloet et al., 2005; Koolhaas et al., 2011).

A number of researchers publicly disagreed with Selye and Cannon and their proposals of stress and homeostasis. Most by focusing their attention on what they believe to be the vagueness of the definition and the fallacious notion that stress is a risk to maintaining homeostasis (Day, 2005; Dickerson & Kemeny, 2004; Koolhaas et al., 2011; Levine, 2005; McEwen, 2007; Romero, Dickens, & Cyr, 2009). Cannon's notion was that there are many functioning systems within the human body. For example, blood pressure, which has preferred ranges of normality. An increase or decrease outside of that normal range induces an automatic physiological action against it to restore the system to its optimal level. Other researchers make the valid point that all physiological activity relates either directly or indirectly to homeostasis (Day, 2005; Levine, 2005; McEwen, 1998; Romero et al., 2009). Therefore, understanding stress as threatening to homeostasis lacks real meaning.

Some researchers add the idea that the perceptual processing of the stimulus is extremely significant (Levine, 2005). Many studies neglect this aspect of the stress response that includes the mental action or process of acquiring knowledge and filtering understanding through thought, experience, and the senses. In fact, for many studies the occurrence of a stress response is the evidence that implies an exposure (Armario, 2006). Other studies and researchers, however, look at stress and the stress response as being dependent on not only the exposure to stressors but also of the essential qualities of the stressor like predictability, controllability and even intensity (Armario et al., 2012; Belda, Fuentes, Nadal, & Armario, 2008; Koolhaas et al., 2011). This research agrees that the stress response is contingent on and determined by the essential qualities of the predictability, controllability and even intensity of stimuli.

For example, some of these researchers portrayed stress as one of two types of mentally and physically demanding encounters. These can be characterized by a positive variety, which can be personally controlled and overcome, and a negative variety, which is grim and depleting to the point of being able to cause some form of harm or injury to the person experiencing it (McEwen, 2007). Many of these researchers also publicly state their preference and bias for supplanting the terms stress and homeostasis with their own terms (Karatsoreos & McEwen, 2011; Koolhaas et al., 2011; Levine, 2005; McEwen, 2007; Romero et al., 2009).

Other researchers however, even though they do not agree entirely with Seyle, encourage the continued use and evidentiary growth of the terms stress and homeostasis (Bijlsma & Loeschcke, 2005; Day, 2005; Dickerson & Kemeny, 2004; de Kloet et al., 2005). For example, many subscribe to the well substantiated notion that a reaction to stress is distinguished by two sets of mediating features characterized by the timing of their action (Joëls & Baram, 2009). One has a rapid onset and fosters personal attentiveness in an effort to reduce risk from a potential menace (Bijlsma & Loeschcke, 2005; Day, 2005; Dickerson & Kemeny, 2004; de Kloet et al., 2005). It also stimulates a critical assessment of the circumstances and the selection of the most favorable plan of action to confront and deal with the problem (Joëls & Baram, 2009; Karatsoreos & McEwen, 2011).

However, these fast-acting stress mediators are regulated to brief effects, and therefore are not favorable for providing an extended relief from stress (Bijlsma & Loeschcke, 2005; Joëls & Baram, 2009). The long-term reaction occurs by means of corticosteroids producing adaptation adjustments in specific genes and modifications in cell activity and purpose (Day, 2005; Dickerson & Kemeny, 2004; Joëls & Baram, 2009; Karatsoreos & McEwen, 2011; de Kloet et al., 2005). This chronic form of stress reaction can potentially foster a change in function that results in the emergence of a new disease process such as elevated blood pressure/hypertension from repeated exposure to stressful stimuli.

One thing not disputed is that it was Selye who developed the meaning for the prevailing stress concept (Koolhaas et al., 2011). He was active and influential in forming popular opinion about stress. This was so successful that soon stress became commonly understood to be the physiological and behavioral responses to a stressor (Selye, 1950). His notion ultimately grew into a theory of a common human adjustment to stress (Koolhaas et al., 2011). Physiologically, this adaptation has consistent hallmarks that are always present despite the specific origin or nature of the stress. The most important of which are growth of the adrenal gland; degeneration of the thymus, lymph nodes, and spleen; and disruption of the protective mucous membrane of the gastrointestinal tract (Marine, Ruotsalainen, Serra, & Verbeek, 2006).

Additionally, the idea of the predictability, controllability, and intensity of stimuli are central to the understanding of the stress response. This notion comes from experiments with rats 44 years ago (Koolhaas et al., 2011; Weiss, 1972). The research conclusion is that it is the degree of intensity, controllability, and predictability of the stimulus, not its actual nature that produces the stress response and generates stress pathology (Koolhaas et al., 2011; Weiss, 1972).

The available body of facts from human research adds a concept by making a distinct differentiation between the actual and perceived predictability and controllability of stimuli (Koolhaas et al., 2011; Salvador, 2005). Additionally, human research demonstrates that perception is the significant factor in the stress response (Koolhaas et al., 2011; Salvador, 2005). The understanding from reasoning through this knowledge is that different individuals can perceive any stimulus as either stressful or not and as one that they have the capability and resources to adapt to or not.

One activity that makes this finding significant is animal experimentation findings which may not equate to humans because perception could be considered different from an animal point of view. Finally, not only is the stressful stimulus personally assessed by its qualities of predictability and controllability, but it requires an additional evaluation regarding its intensity. These truths demonstrate that determining the severity of a stressor varies by the subjective nature of one’s perception and emotional state. These personal features additionally predict the severity and pathological consequences of the effects of stressful stimuli. A thorough understanding about the conceptual framework of stress results in the idea that a stimulus produces a stressful response because the person perceives the demand of that stimulus to exceed their capacity to adapt. This framework of stress comprehension additionally means that if there is no personal perception of excessive demand, then the response to the stimulus is normal homeostatic physiologic behavior.

Literature Review Related to Key Variables and/or Concepts

Physiology of Stress

Stress is universal and inescapable (Bijlsma & Loeschcke, 2005; Dallman, 2003). Sooner or later it has an effect on everyone (Chrousos, 2009; Day, 2005; de Kloet et al., 2005). It can create real and substantial consequences (de Kloet et al., 2005). Stress can adversely affect mental, emotional, and physical states (Charney, 2004; Chrousos, 2009; Cohen, Janicki-Deverts, & Miller, 2007; Joëls & Baram, 2009).

Military service members deployed to areas defined as combat zones can be confronted with the unpleasantness of real and extensive loss, and the very intense and personal experience of a deliberately planned threat on one's own life (Bruner & Woll, 2011). Additionally, there is an awareness of being in a position in which the chances of winning or losing are evenly balanced. These realities intensify the physical and psychological stress effect and generate powerful ethical dilemmas. Moreover, the circumstances of exposure to the field of battle are often sources of great personal agony and are experienced in an isolated context despite the presence and proximity of many others (Bruner & Woll, 2011). Exposure to these stressful circumstances can result in adaptation in unusual and sometimes physically costly ways (Bruner & Woll, 2011).

This adaptation to stress occurs through the sympathetic nervous system which is initially set in motion by mental and emotional stressors recognized through visceral and sensory stimuli in a variety of ways and can be distinguished very generally by the temporal aspect of their action—fast and slow or short and long term (Joëls & Baram, 2009; de Kloet et al., 2005). The slower stress adaptation pathway is more geared toward fueling the adaptation process and recuperating afterwards (Dickerson & Kemeny, 2004; Joëls & Baram, 2009). While the fast, short term pathway produces immediate actions originally conceptualized as the fight or flight response by Walter Cannon (Bracha, Ralston, Matsukawa, Williams, & Bracha, 2004; Griffin & Thomson, 1998; Neylan, 1998).

This fight or flight response, occasionally described with the addition of a paralysis, is a reaction to self-perceived harm (Bracha et al., 2004). It happens via the sympathetic nervous system (SNS), which also controls other functions, such as size of pupils and speed of digestion (Dickerson & Kemeny, 2004; Joëls & Baram, 2009). Principally, however, it is the cardiovascular system which is affected directly through SNS impulses and indirectly by hormones and neurotransmitters secreted from the adrenals (Dickerson & Kemeny, 2004). Corticotropin hormones also play a role and are a part of a cascade of effects that stimulate the adrenals to produce adrenaline (epinephrine) and noradrenaline (norepinephrine; Dickerson & Kemeny, 2004; de Kloet et al., 2005). Fight or flight may be the most commonly recognized of all the physiological stress responses, but it is not the only one.

Elevated Blood Pressure/Hypertension

For the past 30 plus years, the parameters of normal adult blood pressure in the United States have been decided upon by medical consensus through a national committee (Chobanian et al., 2003a; James et al., 2014; Kaplan, 1998). One of the most recent determinations is found in the "2014 Evidence-Based Guideline for the Management of High Blood Pressure in Adults: Report from the Panel Members Appointed to the Eighth Joint National Committee (JNC 8)” (James et al., 2014). The Joint National Committee (JNC) long ago established the parameters of elevated blood pressure/hypertension as the mean of two blood pressure measurements equal to or greater than 120 mm Hg systolic or 80 mm Hg diastolic (Chiong, 2008; Kaplan, 1998).

The report from JNC 7 in 2003, determined a new category of high blood pressure designated as prehypertension, to range from 120 to 139 mm Hg systolic or 80 to 89 mm Hg diastolic (Chobanian et al., 2003a, 2003b). In addition to delineating the boundaries of prehypertension, JNC 7 also clearly declared that risk for cardiovascular disease begins at 115/75 mm Hg—which is well below prehypertension pressures, and that risk increases two-fold with each gain of 20/10 mm Hg (Chiong, 2008; Chobanian et al., 2003a, 2003b). Furthermore, the risk in the United States of any person's blood pressure reaching 120/80 mm Hg or greater at some point during their lifetime is 90% (Chobanian et al., 2003a, 2003b).

However, the most recent change occurred in May 2018 when the American College of Cardiology and the American Heart Association Task Force on Clinical Practice Guidelines published their hypertension clinical practice guidelines (Whelton et al., 2017). The blood pressure categories include normal (less than 120 mm Hg systolic and less than 80 mm Hg diastolic), elevated (120 to 129 mm Hg systolic and less than 80 mm Hg diastolic), stage 1 hypertension (130 to 139 mm Hg systolic or 80 to 89 mm Hg diastolic), and stage 2 hypertension (equal to or greater than 140 mm Hg systolic or equal to or greater than 90 mm Hg diastolic; Whelton et al., 2017). Based on these new guidelines the definition of elevated blood pressure for use by this research was updated to either systolic blood pressure equal to or greater than 120 mm/hg or diastolic blood pressure equal to or greater than 80 mm/hg.

The cause of elevated blood pressure/hypertension is unknown most of the time and when this is true it is called essential (Chiong, 2008). This name means that there is no obvious source for the origin of the elevated blood pressure/hypertension. However, there are potential sources of elevated blood pressure/hypertension that occur as a function of separate disease processes or cardiovascular risks (Oparil, Zaman, & Calhoun, 2003). Their connection to elevated blood pressure/hypertension has been well documented and they include obesity/overweight, tobacco use, age, gender, sleep disordered breathing, hyperlipidemia, kidney disease, cardiovascular disease, excess salt intake, diabetes, and a persistent state of mental stress from such things as job strain, anxiety, depression, and combat exposure/deployment (Abouzeid, Kelsall, Forbes, Sim, & Creamer, 2012; al'Absi & Wittmers, 2003; Artinian et al., 2006; Babu et al., 2014; Baxi, Jackson, Ritter, & Sessums, 2011; Cheung et al., 2006; Chiong, 2008; Chobanian et al., 2003a, 2003b; Cutler et al., 2008; Devereux et al., 1983; Din-Dzietham et al., 2004; Egan et al., 2010; Jorgensen, 2009; Kannel, 2000; Kaplan, 1998; Kottke, Stroebel, & Hoffman, 2003; Landsberg et al., 2013; Leone, 2011; De Marco et al., 2009; Matthews, Salomon, Brady, & Allen, 2003; Mustacchi, 1990; Oparil et al., 2003; Pickering, 2001; Primatesta, Falaschetti, Gupta, Marmot, & Poulter, 2001; Ray et al., 2011; Scalco,

Scalco, Azul, & Lotufo Neto, 2005; Shohat et al., 1989; Smoley et al., 2008; Stamler, Stamler, Riedlinger, Algera, & Roberts, 1978; Stevens et al., 2001; Talukder et al., 2011).

Although there are many factors linked with elevated blood pressure/hypertension, it appears that impairments in kidney and sympathetic nervous system function are the predominant contributors (al'Absi & Wittmers, 2003; Oparil et al., 2003). Therefore it is the physiological activity occurring in both the sympathetic nervous system and adrenocortical system in response to psychosocial stress that plays the primary role in the genesis and continuation of blood pressure elevations in service members during combat deployment (al'Absi & Wittmers, 2003; al'Absi et al., 2002; Artinian et al., 2006; Babu et al., 2014; Bagnell et al., 2013; Bruner & Woll, 2011; Dickerson & Kemeny, 2004; Din-Dzietham et al., 2004; De Marco et al., 2009).

However, from a strictly physiological perspective, elevated blood pressure/hypertension is present only when both cardiac output is increased and peripheral resistance is raised (Chiong, 2008; Omvik, 2015). Behind these two principal physiologic features are an assortment of other components that influence or trigger their action (al'Absi & Wittmers, 2003; al'Absi et al., 2002; Babu et al., 2014; Bowman, Gaziano, Buring, & Sesso, 2007; Brooks, Horner, Kozar, Render-Teixeira, & Phillipson, 1997; Carroll, Phillips, Gale, & Batty, 2010; Cheung et al., 2005; Clark, 2000; Gelber, Gaziano, Manson, Buring, & Sesso, 2007; Granado et al., 2009; Grimsrud, Stein, Seedat, Williams, & Myer, 2009; Hall, 2003; Holman et al., 2008; Johannessen, Strudsholm, Foldager, & Munk-Jørgensen, 2006; Jonas & Lando, 2000; Kario, 2012; Landsberg et al., 2013; Leone, 2011; Ray et al., 2011; WenzelI et al., 2009; Wilson, Kliewer, & Sica, 2004). These integrate in ways that cause elevated blood pressure/hypertension to occur distinctly in different people. Moreover, because of its complexity in development, the occurrence of elevated blood pressure/hypertension is a process of gradual transformation (Oparil et al., 2003).

Risks of Elevated Blood Pressure/Hypertension

Most diseases that involve the heart and blood vessels, including coronary artery disease, stroke, and elevated blood pressure/hypertension, among others, appear to progress slowly over a period of many years. Moreover, though these diseases may begin relatively early, the clinical manifestations do not usually become evident until later in life (Flaa, Eide, Kjeldsen, & Rostrup, 2008; Treiber et al., 2003). Furthermore, these diseases produce significant morbidity and mortality in the military and worldwide. The preponderance of and most significant contributor to cardiovascular risk globally is high blood pressure, being responsible for approximately 50% of heart vessel disease and 65% of brain vessel disease (Cutler et al., 2008).

Moreover, though the causes of these diseases vary, it is important to know that most of them are preventable (Tofler & Muller, 2006). The understanding that a person's risk is estimated on the presence of specific risk factors is also important. However, along with that knowledge one must be cognizant of just how well the presence of those risk factors predicts risk (Cook, 2007). This is more important than knowing what the sensitivity and specificity are during diagnostic testing. Because, even when diagnostic testing is being performed, what is most important is whether the person has the disease or not rather than their chances of having a positive test despite whether or not they have the disease.

If a person has high blood pressure, they probably are not concerned about whether people who have a heart attack also have high blood pressure. Rather, what they want to know is what is their probability or risk of having a heart attack and dying. How well these risk factors predict the risk or probability, is more relevant information. The important and significant question to ask is: What are the chances of developing a disease in the future given the current risk factors? However, the problem is that these long-established risks such as overweight/obesity, tobacco use, elevated cholesterol and family history are unreliable at forecasting these diseases all the time. Consequently, significant work has gone into further supplementing these known risks by identifying new alternatives to advance knowledge and increase prevention.

This research was set in motion to explore risk of exposure to combat deployment on otherwise normal blood pressure of active duty military personnel. Maintenance of normal blood pressure is critical to human survival (Chiong, 2008; Chobanian et al., 2003). Dysfunction of normal blood pressure is a perpetual possibility and can occur associated with or as a consequence of an attribute or the actions of an array of events, that can best be described as risks for elevated blood pressure/hypertension (Chiong, 2008; Garrison, Kannel, Stokes, & Castelli, 1987; Gelber el al., 2007; Granado et al., 2009; Green, Jucha, & Luz, 1986; Grimsrud, Stein, Seedat, Williams, & Myer, 2009; Holman et al., 2008; Johannessen et al., 2006; Jonas & Lando, 2000; Kannel, 2000; Kannel, Brand, Skinner, Dawber, & McNamara, 1967; Kibler, Joshi, & Ma, 2009;

Landsberg et al., 2013; Lavie, Herer, & Hoffstein, 2000; Li, Law, & Power, 2007; Steptoe, 2008; Stamler et al., 1978; Vasan, Larson, Leip, Kannel, & Levy, 2001; Virdis, Giannarelli, Fritsch Neves, Taddei, & Ghiadoni, 2010; WenzelI, Pacheco de SouzaI, & Buongermino de Souza, 2009; Wilson, Kliewer, & Sica, 2004; Young et al., 1997). Therefore, any risk to elevated blood pressure/hypertension is a real hazard to human survival.

Despite knowledge of these risks, a specific risk cannot always be positively pointed to as the cause for an individual's elevated blood pressure/hypertension. Additionally, about 20% of the military population has elevated blood pressure/hypertension and near 60% have prehypertension (Granado et al., 2009; Shohat, Shohat, Mimouni, Nitzan, & Danon, 1989; Smoley, Smith, & Runkle, 2008; Wenzell et al., 2009). Moreover, the finding of this literature search reveals a scarcity of studies examining elevated blood pressure/hypertension and its associated risks among military personnel (Granado et al., 2009; Shohat et al., 1989; Smoley et al., 2008; Wenzell et al., 2009). Nevertheless, there are elevated blood pressure/hypertension risk relationships that, though not yet confirmed in the literature as specifically associated with military personnel, are still informative to this elevated blood pressure/hypertension research.

Following is a description of several of these elevated blood pressure/hypertension risk relationships. The first four relationships are well established in the scientific literature and correspond to the three research questions (al'Absi, Lovallo, McKey, & Pincomb, 1994; al'Absi et al., 1998; al'Absi & Wittmers, 2003; Bevan, Tsuru, & Bevan, 1983; Buckley, Holohan, Greif, Bedard, & Suvak, 2004; Carroll, Phillips, Gale, & Batty, 2010; Cheung et al., 2005; Coughlin, 2011; Flaa et al., 2008; Grimsrud et al., 2009; Hart, Heistad, & Brody, 1980; Johannessen et al., 2006; Jonas, & Lando, 2000; Kibler, Joshi, & Ma, 2009; Light et al., 1999; Pickering, 2001; Rutledge, 2002; Scalco, Scalco, Azul, & Lotufo Neto, 2005; Scott et al., 2007). What is the extent of the association between stress and elevated blood pressure/hypertension among military personnel who have completed a combat deployment? Does overweight/obesity (BMI equal to or greater than 25) alter the relationship between stress and elevated blood pressure/hypertension among military personnel who have completed a combat deployment? Does age alter the relationship between stress and elevated blood pressure/hypertension among military personnel who have completed a combat deployment? The last four relationships are commonly and anecdotally considered as risks among health care providers but only one (sleep disordered breathing) is well established as a risk in the literature (Brooks, Horner, Kozar, Render-Teixeira, & Phillipson, 1997; Clark, 2000; Grote, Hedner, & Peter, 2000; Lavie et al., 2000; Nieto et al., 2000; Peppard, Young, Palta, & Skatrud, 2000; Somers et al., 2008; Young et al., 1997).

Risk Relation of Stress (including Mental Conditions e.g., PTSD, Depression, Anxiety) to Elevated Blood Pressure/Hypertension

People at risk for elevated blood pressure/hypertension may demonstrate greater responses to physical and psychological stress. This exaggerated ability to react to stress may be the thing that imputes their heightened risk for elevated blood pressure/hypertension (Light et al., 1999). Ordinarily the hypothalamic-pituitary-adrenal

(HPA) coalition of glands are energized and set in motion after an exposure to an unfamiliar or stressful circumstance (Armario, 2006; Armario, et al., 2012; Koolhaas et al., 2011). This causes these glands to secrete chemicals with cascading effects on the sympathetic nervous system. This HPA activation appears amplified in some people and that amplification of response to stress may be a risk for elevated blood pressure/hypertension (al'Absi et al., 1994; al'Absi et al., 1998; Armario, 2006; Armario, et al., 2012; Koolhaas et al., 2011).

The relationship of intermittent and sporadic high blood pressure to an increased activity of the HPA glands through the production of mineralocorticoids and glucocorticoids, such as aldosterone, cortisol and androgen is familiar, but the extent to which this occurs in response to stress is not completely understood (Charney, 2004; Chrousos, 2009). Some studies have demonstrated a relationship of episodic elevations of blood pressure to a heightened functional activity of the adrenal system, which supports the possibility that a recurrent adrenal gland reactivity to stress may represent a risk for elevated blood pressure/hypertension (al'Absi & Wittmers, 2003; Charney, 2004; Flaa et al., 2008). Studies focusing on this inflated adrenal gland responsiveness, as a predictor that elevated blood pressure/hypertension will occur later, have revealed that if a person watchfully waits for enough years the association not only appears but grows stronger (Charney, 2004; Chrousos, 2009; Flaa et al., 2008). It is this increase of the response to stress itself that is the hypothesis.

It is a state of being that is characterized by recurrent and over exaggerated responses to stress—whether they are physical or psychological—that after enough time is associated with an increased risk for elevated blood pressure/hypertension (Armario, 2006; Armario et al., 2012; Koolhaas et al., 2011). Of course, this could be just an incidental finding rather than a causal relationship. One side of the concept for this condition of amped-up responsiveness states that it probably only produces episodic blood pressure elevations. However, the other side favors the notion that these intermittent elevations of blood pressure could certainly bring about a new and permanent change in vascular structure and function resulting in permanent elevated blood pressure/hypertension (Charney, 2004; Chrousos, 2009; Flaa et al., 2008). Nevertheless, animal experiments attempting to induce elevated blood pressure/hypertension exclusively from those intermittent elevations of blood pressure have not been successful (Flaa et al., 2008).

Another possible connection between this adrenal gland hyper-responsiveness to stress and the generation of actual elevated blood pressure/hypertension is the frank action and influence of epinephrine and norepinephrine (otherwise known as adrenaline and noradrenaline), and dopamine, all of which are circulated during the fight-flight response (al'Absi & Wittmers, 2003; Armario, 2006; Armario et al., 2012; Charney, 2004; Chrousos, 2009; Flaa et al., 2008; Koolhaas et al., 2011). Additionally, this recurrent adrenalized reactivity may be associated with a perpetual increase in total peripheral resistance from structural changes occurring in arterial vasculature which furthers enhances the environment for the evolution and establishment of elevated blood pressure/hypertension (Bevan, Tsuru, & Bevan, 1983; Hart, Heistad, & Brody, 1980).

The arterial wall growth found materializing after this repeated adrenal responsiveness, has also demonstrated a significant capacity to protect people from a stroke when they are experiencing extremely high elevations of blood pressure (Bevan et al., 1983; Hart et al., 1980). This protection happens as a direct result of that arterial wall development which then is able to handle and prevent damage from those extremes of blood pressure occurring in brain vessels. Moreover, the opposite of the original concept is also true: that a lack of reactivity to stress attenuates arterial wall growth and therefore avoids an increase in peripheral vessel resistance thereby subverting the onset of elevated blood pressure/hypertension (Bevan et al., 1983; Hart et al., 1980).

A persistent state of mental stress can be caused by a variety of factors. These factors may be external to an individual or emanate from within. Things like natural disasters, stressful working circumstances, and a gloomy, pessimistic frame of mind are all associated with elevated blood pressure/hypertension (Jonas & Lando, 2000; Pickering, 2001). Additionally, people who generally express themselves in defensiveness and anger also have an increased risk of elevated blood pressure/hypertension (Jonas & Lando, 2000; Pickering, 2001). Chronic gloom and pessimism can become apparent in an individual through symptoms of anxiety, depression, or enmity. A prevailing connection between all these influences and elevated blood pressure/hypertension is a sense of powerlessness that arises from the erosion of personal autonomy (Jonas & Lando, 2000; Pickering, 2001).

Some researchers have found that the normal blood pressure and heart rate response to stress is significantly intensified in the presence of post-traumatic stress disorder (PTSD; Buckley et al., 2004). Moreover, PTSD appears to be associated with a greater prevalence of elevated blood pressure/hypertension when compared to other mental conditions (Kibler, Joshi, & Ma, 2009). Some have demonstrated that PTSD is related to elevated blood pressure/hypertension separate from depression (Kibler et al., 2009). Others imply that the integral anxiety within PTSD is the key connection to elevated blood pressure/hypertension (Coughlin, 2011; Grimsrud et al., 2009). While still others have indicated that the most commonly associated mental condition related to elevated blood pressure/hypertension is depression (Pickering, 2001; Scalco, Scalco, Azul, & Lotufo Neto, 2005).

Independently, anxiety and depression have relationships with elevated blood pressure/hypertension but jointly, they have a stronger relationship and weightier health significance (Carroll, Phillips, Gale, & Batty, 2010; Scott et al., 2007). An analysis of 15 cohort studies that were each followed forward past one year demonstrated that symptoms of chronic anger, anxiety, and depression frequently precede the incidence of elevated blood pressure/hypertension (Rutledge, 2002). Nevertheless, the relationship with elevated blood pressure/hypertension is even more potent if the individual has a family history of elevated blood pressure/hypertension, is overweight or older (Cheung et al., 2005).

However, to report the literature fairly, there is research that found no real relationship between mental conditions and elevated blood pressure/hypertension (Friedman et al., 2001). One large long-term study even demonstrated that people with symptoms of anxiety and depression were found to have low blood pressure more than a decade later (Hildrum, Mykletun, Holmen, & Dahl, 2008). But most of the research literature regarding this subject has demonstrated at least some association between certain mental conditions and elevated blood pressure/hypertension (Johannessen et al., 2006).

Risk Relation of Combat Exposure and Number of Deployments to Elevated Blood Pressure/Hypertension

On the same note as the mental conditions (Stress, PTSD, Depression, and Anxiety), scientific researchers have made two crucial discoveries about the relationship between trauma experienced and a medical disorder. First, there is an incremental association between the amount of trauma experienced and the risk of a medical disorder (Sledjeski, Speisman, & Dierker, 2008). Second, experiencing a trauma such as direct life-threatening combat and the occurrence of a medical disorder such as elevated blood pressure/hypertension is best explained by the number of traumas that individual has encountered (Sledjeski et al., 2008). Together these research findings support the notion that repetitive traumas have a synergistically detrimental effect on physical and psychological health.

Risk Relation of Overweight to Elevated Blood Pressure/Hypertension

The connection between the presence of elevated blood pressure/hypertension and the state of being overweight in an individual has been well documented for more than 40 years (Bogers et al., 2007; Chiong, 2008; DeMarco et al., 2009; Drukteinis et al., 2007; Fields et al., 2004; Gelber et al., 2007; Hall, 2003; Julius, Valentini, & Palatini, 2000; Kannel et al., 1967; Kannel, 2000; Landsberg et al., 2013; Mensah & Brown, 2007;

McNiece et al., 2007; Moore et al., 2005; Must et al., 1999; Ray et al., 2011; Rosmond, 2005; Salvadori et al., 2008; Stamler et al., 1978; Stevens et al., 2001; Torrance, McGuire, Lewanczuk, & McGavock, 2007; Whelton et al., 2002; Williams et al., 1992; Wyatt et al., 2008). Researchers have demonstrated a strong relationship of correspondence between a person's weight and their blood pressure. The association appears to function with whichever of the alternatives is the case; if weight is gained increases in blood pressure are frequently the result and if weight is lost the relationship often is with a decrease in blood pressure. However, there is research demonstrating that elevated blood pressure/hypertension precedes weight gain, which may seem contradictory to the usual concept that weight gain precedes elevated blood pressure/hypertension (Julius et al., 2000).

These discoveries imply the existence of a physiological process that results in a response associated with alterations in blood pressure and in weight (Bogers et al., 2007; Chiong, 2008; DeMarco et al., 2009; Drukteinis et al., 2007; Fields et al., 2004; Gelber et al., 2007; Hall, 2003; Julius et al., 2000; Kannel et al., 1967; Kannel, 2000; Landsberg et al., 2013; Mensah & Brown, 2007; McNiece et al., 2007; Moore et al., 2005; Must et al., 1999; Ray et al., 2011; Rosmond, 2005; Salvadori et al., 2008; Stamler et al., 1978; Stevens et al., 2001; Torrance et al., 2007; Whelton et al., 2002; Williams et al., 1992; Wyatt et al., 2008). What is not known is whether this relationship between weight and blood pressure is causal or not. Moreover, if it is a causal relationship, from which element does the causation originate? Or do each of the components have the ability to produce a separate physiological response?

Risk Relation of Age to Elevated Blood Pressure/Hypertension

Some researchers have characterized age related variation in blood pressure in the general population as both normal blood pressure and high blood pressure. The results of that characterization demonstrate a progressive increase in blood pressure as people age (Franklin et al., 1997; Kahn, Medalie, Neufeld, Riss, & Goldbourt, 1972; Rodriguez, Labarthe, Huang, & Lopez-Gomez, 1994; Vasan et al., 2001). Another feature of age-related blood pressure change is that those with the highest initial systolic blood pressure measurements have chronically higher and more significant elevated blood pressure/hypertension than those with only mildly elevated initial blood pressures. This is consistent with the occurrence of the gradual expansion of large artery stiffness (Franklin et al., 1997). Other researchers show that people who maintain a normal blood pressure even up to the age of 55, are subject to a 90% risk of elevated blood pressure/hypertension throughout the remainder of their life (Chobanian et al., 2003; Kottke et al., 2003).

Risk Relation of Sleep Disordered Breathing to Elevated Blood Pressure/Hypertension

The relationship between sleep disordered breathing (which includes obstructive sleep apnea) and elevated blood pressure/hypertension has also been well substantiated (Brooks, Horner, Kozar, Render-Teixeira, & Phillipson, 1997; Clark, 2000; Grote et al., 2000; Lavie et al., 2000; Nieto et al., 2000; Peppard et al., 2000; Somers et al., 2008; Young et al., 1997). This association has also been demonstrated to be independent of other potentially confounding variables (Brooks et al., 1997; Peppard et al., 2000; Somers et al., 2008; Young et al., 1997). The correspondence revealed between the two is one in which the effect of elevation of blood pressure relates directly to the amount of time a person is exposed to sleep disordered breathing (Brooks et al., 1997; Peppard et al., 2000; Somers et al., 2008; Young et al., 1997). Moreover, sleeping 5 or less hours a night also correlates with an increased risk of elevated blood pressure/hypertension (Cappuccio et al., 2007).

Risk Relation of Tobacco Use to Elevated Blood Pressure/Hypertension

Researchers have been unable to explicitly substantiate the relationship between tobacco use and elevated blood pressure/hypertension (Thuy et al., 2010; Virdis et al., 2010). The crucial question is whether tobacco use precipitates elevated blood pressure/hypertension along with its accompanying adverse effects or not. The question of causality in this relationship has been explored, but its answer remains unsettled (Thuy et al., 2010; Virdis et al., 2010). Some researchers demonstrate, rather counter intuitively, that tobacco use is predominantly associated with levels of blood pressure that are comparatively lower than what is associated with those who do not use tobacco (Green et al., 1986; Omvik, 2015; Thuy et al., 2010). This is despite the common physiological finding that tobacco use typically produces a significant although short lived rise in blood pressure (Leone, 2011; Minami, Ishimitsu, & Matsuoka, 1999; Primatesta, Falaschetti, Gupta, Marmot, & Poulter, 2001; Thuy et al., 2010; Virdis et al., 2010).

Other researchers only partially agree, by demonstrating that the normal physiological findings associated with tobacco use are ephemeral (Groppelli, Giorgi, Omboni, Parati, & Mancia, 1992; Virdis et al., 2010). However, that does not signify that all tobacco users have lower blood pressure than non-tobacco users. Some researchers are unable to identify any significant independent association between tobacco use and elevated blood pressure/hypertension (Primatesta, 2001). While others have established that persistent tobacco use over the long term characteristically produces not only a persistent elevation of blood pressure but also a fixed or permanent hypertensive state (Doroszko, Andrzejak, & Szuba, 2011; Giles, Sander, Nossaman, & Kadowitz, 2012; Groppelli, Giorgi, Omboni, Parati, & Mancia, 1992; Talukder et al., 2011; Thuy et al., 2010).

Risk Relation of Gender to Elevated Blood Pressure/Hypertension

Research findings on the relationship of gender to the control of elevated blood pressure/hypertension have lacked consistency. Some researchers have found gender to be a characteristic that is associated with differences in blood pressure control (Knight et al., 2001). Some even learn that control of elevated blood pressure/hypertension is more strongly associated with being female (Knight et al., 2001; Ornstein, Nietert, & Dickerson, 2004). However, others report that being female decreases the likelihood of control of elevated blood pressure/hypertension (Majernick, Zacker, Madden, Belletti, & Arcona, 2004). While still other researchers find no gender differences (Hicks et al., 2004). This leaves a data record on this point that is paradoxical and incongruous (Chiong, 2008).

Risk Relation of Pain Syndromes to Elevated Blood Pressure/Hypertension

Researchers have reported that blood pressure reacts in the opposite direction of perception and sensitivity to pain in healthy individuals (Bruehl, Chung, Ward, Johnson,

& McCubbin, 2002; Campbell, Hughes, Girdler, Maixner, & Sherwood, 2004; France, 1999). Elevated blood pressure appears to be associated with significantly higher pain thresholds, lower pain ratings and lower pain sensitivity (Bruehl et al., 2002; Campbell et al., 2004; France, 1999). This hypoalgesia may have a mutual relationship with an abnormality in how the central nervous system regulates the heart and blood vessels and pain perception (France, 1999). Implying that hypoalgesia precedes and is therefore not an effect of elevated blood pressure/hypertension and possibly making it useful in identifying individuals at risk for elevated blood pressure/hypertension.

Summary

The concept of stress started with an understanding that exposures cause us to alter the way we live (Griffin & Thomson, 1998). Eventually researchers found that stress causes a release of chemicals that cause other effects on specialized cells (Joëls & Baram, 2009). These physiological actions and reactions are thought to provide humans the means to adapt (Joëls & Baram, 2009). Repetitive exposures to stress may even accumulate their effects over the time. These powerful stressful circumstances may generate diseases such as elevated blood pressure/hypertension (Lester et al., 2010; Ray et al., 2011; Sharp et al., 2008; Thomsen, Stander, McWhorter, Rabenhorst, & Milner, 2011).

Elevated blood pressure/hypertension exists in the U.S. military and contributes to the development of serious diseases and premature deaths (Granado et al., 2009; Kearney et al., 2005; Lopez et al., 2006; Mensah & Brown, 2007; Smith et al., 2008; Smoley et al., 2008; Wenzell et al., 2009). Unfortunately, there are not many studies on elevated blood pressure/hypertension in the military and only one about stress and elevated blood pressure/hypertension (Granado et al., 2009; Smith et al., 2008; Smoley et al., 2008; Wenzell et al., 2009). The knowledge gap is clear—no researcher to date had gathered appropriately time sequenced blood pressure data regarding the relationship between elevated blood pressure/hypertension and the stressful exposure of military personnel to a combat deployment. This research addressed the issue of temporality, which is fundamental to a posteriori knowledge that proceeds from observations to the deduction of probable causes.

Beyond stress is the finding that many other factors are risks to the development of elevated blood pressure/hypertension (Chiong, 2008; Fields et al., 2004; Guo et al., 2012; Landsberg et al., 2013; De Marco et al., 2009; Qureshi, Pyne, Magruder, Schulz, & Kunik, 2009; Ray et al., 2011). These associated hazards are described in scholarly literature and they include obesity, tobacco use, age, gender, sleep disordered breathing, hyperlipidemia, kidney disease, cardiovascular disease, excess salt intake, diabetes, and a persistent state of mental stress from such things as job strain, anxiety, depression, and combat exposure/deployment (Abouzeid, Kelsall, Forbes, Sim, & Creamer, 2012; al'Absi & Wittmers, 2003; Artinian et al., 2006; Babu et al., 2014; Baxi, Jackson, Ritter, & Sessums, 2011; Cheung et al., 2006; Chiong, 2008; Chobanian et al., 2003a, 2003b; Cutler et al., 2008; Devereux et al., 1983; Din-Dzietham et al., 2004; Egan et al., 2010; Jorgensen, 2009; Kannel, 2000; Kaplan, 1998; Kottke et al., 2003; Landsberg et al., 2013; Leone, 2011; De Marco et al., 2009; Matthews, Salomon, Brady, & Allen, 2003; Mustacchi, 1990; Oparil et al., 2003; Pickering, 2001; Primatesta, Falaschetti, Gupta, Marmot, & Poulter, 2001; Ray et al., 2011; Scalco, Scalco, Azul, & Lotufo Neto, 2005; Shohat et al., 1989; Smoley et al., 2008; Stamler et al., 1978; Stevens et al., 2001; Talukder et al., 2011). Chapter 3 provides information about the research methods used for this study.

Chapter 3: Research Method

Introduction

The purpose of this research was to describe the extent of association between exposure to combat deployment, being 40 or older, or being overweight/obese, and elevated blood pressure among U.S. military personnel who were deployed to an area of combat between 2012 and 2017. This chapter contains the relevant research design and rationale of the study. It additionally includes the methodology, identification of the research population, and procedures carried out to obtain the desired population sample. Finally, this chapter records potential threats to the validity of the research and important ethical issues that were considered during research and a summary.

Research Design and Rationale

This study was an analysis of the incidence of elevated blood pressure occurring over time in a source population of active duty service members and DOD personnel. The epidemiologic advantage and strength of an incidence study design such as this is that it initially identifies an outcome free source population, thereby creating a homologous baseline population characteristic. Additionally, and still prior to any occurrence of outcome, it categorizes this source population as a cohort with distinct exposures, e.g. combat deployment, BMI ≥ 25, and age ≥ 40. Finally, it quantifies the occurrence of the outcome concurrent with the passage of time in each of the groups. These design elements are crucial to establishing a direct temporal relationship of exposure to outcome and thereby providing powerful information for the deduction and discovery of causality.

The single dependent variable was soldiers' blood pressure. This investigation considered several independent variables, primarily deployment status, but secondarily BMI and age. These other explanatory or predictor independent variables were collected from the MDR and MART or M2 database, MODS, and AHLTA electronic medical record. They also had time series collections (T1 and T2). However, unlike the dependent variable, dual measurements at each time series were not required. T1 was predeployment and T2 post deployment. This time series collection of the independent variables allowed for a more detailed influence comparison between each of the variables as an effect on the change noted in the dependent variable.

The presence of multiple independent variables and their impact on outcomes could introduce doubt and confusion. When the effects of different variables cannot be parsed, the variables are said to be confounded. In the performance of observational research, all variables are confounded with one another to some extent and the important analytical task is to untangle the comingled influence of several of these variables that may be measured at the same time.

The data representing dependent, independent, and confounding variables were collected electronically from the MDR and MART or MDR/M2 database, MODS, and AHLTA electronic medical record. This research was the first and only study to date that specifically included actual blood pressure measurements performed and recorded at pre and post deployment times by qualified healthcare personnel rather than from a self-related questionnaire answer. What the acquisition of actual blood pressure measurements accomplished was an increase in validity and reliability compared to previous studies that only had access to subjective information from questionnaires. The primary time and resource constraints found in conducting this research were the single student researcher performing all the research tasks and the additional bureaucracy encountered when dealing with the DOD which required multiple organizational contacts, filling of government forms, and obtaining multiple layers of permission, all of which added considerably to the time burden of performing the study.

Methodology

Population

The source population consisted of U.S. Army active duty service members deployed between 2012 and 2017. According to the Office of the Deputy Under Secretary of Defense (2012), the U.S. Army had 561,000 service members on active duty as of November 2012. This research uses the U.S. Central Command's definition of combat deployment for medical purposes, which was: "travel to or through the U.S. Army Central Command's area of responsibility (AOR), with expected or actual time in country (AKA "boots on ground") for a period of greater than 30 days" (Office of the Secretary of Defense, 2011, para.15.A.1). For a reference population, I used the average number of active duty service members deployed to a combat area of operations during 2012. I also used the Congressional Research Service Report for Congress which provided the average monthly boots on the ground population in Afghanistan and Iraq for FY 2012 was 67,500 (Belasco, 2009). Extrapolating the size of the deployed military population over 5 years, based upon the further assumption that the average deployment lasts 12 months, the final approximate tally was 67,500 × 5 = 337,500 total subjects deployed over the 5-year period between 2012 and 2017.

Sample and Sampling Procedures

The repository of information for the population and variables analyzed in this research ultimately was collected from the DMDC database, the MDR, a U.S. Army Medical Department (AMEDD) Patient Administration Systems and Biostatistics Activity (PASBA) database and the AHLTA electronic health record, all of which are maintained by the DOD. The sample population was initially derived from a population of individual active duty service members selected from the DMDC database who met DOD deployment qualifications and who ultimately deployed to a geographical area of declared combat during the years of 2012-2017 (Office of the Secretary of Defense, 2011). From that initial selection the MDR/M2 database, MODS and AHLTA were utilized to ensure each individual also had recordings of blood pressure measurements (two performed pre and two post deployment), height and weight measurements, and date of birth. This allowed the formation of a cohort of medically qualified military personnel who ultimately deployed to combat and also had recorded data points necessary for answering the three research questions.

The sample size was determined by computer calculation using statistical power, level of probability or statistical significance (*p*), and effect size or practical significance. The statistical power was set at 0.8, which is an 80% probability of correctly rejecting the null hypothesis when it is false or by implication an 80% probability of correctly detecting a relationship between independent and dependent variables. The level of probability or statistical significance (*p*) was set at 0.05, which is a 5% probability of incorrectly rejecting the null hypothesis when it is true or by implication a 5% probability of incorrectly detecting a relationship between independent and dependent variables when none exists. The practical significance or effect size was set at 0.2, which is the magnitude of difference or correlation occurring between independent and dependent variables. A small effect would only take 1/5 of a standard deviation unit to demonstrate a difference or correlation. Based upon a G*Power Version 3.1.6 estimate of multiple regression with power set at .80 and probability set at .05, using 3 predictors (deployment, age, and body mass index), a small size effect (.02) would be detectable using a sample size of 395.

Inclusion and Exclusion Criteria

The inclusion criteria for the source population required that the individual subjects be active duty service members and normotensive as evidenced by the mean of two recorded blood pressure measurements less than 120 mm Hg systolic and 80 mm Hg diastolic performed prior to deployment and closest to the time of their predeployment medical evaluation. Additionally, the deployment group must subsequently have completed greater than 30 consecutive days of deployment at least once during the years 2012-2017 to a geographical area of declared combat as defined by the U.S. Army's Central Command in its deployment publication (Office of the Secretary of Defense, 2011). Source population subjects were excluded if at the time of their predeployment medical evaluation the mean of their two recorded blood pressure measurements was equal to or greater than 120 mm Hg systolic or 80 mm Hg diastolic.

Data Collection

The DHCAPE Division promotes and sustains an evidence founded approach to management for the DHA. The goal of the DHCAPE is to provide analyzed data to the MHS to improve the delivery of healthcare to DOD recipients. The AMEDD, PASBA provides access to that information through the MDR and MART or M2, which are the database and analysis tools used by DHCAPE to perform these functions. Moreover, MDR/M2 provides the AMEDD with a consolidated on-line computerized application that comprehensively handles all aspects of the medical data pertaining to the life of a soldier during war and peace.

The population for this study was followed forward from predeployment until at least the time of their post deployment medical evaluation in order to establish the incidence of elevated blood pressure, and additionally to untangle the co-mingled influence of the independent and extraneous variables that were measured at the same time. The data for this research was secondary data that was previously collected for other reasons in the MDR/M2 database. This data was accessed only after approval was granted through a DoD Data Sharing Agreement and a Walden IRB was accomplished. A PASBA statistician de-identified all data records prior to release to the primary investigator, thereby negating any risk of inadvertent public release of private medical information.

Operationalization of Variables

Blood Pressure

The dependent variable was a deployed service member's blood pressure. More specifically it was the mean of two blood pressure measurements taken temporally near each of the respective time series: T1 (time series one) a and b and T2 (time series 2) a and b. This arithmetic mean calculated from the double collection of blood pressure measurements for each of the time series was required for the proper classification of elevated blood pressure/hypertension or normotension (Chobanian et al., 2003a; Whelton et al., 2017). T1 measurements consisted of one measurement (T1a) performed prior to and one (T1b) during the predeployment medical evaluation. T2 measurements were one measurement (T2a) performed during and one (T2b) after the post deployment medical evaluation.

Blood pressure data originated from recordings of objective physical measurement accomplished by health care personnel. It was collected in a continuous data form, as a result of a query of the AHLTA electronic health record for uniformed services members, from the MDR and MART or M2 commonly designated MDR/M2 as Vitals outpatient data. Blood pressure data was recorded in categorical form and designated as either: normotension (systolic BP less than 120 mm Hg and diastolic BP less than 80 mm Hg) or elevated blood pressure/hypertension (systolic blood pressure 120 mm/hg or greater or a diastolic blood pressure 80 mm/hg or greater; Chobanian et al., 2003a; Whelton et al., 2017).

Combat Deployment

The independent variable, combat deployment, was confirmed objectively through a query of the Contingency Tracking System (CTS) File from MDR/M2 as Comprehensive Ambulatory/Professional Encounter Record (CAPER) outpatient data. This data represents exposure to a combat deployment. This exposure classification was collected and recorded as categorical data and designated as either: combat deployment or no combat deployment. Combat deployment was confirmation of travel to or through a U.S. Army Central Command (USCENTCOM) area of responsibility (AOR), and more specifically to or through a USCENTCOM geographical area of declared combat, such as Afghanistan, with actual time in country (AKA "Boots On Ground") for a period of greater than 30 days, at least once during the years 2012-2017 (Office of the Secretary of Defense, 2011).

Age in Years

The independent variable, age, was collected as a result of a query of the Armed Forces Health Longitudinal Technology Application (AHLTA- the electronic health record for uniformed services members), from the MDR/M2 as Comprehensive Ambulatory/Professional Encounter Record (CAPER) outpatient data. This data represents exposure to advancing age in a military environment as noted by cardiovascular screening required at age 40 in Army Regulation 40-501: Medical Services-Standards of Medical Fitness and Modification 11 to CENTCOM's Individual/Unit protection and deployment policy (U S Army, 2011; Office of the Secretary of Defense, 2011). It was collected in a continuous data form as age in whole years determined at the time of the predeployment physical examination by the service member's date of birth. This exposure classification was recorded in a categorical data form and designated as either: less than 40 years old, or equal to or greater than 40 years old.

Body Mass Index

The independent variable, height and weight or body mass index (BMI), was collected as a result of a query of the Armed Forces Health Longitudinal Technology Application (AHLTA- the electronic health record for uniformed services members), from the MDR/M2 as Comprehensive Ambulatory/Professional Encounter Record (CAPER) outpatient data. This data represents exposure to a specific body mass index. This exposure classification was recorded as categorical and designated as: normal (BMI less than 25), or overweight/obese (BMI equal to or greater than 25) in accordance with National Heart Lung and Blood Institute guidelines (Pi-Sunyer et al., 1998).

Data Analysis Plan

All of the statistical data analysis performed during the process of this research was conducted on the IBM computer software program called Statistical Package for the Social Sciences (SPSS) version 21. Primarily this study allowed a determination of the incidence of elevated blood pressure in the source population cohort that was exposed to a combat deployment. This was accomplished by discovering if elevated blood pressure was statistically associated with exposure to a combat deployment. Additional data analysis enabled the establishment of whether elevated blood pressure was statistically associated with advancing age or an elevated body mass index.

Univariate Analysis

Initially I examined of the all study variables at the univariate level providing descriptive statistics. Next, I performed categorical analysis on the variables of combat deployment, age, BMI, and blood pressure essentially describing how many (number and percentage) study participants fell into each category within the variable. No continuous variable analysis was conducted even though age was collected in continuous data form because I recorded and analyzed in categorical data form.

Bivariate Analysis

After the univariate analysis, I conducted bivariate analysis. This enabled the examination of single relationships between independent and dependent variables without the influence of other variables. This was done so I could identify the need of whether to include those variables as covariates in multivariate testing, and to enable statistical control for the influence of that variable in the final stage of analysis, multivariate testing. There was suspicion that the covariate independent variables (age and BMI), not just the primary independent variable (combat deployment), were also associated with the dependent variable (blood pressure).

It was important to identify if these covariate independent variable relationships existed and to then account for their effects on the dependent variable by including them in the final multivariate model after bivariate analysis. When the effects of a primary independent variable and a covariate independent variable cannot be separated, the variables are said to be confounded. In observational studies, all variables are generally confounded with one another to some extent and the analytical task is to untangle the comingled influence of many variables that are measured at the same time with the use of bivariate and then multivariate testing.

Bivariate analysis determines how two variables associate or correlate. The choice regarding which bivariate test to use is based on the specific structural mix of each variable set—continuous and/or categorical. Chi-square testing is used when there are two categorical variables (Forthofer et al., 2007). In the current study, all of the variables were categorical, which means that Chi-square analysis met the data analysis needs. The application and interpretation of Chi-square testing for each research question follows.

RQ1: What was the extent of the association between exposure to combat deployment (completing greater than 30 consecutive days of deployment to a geographic area of declared combat) and new onset elevated blood pressure/hypertension (the mean of two recorded blood pressure measurements demonstrating either a systolic blood pressure equal to or greater than 120 mm/hg or a diastolic blood pressure equal to or greater than 80 mm/hg) among active duty military personnel?

$H_0 1$: Active duty military personnel exposed to combat deployment have the same or lower prevalence of elevated blood pressure/hypertension as those active duty military personnel not exposed to combat deployment.

$H_A 1$: Active duty military personnel exposed to combat deployment have a higher prevalence of elevated blood pressure/hypertension than active duty military personnel not exposed to combat deployment.

For *RQ1*, bivariate analysis using Chi-square testing determined if and how (which direction—positive or negative) blood pressure was significantly associated with combat deployment. Chi-square testing was selected because it is the appropriate test for two categorical variables. In Chi-square correlation, there are two crucial details, the statistical significance and the distribution of cases within categories. It was essential first to identify if the relationship was statistically significant. In the Statistical Package for the Social Sciences (SPSS), statistical significance for Chi-Square is found on the Pearson Chi-Square line in the column labeled "Asymp. Sig. (2-sided)". This data can either confirm or reject the relationship. To confirm a statistically significant relationship the value must be less than .05.

Subsequently, it was important to analyze how the cases within the categories were distributed to determine the nature of their relationship. SPSS has a "Cross-tabulation" box where each variable is listed and the sections inside each variable are cross-tabulated. Here it was critical to ascertain how the cases and percentages varied within these subsections of cross-tabulated variables. This data can confirm and specify the direction of the relationship between the variables.

If cross tabulation revealed a higher percentage of those who were exposed to combat deployment were hypertensive relative to those not exposed to combat deployment, then this would specify a positive relationship between combat deployment exposure and elevated blood pressure/hypertension. It would also confirm the alternative hypothesis, H_A1: Active duty military personnel exposed to combat deployment, have a higher prevalence of elevated blood pressure/hypertension than do those active duty military personnel not exposed to combat deployment.

On the other hand, if cross tabulation revealed that a higher percentage of those exposed to combat deployment were normotensive relative to those not exposed to combat deployment, then this would indicate a negative relationship between combat deployment and elevated blood pressure/hypertension. It would also confirm the null hypothesis, H_01: Active duty military personnel exposed to combat deployment have the same or lower prevalence of elevated blood pressure/hypertension as those active duty military personnel not exposed to combat deployment.

RQ2: What was the extent of the association between being overweight/obese (BMI equal to or greater than 25) and new onset elevated blood pressure/hypertension (the mean of two recorded blood pressure measurements demonstrating either a systolic blood pressure equal to or greater than 120 mm/hg or a diastolic blood pressure equal to or greater than 80 mm/hg) among active duty military personnel?

H_02: Active duty military personnel exposed to being overweight/obese have the same or lower prevalence of elevated blood pressure/hypertension as those active duty military personnel not exposed to being overweight/obese.

H_A2: Active duty military personnel exposed to being overweight/obese have a higher prevalence of elevated blood pressure/hypertension than those active duty military personnel not exposed to being overweight/obese.

For *RQ2*, bivariate analysis using Chi-square testing determined if and how (which direction—positive or negative) blood pressure was significantly associated with BMI. Chi-square testing was selected because it is the appropriate test for two categorical variables. Again, it was essential first to identify if the relationship was statistically significant. To confirm a statistically significant relationship between BMI and blood pressure, the value found in SPSS on the Pearson Chi-Square line in the column labeled “Asymp. Sig. (2-sided)” must be less than .05.

Next, it was important to determine the nature of the relationship between BMI and blood pressure. If cross tabulation revealed a higher percentage of those who were overweight/obese (BMI equal to or greater than 25) were also hypertensive relative to those who were normal weight (BMI less than 25), then this would specify a positive relationship between overweight/obese (BMI equal to or greater than 25) and elevated blood pressure/hypertension. It would also confirm the alternative hypothesis, H_A2: Active duty military personnel exposed to being overweight/obese (BMI equal to or greater than 25) have a higher prevalence of elevated blood pressure/hypertension than do those not exposed to being overweight/obese (BMI equal to or greater than 25).

On the other hand, if cross tabulation revealed a higher percentage of those overweight/obese (BMI equal to or greater than 25) service members were normotensive relative to those who were normal weight (BMI less than 25), then this would indicate a negative relationship between overweight/obese (BMI equal to or greater than 25) and elevated blood pressure/hypertension. It would also confirm the null hypothesis, H_02: Active duty military personnel exposed to being overweight/obese (BMI equal to or greater than 25) have the same or lower prevalence of elevated blood pressure/hypertension as those not exposed to being overweight/obese (BMI equal to or greater than 25).

RQ3: What was the extent of the association between being 40 years of age or older and new onset elevated blood pressure/hypertension (the mean of two recorded blood pressure measurements demonstrating either a systolic blood pressure equal to or greater than 120 mm/hg or a diastolic blood pressure equal to or greater than 80 mm/hg) among active duty military personnel?

$H_0 3$: Active duty military personnel exposed to being 40 years of age or older have the same or lower prevalence of elevated blood pressure/hypertension as those active duty military personnel not exposed to being 40 years of age or older.

$H_A 3$: Active duty military personnel exposed to being 40 years of age or older have a higher prevalence of elevated blood pressure/hypertension than those active duty military personnel not exposed to being 40 years of age or older.

For *RQ3*, bivariate analysis using Chi-square testing determined if and how (which direction—positive or negative) blood pressure was significantly associated with age. Chi-square testing was selected because it is the appropriate test for two categorical variables. Once again, it was essential first to identify if the relationship was statistically significant. To confirm a statistically significant relationship between age and blood pressure, the value found in SPSS on the Pearson Chi-Square line in the column labeled "Asymp. Sig. (2-sided)" must be less than .05.

Next, it was important to determine the nature of the relationship between age and blood pressure. If cross tabulation revealed a higher percentage of those 40 years of age or older were hypertensive relative to those less than 40 years of age, then this would specify a positive relationship between being 40 years of age or older and elevated blood pressure/hypertension. It would also confirm the alternative hypothesis, H_A3: Active duty military personnel exposed to stress by being 40 years of age or older have a higher prevalence of elevated blood pressure/hypertension than do those not exposed to stress by being 40 years of age or older.

On the other hand, if cross tabulation revealed that a higher percentage of those 40 years of age or older were normotensive relative to those were less than 40 years of age, then this would indicate a negative relationship between being 40 years of age or older and elevated blood pressure/hypertension. It would also confirm the null hypothesis, H_03: Active duty military personnel exposed to stress by being 40 years of age or older, have the same or lower prevalence of elevated blood pressure/hypertension as those not exposed to stress by being 40 years of age or older.

Multivariate Analysis

It is important to note that bivariate testing cannot establish whether the primary independent variable or the other secondary covariate variables are the strongest predictors of the dependent variable. This requires analysis of the influences of all the independent variables at once on the dependent variable. Multivariate analysis can accomplish this and is considered a distinctive feature of modern empirical research.

During the multivariate part of the data analysis, binary logistic regression was used to assess odds ratios, of exposure to combat deployment, being overweight/obese (BMI equal to or greater than 25), and being 40 years of age or older, among military personnel to have elevated blood pressure. Odds Ratio assessments were formulated into statements that the presence of the variable in question was x times more or less likely to result in elevated blood pressure relative to its absence. Subsequent to this multivariate analysis, relative comparisons were made of which were the strongest predictors of elevated blood pressure in military personnel.

Fundamental to proper interpretation of binary logistic regression analysis was the identification of the statistical significance of the model. In the SPSS, this statistical significance is found in the “Omnibus Tests of Model Coefficients” box. To confirm a statistically significant regression model the value in the intersection of the last row “Model” and the last column “Sig.” had to be less than .05.

It was equally important to understand the relationships between the independent and dependent variables in the model. The details of these relationships are found in the SPSS “Variables in the Equation” box. Again, it was crucial to initially ascertain that the significance for each predictor in the “Sig.” column was less than .05. Subsequently the “B” (beta) column was analyzed for the values of each independent variable, recognizing that positive and negative values represented positive and negative relationships with the dependent variable.

Finally, the researcher examined the “Exp (B)” column, which contains the odds ratios. This represents how many times more or less likely elevated blood pressure/hypertension occurred based on the presence of one of the independent variables. Negative relationships noted in the “B” column with a “-” corresponded to “Exp (B)” values less than 1. In those cases, dividing the number 1 by the “Exp (B)” value that was less than 1 provided the Odds Ratio.

The preparation of data prior to data analysis included that all test assumptions for regression were met, including checks of the linearity between variables, multicollinearity, heteroscedasticity, and normal distributions among continuous variables.

Threats to Validity

Validity refers to the trustworthiness of research findings. However, validity is not an exclusive either-or condition but one of scope (Messick, 1987). Therefore, valid research conclusions are reliable in the sense that to the degree currently possible for the researcher they represent reality as it exists. This, by logical extension, also conveys the idea that factors exist that are able to diminish the validity of a research conclusion.

Internally, this research was validated through the strength of inference of causality between combat deployment and elevated blood pressure/hypertension. Initially, causality was substantiated by application of the exclusion criteria which established the temporal occurrence of combat deployment antecedent to elevated blood pressure/hypertension. Subsequently, the covariation of combat deployment and elevated blood pressure/hypertension was confirmed through bivariate analysis using Chi-square testing and multivariate analysis through the application of binary logistic regression. Finally, a spurious relationship was rebutted by controlling for the most plausible alternate relationships with age and body mass index.

The research obtained external validation by confirming that the new onset of elevated blood pressure/hypertension, observed and measured under ordinary combat deployment circumstances, in a group of 308 U.S. military personnel, during and after their exposure to a combat deployment, from 2012-2017, could be broadly inferred to transpire in other deployment locations, military personnel, and times. Credence to this inference was found in the representative nature of the sample population. The uniformity of the sample population as compared to the entire U.S. military's deployable population was established through the unvarying requirement for all deploying service members to satisfactorily meet the U.S. Army Central Command's (USACENTCOM) physical and medical standards for deployment (Office of the Secretary of Defense, 2011).

Statistical conclusions resulting from this research were substantiated by demonstrating that a statistically significant relationship existed or did not exist between the proposed cause (combat deployment) and effect (elevated blood pressure/hypertension). This relationship demonstration was only fitting subsequent to appropriate inference from an evaluation of the interactions between several statistical elements. These included the sample group size (*N)*, the estimate of how much the explanation that no relationship existed was believed to be false or effect size (f^2) [small $f^2 \geq 0.02$, medium $f^2 \geq 0.15$, and large $f^2 \geq 0.35$], the risk of wrongly abandoning the explanation that no relationship existed or probability (α), and the probability of rightly discarding the null explanation that no relationship existed or power (β; Cohen, 1992).

In this study, that inference was accomplished by following the recommendations of the power analyses. The power analysis recommended that *N* needed to be 395 to be able to use multiple regression on 3 predictors (deployment, age, and body mass index) with β set at .80, and α set at .05, to detect a small f^2 of .02. This meant that it would only take 1/50 of a standard deviation unit to demonstrate a difference or correlation.

Additionally, the most significant threats to the validity of this research occurred consequent to its design. This was an observational retrospective cohort design in which the population of interest was chosen at the outset for the lack of the outcome of interest and the presence of the exposure of interest. Subsequently, that population was analyzed over time for the presence of a relationship between several of the exposures and the outcome of interest. This study therefore examined people in their natural setting. No intervening force was deliberately applied. This meant that the things that happened to those people happened because of their own individual preference, random occurrence, or some other outside force.

Preference, randomness, and outside forces can create confounding results in uncertainty when the researcher attempts to impute cause. The measure taken to counter this uncertainty was the use of binary logistic regression. Subsequent to this multivariate form of analysis, comparisons of the different variables could be performed, relative to each other. The results revealed the relative strength of each of the predictors of elevated blood pressure in military personnel.

Another threat occurred because of the timing and reason for which the research information was gathered. In this incidence study, the data was initially collected by the DOD for their own set of purposes. However, this information was then going to be used to examine a different research question. The primary issue with using previously collected data is that the second user is not in control of which variables were collected. This added uncertainty and was therefore a threat to validity.

Variable data integrity checks could be performed, including checks for data randomness. e.g., a logistic regression test predicting missingness (0=not missing, 1=missing) from all other variables or an ANOVA to see if individuals with missing data on one variable are significantly different on other, similar variable. Although not definitive, this sort of analysis of the data could give support to an inference of randomness or non-randomness regarding missing data (Rubin, 1976). More significantly however, because obtaining and recording pre and post deployment blood pressure measurement data is a mandatory DoD requirement the most reasonable inference was that missing blood pressure measurement data were missing at random. Therefore, missing blood pressure data were considered ignorable and list-wise deletion performed on cases with missing values on the blood pressure variable and only records with completed T1a, T1b, T2a, and T2b blood pressure measurements were utilized for data analysis.

Ethical Procedures

The performance of this research required approval from the Walden University Institutional Review Board (IRB). The IRB approval number for this study is 03-27-17-0336503. A request for change in research procedures was additionally approved by the Walden University IRB on 07-11-17.

This research presented no more than minimal risk of harm to study subjects and involved no procedures for which written consent was normally required outside of the research context. The risks involved in conducting this and any research performed by extracting data from a medical data repository lie in the possibility of inadvertent public release of private medical information or breaking patient confidentiality through inadvertent identification of previously seen or treated patients. To prevent this occurrence, a Data Sharing Agreement (DSA) was signed with the DHA for the required research data to be deidentified and delivered via secure e-mail.

Once the physical data was obtained, I completed the research tasks utilizing and storing the data in encrypted electronic files, utilizing password protection. This all occurred on my personal computer, thereby negating any risk of inadvertent public release of private medical information. I was the only person with access to the data.

Furthermore, because this was retrospective data file research, risk to the study population could only occur as a result of breaches of privacy and confidentiality. Consequently, a waiver of the requirement to document informed consent did not adversely affect the rights and welfare of the subjects because the only record linking the study subject and the research would be the informed consent document and the principal risk would be the potential harm resulting from a breach of confidentiality. Additionally, the research could not practicably be carried out without the waiver inasmuch as it was impossible to contact the sample population subjects since their contact information was not part of the source data file and therefore not available. As a result, this retrospective data file research involves no more than minimal risk to the study population subjects.

Summary

The historical tenet of this quantitative research was Selye's original finding that the principal autonomic human response to an awareness of or exposure to imminent danger is both a prompt rise in heart rate and pronounced vasoconstriction, which can precipitate elevated blood pressure/hypertension (Selye 1950; Viner, 1999). The purpose of this study was to describe the extent of association between exposure to combat deployment, being overweight/obese, being older, and hypertension among U.S. military personnel who deployed to an area of declared combat during the years 2012-2017.

The nature of the research was a quantitative investigation into the incidence of elevated blood pressure/hypertension among previously deployed active duty service members (Kuchel, 2003; Melby, 1983; Milliken et al., 2007; Polusny et al., 2011; Thomsen et al., 2011). The time series data representing the variables were from secondarily collected data maintained by the DOD. This study was the first to include previously recommended pre and post deployment blood pressure data (Granado et al., 2009; Jorgensen, 2009). Chapter 4 is a documentation of the statistical results of this research study.

Chapter 4: Results

Introduction

The purpose of this research was to describe the extent of association between exposure to combat deployment, being overweight, being old, and hypertension among U.S. military personnel who deployed to an area of declared combat between 2012 and 2017. The three independent variables were combat deployment status (completing greater than 30 consecutive days of deployment to a geographic area of declared combat), BMI of 25 or higher, and age of 40 years or older. The dependent variable was the new onset of elevated blood pressure (the mean of two recorded blood pressure measurements demonstrating either a systolic blood pressure equal to or greater than 120 mm/hg or diastolic blood pressure equal to or greater than 80 mm/hg). The research questions and hypotheses that guided this study were

RQ1: What was the extent of the association between exposure to combat deployment (completing greater than 30 consecutive days of deployment to a geographic area of declared combat) and new onset elevated blood pressure/hypertension (the mean of two recorded blood pressure measurements demonstrating either a systolic blood pressure equal to or greater than 120 mm/hg or a diastolic blood pressure equal to or greater than 80 mm/hg) among active duty military personnel?

H_01: Active duty military personnel exposed to combat deployment have the same or lower prevalence of elevated blood pressure/hypertension as those active duty military personnel not exposed to combat deployment.

H_A1: Active duty military personnel exposed to combat deployment have a higher prevalence of elevated blood pressure/hypertension than active duty military personnel not exposed to combat deployment.

RQ2: What was the extent of the association between being overweight/obese (BMI equal to or greater than 25) and new onset elevated blood pressure/hypertension (the mean of two recorded blood pressure measurements demonstrating either a systolic blood pressure equal to or greater than 120 mm/hg or a diastolic blood pressure equal to or greater than 80 mm/hg) among active duty military personnel?

H_02: Active duty military personnel exposed to being overweight/obese have the same or lower prevalence of elevated blood pressure/hypertension as those active duty military personnel not exposed to being overweight/obese.

H_A2: Active duty military personnel exposed to being overweight/obese have a higher prevalence of elevated blood pressure/hypertension than those active duty military personnel not exposed to being overweight/obese.

RQ3: What was the extent of the association between being 40 years of age or older and new onset elevated blood pressure/hypertension (the mean of two recorded blood pressure measurements demonstrating either a systolic blood pressure equal to or greater than 120 mm/hg or a diastolic blood pressure equal to or greater than 80 mm/hg) among active duty military personnel?

H_03: Active duty military personnel exposed to being 40 years of age or older have the same or lower prevalence of elevated blood pressure/hypertension as those active duty military personnel not exposed to being 40 years of age or older.

H_A3: Active duty military personnel exposed to being 40 years of age or older have a higher prevalence of elevated blood pressure/hypertension than those active duty military personnel not exposed to being 40 years of age or older.

This chapter presents the process of collecting the data. This chapter additionally presents the results of the statistical analysis. Finally, the chapter provides a summary of the results.

Data Collection

At the start of this research, the parameters I used for the categorization of normal versus elevated blood pressure were recommended by the Seventh Joint National Committee (JNC) on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (Chobanian et al., 2003). The Seventh JNC blood pressure categorization was recognized as reasonably sound dogma and accepted by the majority of the U.S. medical community as established. Their categorization states that elevated blood pressure exists when either systolic blood pressure is equal to or greater than 140 mm/hg or diastolic blood pressure is equal to or greater than 90 mm/hg (Chobanian et al., 2003).

However, the JNC categorization was challenged in May 2018 when the American College of Cardiology and American Heart Association Task Force on Clinical Practice Guidelines published their most recent elevated blood pressure/hypertension clinical practice guidelines (Whelton et al., 2017). Their blood pressure categories include normal (less than 120 mm Hg systolic and less than 80 mm Hg diastolic), elevated (120-129 mm Hg systolic and less than 80 mm Hg diastolic), stage 1 hypertension (130-139 mm Hg systolic or 80-89 mm Hg diastolic), and stage 2 hypertension (equal to or greater than 140 mm Hg systolic or equal to or greater than 90 mm Hg diastolic; Whelton et al., 2017). Based on this new determination, the category I chose to use for elevated blood pressure in this research was updated to either systolic blood pressure equal to or greater than 120 mm/hg or diastolic blood pressure equal to or greater than 80 mm/hg.

I initially used the DMDC database and MDR, a DHA PASBA database, to obtain a study population of active duty service members who were eligible to deploy between 2012 and 2017. First, I had to gain permission from the DHA in order to be able to request the data from the two separate databases owned by the DMDC and PASBA. Next, the DMDC mandated that I complete a data request for the identification of a population (with identifiers) of service members who met the deployment requirement.

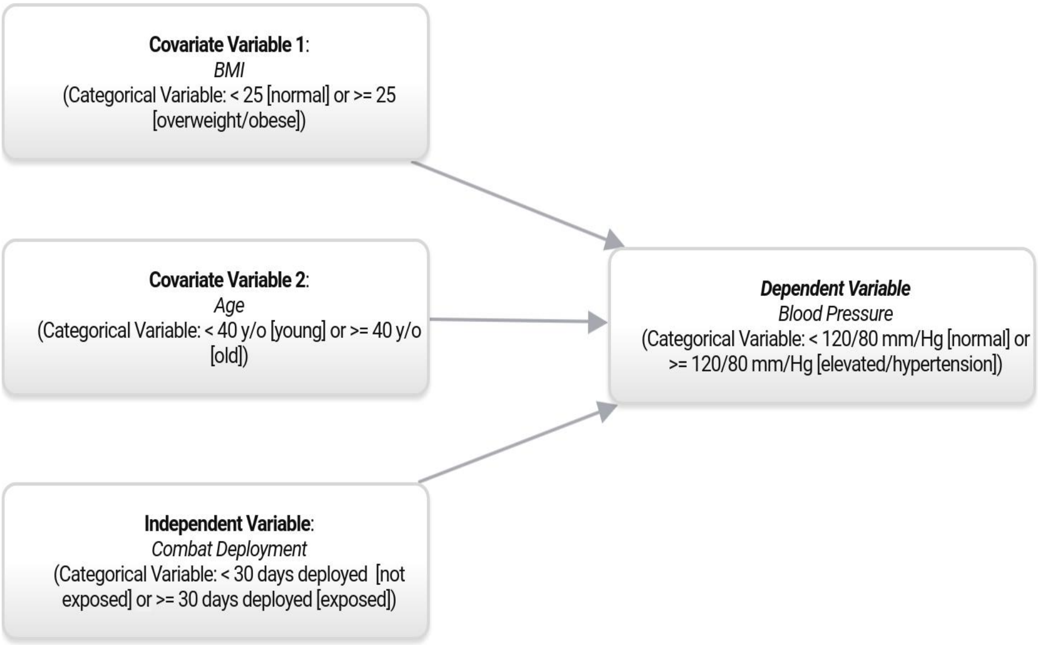
On the execution of this first data extraction, the DMDC identified, collected, and sent 160,000 records of qualified previously deployed service members to PASBA. This is the standard maximum number of records that DMDC can send to another agency because of the risk of data loss. Once the data extraction was sent to PASBA, their statisticians used the DMDC-provided service member identifiers to locate the medical records.

Next, PASBA statisticians were able to program a computer action to correlate how many of those 160,000 DMDC identified deployed service members also had two predeployment blood pressure measurements and two post-deployment blood pressure measurements, along with measurements of height, weight, and age. However, this action established a mutual relationship or connection with only 20 qualified service members.

This shortfall required another data request from DMDC to identify additional deployers. This second DMDC data extraction produced 267,000 additional records of qualified previously deployed service members. The higher number of records required a special request and special permission to send from DMDC to PASBA. This time when the PASBA statistician received the data from DMDC and ran the computer action, 308 qualified service members were identified. The data did not include identification of gender, but service member ages spanned from 17 to 58 years old, deployment lengths varied from 1 to 485 days during the years 2012 to 2017, and body mass indexes fluctuated from 17 to 46.

Results of Analysis

I used the DMDC database and the MDR collectively to obtain a study population that consisted of 308 U.S. active duty service members eligible to deploy to combat during the years 2012-2017. Figure 1 depicts the type, description and relationship of each of the variables used during the testing of this study population.



created with www.bubbl.us

Figure 1. Variable study map.

Univariate Analysis

I performed univariate analysis to describe characteristics of the study population. Tables 1 and 2 detail number and percentage of participants in each category within each variable. Concerning continuous variable analysis, though the variable age was initially collected in continuous data form, I recorded and analyzed it in categorical data form.

Table 1

Demographic Characteristics of Study Population

Characteristic	n	%
Combat deployment category		
Not exposed to combat deployment (≤30 days)	66	21.4
Exposed to combat deployment > 30 days)	242	78.6
Total	308	100
BP category (not exposed to combat deployment)		
Normal (< 120/80 mm/Hg)	37	56.1
Elevated blood pressure/hypertension (≥ 120/80 mm/Hg)	29	43.9
Total	66	100
BP category (precombat deployment exposure)		
Normal (< 120/80 mm/Hg)	123	50.8
Elevated blood pressure/hypertension (≥ 120/80 mm/Hg)	119	49.2
Total	242	100
BP category (post-combat deployment exposure)		
Normal (< 120/80 mm/Hg)	124	51.2
Elevated blood pressure/hypertension (≥ 120/80 mm/Hg)	118	48.8
Total	242	100

Table 2

Demographic Characteristics of Study Population-Continued

Characteristic	*n*	%
BMI category (not exposed to combat deployment)		
Normal (< 25)	25	37.9
Overweight/Obese (≥ 25)	41	62.1
Total	66	100
BMI category (precombat deployment exposure)		
Normal (< 25)	114	47.1
Overweight/Obese (≥ 25)	128	52.9
Total	242	100
BMI category (post-combat deployment exposure)		
Normal (< 25)	96	39.7
Overweight/Obese (≥ 25)	146	60.3
Total	242	100
Age category (not exposed to combat deployment)		
Young (< 40 y/o)	56	84.8
Old (≥ 40 y/o)	10	15.2
Total	66	100
Age category (precombat deployment exposure)		
Young (< 40 y/o)	219	90.5
Old (≥ 40 y/o)	23	9.5
Total	242	100
Age category (post-combat deployment exposure)		
Young (< 40 y/o)	203	83.9
Old (≥ 40 y/o)	39	16.1
Total	242	100

Bivariate Analysis

Bivariate analysis enables the examination of single relationships between independent and dependent variables without the influence of other variables. Chi-square testing should be used when there are two categorical variables (Forthofer et al., 2007). In the current study, the variables are categorical, which means that Chi-square analysis met the data analysis needs. The application and results of Chi-square testing for each research question follows.

In Chi-square correlation there are two crucial details: the statistical significance and the distribution of cases within categories (Forthofer et al., 2007). The statistical significance for Chi-Square is labeled “Asymptotic Significance”. I set the confirmation of a statistically significant relationship using Chi-Square testing in this research at an “Asymptotic Significance” level of less than .05.

RQ1: What was the extent of the association between exposure to combat deployment (completing greater than 30 consecutive days of deployment to a geographic area of declared combat) and new onset elevated blood pressure/hypertension (the mean of two recorded blood pressure measurements demonstrating either a systolic blood pressure equal to or greater than 120 mm/hg or a diastolic blood pressure equal to or greater than 80 mm/hg) among active duty military personnel?

H_01: Active duty military personnel exposed to combat deployment have the same or lower prevalence of elevated blood pressure/hypertension as those active duty military personnel not exposed to combat deployment.

H_A1: Active duty military personnel exposed to combat deployment have a higher prevalence of elevated blood pressure/hypertension than active duty military personnel not exposed to combat deployment.

For *RQ1*, the "Asymptotic Significance" test result was .487 (see Table 3 under *p* column), which is greater than .05. This result indicates there is no statistically significant relationship between the independent variable exposure to combat deployment (completing greater than 30 consecutive days of deployment to a geographic area of declared combat), and the dependent variable new onset elevated blood pressure (the mean of two recorded blood pressure measurements demonstrating either a systolic blood pressure equal to or greater than 120 mm/hg or diastolic blood pressure equal to or greater than 80 mm/hg) among active duty military personnel.

Subsequently, I analyzed the SPSS "Cross-tabulation" box for the distribution of cases within categories (see Table 4). This revealed a higher percentage (48.8%) of those exposed to combat deployment had elevated blood pressure/hypertension relative to those with elevated blood pressure/hypertension not exposed to combat deployment (43.9%). These results would connote a positive relationship between combat deployment and elevated blood pressure/hypertension. However, the lack of significance confirms the null hypothesis, *$H_0$1*: Active duty military personnel exposed to combat deployment have the same or lower prevalence of elevated blood pressure as those not exposed to combat deployment.

Table 3

Normal vs Elevated BP in Service Members Exposed to Combat Deployment, Being Overweight, and Being Old

	Normal BP/Normotension		Elevated BP/Hypertension			
Exposure	*n*	%	*N*	%	χ^2	*p*
Combat Deployment (≥ 30 days)	124	51.2	118	48.8	0.483	0.487
BMI ≥ 25 (Overweight/Obese)	77	41.2	110	58.8	23.492	0.000
Age ≥ 40 y/o (Old)	18	36.7	31	63.3	5.639	0.018

Table 4

Normal vs Elevated BP in Service Members Exposed and Not Exposed to Combat Deployment

	Normal BP/Normotension		Elevated BP/Hypertension		Total	
Exposure	*n*	%	*N*	%	*n*	%
No Combat Deployment (< 30 days)	37	56.1	29	43.9	66	100
Combat Deployment (≥ 30 days)	124	51.2	118	48.8	242	100
Total	161	52.3	147	47.7	308	100

RQ2: What was the extent of the association between being overweight/obese (BMI equal to or greater than 25) and new onset elevated blood pressure/hypertension (the mean of two recorded blood pressure measurements demonstrating either a systolic blood pressure equal to or greater than 120 mm/hg or a diastolic blood pressure equal to or greater than 80 mm/hg) among active duty military personnel?

$H_0$2: Active duty military personnel exposed to being overweight/obese have the same or lower prevalence of elevated blood pressure/hypertension as those active duty military personnel not exposed to being overweight/obese.

H_A2: Active duty military personnel exposed to being overweight/obese have a higher prevalence of elevated blood pressure/hypertension than those active duty military personnel not exposed to being overweight/obese.

For *RQ2*, the “Asymptotic Significance” was less than .05 at .000, (see Table 5 under *p* column). This result connotes a statistically significant relationship between BMI equal to or greater than 25 (overweight/obese), and new onset elevated blood pressure (the mean of two recorded blood pressure measurements demonstrating either a systolic blood pressure equal to or greater than 120 mm/hg or diastolic blood pressure equal to or greater than 80 mm/hg) among active duty military personnel.

SPSS “Cross-tabulation” box analysis (see Table 6) revealed 58.8% of those exposed to a BMI equal to or greater than 25 (overweight/obese) had elevated blood pressure/hypertension relative to 30.6% of those with a BMI less than 25 (normal). This describes a positive relationship between BMI equal to or greater than 25 (overweight/obese) and elevated blood pressure/hypertension. It also confirms the alternative hypothesis, H_A2: Active duty military personnel who have been exposed to stress by virtue of being overweight/obese will have higher prevalence of elevated blood pressure/hypertension.

Table 5

Normal vs Elevated BP in Service Members Exposed to Combat Deployment, Being Overweight, and Being Old

	Normal BP/Normotension		Elevated BP/Hypertension			
Exposure	*n*	%	*n*	%	χ^2	*p*
Combat Deployment (≥ 30 days)	124	51.2	118	48.8	0.483	0.487
BMI ≥ 25 (Overweight/Obese)	77	41.2	110	58.8	23.492	0.000
Age ≥ 40 y/o (Old)	18	36.7	31	63.3	5.639	0.018

Table 6

Normal vs Elevated BP in Service Members Exposed and Not Exposed to Being Overweight

	Normal BP/Normotension		Elevated BP/Hypertension		Total	
Exposure	n	%	n	%	n	%
BMI < 25 (Normal)	84	69.4	37	30.6	121	100
BMI ≥ 25 (Overweight/Obese)	77	41.2	110	58.8	187	100
Total	161	52.3	147	47.7	308	100

RQ3: What was the extent of the association between being 40 years of age or older and new onset elevated blood pressure/hypertension (the mean of two recorded blood pressure measurements demonstrating either a systolic blood pressure equal to or greater than 120 mm/hg or a diastolic blood pressure equal to or greater than 80 mm/hg) among active duty military personnel?

$H_0$3: Active duty military personnel exposed to being 40 years of age or older have the same or lower prevalence of elevated blood pressure/hypertension as those active duty military personnel not exposed to being 40 years of age or older.

H_A3: Active duty military personnel exposed to being 40 years of age or older have a higher prevalence of elevated blood pressure/hypertension than those active duty military personnel not exposed to being 40 years of age or older.

Finally, *RQ3*, analyzed if and how blood pressure is associated with age. The “Asymptotic Significance” test result was .018, which like *RQ2*, is less than .05 (see Table 7 under *p* column). This result confirms a statistically significant relationship between age equal to or greater than 40 years, and elevated blood pressure/hypertension (the mean of two blood pressure measurements at either a systolic blood pressure equal to or greater than 120 mm/hg or diastolic blood pressure equal to or greater than 80 mm/hg) among active duty military personnel.

Next, SPSS “Cross-tabulation” was analyzed and reveals in Table 8, 63.3% of those exposed to age equal to or greater than 40 years old, had elevated blood pressure/hypertension relative to 44.8% of those who were age less than 40 years old. This indicates a positive relationship between age equal to or greater than 40 years and having elevated blood pressure/hypertension. It also confirms the alternative hypothesis, *H_A3*: Military personnel who have been exposed to stress, by being older, will have higher prevalence of elevated blood pressure/hypertension.

Table 7

Normal vs Elevated BP in Service Members Exposed to Combat Deployment, Being Overweight, and Being Old

	Normal BP/Normotension		Elevated BP/Hypertension			
Exposure	*n*	%	*n*	%	χ^2	*p*
Combat Deployment (≥ 30 days)	124	51.2	118	48.8	0.483	0.487
BMI ≥ 25 (Overweight/Obese)	77	41.2	110	58.8	23.492	0.000
Age ≥ 40 y/o (Old)	18	36.7	31	63.3	5.639	0.018

Table 8

Normal vs Elevated BP in Service Members Exposed and Not Exposed to Being Old

	Normal BP/Normotension		Elevated BP/Hypertension		Total	
Exposure	*n*	%	*n*	%	*n*	%
Age < 40 (Young)	143	55.2	116	44.8	259	100
Age ≥ 40 y/o (Old)	18	36.7	31	63.3	49	100
Total	161	52.3	147	47.7	308	100

In summary, Chi-square correlation analyses, revealed that being older (age equal to or greater than 40 years), and being overweight/obese (BMI equal to or greater than 25) both have statistically significant relationships in a positive direction with the new onset of elevated blood pressure among active duty military personnel. These analyses also confirm alternative hypotheses, H_A2: Active duty military personnel who have been exposed to being overweight/obese (BMI equal to or greater than 25), will have higher prevalence of elevated blood pressure than do active duty military personnel who have not been exposed to being overweight/obese (BMI equal to or greater than 25), and H_A3: Active duty military personnel who have been exposed to being 40 years of age or older, will have higher prevalence of elevated blood pressure than do active duty military personnel who have not been exposed to being 40 years of age or older. However, the relationship of experiencing combat deployment, with new onset elevated blood pressure among active duty military personnel, although positive in its direction, was not found to be statistically significant. This lack of significance related to combat deployment confirms the null hypothesis, $H_0 1$: Active duty military personnel who have been exposed to experiencing combat deployment will have the same or lower prevalence of elevated blood pressure as those active duty military personnel who have not been exposed to experiencing combat deployment.

Multivariate Analysis

Binary logistic regression was used to assess odds ratios, of exposure to combat deployment, being overweight/obese, and being old, among the 308 qualified service member study population to have elevated blood pressure. Odds Ratio assessments are formulated into statements that the presence of the variable in question is x times more or less likely to result in elevated blood pressure relative to its absence. Subsequent to this multivariate analysis, comparisons can be made to determine the strongest predictors of elevated blood pressure in military personnel.

Fundamental to proper interpretation of binary logistic regression analysis is the identification of the statistical significance of the model. In SPSS, this statistical significance is found in the “Omnibus Tests of Model Coefficients” box (Table 9). To confirm a statistically significant regression model the value in the intersection of the last row “Model” and the last column “Significance” must be less than .05. This binary logistic regression model indicates a statistical significance of .000.

It is equally important to understand the relationships between the independent and dependent variables in the model. The details of these relationships are found in the SPSS “Variables in the Equation” box, (Table 10). It is crucial to note that only the Research Question 2 predictor overweight/obese (BMI equal to or greater than 25) in the “*p*” column is less than .05. Both the Research Question 1 predictor combat deployment (deployment length equal to or greater than 30 days) and the Research Question 3 predictor old (age equal to or greater than 40 years) are greater than .05. Subsequently the “B” column values of each independent variable in the model were analyzed and it is noted that all predictors have positive values representing positive relationships with the dependent variable.

Finally, the “OR” column was examined, which contains the odds ratios. This revealed that elevated blood pressure/hypertension is greater than three times more likely occur in the presence of only the Research Question 2 overweight/obese predictor (BMI equal to or greater than 25) independent variable.

Table 9

Binary Logistic Regression

Omnibus Tests of Model Coefficients				
		Chi-Square	df	Significance
Step 1	Step	27.246	3	0.000
	Block	27.246	3	0.000
	Model	27.246	3	0.000

Table 10

Summary of Logistic Regression Analysis Predicting Elevated BP

				95% CI for OR			
Variables in the Equation	*B*	*S.E.*	OR	Lower	Upper	*Wald*	*p*
Combat Deployment	0.226	0.291	1.254	0.708	2.219	0.603	0.437
Overweight/Obese	1.123	0.250	3.074	1.883	5.020	20.14	0.000
Old	0.537	0.334	1.711	0.889	3.292	8	0.108

This binary logistic regression model indicates that while controlling for exposure to combat deployment (*RQ1* - deployment length equal to or greater than 30 days), and being older (*RQ3* - age equal to or greater than 40 years), among military personnel to have elevated blood pressure/hypertension, only one statistically significant finding exists, which is that those military personnel who are exposed to being overweight/obese (*RQ2* - BMI equal to or greater than 25) are greater than three times (OR=3.074) more likely to have elevated blood pressure/hypertension relative to those who have a normal weight (BMI less than 25).

Summary

Analysis of the data reveals that though exposure to a combat deployment does produce an increase in the prevalence of the new onset of elevated blood pressure among active duty military personnel, it is not statistically significant. Being overweight/obese produces an increase in the prevalence of the new onset of elevated blood pressure among active duty military personnel that is statistically significant. Additionally, binary logistic regression indicates that even when controlling for exposure to combat deployment and of being older, those military personnel who are exposed to being overweight/obese are greater than three times more likely to have elevated blood pressure.

Analysis of the data also shows that being older produces both a positive and a statistically significant increase in the prevalence of the new onset of elevated blood pressure among active duty military personnel. However, when controlling for exposure to combat deployment and being overweight/obese, being older does not produce a statistically significant increase in the prevalence of the new onset of elevated blood pressure among active duty military personnel. Chapter 5 provides an interpretation of these results based upon the literature, stipulates limitations to those interpretations, posits practical implications, and then imparts appropriate recommendations for future research.

Chapter 5: Discussion, Conclusions, and Recommendations

Introduction

The purpose of this research was to describe the extent of association between exposure to combat deployment, being older, being overweight/obese and hypertension among U.S. military personnel who deployed to an area of declared combat between 2012 and 2017. I made three noteworthy discoveries during the analysis of this study. First, exposure to combat deployment produced an increase in the prevalence of the new onset of elevated blood pressure among active duty military personnel, but it was not statistically significant. Second, being older produced a statistically significant increase in the prevalence of the new onset of elevated blood pressure among active duty military personnel; however, when controlling for exposure to combat deployment and being overweight/obese, being older was not statistically significant. Third, being overweight/obese produced an increase in the prevalence of the new onset of elevated blood pressure among active duty military personnel that was statistically significant, and it remained significant even when controlling for combat deployment and being older. Furthermore, the significance of this third discovery was magnified by the finding that those military personnel who were overweight/obese were greater than three times more likely to have elevated blood pressure/hypertension than those exposed to combat deployment or who were older.

Interpretation of the Findings

Hypertension is one hazard that accompanies life in the U.S. military and contributes to the development of serious diseases and premature deaths (Granado et al., 2009; Smoley et al., 2008). Multiple exposures such as combat deployment, age, and obesity, may lead to the development of hypertension (Chiong, 2008; Landsberg et al., 2013; De Marco et al., 2009).

My initial key finding from this study was that exposure to combat deployment produced a statistically insignificant increase in the prevalence of the new onset of elevated blood pressure among military personnel. This finding is similar with the weak and varied literature findings regarding recurrent adrenal gland reactivity to stress being a possible risk for hypertension (al'Absi & Wittmers, 2003; Charney, 2004; Flaa et al., 2008). Adrenalized overresponsiveness to stress only produces episodic blood pressure elevations (al'Absi & Wittmers, 2003). However, these intermittent elevations of blood pressure could bring about a new and permanent change in vascular structure and function resulting in permanent hypertension (Charney, 2004). Flaa (2008) found hypertension was not able to be exclusively induced from those intermittent elevations of blood pressure. Additionally, Buckley (2004) states that blood pressure responses to stress are significantly intensified in the presence of post-traumatic stress disorder. However, Friedman (2001) found no real relationship between mental conditions and hypertension. Hildrum (2008) associated low blood pressure with mental stress disorders (Hildrum et al., 2008), but Johannessen (2006) demonstrates at least some association between certain mental conditions and hypertension.

My second key finding from this research study was a statistically significant increase in the prevalence of new onset elevated blood pressure among active duty military personnel who were older. However, this finding became nonsignificant when controlling for the stresses of combat deployment and being overweight/obese. This was similar to the variation of age-related findings in the literature. Franklin (1997) found that those individuals with the highest initial systolic blood pressure measurements have chronically higher and more significant hypertension than those individuals with only mildly elevated initial blood pressures. Chobanian (2003) states that people who maintain a normal blood pressure even up to the age of 55 are subject to a 90% risk of hypertension throughout the remainder of their life.

The third key finding from this study was that being overweight/obese produced an increase in the prevalence of the new onset of elevated blood pressure among active duty military personnel and it remained statistically significant even when controlling for the stresses of combat deployment and being older. Military personnel who were overweight/obese were greater than three times more likely than those exposed to the stresses of combat deployment and older individuals to have elevated blood pressure. This confirms the similar findings in the literature between the association of hypertension and the state of being overweight in an individual (Bogers et al., 2007; Chiong, 2008; DeMarco et al., 2009; Drukteinis et al., 2007; Fields et al., 2004; Gelber et al., 2007; Hall, 2003; Julius et al., 2000; Kannel, 2000; Kannel et al., 1967; Landsberg et al., 2013; Mensah & Brown, 2007; McNiece et al., 2007; Moore et al., 2005; Must et al., 1999; Ray et al., 2011; Rosmond, 2005; Salvadori et al., 2008; Stamler et al., 1978; Stevens et al., 2001; Torrance et al., 2007; Whelton et al., 2002; Williams et al., 1992; Wyatt et al., 2008). The association appears to function with whichever of the alternatives is the case; if weight is gained, increases in blood pressure are frequently the result, and if weight is lost, the result is often a decrease in blood pressure (Julius, Valentini, & Palatini , 2000). These associations imply the existence of a physiological process that results in a response associated with alterations in blood pressure and weight. What is not known is whether this relationship between weight and blood pressure is causal or not.

Limitations of the Study

One limitation was that the only blood pressure measurements used were performed pre and post deployment, instead of additionally using measurements made during the deployment. These additional blood pressure measurements could potentially allow for recognition of changes that may be tied to very specific events or circumstances during the deployment rather than subject to the combat deployment in general. This limitation exists because there were not enough during deployment blood pressure measurements available. Another closely related limitation was not parsing deployment exposure into much more specific combat and noncombat experiences to enable identification of blood pressure changes associated with individual experiences, specific events or circumstances. Again, though this information is desirable it is not currently collected in a consistent or useful form for study. A further limitation was not measuring the total number of deployments that an individual service member had completed, thereby affecting the ability to discern differences in exposure type and amount. Though this information about the number of deployments is available, there was not enough qualifying data to make correlation viable. Any of these limitations could have a significant impact alone or together, and therefore they could bias this study’s findings and produce conclusions that may not be as widely or generally applicable as hoped.

Additionally, my original calculation for this study, using G*Power estimates of multiple regression with power set at .80 and probability set at .05, with the three predictors (deployment, age, and body mass index), and looking for a small sized effect (.02), was a population sample size of 395. However, the DMDC and the MDR databases were collectively only able to obtain an eligible study population of 308 U.S. active duty service members. This fact decreases the power, generalizability, trustworthiness, validity, and reliability of the findings that arose during this study.

Recommendations

The central theoretical construct of this research was that psychosocial stress can induce and intensify high blood pressure (al'Absi & Wittmers, 2003; Artinian et al., 2006; Babu et al., 2014; Bruner & Woll, 2011; Din-Dzietham et al., 2004). Assenting to this construct does not challenge or oppose a multifactorial causality of hypertension that includes other factors such as obesity and age (Chiong, 2008; Cutler et al., 2008; Fields et al., 2004; De Marco et al., 2009). The multifactorial examination of causality performed in this research by regressing deployment, obesity, and age on hypertension to provide a comparison of strength of association revealed that obesity was the strongest association followed by age.

Although confirmation that psychosocial stress resulting from a combat deployment induces and intensifies high blood pressure was not accomplished with this research, I did determine that an elevation in blood pressure occurs among those who were normotensive prior to deployment, just not in significant numbers. The temporality of this information still points toward the potential of causality by supplying some evidence supporting the validation of the theory that stressors of combat deployment produce hypertension.

Additionally, because this is the first research to use actual blood pressure measurement data, which is fundamental to a knowledge that proceeds from observations to the deduction of probable causes, there should be a continuation of research for further clarification. My recommendations would initially address the limitations already stated. One recommendation would be to design research with mid deployment and not just pre and post deployment blood pressure measurements. Another would be to design research with the ability to parse deployment exposure into combat and noncombat experiences and to measure the total number of deployments that an individual subject has completed, thereby identifying whether differences in exposure type and amount are significant (Granado et al., 2009). The last and most important recommendation would be to obtain research with greater power by increasing population sample size.

Implications

The most important finding of this study is that being overweight/obese produced a statistically significant increase in the prevalence of new onset elevated blood pressure among military personnel and that this increase remained statistically significant even when controlling for exposure to combat deployment and being older. This finding was bolstered by the discovery that military personnel exposed to being overweight/obese were greater than 3 times more likely than those exposed to combat deployment and being older to have elevated blood pressure/hypertension.

A concern already established in the literature is that maintenance of normal blood pressure is critical to human survival (Chiong, 2008; Chobanian et al., 2003). The implication from this research is that military personnel exposed to being overweight/obese are at a much greater risk of sustaining and succumbing to the adverse effects of hypertension. The application of information from this research should raise awareness among individual military personnel and within the community of the DOD about how being overweight/obese can affect a soldier's health. This heightened awareness of the increased hypertension risk concomitant with being overweight/obese will hopefully encourage individual service members to alter personal risk factors able to generate hypertension.

The potential impact for positive social change in the DOD would be in the acknowledgement of the importance and seriousness of concern in sustaining proper body mass indexes for all employees because of the significance of the risk for hypertension. The DOD should proactively emplace employee and family wellness centers on all installations and require enforcement of medically derived appropriate weight maintenance for all employees. These adaptations could be instrumental in improving wellness through the reduction of hypertension risk among military personnel.

Conclusion

A normal life is not static, but instead is perpetually adapting to life's exposures. Combat deployment, being old, and being overweight/obese are life exposures for some military personnel that can be associated with stress and hypertension. This research demonstrates that exposure to a combat deployment does not by itself significantly increase the prevalence of new onset elevated blood pressure among active duty military personnel. Likewise, being older, though initially associated with a significant increase in elevated blood pressure, subsequently vanishes when controlling for exposure to combat deployment and being overweight/obese.

However, the singular key finding of this study is that being overweight/obese significantly increased the prevalence of new onset elevated blood pressure. Notably this finding was corroborated even when controlling for exposure to combat deployment and being older. Even more remarkable was that the odds ratio found by regressing deployment, obesity, and age on hypertension to provide a comparison of strength of association demonstrated military personnel who were exposed to being overweight/obese were greater than three times more likely to have elevated blood pressure/hypertension.

This study advances knowledge of the extent of the association of being overweight/obese to new onset hypertension or possibly even exacerbation of previously controlled hypertension in military personnel. The real import of this awareness is found in the recognition of the danger of not knowing that hypertension has occurred. Hypertension can cause serious harm without experiencing symptoms. Therefore, if hypertension can progress unrecognized and untreated, then disability or death or both are reasonable and predictable results.

Hence the significance of increasing awareness of inherent hypertension risk for military personnel is both conspicuous and salient. Moreover, this study should boost concern about how being overweight/obese can affect a soldier’s health, encouraging individual service members to alter their risk factors for hypertension, and inspiring military organizations and military communities to demand expansion of employee wellness centers on all installations and require enforcement of medically derived appropriate weight maintenance for all employees. These social adaptations could be instrumental in improving wellness among military personnel.

References

Abouzeid, M., Kelsall, H. L., Forbes, A. B., Sim, M. R., & Creamer, M. C. (2012). Posttraumatic stress disorder and hypertension in Australian veterans of the 1991 gulf war. *Journal of Psychosomatic Research*, *72*(1), 33-8. doi:10.1016/j.jpsychores.2011.08.002

al'Absi, M., & Wittmers, L. E. (2003). Enhanced adrenocortical responses to stress in hypertension-prone men and women. *Annals of Behavioral Medicine*, *25*(1), 25-33.

al'Absi, M., Devereux, R. B., Lewis, C. E., Kitzman, D. W., Rao, D. C., Hopkins, P., . . . Arnett, D. K. (2002). Blood pressure responses to acute stress and left ventricular mass (the hypertension genetic epidemiology network study). *The American Journal of Cardiology*, *89*(5), 536-540.

al'Absi, M., Lovallo, W. R., McKey, B. S., & Pincomb, G. A. (1994). Borderline hypertensives produce exaggerated adrenocortical responses to mental stress. *Psychosomatic Medicine*, *56*(3), 245.

al'Absi, M., Lovallo, W. R., McKey, B., Sung, B. H., Whitsett, T. L., & Wilson, M. F. (1998). Hypothalamic-pituitary-adrenocortical responses to psychological stress and caffeine in men at high and low risk for hypertension. *Psychosomatic Medicine*, *60*(4), 521-527.

Armario, A. (2006). The hypothalamic-pituitary-adrenal axis: What can it tell us about stressors? *CNS & Neurological Disorders - Drug Targets (Formerly Current Drug Targets, 5(*5). doi:10.2174/187152706778559336

Armario, A., Daviu, N., Muñoz-Abellán, C., Rabasa, C., Fuentes, S., Belda, X., . . . Nadal, R. (2012). What can we know from pituitary–adrenal hormones about the nature and consequences of exposure to emotional stressors? *Cellular and Molecular Neurobiology, 32*(5), 749-758. doi:10.1007/s10571-012-9814-6

Artinian, N. T., Washington, O. G., Flack, J. M., Hockman, E. M., & Jen, K. -L. C. (2006). Depression, stress, and blood pressure in urban African-American women. *Progress in Cardiovascular Nursing*, *21*(2), 68-75.

Babu, G. R., Jotheeswaran, A. T., Mahapatra, T., Mahapatra, S., Kumar, A., Detels, R., & Pearce, N. (2014). Is hypertension associated with job strain? A meta-analysis of observational studies. *Occupational and Environmental Medicine*, *71*(3), 220-7. doi:10.1136/oemed-2013-101396

Bagnell, M. E., Leardmann, C. A., McMaster, H. S., Boyko, E. J., Smith, B., Granado, N. S., & Smith, T. C. (2013). The association of predeployment and deployment-related factors on dimensions of postdeployment wellness in U.S. Military service members. *American Journal of Health Promotion: AJHP*, *28*(2), 56-66. doi:10.4278/ajhp.120727-QUAN-366

Banerjee, D., Chung, S., Wong, E. C., Wang, E. J., Stafford, R. S., & Palaniappan, L. P. (2012). Underdiagnosis of hypertension using electronic health records. *American Journal of Hypertension*, *25*(1), 97-102. doi:10.1038/ajh.2011.179

Baxi, N. S., Jackson, J. L., Ritter, J., & Sessums, L. L. (2011). How well do the Framingham risk factors correlate with diagnoses of ischemic heart disease and cerebrovascular disease in a military beneficiary cohort? *Military Medicine*, *176*(4), 408-413.

Belasco, A. (2009). Troop levels in the Afghan and Iraq wars, FY2001-FY2012: Cost and other potential issues. Washington, DC: Congressional Research Service. Retrieved from http://www.fas.org/sgp/crs/natsec/R40682.pdf

Bevan, R. D., Tsuru, H., & Bevan, J. A. (1983). Cerebral artery mass in the rabbit is reduced by chronic sympathetic denervation. *Stroke; A Journal of Cerebral Circulation*, *14*(3), 393-396. doi:10.1161/01.str.14.3.393

Bijlsma, R., & Loeschcke, V. (2005). Environmental stress, adaptation and evolution: An overview. *Journal of Evolutionary Biology*, *18*(4), 744-9. doi:10.1111/j.1420-9101.2005.00962.x

Bogers, R. P., Bemelmans, W. J., Hoogenveen, R. T., Boshuizen, H. C., Woodward, M., Knekt, P., . . . Menotti, A. (2007). Association of overweight with increased risk of coronary heart disease partly independent of blood pressure and cholesterol levels: A meta-analysis of 21 cohort studies including more than 300 000 persons. *Archives of Internal Medicine*, *167*(16), 1720-1728.

Bowman, T. S., Gaziano, J. M., Buring, J. E., & Sesso, H. D. (2007). A prospective study of cigarette smoking and risk of incident hypertension in women. *Journal of the American College of Cardiology*, *50*(21), 2085-92. doi:10.1016/j.jacc.2007.08.017

Bracha, H. S., Ralston, T. C., Matsukawa, J. M., Williams, A. E., & Bracha, A. S. (2004). Does fight or flight need updating? *Psychosomatics*, *45*(5), 448-449.

Brener, N. D., Billy, J. O., & Grady, W. R. (2003). Assessment of factors affecting the validity of self-reported health-risk behavior among adolescents: Evidence from the scientific literature. *Journal of Adolescent Health*, *33*(6), 436-457. doi:10.1016/s1054-139x(03)00052-1

Brooks, D., Horner, R. L., Kozar, L. F., Render-Teixeira, C. L., & Phillipson, E. A. (1997). Obstructive sleep apnea as a cause of systemic hypertension. Evidence from a canine model. *Journal of Clinical Investigation*, *99*(1), 106.

Bruehl, S., Chung, O. Y., Ward, P., Johnson, B., & McCubbin, J. A. (2002). The relationship between resting blood pressure and acute pain sensitivity in healthy normotensives and chronic back pain sufferers: The effects of opioid blockade. *Pain*, *100*(1), 191-201. doi:10.1016/s0304-3959(02)00295-6

Bruner, V. E., & Woll, P. (2011). The battle within: Understanding the physiology of war-zone stress exposure. *Social Work in Health Care*, *50*(1), 19-33. doi:10.1080/00981389.2010.513915

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*(2), 163-171.

Campbell, T. S., Hughes, J. W., Girdler, S. S., Maixner, W., & Sherwood, A. (2004). Relationship of ethnicity, gender, and ambulatory blood pressure to pain sensitivity: Effects of individualized pain rating scales. *The Journal of Pain: Official Journal of the American Pain Society*, *5*(3), 183-91. doi:10.1016/j.jpain.2004.02.305

Cannon, W. B. (1932). *The wisdom of the body.* New York, NY, US: W.W. Norton & Co.

Cappuccio, F. P., Stranges, S., Kandala, N. B., Miller, M. A., Taggart, F. M., Kumari, M., . . . Marmot, M. G. (2007). Gender-specific associations of short sleep duration with prevalent and incident hypertension: The Whitehall II study. *Hypertension*, *50*(4), 693-700. doi:10.1161/HYPERTENSIONAHA.107.095471

Carroll, D., Phillips, A. C., Gale, C. R., & Batty, G. D. (2010). Generalized anxiety and major depressive disorders, their comorbidity and hypertension in middle-aged men. *Psychosomatic Medicine*, *72*(1), 16-9. doi:10.1097/PSY.0b013e3181c4fca1

Charney, D. S. (2004). Psychobiological mechanisms of resilience and vulnerability implications for successful adaptation to extreme stress. *FOCUS: The Journal of Lifelong Learning in Psychiatry*, *2*(3), 368-391.

Cheung, B. C., Au, T., Chan, S., Lam, C., Lau, S., Lee, R., . . . Tsang, H. (2005). The relationship between hypertension and anxiety or depression in Hong Kong Chinese. *Experimental Clinical Cardiology*, *10*(1), 21-24.

Cheung, B. M., Ong, K. L., Man, Y. B., Lam, K. S., & Lau, C. -P. (2006). Prevalence, awareness, treatment, and control of hypertension: United States national health and nutrition examination survey 2001--2002. *The Journal of Clinical Hypertension*, *8*(2), 93-98.

Chiong, J. R. (2008). Controlling hypertension from a public health perspective. *International Journal of Cardiology*, *127*(2), 151-6. doi:10.1016/j.ijcard.2007.10.039

Chobanian, A. V., Bakris, G. L., Black, H. R., Cushman, W. C., Green, L. A., Izzo, J. L., . . . National High Blood Pressure Education Program Coordinating Committee.

(2003a). Seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. *Hypertension*, *42*(6), 1206-52. doi:10.1161/01.HYP.0000107251.49515.c2

Chobanian, A. V., Bakris, G. L., Black, H. R., Cushman, W. C., Green, L. A., Izzo Jr, J. L., . . . Wright Jr, J. T. (2003b). The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure: The JNC 7 report. *JAMA: The Journal of the American Medical Association*, *289*(19), 2560-2571.

Chrousos, G. P. (2009). Stress and disorders of the stress system. *Nature Reviews Endocrinology, 5*(7), 374-381. doi:10.1038/nrendo.2009.106

Clark, K. (2000). Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study. *Journal of Insurance Medicine*, *32*, 195-197.

Cohen J. E. (1988). Statistical power analysis for the behavioral sciences. Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

Cohen, J. (1992). A power primer. *Psychological Bulletin*, *112*(1), 155-159.

Cohen, S., Janicki-Deverts, D., & Miller, G. E. (2007). Psychological stress and disease. *JAMA: The Journal of the American Medical Association, 298*(14), 1685-1687.

Cook, N. R. (2007). Use and misuse of the receiver operating characteristic curve in risk prediction. *Circulation, 115*(7), 928-35. doi:10.1161/CIRCULATIONAHA.106.672402.

Coughlin, S. S. (2011). Post-traumatic stress disorder and cardiovascular disease. *The*

Open Cardiovascular Medicine Journal, *5*, 164.

Cutler, J. A., Sorlie, P. D., Wolz, M., Thom, T., Fields, L. E., & Roccella, E. J. (2008). Trends in hypertension prevalence, awareness, treatment, and control rates in United States adults between 1988-1994 and 1999-2004. *Hypertension*, *52*(5), 818-27. doi:10.1161/HYPERTENSIONAHA.108.113357

Dallman, M. F. (2003). Stress by any other name …? *Hormones and Behavior, 43*(1), 18-20. doi:10.1016/S0018-506X(02)00034-X

Day, T. A. (2005). Defining stress as a prelude to mapping its neurocircuitry: No help from allostasis. *Progress in Neuro-psychopharmacology & Biological Psychiatry*, *29*(8), 1195-200. doi:10.1016/j.pnpbp.2005.08.005

De Marco, M., de Simone, G., Roman, M. J., Chinali, M., Lee, E. T., Russell, M., . . . Devereux, R. B. (2009). Cardiovascular and metabolic predictors of progression of prehypertension into hypertension: The strong heart study. *Hypertension*, *54*(5), 974-80. doi:10.1161/HYPERTENSIONAHA.109.129031

Devereux,. B., Pickering,. G., Harshfield,. A., Kleinert,. D., Denby, L., Clark, L., . . . Laragh, H. (1983). Left ventricular hypertrophy in patients with hypertension: Importance of blood pressure response to regularly recurring stress. *Circulation*, *68*(3), 470-476. doi:10.1161/01.CIR.68.3.470

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, *130*(3), 355-91. doi:10.1037/0033-2909.130.3.355

Din-Dzietham, R., Nembhard, W. N., Collins, R., & Davis, S. K. (2004). Perceived stress following race-based discrimination at work is associated with hypertension in African–Americans. The metro Atlanta heart disease study, 1999–2001. *Social Science & Medicine*, *58*(3), 449-461. doi:10.1016/S0277-9536(03)00211-9

Doroszko, A., Andrzejak, R., & Szuba, A. (2011). Role of the nitric oxide metabolic pathway and prostanoids in the pathogenesis of endothelial dysfunction and essential hypertension in young men. *Hypertension Research: Official Journal of the Japanese Society of Hypertension*, *34*(1), 79-86. doi:10.1038/hr.2010.169

Drukteinis, J. S., Roman, M. J., Fabsitz, R. R., Lee, E. T., Best, L. G., Russell, M., & Devereux, R. B. (2007). Cardiac and systemic hemodynamic characteristics of hypertension and prehypertension in adolescents and young adults: The strong heart study. *Circulation*, *115*(2), 221-7. doi:10.1161/CIRCULATIONAHA.106.66892

Egan, B. M., Zhao, Y., & Axon, R. N. (2010). US trends in prevalence, awareness, treatment, and control of hypertension, 1988-2008. *JAMA: The Journal of the American Medical Association*, *303*(20), 2043-2050.

Ezzati, M., Lopez, A. D., Rodgers, A., Vander Hoorn, S., & Murray, C. J. (2002). Selected major risk factors and global and regional burden of disease. *The Lancet*, *360*(9343), 1347-1360.

Fauci, A. S. (2001). Infectious diseases: Considerations for the 21st century. *Clinical Infectious Diseases*, *32*(5), 675-685.

Fields, L. E., Burt, V. L., Cutler, J. A., Hughes, J., Roccella, E. J., & Sorlie, P. (2004). The burden of adult hypertension in the United States 1999 to 2000: A rising tide.

Hypertension, *44*(4), 398-404. doi:10.1161/01.HYP.0000142248.54761.56

Flaa, A., Eide, I. K., Kjeldsen, S. E., & Rostrup, M. (2008). Sympathoadrenal stress reactivity is a predictor of future blood pressure: An 18-year follow-up study. *Hypertension*, *52*(2), 336-41. doi:10.1161/HYPERTENSIONAHA.108.11162

Fletcher, R. D., Amdur, R. L., Kolodner, R., McManus, C., Jones, R., Faselis, C., . . . Papademetriou, V. (2012). Blood pressure control among US veterans: A large multiyear analysis of blood pressure data from the veterans administration health data repository. *Circulation*, *125*(20), 2462-8. doi:10.1161/CIRCULATIONAHA.111.029983

Foraster, M., Künzli, N., Aguilera, I., Rivera, M., Agis, D., Vila, J., . . . Basagaña, X. (2014). High blood pressure and long-term exposure to indoor noise and air pollution from road traffic. *Environmental Health Perspectives, 122*(11), 1193-200. doi:10.1289/ehp.1307156

Forthofer, R. N., Lee, E. S., & Hernandez, M. (2007). *Biostatistics: A guide to design, analysis, and discovery*. Retrieved from http://site.ebrary.com/id/10158361

France, C. R. (1999). Decreased pain perception and risk for hypertension: Considering a common physiological mechanism. *Psychophysiology*, *36*(06), 683-692.

Franklin, S. S., Gustin, W., Wong, N. D., Larson, M. G., Weber, M. A., Kannel, W. B., & Levy, D. (1997). Hemodynamic patterns of age-related changes in blood pressure. *Circulation*, *96*(1), 308-315. doi:10.1161/01.CIR.96.1.308

Friedman, R., Schwartz, J. E., Schnall, P. L., Landsbergis, P. A., Pieper, C., Gerin, W., & Pickering, T. G. (2001). Psychological variables in hypertension: Relationship to casual or ambulatory blood pressure in men. *Psychosomatic Medicine*, *63*(1), 19-31. doi:10.1097/00006842-200101000-0000

Garrison, R. J., Kannel, W. B., Stokes, J., & Castelli, W. P. (1987). Incidence and precursors of hypertension in young adults: The Framingham offspring study. *Preventive Medicine*, *16*(2), 235-251. doi:10.1016/0091-7435(87)90087-9

Gelber, R. P., Gaziano, J. M., Manson, J. E., Buring, J. E., & Sesso, H. D. (2007). A prospective study of body mass index and the risk of developing hypertension in men. *American Journal of Hypertension*, *20*(4), 370-7. doi:10.1016/j.amjhyper.2006.10.011

Giles, T. D., Sander, G. E., Nossaman, B. D., & Kadowitz, P. J. (2012). Impaired vasodilation in the pathogenesis of hypertension: Focus on nitric oxide, endothelial-derived hyperpolarizing factors, and prostaglandins. *Journal of Clinical Hypertension*, *14*(4), 198-205. doi:10.1111/j.1751-7176.2012.00606.x

Gordon Jr, R. S. (1983). An operational classification of disease prevention. *Public Health Reports*, *98*(2), 107.

Granado, N. S., Smith, T. C., Swanson, G. M., Harris, R. B., Shahar, E., Smith, B., . . . Millennium Cohort Study Team. (2009). Newly reported hypertension after military combat deployment in a large population-based study. *Hypertension*, *54*(5), 966-73. doi:10.1161/HYPERTENSIONAHA.109.132555

Green, M. S., Jucha, E., & Luz, Y. (1986). Blood pressure in smokers and nonsmokers: Epidemiologic findings. *American Heart Journal*, *111*(5), 932-940. doi:10.1016/0002-8703(86)90645-9

Griffin, J. F. T., & Thomson, A. J. (1998). Farmed deer: A large animal model for stress. *Domestic Animal Endocrinology*, *15*(5), 445-456.

Grimsrud, A., Stein, D. J., Seedat, S., Williams, D., & Myer, L. (2009). The association between hypertension and depression and anxiety disorders: Results from a nationally-representative sample of South African adults. *PloS One*, *4*(5), e5552. doi:10.1371/journal.pone.0005552

Groppelli, A., Giorgi, D. M. A., Omboni, S., Parati, G., & Mancia, G. (1992). Persistent blood pressure increase induced by heavy smoking. *Journal of Hypertension*, *10*(5), 495-499. doi:10.1097/00004872-199205000-00014

Gross, . G. (1998). Claude Bernard and the constancy of the internal environment. *The Neuroscientist*, *4*(5), 380-385. doi:10.1177/107385849800400520

Gross, C. G. (2009a). Three before their time: Neuroscientists whose ideas were ignored by their contemporaries. *Experimental Brain Research*, *192*(3), 321-34. doi:10.1007/s00221-008-1481-y

Gross, C. G. (2009b). Three before their time: Neuroscientists whose ideas were ignored by their contemporaries. *Experimental Brain Research*, *192*(3), 321-34. doi:10.1007/s00221-008-1481-y

Grote, L., Hedner, J., & Peter, J. H. (2000). Sleep-related breathing disorder is an independent risk factor for uncontrolled hypertension. *Journal of Hypertension*, *18*(6), 679-85.

Gu, Q., Dillon, C. F., Burt, V. L., & Gillum, R. F. (2010). Association of hypertension treatment and control with all-cause and cardiovascular disease mortality among

US adults with hypertension. *American Journal of Hypertension*, *23*(1), 38-45. doi:10.1038/ajh.2009.191

Guo, F., He, D., Zhang, W., & Walton, R. G. (2012). Trends in prevalence, awareness, management, and control of hypertension among United States adults, 1999 to 2010. *Journal of the American College of Cardiology*, *60*(7), 599-606. doi:10.1016/j.jacc.2012.04.026

Hall, J. E. (2003). The kidney, hypertension, and obesity. *Hypertension*, *41*(3 Pt 2), 625-33. doi:10.1161/01.HYP.0000052314.95497.78

Hall, J. E., Granger, J. P., do Carmo, J. M., da Silva, A. A., Dubinion, J., George, E., . . . Hall, M. E. (2012). Hypertension: Physiology and pathophysiology. *Comprehensive Physiology,2*, 2393-2442. doi:10.1002/cphy.c110058

Hakre, S., Peel, S. A., O'Connell, R. J., Sanders-Buell, E. E., Jagodzinski, L. L., Eggleston, J. C., . . . Scott, P. T. (2010). Transfusion-transmissible viral infections among U.S. military recipients of whole blood and platelets during operation enduring freedom and operation iraqi freedom. *Transfusion*, *51*(3), 473-485. doi:10.1111/j.1537-2995.2010.02906.x

Hart, M. N., Heistad, D. D., & Brody, M. J. (1980). Effect of chronic hypertension and sympathetic denervation on wall/lumen ratio of cerebral vessels. *Hypertension*, *2*(4), 419-423. doi:10.1161/01.hyp.2.4.419

Helmer, D. A., Rossignol, M., Blatt, M., Agarwal, R., Teichman, R., & Lange, G. (2007). Health and exposure concerns of veterans deployed to Iraq and Afghanistan. *Journal of Occupational and Environmental Medicine*, *49*(5), 475.

doi:10.1097/JOM.0b013e318042d682

Hicks, L.S., Fairchild, D. G., Horng, M.S., Orav, E.J., Bates, D.W., & Ayanian, J.Z. (2004). Determinants of JNC VI guideline adherence, intensity of drug therapy, and blood pressure control by race and ethnicity. *Hypertension*, *44*(4), 429-34. doi:10.1161/01.HYP.0000141439.34834.84

Hildrum, B., Mykletun, A., Holmen, J., & Dahl, A. A. (2008). Effect of anxiety and depression on blood pressure: 11-year longitudinal population study. *The British Journal of Psychiatry: The Journal of Mental Science*, *193*(2), 108-13. doi:10.1192/bjp.bp.107.04501

Holman, E. A., Silver, R. C., Poulin, M., Andersen, J., Gil-Rivas, V., & McIntosh, D. N. (2008). Terrorism, acute stress, and cardiovascular health: A 3-year national study following the September 11th attacks. *Archives of General Psychiatry*, *65*(1), 73-80.

James, P. A., Oparil, S., Carter, B. L., Cushman, W. C., Dennison-Himmelfarb, C., Handler, J., . . . Ortiz, E. (2014). 2014 evidence-based guideline for the management of high blood pressure in adults: Report from the panel members appointed to the eighth joint national committee (JNC 8). *JAMA: The Journal of the American Medical Association*, *311*(5), 507-20. doi:10.1001/jama.2013.284427

Joëls, M., & Baram, T. Z. (2009). The neuro-symphony of stress. *Nature Reviews. Neuroscience*, *10*(6), 459-66. doi:10.1038/nrn2632

Johannessen, L., Strudsholm, U., Foldager, L., & Munk-Jørgensen, P. (2006). Increased risk of hypertension in patients with bipolar disorder and patients with anxiety compared to background population and patients with schizophrenia. *Journal of Affective Disorders*, *95*(1-3), 13-7. doi:10.1016/j.jad.2006.03.027

Jonas, B. S., & Lando, J. F. (2000). Negative affect as a prospective risk factor for hypertension. *Psychosomatic Medicine*, *62*(2), 188-196. doi:10.1097/00006842-200003000-00006

Jorgensen, R. S. (2009). Newly reported hypertension after military combat deployment: Research implications from a biopsychosocial perspective. *Hypertension*, *54*(5), 956-7. doi:10.1161/HYPERTENSIONAHA.109.134478

Julius, S., Valentini, M., & Palatini, P. (2000). Overweight and hypertension: A 2-way street? *Hypertension*, *35*(3), 807-813. doi:10.1161/01.hyp.35.3.80

Kahn, H. A., Medalie, J. H., Neufeld, H. N., Riss, E., & Goldbourt, U. (1972). The incidence of hypertension and associated factors: The Israel ischemic heart disease study. *American Heart Journal*, *84*(2), 171-182. doi:10.1016/0002-8703(72)90331-6

Kannel, W. B., Brand, N., Skinner, J. J., Dawber, T. R., & McNamara, P. M. (1967). The relation of adiposity to blood pressure and development of hypertension (the Framingham study). *Annals of Internal Medicine*, *67*(1), 48-59.

Kannel, W. B. (2000). Risk stratification in hypertension: New insights from the Framingham study. *American Journal of Hypertension*, *13*(S1), 3S-10S. doi:10.1016/S0895-7061(99)00252-6

Kaplan, N. M. (1998). The 6th joint national committee report (JNC-6): New guidelines for hypertension therapy from the USA. *The Keio Journal of Medicine*, *47*(2), 99-105.

Karatsoreos, I. N., & McEwen, B. S. (2011). Psychobiological allostasis: Resistance, resilience and vulnerability. *Trends in Cognitive Sciences*, *15*(12), 576-84. doi:10.1016/j.tics.2011.10.005

Kario, K. (2012). Disaster hypertension. *Circulation Journal*, *76*(3), 553-562. doi:10.1253/circj.cj-11-1510

Kearney, P. M., Whelton, M., Reynolds, K., Whelton, P. K., & He, J. (2004). Worldwide prevalence of hypertension: A systematic review. *Journal of Hypertension*, *22*(1), 11-19.

Kearney, P. M., Whelton, M., Reynolds, K., Muntner, P., Whelton, P. K., & He, J. (2005). Global burden of hypertension: Analysis of worldwide data. *The Lancet*, *365*(9455), 217-223.

Kibler, J. L., Joshi, K., & Ma, M. (2009). Hypertension in relation to posttraumatic stress disorder and depression in the U.S. national comorbidity survey. *Behavioral Medicine*, *34*(4), 125-132.

King, M. S., Eisenberg, R., Newman, J. H., Tolle, J. J., Harrell Jr, F. E., Nian, H., . . . Johnson, J. E. (2011). Constrictive bronchiolitis in soldiers returning from Iraq and Afghanistan. *New England Journal of Medicine*, *365*(3), 222-230.

de Kloet, E. R., Joëls, M., & Holsboer, F. (2005). Stress and the brain: From adaptation to disease. *Nature Reviews. Neuroscience*, *6*(6), 463-75. doi:10.1038/nrn1683

Knight, E. L., Bohn, R. L., Wang, P. S., Glynn, R. J., Mogun, H., & Avorn, J. (2001).

Predictors of uncontrolled hypertension in ambulatory patients. *Hypertension*, *38*(4), 809-814. doi:10.1161/hy0901.091681

Koolhaas, J. M., Bartolomucci, A., Buwalda, B., de Boer, S. F., Flügge, G., Korte, S. M., . . . Fuchs, E. (2011). Stress revisited: A critical evaluation of the stress concept. *Neuroscience and Biobehavioral Reviews*, *35*(5), 1291-301. doi:10.1016/j.neubiorev.2011.02.003

Kottke, T. E., Stroebel, R. J., & Hoffman, R. S. (2003). JNC 7--it's more than high blood pressure. *JAMA: The Journal of the American Medical Association*, *289*(19), 2573-5. doi:10.1001/jama.289.19.2573

Kuchel, O. (2003). Mental stress and hypertension, an evolutionary framework: Some historical perspectives of the 1960 World Health Organization Prague hypertension meeting. *Journal of Hypertension*, *21*(3). Retrieved from http://journals.lww.com/jhypertension/Fulltext/2003/03000/Mental_stress_and_hypertension,_an_evolutionary.32.aspx

Landsberg, L., Aronne, L. J., Beilin, L. J., Burke, V., Igel, L. I., Lloyd-Jones, D., & Sowers, J. (2013). Obesity-related hypertension: Pathogenesis, cardiovascular risk, and treatment--a position paper of the obesity society and the American society of hypertension. *Obesity (Silver Spring, Md.)*, *21*(1), 8-24. doi:10.1002/oby.20181

Lavie, P., Herer, P., & Hoffstein, V. (2000). Obstructive sleep apnoea syndrome as a risk factor for hypertension: Population study. *BMJ (Clinical Research Ed.)*, *320*(7233), 479-482.

Leone, A. (2011). Smoking and hypertension: Independent or additive effects to determining vascular damage? *Current Vascular Pharmacology*, *9*(5), 585-593.

Lester, M. E., Knapik, J. J., Catrambone, D., Antczak, A., Sharp, M. A., Burrell, L., & Darakjy, S. (2010). Effect of a 13-month deployment to Iraq on physical fitness and body composition. *Military Medicine*, *175*(6), 417-423.

Levine, S. (2005). Developmental determinants of sensitivity and resistance to stress. *Psychoneuroendocrinology*, *30*(10), 939-46. doi:10.1016/j.psyneuen.2005.03.013

Li, L., Law, C., & Power, C. (2007). Body mass index throughout the life-course and blood pressure in mid-adult life: A birth cohort study. *Journal of Hypertension*, *25*(6), 1215. doi:10.1097/HJH.0b013e3280f3c01a

Light, K. C., Girdler, S. S., Sherwood, A., Bragdon, E. E., Brownley, K. A., West, S. G., & Hinderliter, A. L. (1999). High stress responsivity predicts later blood pressure only in combination with positive family history and high life stress. *Hypertension*, *33*(6), 1458-1464. doi:10.1161/01.hyp.33.6.1458

Lopez, A. D., Mathers, C. D., Ezzati, M., Jamison, D. T., & Murray, C. J. (2006). Global and regional burden of disease and risk factors, 2001: Systematic analysis of population health data. *The Lancet*, *367*(9524), 1747-1757.

Lundin, R. L., & Steinthorsson, R. S. (2003). Studying organizations as temporary. *Scandinavian Journal of Management*, *19*, 233-250.

Majernick, T. G., Zacker, C., Madden, N. A., Belletti, D. A., & Arcona, S. (2004). Correlates of hypertension control in a primary care setting. *American Journal of Hypertension*, *17*(10), 915-20. doi:10.1016/j.amjhyper.2004.05.016

Manual Calculation Method. (n.d.). Manual calculation method. [Web page]. Retrieved from http://bphc.hrsa.gov/policiesregulations/performancemeasures/patientsurvey/calculating.html

Marine, A., Ruotsalainen, J., Serra, C., & Verbeek, J. (2006). Preventing occupational stress in healthcare workers. *The Cochrane Database of Systematic Reviews*, (4), CD002892. doi:10.1002/14651858.CD002892.pub2

Matthews, K. A., Salomon, K., Brady, S. S., & Allen, M. T. (2003). Cardiovascular reactivity to stress predicts future blood pressure in adolescence. *Psychosomatic Medicine*, *65*(3), 410-415. doi:10.1097/01.psy.0000057612.94797.5f

Matthews, K. A., Katholi, C. R., McCreath, H., Whooley, M. A., Williams, D. R., Zhu, S., & Markovitz, J. H. (2004). Blood pressure reactivity to psychological stress predicts hypertension in the CARDIA study. *Circulation, 110*(1), 74-8. doi:10.1161/01.CIR.0000133415.37578.E4

McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews*, *87*(3), 873-904. doi:10.1152/physrev00041.2006

McNiece, K. L., Poffenbarger, T. S., Turner, J. L., Franco, K. D., Sorof, J. M., & Portman, R. J. (2007). Prevalence of hypertension and pre-hypertension among adolescents. *The Journal of Pediatrics*, *150*(6), 640-4, 644.e1. doi:10.1016/j.jpeds.2007.01.052

Melby, C. L. (1983). Inhibition of prostaglandin synthesis: A possible mechanism for stress-induced hypertension. *Medical Hypotheses*, *10*(4), 445-449. doi:/10.1016/0306-9877(83)90010-5

Mensah, G. A., & Brown, D. W. (2007). An overview of cardiovascular disease burden in the United States. *Health Affairs (Project Hope)*, *26*(1), 38-48. doi:10.1377/hlthaff.26.1.38

Messick, S. (1987). Validity. *ETS Research Report Series*, *1987*(2), i-208.

Milliken, C. S., Auchterlonie, J. L., & Hoge, C. W. (2007). Longitudinal assessment of mental health problems among active and reserve component soldiers returning from the Iraq war. *JAMA: The Journal of the American Medical Association, 298*(18), 2141-2148. doi:10.1001/jama.298.18

Minami, J., Ishimitsu, T., & Matsuoka, H. (1999). Effects of smoking cessation on blood pressure and heart rate variability in habitual smokers. *Hypertension*, *33*(1), 586-590. doi:10.1161/01.hyp.33.1.586

Moore, L. L., Visioni, A. J., Qureshi, M. M., Bradlee, M. L., Ellison, R. C., & D'Agostino, R. (2005). Weight loss in overweight adults and the long-term risk of hypertension: The Framingham study. *Archives of Internal Medicine*, *165*(11), 1298-1303. doi:10.1001/archinte.165.11.1298

Muller, E., Tofler, H., & Stone, H. (1989). Circadian variation and triggers of onset of acute cardiovascular disease. *Circulation*, *79*(4), 733-743. doi:10.1161/01.CIR.79.4.733

Must, A., Spadano, J., Coakley, E. H., Field, A. E., Colditz, G., & Dietz, W. H. (1999). The disease burden associated with overweight and obesity. *JAMA: The Journal* *of the American Medical Association*, *282*(16), 1523-1529.

Mustacchi, P. (1990). Stress and hypertension. *Western Journal of Medicine*, *153*(2), 180-185.

Neylan, T. C. (1998). Hans Selye and the field of stress research. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *10*(2), 230-231.

Nieto, F. J., Young, T. B., Lind, B. K., Shahar, E., Samet, J. M., Redline, S., . . . Pickering, T. G. (2000). Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study. *JAMA: The Journal of the American Medical Association*, *283*(14), 1829-1836.

Office of the Deputy Under Secretary of Defense. (2012). *2011 Demographics Profile of the Military Community*. Office of the Deputy Under Secretary of Defense. http://www.icfi.com/workforce:http://www.militaryonesource.mil/12038/MOS/Reports/2011_Demographics_Report.pdf.

Office of the Secretary of Defense. (2011). USCENTCOM 021922Z DEC 11 MOD ELEVEN TO USCENTCOM INDIVIDUAL PROTECTION AND INDIVIDUAL-UNIT DEPLOYMENT POLICY. *USCENTCOM*. Retrieved from https://www.cpms.osd.mil/expeditionary/pdf/MOD11-USCENTCOM-Indiv-Protection-Indiv-Unit-Deployment-Policy-Incl-Tab-A-and-B.pdf

Omvik, P. (2015). How smoking affects blood pressure. *Blood Pressure.* doi:10.3109/08037059609062111

Ong, K. L., Cheung, B. M., Man, Y. B., Lau, C. P., & Lam, K. S. (2007). Prevalence, awareness, treatment, and control of hypertension among United States adults

1999-2004. *Hypertension*, *49*(1), 69-75. doi:10.1161/01.HYP.0000252676.46043.18

Oparil, S., Zaman, M. A., & Calhoun, D. A. (2003). Pathogenesis of hypertension. *Annals of Internal Medicine*, *139*(9), 761-776.

Ornstein, S. M., Nietert, P. J., & Dickerson, L. M. (2004). Hypertension management and control in primary care: A study of 20 practices in 14 states. *Pharmacotherapy*, *24*(4), 500-507. doi:10.1592/phco.24.5.500.33359

Pearlin, L. I., Schieman, S., Fazio, E. M., & Meersman, S. C. (2005). Stress, health, and the life course: Some conceptual perspectives. *Journal of Health and Social Behavior, 46*(2), 205-219.

Peppard, P. E., Young, T., Palta, M., & Skatrud, J. (2000). Prospective study of the association between sleep-disordered breathing and hypertension. *New England Journal of Medicine*, *342*(19), 1378-84. doi:10.1056/NEJM200005113421901

Petruccelli, B. P., Goldenbaum, M., Scott, B., Lachiver, R., Kanjarpane, D., Elliott, E., . . . Deeter, D. (1999). Health effects of the 1991 Kuwait oil fires: A survey of US army troops. *Journal of Occupational and Environmental Medicine*, *41*(6), 433.

Pickering, T. G. (2001). Mental stress as a causal factor in the development of hypertension and cardiovascular disease. *Current Hypertension Reports*, *3*(3), 249-254.

Pi-Sunyer, F. X., Becker, D. M., Bouchard, C., Carleton, R. A., Colditz, G. A., Dietz, W. H., . . . Wilson, G. T. (1998). NHLBI obesity education initiative expert panel on the identification, evaluation, and treatment of overweight and obesity in adults

(US). Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: The Evidence Report. Bethesda (MD): National Heart, Lung, and Blood Institute; [Pamphlet] National Institutes of Health. NIH Publication (98-4083). Retrieved from http://www.ncbi.nlm.nih.gov/books/NBK2003/

Polusny, M. A., Erbes, C. R., Murdoch, M., Arbisi, P. A., Thuras, P., & Rath, M. B. (2011). Prospective risk factors for new-onset post-traumatic stress disorder in national guard soldiers deployed to Iraq. *Psychological Medicine, 41*(4), 687-98. doi:10.1017/S0033291710002047

Primatesta, P., Falaschetti, E., Gupta, S., Marmot, M. G., & Poulter, N. R. (2001). Association between smoking and blood pressure: Evidence from the health survey for England. *Hypertension*, *37*(2), 187-193. doi:10.1161/01.hyp.37.2.187

Qureshi, S. U., Pyne, J. M., Magruder, K. M., Schulz, P. E., & Kunik, M. E. (2009). The link between post-traumatic stress disorder and physical comorbidities: A systematic review. *The Psychiatric Quarterly*, *80*(2), 87-97. doi:10.1007/s11126-009-9096-4

Ray, S., Kulkarni, B., & Sreenivas, A. (2011). Prevalence of prehypertension in young military adults & its association with overweight & dyslipidaemia. *The Indian Journal of Medical Research*, *134*, 162-7.

Rimer, B. K., & Glanz, K. (2005). Theory at a glance: A guide for health promotion practice (Second edition). [Bethesda Maryland] National Institutes of Health [NIH] National Cancer Institute 2005. NIH Publication (05-3896). Retrieved from http://www.popline.org/node/276257

Rodriguez, B. L., Labarthe, D. R., Huang, B., & Lopez-Gomez, J. (1994). Rise of blood pressure with age. New evidence of population differences. *Hypertension*, *24*(6), 779-785. doi:10.1161/01.hyp.24.6.779

Romero, L. M., Dickens, M. J., & Cyr, N. E. (2009). The reactive scope model - a new model integrating homeostasis, allostasis, and stress. *Hormones and Behavior*, *55*(3), 375-89. doi:10.1016/j.yhbeh.2008.12.009

Rosmond, R. (2005). Role of stress in the pathogenesis of the metabolic syndrome. *Psychoneuroendocrinology*, *30*(1), 1-10. doi:10.1016/j.psyneuen.2004.05.007

Roy, S. S., Foraker, R. E., Girton, R. A., & Mansfield, A. J. (2015). Posttraumatic stress disorder and incident heart failure among a community-based sample of US veterans. *American Journal of Public Health*, *105*(4), 757-763. doi:10.2105/AJPH.2014.302342

Rubin, D. B. (1976). Inference and missing data. *Biometrika*, *63*(3), 581-592. doi:10.1093/biomet/63.3.581

Rutledge, T. (2002). A quantitative review of prospective evidence linking psychological factors with hypertension development. *Psychosomatic Medicine*, *64*(5), 758-766. doi:10.1097/01.psy.0000031578.42041.1

Salvadori, M., Sontrop, J. M., Garg, A. X., Truong, J., Suri, R. S., Mahmud, F. H., . . . Clark, W. F. (2008). Elevated blood pressure in relation to overweight and obesity among children in a rural Canadian community. *Pediatrics*, *122*(4), e821-e827. doi:10.1542/peds.2008-095

Satcher, D. (1995). Emerging infections: Getting ahead of the curve. *Emerging Infectious Diseases*, *1*(1), 1.

Scalco, A. Z., Scalco, M. Z., Azul, J. B. S., & Lotufo Neto, F. (2005). Hypertension and depression. *Clinics*, *60*(3), 241-250.

Scott, K. M., Bruffaerts, R., Tsang, A., Ormel, J., Alonso, J., Angermeyer, M. C., . . . Von Korff, M. (2007). Depression-anxiety relationships with chronic physical conditions: Results from the world mental health surveys. *Journal of Affective Disorders*, *103*(1-3), 113-20. doi:10.1016/j.jad.2007.01.015

Selye, H. (1950). Stress and the general adaptation syndrome. *British Medical Journal*, *1*(4667), 1383.

Sharp, M. S., Knapik, J. K., Walker, L. W., Burrell, L. B., Frykman, P. F., Darakjy, S. D., . . . Marin, R. M. (2008). Physical fitness and body composition after a 9-month deployment to Afghanistan. *Medicine & Science in Sports & Exercise.* doi:10.1249/MSS.0b013e318176b978

Shohat, M., Shohat, T., Mimouni, M., Nitzan, M., & Danon, Y. L. (1989). Hypertension in Israeli adolescents: Prevalence according to weight, sex and parental origin. *American Journal of Public Health*, *79*(5), 582-585.

Sledjeski, E. M., Speisman, B., & Dierker, L. C. (2008). Does number of lifetime traumas explain the relationship between PTSD and chronic medical conditions? Answers from the national comorbidity survey-replication (NCS-R). *Journal of Behavioral Medicine*, *31*(4), 341-9. doi:10.1007/s10865-008-9158-3

Smith, B., Chu, L. K., Smith, T. C., Amoroso, P. J., Boyko, E. J., Hooper, T. I., . . .

Millennium Cohort Study Team. (2008). Challenges of self-reported medical conditions and electronic medical records among members of a large military cohort. *BMC Medical Research Methodology*, *8*, 37. doi:10.1186/1471-2288-8-37

Smith, T. C., Ryan, M. A., Wingard, D. L., Slymen, D. J., Sallis, J. F., Kritz-Silverstein, D., & Millennium Cohort Study Team. (2008). New onset and persistent symptoms of post-traumatic stress disorder self reported after deployment and combat exposures: Prospective population based US military cohort study. *BMJ (Clinical Research Ed.)*, *336*(7640), 366-71. doi:10.1136/bmj.39430.638241.AE

Smith, T. C. (2011). Linking exposures and health outcomes to a large population-based longitudinal study: The millennium cohort study. *Military Medicine*, *176*(7S), 56-63.

Smoley, B. A., Smith, N. L., & Runkle, G. P. (2008). Hypertension in a population of active duty service members. *Journal of the American Board of Family Medicine: JABFM*, *21*(6), 504-11. doi:10.3122/jabfm.2008.06.070182

Somers, V. K., White, D. P., Amin, R., Abraham, W. T., Costa, F., Culebras, A., . . . Young, T. (2008). Sleep apnea and cardiovascular disease: An American Heart Association/American College of Cardiology foundation scientific statement from the American Heart Association council for high blood pressure research professional education committee, council on clinical cardiology, stroke council, and council on cardiovascular nursing in collaboration with the National Heart, Lung, and Blood Institute National Center on Sleep Disorders Research (National Institutes of Health). *Circulation*, *118*(10), 1080-1111.

doi:10.1161/circulationaha.107.189420

Sparrenberger, F., Cichelero, F. T., Ascoli, A. M., Fonseca, F. P., Weiss, G., Berwanger, O., . . . Fuchs, F. D. (2009). Does psychosocial stress cause hypertension? A systematic review of observational studies. *Journal of Human Hypertension, 23*(1), 12-9. doi:10.1038/jhh.2008.74

Spelman, J. F., Hunt, S. C., Seal, K. H., & Burgo-Black, A. L. (2012). Post deployment care for returning combat veterans. *Journal of General Internal Medicine*, *27*(9), 1200-1209.

Spera, C. S., Thomas, R. K., Barlas, F., Szoc, R., & Cambridge, M. H. (2011). Relationship of military deployment recency, frequency, duration, and combat exposure to alcohol use in the Air Force. *Journal of Studies on Alcohol and Drugs*, *72*, 5-14.

Stamler, R., Stamler, J., Riedlinger, W. F., Algera, G., & Roberts, R. H. (1978). Weight and blood pressure: Findings in hypertension screening of 1 million Americans. *JAMA: The Journal of the American Medical Association*, *240*(15), 1607-1610. doi:10.1001/jama.1978.03290150053024

Steptoe, A. (2008). Psychophysiological stress reactivity and hypertension. *Hypertension*, *52*(2), 220-1. doi:10.1161/HYPERTENSIONAHA.108.11547

Stevens, V. J., Obarzanek, E., Cook, N. R., Lee, I. -M., Appel, L. J., West, D. S., . . . Bragg, C. (2001). Long-term weight loss and changes in blood pressure: Results of the trials of hypertension prevention, phase II. *Annals of Internal Medicine*, *134*(1), 1-11.

Syme, S. L. (2004). Social determinants of health: The community as an empowered partner. *Preventing Chronic Disease*, *1*(1).

Talukder, M. A., Johnson, W. M., Varadharaj, S., Lian, J., Kearns, P. N., El-Mahdy, M. A., . . . Zweier, J. L. (2011). Chronic cigarette smoking causes hypertension, increased oxidative stress, impaired NO bioavailability, endothelial dysfunction, and cardiac remodeling in mice. *American Journal of Physiology. Heart and Circulatory Physiology*, *300*(1), H388-96. doi:10.1152/ajpheart.00868.2010

Thomsen, C. J., Stander, V. A., McWhorter, S. K., Rabenhorst, M. M., & Milner, J. S. (2011). Effects of combat deployment on risky and self-destructive behavior among active duty military personnel. *Journal of Psychiatric Research*, *45*(10), 1321-31. doi:10.1016/j.jpsychires.2011.04.003

Thuy, A. B., Blizzard, L., Schmidt, M. D., Luc, P. H., Granger, R. H., & Dwyer, T. (2010). The association between smoking and hypertension in a population-based sample of Vietnamese men. *Journal of Hypertension*, *28*(2), 245-50. doi:10.1097/HJH.0b013e32833310e0

Tofler, G. H., & Muller, J. E. (2006). Triggering of acute cardiovascular disease and potential preventive strategies. *Circulation*, *114*(17), 1863-72. doi:10.1161/CIRCULATIONAHA.105.596189

Torrance, B., McGuire, K. A., Lewanczuk, R., & McGavock, J. (2007). Overweight, physical activity and high blood pressure in children: A review of the literature. *Vascular Health and Risk Management*, *3*(1), 139-149.

Treiber, F. A., Kamarck, T., Schneiderman, N., Sheffield, D., Kapuku, G., & Taylor, T.

(2003). Cardiovascular reactivity and development of preclinical and clinical disease states. *Psychosomatic Medicine*, *65*(1), 46-62.

Unger, T., & Parati, G. (2005). Acute stress and long-lasting blood pressure elevation: A possible cause of established hypertension? *Journal of Hypertension, 23*(2), 261.

U S Army. (2011). Army regulation 40-501: Medical services standards of medical fitness. *Washington, DC: US Government Printing Office.* https://army.com/sites/army.com/files/Standards_of_Medical_Fitness_r40_501.pdf.

VanLeeuwen, J. A., Waltner-Toews, D., Abernathy, T., & Smit, B. (1999). Evolving models of human health toward an ecosystem context. *Ecosystem Health*, *5*(3), 204-219.

Vasan, R. S., Larson, M. G., Leip, E. P., Kannel, W. B., & Levy, D. (2001). Assessment of frequency of progression to hypertension in non-hypertensive participants in the Framingham heart study: A cohort study. *The Lancet*, *358*(9294), 1682-1686.

Viner, R. (1999). Putting stress in life: Hans Selye and the making of stress theory. *Social Studies of Science*, *29*(3), 391-410. doi:10.1177/030631299029003003

Virdis, A., Giannarelli, C., Fritsch Neves, M., Taddei, S., & Ghiadoni, L. (2010). Cigarette smoking and hypertension. *Current Pharmaceutical Design*, *16*(23), 2518-2525.

Walter, P., & Ron, D. (2011). The unfolded protein response: From stress pathway to homeostatic regulation. *Science (New York, N.Y.), 334*(6059), 1081-6. doi:10.1126/science.1209038

Wenzell, D. W., Pacheco de SouzaI, J. P., & Buongermino de Souza, S. B. (2009). Prevalence of arterial hypertension in young military personnel and associated factors. *Rev Saúde Pública*, *43*(5), 1-7.

Whelton, P. K., He, J., Appel, L. J., Cutler, J. A., Havas, S., Kotchen, T. A., . . . Winston, M. C. (2002). Primary prevention of hypertension: Clinical and public health advisory from the national high blood pressure education program. *JAMA: The Journal of the American Medical Association*, *288*(15), 1882-1888.

Whelton, P. K., Carey, R. M., Aronow, W. S., Casey, D. E., Collins, K. J., Himmelfarb, C. D., . . . Wright, J. T. (2018). 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/apha/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults. *Journal of the American College of Cardiology, 71*(19), e127-e248. doi:10.1016/j.jacc.2017.11.006

Williams, D. P., Going, S. B., Lohman, T. G., Harsha, D. W., Srinivasan, S. R., Webber, L. S., & Berenson, G. S. (1992). Body fatness and risk for elevated blood pressure, total cholesterol, and serum lipoprotein ratios in children and adolescents. *American Journal of Public Health*, *82*(3), 358-363.

Wilson, D. K., Kliewer, W., & Sica, D. A. (2004). The relationship between exposure to violence and blood pressure mechanisms. *Current Hypertension Reports*, *6*(4), 321-326.

Wyatt, S. B., Akylbekova, E. L., Wofford, M. R., Coady, S. A., Walker, E. R., Andrew, M. E., . . . Jones, D. W. (2008). Prevalence, awareness, treatment, and control of hypertension in the Jackson heart study. *Hypertension*, *51*(3), 650-6. doi:10.1161/HYPERTENSIONAHA.107.100081

Young, T., Peppard, P., Palta, M., Hla, K. M., Finn, L., Morgan, B., & Skatrud, J. (1997). Population-Based study of sleep-disordered breathing as a risk factor for hypertension. *Archives of Internal Medicine*, *157*(15), 1746-1752. doi:10.1001/archinte.1997.00440360178019