IMPACTS OF POST-TRAUMATIC STRESS DISORDER ON PHYSICAL HEALTH IN MILITARY FAMILIES

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Abstract: Existing studies have documented the adverse impacts of PTSD both in military service members as well as their families. The impacts of PTSD on physical health were only observed among the service members and veterans. There are major gaps in the field including in the understanding of mechanism that connects PTSD and physical health consequences, as well as evidence of spillover impacts of PTSD on physical health within the family of service members.The current study addresses this gap through investigation on the role of physiological functions including physiological responses (e.g., blood pressure), pre-disease biomarkers (e.g., inflammation), and evaluating risk for and the impacts of PTSD. The current study also proposed a mechanism to understand the connection between PTSD and pathology through those variables. Further, comparisons of the proposed model across different sociodemographic groups were also conducted. A secondary data analysis from CERNER Health Facts, specifically using path and multiple group path analyses, was utilized to attain the goal of the study. Findings yielded that physiological profiles, including pulse rate and leukocyte count, can provide insight into the risk of PTSD. Following PTSD diagnosis, alterations in pulse rate and N/L ratio were observed, confirming that PTSD altered different physiological functions. PTSD was also directly connected to pathology. Further, these physiological responses following PTSD were found to be connected and provided insights on the mechanisms of the connection between PTSD and pathology. However, comparisons of proposed model across different sociodemographic groups indicated that the proposed model needs to be used with caution as it did not capture the mechanisms equally across different groups. Nevertheless, it provides insights into the unexplored area. The findings from this study contributed to important movement for the integration between mental and physical health services especially in PTSD treatments. Monitoring of physiological markers of individuals at risk or diagnosed with PTSD can help minimize further damaged on physical health.

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CHAPTER I

INTRODUCTION

Post-traumatic Stress Disorder (PTSD) is a debilitating mental health problem. Individuals with PTSD often repeatedly re-live their traumatic memories, which is a core feature of PTSD. These repeated flashbacks of intrusive and unwanted memories have been reported to disrupt relationships, increase mortality and morbidity, and derange mental health (Allen et al., 2018; Nichter et al., 2019; Britvic et al., 2015; Schlenger et al., 2015).

The current study will focus on the relationship between PTSD and physical health. Studies report PTSD enhances the risk of diseases such as cardiovascular disease and sleep-related disorders (Beristianos et al, 2016; Violanti et al., 2006; Boscarino, 2008; Williams et al., 2015; El-Gabalawy et al., 2018; Krakow et al., 2001). The potential connection between PTSD and pathology raises concern as it could increase disability, lower quality of life, and impact distinct functions of those with PTSD. Unfortunately, the mechanism that connects the two are not fully understood. For this reason, there are numerous challenges facing prevention and intervention. Nevertheless, existing studies provide evidence of physiological alterations that could offer insights into the mechanisms underlying associations between PTSD and pathology. A set of studies documented alterations in blood pressure, pulse rate, and inflammation following PTSD

(Buckley et al., 2004; Lindqvist et al., 2014). On the contrary, other studies have found that certain physiological response characteristics (e.g.,blood pressure) predict the development of PTSD. Heart rate was also reported to be predictive of later development of PTSD (Morris et al., 2016; Bryant et al., 2011). This evidence raises the question of temporal precedence in the relationship between physiological markers and PTSD, and there is no study to date with extensive data to delineate the effect. The allostatic load model proposed a systemic adaptation mechanism that can be applied to understand the connection between PTSD and pathology. The mechanism ranges from adaptive to maladaptive that could gradually become detrimental to the system. Further, the current study will evaluate bi-directional relationships between PTSD and physiological markers.

PTSD is a growing concern in the military population. An estimated 5-15% of active service members received a clinical diagnosis of PTSD during or after completing their military assignment in the past decade (Ramchand et al., 2008). The estimated prevalence of PTSD in the military population is higher than in the general population (APA, 2013). Studies also report increased morbidity among service members with PTSD (Beristianos et al., 2016). These studies illustrate the prevalence of PTSD and associations with poorer physical health among this population.

Further evidence points to another detrimental impact of PTSD beyond service members. Previous studies indicate a higher risk of marital problems and mental health issues for the spouse and children of military service members (Dirkzwager et al., 2005; Lester et al., 2010). It is worth noting that physical health risks for this population havenot been examined yet. However, deriving from existing evidence, this population may also be at an increased risk for physical health problems. To evaluate physical health

impacts on military family members, Figley's work (1995) on compassion fatigue offered early evidence of a contemporary approach of systemic impact on psychological trauma. Evidence showed that secondary trauma can be acquired from interaction with traumatized individuals (Boscarino, et al., 2010). This perspective can advance our understanding of the detrimental effect of PTSD beyond the traumatized individuals.

The current study has three major aims. First, the current study will evaluate whether physiological markers predict PTSD. Secondly, the impacts of PTSD on physiological marker alteration following a PTSD diagnosis will be examined. Finally, the relationship between PTSD and pathology will be evaluated through physiological markers. To accomplish this, a secondary dataset, Cerner Health Facts® was obtained from the Center for Health System Innovation (CHSI) at Oklahoma State University. Cerner Health Facts® is a HIPAA compliant electronic health record (EHR) that ICD diagnosis codes for mental and physical health and physiological markers collectedthrough examination and laboratory tests, as well as several sociodemographic factors.

The current study will fill several gaps within the field. Existing studies have been limited to PTSD impacts on service members (Beristianos et al., 2016; O'Donovan et al., 2015; Yaffe et al., 2010). This study will extend the knowledge on the impact of PTSD on physical health to include family members of service men and women. Second, the current study will be used to test a mechanistic model to better understand underlying mechanisms of PTSD and physical health. Furthermore, findings from this study have the potential to extend and improve theoretical insight regarding complexities in the PTSD-

physical health association. It could also be utilized to inform and design prevention and intervention programs to serve service members and their families.

CHAPTER II

REVIEW OF THE LITERATURE

Problem Statement

Studies on the impacts of PTSD have been done quite extensively. Yet, there are two major existing gaps in the study of PTSD. First, the underlying mechanisms between PTSD and physical health are not well understood. However, there is some initial evidence of physiological marker alterations relevant to PTSD (Buckley et al., 2004; Muraoka et al., 1998; Vieweg et al., 2006) and inflammation (Lindqvist et al., 2017; Kagan et al., 1999). Another set of evidence also notes strong associations between PTSD and pathology development (CVD, Ahmadi et al., 2011; Sleep, El-Gabalawy et al., 2018). This initial evidence on some variables that are potentially involved in the process will be further reviewed. The integration and evaluation of a model that includes these variables may offer explanation to the connection of PTSD and physical health. Secondly, there is accumulated evidence on the functional and mental health impact, but not physical health of family members of those with PTSD (Allen et al., 2018; Dirkzwager et al., 2005; Herzog et al., 2011; Lester et al., 2010). Exploring the impact of transmission to family members is an important step to advance understanding of PTSD impact and initiate protection programs to minimize the impacts. The proposed study will fill these previously identified gaps.

Conceptualizing and Diagnosing Trauma

Existing literature includes different definitions and conceptualizations of psychological trauma. Two major guidelines exist for psychological trauma; the Diagnostic and Statistic Manual of Mental Disorder 5th Edition (DSM-5) published by American Psychiatric Association (APA) and the International Classification of Disease 10 Clinical Modification (ICD-10-CM) issued by the World Health Organization (WHO). Further details of each will be discussed.

According to the DSM-5, psychological trauma is defined as an exposure to actual or threatened death, injury, or actual or sexual violence (APA, 2013). These adverse experiences can be experienced directly or indirectly (APA, 2013). One could witness traumatic events indirectly through learning about a relative's or close friend's experience. Additionally, certain professions, such as first responders, may have elevated risk of being exposed to trauma. To meet DSM-5 criteria for PTSD diagnosis requires the symptoms of intrusion, avoidance, alteration in cognition and mood, and emotional arousal and reactivity (APA, 2013). These symptoms must be present for longer than one month.

Intrusion symptoms include involuntary and distressing memories and dreams, dissociative reactions as if the traumatic event were re-occurring, psychological distress toward reminders relevant to the traumatic event(s), and/or physiological reactions to cues related to the traumatic event(s) (APA, 2013). Whereas an avoidance symptom may include avoiding memory recall or reminders relevant to the traumatic event (APA, 2013). Further, negative alteration symptoms in cognition and mood can vary relative to an inability to remember the event, persistent and exaggerated negative beliefs about oneself,

distorted beliefs about the cause of the event, prolonged negative emotional state, low interest in significant activities, or detachment from others and absence of positive emotion (APA, 2013). Finally, symptoms indicative of an alteration in arousal and reactivity includes irritability and outburst, risk-taking behaviors, hypervigilance and exaggerated startle response, problems with concentration, and sleep disturbance (APA, 2013).

On the other hand, the International Codebook for Diseases (ICD-10) defined psychological trauma as any stressful, threatening, or catastrophic circumstance that can precipitate distress in most people (WHO, 2019). Some symptoms listed for PTSD include intrusive memories, alteration in emotion that affect self and relationship with others, and alteration in arousal. One may relive their trauma through intrusive memories and nightmares (WHO, 2019). Thus, it is common for individuals to avoid trauma-related triggers. Further, the alteration in emotion, such as numbness, could lower interest in things they previously enjoyed (anhedonia), decrease in responsiveness to the environment, and detachment to existing relationships. Finally, alteration in arousal could be manifested in sleep disturbances.

Theoretical and Conceptual Frameworks

Systemic Impact of PTSD

The work of Charles Figley (1997) offered a new lens to view PTSD. Figley highlighted the impact of World War II on veterans' family reintegration process (Figley, 1997). This historical event had significantly disturbed family life with the exit and return of the service members. Many experienced psychological trauma-related symptoms from a significant event that occurred during their service. It was found that the presence of

trauma symptoms then could affect family function and dynamics. Through this mechanism, the impact of individual psychological trauma could be transmitted to the family.

Figley (date) also found that the transmission of psychological trauma extended beyond family. Mental health professionals who provided services for impacted individuals in the World Trade Center disaster reported significant trauma symptoms (Boscarino et al., 2004; Boscarino, Adams & Figley, 2010). Figley and his colleagues' work indicated that psychological trauma is a dynamic concept that can affect others through interaction.

Despite the evidence of trauma transmission, most studies have evaluated PTSD impacts at an individual level, especially in the physical health domain (Ahmadi et al., 2011; O'Donovan et al., 2015). Thus, there is little understanding of the physical health impact on the significant others of those who experienced traumatic events. While isolating other aspects can offer clarity and potentially establish causality for the study, it is important to acknowledge that individuals do not live in a vacuum. Thus, evaluating the impacts of PTSD beyond those who experience it first-hand is an important step to advancing the field and to extending protection and intervention services for those who are impacted.

Allostatic Load Model

PTSD has been linked to altered physiological functioning as well as comorbidity with various health conditions. Therefore, an integrative model is needed to understand the mechanism that connects mental and physical health in the context of PTSD. McEwen and Stellar (1993) introduced a theoretical perspective called the allostatic load

model, which involves a balancing process to meet environmental demands through alterations to maintain the stability of the system. One key assumption of this model is that living organisms are able to regulate and maintain stability of their physiological system, otherwise known as homeostasis (McEwen & Wingfield, 2003).

In the context of the human body, homeostasis is best represented by various regulatory biological indicators including how acidic or basic a system is (pH measure) and body temperature (McEwen & Wingfield, 2003). When a threat presents to the system, the organism attempts to maintain homeostasis by making alterations to the body, referred to as allostasis.

These alterations can create a temporary balance which could be critical for organism survival. Yet, in the long-term, it could cause wear and tear to the body, and this is known as allostatic load (McEwen & Stellar, 1993). Further, if allostatic load persists or accumulates, it can develop into permanent damage to the system, referred to as allostatic overload (McEwen & Stellar, 1993). One study documented evidence of prolonged allostatic alteration load to health problems relevant to the cardiovascular and metabolic systems (Dowd et al., 2009). Allostatic load model is a key determinant to understanding illness and disease (McEwen & Wingfield, 2003).

Military Service and PTSD

The rate of Post-Traumatic Stress Disorder (PTSD) diagnoses among returned military service members is estimated between 5-15% (Ramchand et al., 2008). The number reported is higher than PTSD prevalence in the general U.S. population, which is 3.5% (APA, 2013). One potential explanation for the differences in prevalence is the nature of the job in that military service members have enhanced risk of exposure to

traumatic situations. During their service, military personnel may receive assignments to serve in a high conflict region where they may encounter various threatening attacks. Therefore, it is possible for military service members to be involved in witnessing or experiencing injuries or threats to safety, both to themselves and others. Of those military personnel who were deployed to Iraq and Afghanistan, 49% witnessed a friend be wounded or killed, and 45% witnessed someone other than a friend be injured or killed (Schell & Marshall, 2008).

The underlying connection between traumatic events exposure and the psychopathological development of PTSD is not well understood. However, recent work on the concept of moral injury may be able to explain this connection in the context of military PTSD. Moral injury refers to the concept of violations of moral value that one has (Nash & Litz, 2013). Military service members must follow the orders made by their superior despite their personal values. Hence, the service members are especially at higher risk to develop PTSD if the orders they received contradict their values. Additionally, various stories and interactions have also been documented to serve as a potential mechanism of moral injury to family members (Nash & Litz, 2013). This evidence offers potential insight to the enhanced risk of developing PTSD for military service members and their families.

Existing literature has established detrimental effects of PTSD on military service men and women. In regard to emotion reactivity, military personnel diagnosed with PTSD commonly have higher rates of anger reactivity when presented with trauma-related stimuli compared to those without PTSD diagnosis (Taft et al., 2007). Without proper intervention, these continuous negative emotions experienced by service members

may be an overwhelming experience that can later develop to be dysfunctional behavior including addiction, alcohol abuse (Frazier & Burnett, 1994), and aggressive behaviors (e.g., destroy property, threat) (Jakupcak et al., 2007).

Unfortunately, research has also documented evidence of a spillover effect of PTSD onto the significant others of service members. As service members with PTSD have enhanced risks of persistent negative emotions (Taft et al., 2007), it is reasonable that the dynamic within the family could be affected. Findings on marital research has confirmed associations between PTSD and higher levels of negative communication, delay in attachment and bonding with a spousal partner, and incidences of marital conflict related behavior (e.g., yelling, blaming) (Allen et al., 2010). The continuing pattern of negative interaction and communication can further erode marital relationships over time resulting in reduced marital satisfaction (Allen et al., 2018). Further, spouses were also found to be at a higher risk of experiencing PTSD related symptoms compared to those with spouses without the diagnosis (Dirkzwager et al., 2005). These findings provide insight that close and intimate relationships may mediate the transmission of trauma. Moreover, researchers report that higher risk of both physical assault (e.g., hitting) and psychological aggression (e.g., being spiteful) toward one's romantic or marital partner are associated with PTSD status (Taft et al., 2007).

The impacts of PTSD can also be further transmitted to offspring. When service members experience PTSD symptoms, their available resources, such as energy and time for positive interaction with their children, can become limited (Cozza et al., 2005). In addition, negative interactions between military personnel and their spouses have been linked to diminished cooperation relevant to childcare (Allen et al., 2010). Thus, children

of individuals with PTSD are exposed to distinct challenges that can limit positive interactions between themselves and their parents. Studies have also documented some negative impacts relevant to parenting with PTSD including higher levels of anxiety (Lester et al., 2010). Based on this evidence, spouse and offspring of military service members are at substantial risk for negative impacts associated with PTSD.

PTSD has also been linked to physical health of service members. Existing studies reported enhanced risk for mortality among Vietnam War veterans (Boscarino, 2006). The study highlighted the need for further exploration of the underlying behavioral, psychological, and physical health reasons behind this enhanced mortality risk (Boscarino, 2006). Further, a study on specific causes of mortality relevant to heart disease demonstrated consistent findings. Individuals with PTSD were found to be at higher risk for death caused by cardiovascular disease (Boscarino, 2008). Another study had similar findings that PTSD increased future risk for cardiovascular disease (Beristianos et al., 2016). These health conditions have, in turn, affected various aspects of life, sometimes long after the traumatic event(s) occured. A study reported that PTSD was associated with lower life satisfaction thirty years after service members returned from war (Koenen et al., 2008). PTSD has also impacted the utilization of healthcare. Chan and colleagues discovered that veterans with PTSD or other mental health issues had higher healthcare utilization (Chan et al., 2009). Considering the evidence of spillover impact of PTSD beyond service members to family members, it is likely there is spillover occurring in physical health, too. However, a major gap exists in the field as there is no existing study exploring the impact of military service members' PTSD on the family members' physical health. The current study aims to fill this gap in the field.

Proposed Conceptual Model

Based on the review of existing theory and studies, below is the proposed model of PTSD and physical health (see figure 1). Existing evidence shows a connection between psychological trauma and various physical health consequences (Ahmadi et al., 2011; O'Donovan et al., 2014; Greenberg et al., 2014). Researchers have explored potential underlying mechanisms; however, this connection is not yet fully understood. The proposed model integrates the allostatic load model and the systemic impact model of PTSD to understand the connection between PTSD and physical health in the context of military families. This model has the potential to advance the understanding of the relationship between PTSD and disease pathology, which in turn could aid in developing prevention and intervention programming to minimize the impact of PTSD.

For the current model, the integration of the allostatic load model is intended to help decipher the mechanism of psychological trauma and physical health. Allostasis and allostatic load are two concepts that can be useful to understanding the mechanism. Allostasis represents the adaptive changes that might occur following a traumatic stress exposure, which may result in alterations of basic physical functions including heart rate and blood pressure. Allostatic load refers to the accumulation of changes that occur as a result of a significant event. In this model, allostatic load also represents an early indication of pathology which includes inflammation. Overtime, allostatic load can accumulate and develop to be allostatic overload. The clinical diagnosis of disease represents the allostatic overload concept in the proposed model.

Evidence of the mental health impacts of PTSD on spouse and offspring have been well documented. However, there is an existing gap of the knowledge on the

physical health consequences associated with the PTSD diagnosis. The proposed model aims to explain the interdependent nature of this psychological disorder and physical health through various social interactions within the family. The knowledge gained can provide a foundation for a psychological and physical health integrated program to minimize the impacts of PTSD.

Allostasis: Physiological Responses to Stress

Exposure to trauma can activate the Stress Response System (SRS) in the human body. The SRS serves as a mechanism for adaptation to internal or external threats (Friedman & McEwen, 2003). The goal of the SRS is to enhance the odds of survival, especially in an extreme situation. SRS pathways include: Autonomic Nervous System (ANS) & Hypothalamus-Pituitary-Adrenal (HPA) (Gianaros & Wager, 2015).

The activation of SRS begins with exposure to a potential threat, then an appraisal of the situation (Gianaros & Wager, 2015). During this phase, the individual evaluates the situation and forms a perception relevant to the situation. Therefore, any stress appraisal outcomes may vary from one individual to another, even when two individuals experience the same event. The amygdala is an area of the brain involved in the appraisal process (Hermann et al., 2014). Following the appraisal process, if an individual perceives that there is a threat, then the brain signals either or both ANS and HPA to activate.

The automatic nervous system (ANS) reacts to the presence of a threat by increasing the sympathetic and decreasing parasympathetic activity (Gianaros & Wager, 2015). Sympathetic activity, also known as fight or flight response, is commonly present during stressful situations, accompanied by alterations in heart rate and blood pressure.

On the contrary, parasympathetic activity occurs during rest periods, which includes an increase in energy conservation such as in the digestive system. If a threat is perceived to be present, the amygdala sends a signal to the hypothalamus, which will then activate the sympathetic nervous system (Brottman, Golden & Wittstein, 2007). In the context of PTSD, alteration in arousal and sensitivity to stimuli related to a traumatic event are the core features (APA, 2013). Accumulated evidence supports that veteran with PTSD have higher blood pressure and faster heart rates (Buckley et al., 2004; Muraoka et al., 2005; Bryant et al., 2008).

The other component of SRS is the Hypothalamic-pituitary-adrenal (HPA) axis also begins with the appraisal process. During this process, the amygdala signals the hypothalamus to send message to the pituitary to produce corticotropin releasing factor/hormone (CRF/H) (Friedman & McEwen, 2004; Brottman et al., 2007). CFR then travels to the pituitary gland to bind with the CRF receptor, which then will produce adrenocorticotropic hormone (ACTH) (Brottman et al., 2007). Next, ACTH binds with the designated receptor in the adrenal glands to produce cortisol (Brottman et al., 2007). Cortisol is the hormone that is produced during stressful conditions and travels in the blood to alter various systems in the body, including the immune system (Brottman et al., 2007). Cortisol plays a role as a warning system which notifies various systems to fight or flee in response to a threat. When it reaches an adequate level as required to handle the stress, it sends out signal feedback to stop the ACTH production (Liu et al., 2017).

The exploration of HPA activity following a traumatic event has yielded mixed findings. One study found enhanced levels of cortisol in victims three months following an earthquake compared to baseline levels prior to the event (Kotozaki & Kawasima,

2012). On the contrary, another study following individuals with PTSD showed lower levels of cortisol compared to people without PTSD (Wessa et al., 2005). Additionally, there is evidence of the connection between cortisol and inflammation levels. A study discovered that higher levels of inflammatory agents, including tumor necrosis factor and interleukin 6 (IL-6), were found in individuals with PTSD (Gill et al., 2008). Notably, these individuals showed low levels of cortisol (Gill et al., 2008). Thus, the level of cortisol may play a role in the immune system reaction in the context of PTSD. Further investigation is needed to understand the differential effect following traumatic events.

The reaction to stress described above is designed for the short-term. In a situation where the stress response system is activated for an extended period, it may result in adverse health consequences for the individual (Gianaros & Wager, 2015). The nature of PTSD symptoms, including arousal, hypervigilance, and flashbacks of traumatic memories, may continuously alter the individual stress response system, long-term (Friedman & McEwen, 2003). The extended alteration to SRS relevant to psychological trauma has been associated with elevated risks for various health problems including cardiovascular disease (Ahmadi et al., 2011; Beristianos et al., 2016). Despite the established connection between the two, the relationship between pathology development and PTSD is not well understood. Hence, incorporating the understanding of SRS may contribute to the understanding of the mechanism linking the two.

Allostatic Load: Pre-Disease Biomarkers

Prolonged activation of the stress response system (SRS) can accumulate and cause damage to the cells, tissues, and organs, as it is designed for temporary adaptation. It can further lead to an inflammatory state, which can be a precursor for the later

development of diseases, if it persists. PTSD is a mental health disorder characterized by repeated and magnified fear responses relevant to trauma-related stimuli in the absence of any actual threat (Michopoulos et al., 2017). Due to the nature of the disorder, individuals with PTSD experience consistent activation of the SRS and are more vulnerable to inflammation. Pro-inflammatory agents, including C-reactive protein (CRP) and IL-6, have been well-studied in the context of PTSD investigations. However, these are not the only indicators of inflammation. An alternative to the sophisticated and potentially costly inflammatory agent tests is a blood test.

A blood test is a common procedure and is considerably more affordable compared to most medical testing. It can provide various information including blood cell count. Leukocyte count is one type of inflammation indicator that has been studied. Exploration of other types of cells in conjunction with inflammation is needed. In the current study, we will specifically study leukocytes, lymphocyte, neutrophils, and platelets. Existing studies found increased levels of pro-inflammatory agents including white blood count (leukocyte), specific components of leukocytes including lymphocyte and neutrophils, and platelets among individual with PTSD (Boscarino & Chang, 1999; Lindqvist et al., 2017; O'Donovan et al., 2017). These specific components of inflammation will be further reviewed below.

Leukocytes. Leukocytes or white blood cells are thought to be an indicator of inflammation in the body that is inexpensive and easily accessible to assess (Anderson et al., 2007). Exposure to a psychological trauma can influence the production of cortisol, which has an immunosuppressant quality that then affects the level of leukocyte produced (Boscarino & Chang, 1999). A study reported that Vietnam Veterans with PTSD had a

significantly higher level of leukocytes compared to those without PTSD, even twenty years after trauma exposure (Boscarino & Chang, 1999). Similar findings of significantly higher leukocyte levels were also detected among veterans from Operation Iraqi Freedom and Operation Enduring Freedom (Lindqvist et al., 2017).

Further, leukocytes have also been found to play role in the pathology of diseases. Leukocytes are involved in the inflammation process. The inflammation process could be reparative, meaning it tells the body about damage or injury in certain areas and kickstarts the healing process. However, if the inflammation stage lasts for an extended period of time, it could cause permanent damage to the cells, tissues, and organs. Findings consistently support the connection between leukocytes and various disease including cardiovascular disease. A study reported that leukocyte levels serve as a significant predictor for a cardiovascular event and mortality due to CVD (Horne et al., 2005). The association also extends to peripheral arterial disease (Giugliano et al., 2010)

Despite some evidence that connects PTSD diagnoses, leukocyte levels, and cardiovascular disease, there has been lack of research focused on evaluating the relationship between the three variables together. Leukocyte levels may potentially serve as a mechanism that explains the relationship between PTSD and CVD. It is also important to highlight that if leukocyte levels serve as a mediator between PTSD and CVD, it can also be used as an inexpensive monitoring procedure for at-risk individuals.

Neutrophils and Lymphocyte Ratio (N/L ratio). Leukocytes consist of a variety of different types of cells. Each type of cells serves a distinct role in the inflammation process. Neutrophil, for example, is a type of leukocyte that serves as a first responder in the event of inflammation. It is the first cell that migrates to the inflamed site and attacks

the external pathogens through phagocytosis (Rosales et al., 2017). Lymphocytes are another cell-type that migrates to the injured site to do repair work. This specific type of cell focuses on preventing the pathogen from multiplying (Koyasu & Moro, 2012). Both neutrophils and lymphocytes provide information about the inflammatory site. However, when combined as neutrophils and lymphocyte ratio (N/L ratio), further information on health events, including mortality and morbidity, is given (Fest et al., 2019; Tokgoz et al., 2018; Angkananard et al., 2018). The use of neutrophils and lymphocyte ratio is more common in the medical field, especially in predicting specific outcomes relevant to morbidity such as cardiovascular disease. A study by Hartaigh and colleagues (2012) reported that both neutrophils and neutrophils/lymphocyte ratio were found to be significant predictors for cardiovascular mortality. The same study suggested that measures that involved neutrophils served as a better predictor than a contemporary measure of inflammation such as C-reactive protein (CRP) (Hartaigh et al., 2012).

Despite some exploration of the relationship between PTSD and inflammation, there are limited studies utilizing the neutrophils and lymphocyte ratio as an indicator of inflammation. One study of veterans with PTSD reported significantly higher level of neutrophils compared to those without PTSD (Lindqvist et al., 2017). However, the same study reported a non-significant finding for the level of lymphocytes between groups (Lindqvist et al., 2017). Another study on maltreated children documented different levels of lymphocytes in maltreated children with PTSD compared to those without PTSD (Bielas et al., 2012). Further exploration of the association between the neutrophils and lymphocyte ratio and PTSD is needed.

Platelet. The platelet is another component included in the complete blood count (CBC) procedure. Platelets serve the function of terminating blood loss during internal and external injury through the sealing of the damaged vessels (Willoughby et al., 2002). On the other hand, platelets can also serve as a risk. In abnormal conditions, blood vessels may have lesions as a result of high blood pressure. The lesion activates the inflammation mechanism and recruit platelets to heal the damage. Therefore, information on platelets may serve as an early indicator of underlying conditions and risk relevant to cardiovascular disease. Studies have utilized platelets as a predictor of health outcomes. One study reported that platelets are a significant predictor for cardiac mortality (Goliasch et al., 2013). Another study found a positive association between platelets and cardiovascular risk (Sloan et al., 2015).

Individuals with PTSD are found to have a higher platelet count compared to those without (Lindqvist et al., 2017). As platelets play a role in the blood clotting process during a blood clotting injury, a high count can result in spontaneous blood clotting, which can impede blood and oxygen flow to important organs. Another study reported the alteration in the reactivity of platelets following a traumatic event in a sample of male Croatian veterans (Vidovic et al., 2011). This study found higher platelet reactivity, meaning the platelets become more adhesive to different leukocyte types enhancing the risk of developing atherosclerotic plaque, in those with PTSD compared to those without PTSD (Vidovic et al., 2011). Higher platelet reactivity and the elevated level of platelets can be disastrous as it can spontaneously lead to the development of blood clotting and cause various problems including cardiovascular disease. Hence, individuals with PTSD may potentially be at risk to develop cardiovascular disease.

Allostatic Overload: Clinical Diagnosis of Disease

Prolonged SRS activation due to psychological trauma has been associated with some negative health consequences, including cardiovascular disease (CVD) and sleep disorder (Ahmadi et al., 2010; El-Gabalawy et al., 2018). Further details of each disease diagnosis are provided in the following section.

Cardiovascular Disease (CVD). The World Health Organization (WHO) defined CVD as various problems in the heart or blood vessels (WHO, 2017). Some examples of CVD include coronary artery disease (CAD) and hypercholesterolemia. CVD contributes to approximately 655,000 mortalities annually and is the leading cause of death in the United States (CDC, 2020). Among those who suffer from CVD, many are military service members and veterans. A Veteran Affairs' report named cardiovascular disease as the top leading cause of hospitalizations in the veteran affairs healthcare systems (Veteran affairs, 2016). It raises question as to why the numbers are high among this specific population.

CAD is one of the most common types of CVD to be found (CDC, 2017). It refers to blockage in the artery, known as atherosclerosis, that causes the heart, and potentially the rest of the body, to not receive proper oxygen and nutrient supply (CDC, 2017). When CAD becomes so severe and reaches complete blockage, consequences may include Myocardial Infarction (MI) or a stroke. MI, also known as a heart attack, is a condition where the heart muscle starts to die as a result of blockage in arteries that supply oxygen to the heart (American Heart Association, 2017). A stroke is another condition that can emerge from the development of artery blockage (American Heart Association, 2017). The blockage of blood vessels can result in either the death of brain

cells because the brain does not get enough oxygen supply, which is known as an ischemic stroke, or the vessel can burst as result of high blood pressure, which is known as a hemorrhage stroke (American Heart Association, 2017). Atherosclerosis is also found to consist of various immune system components which may indicate the role of SRS in the pathology of atherosclerosis and CVD (Hansson et al., 2006).

Cardiovascular disease has been linked to various contributing factors from genetic to environmental. Psychological trauma, as a form of extreme stress, is one factor that has been studied. PTSD, a diagnosis relevant to psychological trauma, is associated with an enhanced risk for cardiovascular disease (Ahmadi et al. 2011; Beristianos et al., 2016). Nevertheless, the mechanism underlying disease pathology is not fully understood although previous findings offer some insights. Individuals with PTSD were reported to have higher systolic and diastolic BP and pulse rate (Paulus et al., 2013). In a separate study, systolic BP was linked to enhanced risk of hemorrhagic stroke whereas pulse was a predictor for peripheral arterial disease (Rapsomaniki et al., 2014). Additionally, individuals with PTSD also showed higher inflammation, specifically higher white and red blood cells, as well as a higher platelet count (Lindqvist et al., 2017). Taken together, these alterations following a PTSD diagnosis may suggest key variables that are involved in the pathology of cardiovascular disease linked to psychological trauma.

Sleep disorders. The American Psychiatric Association (APA) defines sleep disorders as any significant disturbances in sleep pattern and quality that impact individual ability to perform normal functions (APA, 2013). Some examples of sleep disorders include insomnia and nightmare disorder (APA, 2013). Inevitably, the quality of sleep and sleep disorder are relevant topics to PTSD, as some of the symptoms overlap.

PTSD symptoms can include intrusion symptoms and alteration of arousal (APA, 2013). Intrusion symptoms can manifest as nightmares, which can disrupt one's sleep pattern and quality. Further, the alteration of arousal symptoms may also be exhibited as sleep disturbances such as insomnia. These symptoms of PTSD can impact the quality of sleep, which, if extended for a period of time, can meet the criteria for sleep disorders.

The connection of PTSD and sleep disorders have been documented in military and veteran populations. Existing literature documented that military service members experienced significant sleep problems (Plumb et al., 2014; Straus et al., 2015), and some received diagnoses of sleep disorders specifically called insomnia (Mysliwiec et al., 2012). One study discovered that significant disturbances of sleep are more profound during and after a deployment period (Seelig et al., 2010). Another study revealed that the severity of PTSD symptoms and sleep problems are related (Plumb et al., 2014). Despite the established association between PTSD and sleep problems, there has been a lack of attempts to systematically understand the relationship between the two. An article points at this gap and further raises the question of whether sleep disorder is a feature of PTSD or an impact of PTSD due to physical changes following psychological trauma (Spoormaker & Montgomery, 2008). Unfortunately, the lack of understanding of the connection between the two hinders the development of an effective treatment. The proposed model in the current study offers the potential to understand the contribution of alteration in physiological responses and inflammation in the development of sleep disorders.

Sociodemographic Characteristics. The influence of sociodemographic characteristics in various outcomes have been well-studied, especially in the field of

social science. These are considered to be key factors, as sociodemographic characteristics provide context for the human experience. Specifically, in the study of psychological trauma, sociodemographic characteristics have a distinct role. In some cases, they contribute to creating an enhanced risk to exposure of trauma. On the contrary, they can also buffer the negative impacts of trauma. The current study will focus on sex, age, and race as key characteristics. In the next section, each sociodemographic characteristic is further discussed.

Sex. There is ample evidence surrounding sex differences in both mental and physical health. In the context of psychological trauma, for example, researchers have reported stronger association between combat exposure and PTSD in military service female than what is found in their male counterparts (Luxton et al., 2010). Another study discovered distinct symptom differences of PTSD between males and females. Males appeared to show more numbing symptoms compared to females (Hourani et al., 2015). The processes that differentiate these trauma symptoms between the two sexes are still unclear. On the physical health side, the evidence of sex differences is also salient. In a study of cardiovascular risk, older women had multiplied risk of mortality due to cardiovascular compared to women (Regnault et al., 2012).

Existing evidence also points to women as having higher vulnerability for both PTSD and health problems, specifically CVD. There is limited research on sex differences in the area of sleep hygiene and sleep disorder. Further investigation for understanding better the role of sex differences in the area of psychological trauma is needed, as well.

Age. Age is another important determinant in one's health functioning. Generally, individuals experience decreases in physical health functioning overtime (Ferraro & Farmer, 1996). As an individual ages, the function as well as structure of various organs also change. For example, in one study the liver showed declined function as individual aged (Zeeh & Platt, 2002). The changes on cells, organs, tissue structures and their functions can alter the relationship between PTSD and physical health. Thus, age can serve as a moderator in the PTSD-physical health association.

Race. Further, race also contributes to our understanding of health. Disparities in health status between white individuals and other minority groups (e.g., African Americans) has been long standing and well-documented (Williams et al., 2016). A study showed that African American individuals experienced more chronic and serious illness across their lifespan in comparison to White Caucasians (Ferraro & Farmer, 1996). The differences in health status stems from the issue of racial discrimination that led to deprived access to health resources. The current study investigates whether the finding of health disparities between races extends to the context of the PTSD and physical health relationship.

Body Mass Index (BMI). BMI is a ratio of weight and height measured in kilogram per square meter (Center for Disease Control, 2020). It is a health indicator commonly used as a proxy to measure of body fat (CDC, 2020). According to the CDC (2020), BMI can be categorized into one of four groups; under 18 is considered underweight, between 18.5 and 24.9 is considered normal, 25.0 to 29.9 is considered overweight, and above 30 is considered obese. BMI has been linked to the general risk of morbidity and mortality (Zhai et al., 2020; Lung et al., 2019; Konnopka et al., 2011;

Zheng et al., 2013). One epidemiology study discovered the BMI trajectory after the age of 51 is a more significant predictor compared to the BMI status at a single point of time (Zheng et al., 2013). Another study also confirmed that individuals with healthy BMI have the lowest mortality risk compared to overweight and obese individuals (de Gonzales et al., 2010). Additionally, Konnopka and colleagues (2011) found that obesity was associated with high healthcare cost, including from cardiovascular disease.

Further, existing literature has established an association between BMI and PTSD. In a study of military veterans, BMI was explored as a potential negative effect of PTSD (Smith et al., 2015). The findings showed that the current positive PTSD status was linked to an increased risk of being overweight or obese, especially for the older adult population (Smith et al., 2015). Another study also found that BMI was increased to meet the overweight or obese category in the presence of PTSD symptoms (Kubzansky et al., 2014). Following a psychological trauma, poor health behaviors including a lack of exercise, unhealthy food consumption, and substance use may emerge as a form of coping response (Hoerster et al., 2019). These types of behaviors could affect one's health status including BMI. Interestingly, there is a lack of research exploring the risk of PTSD for those in certain BMI groups.

Further, BMI has also been linked to specific health problems including cardiovascular diseases. Khan and colleagues (2018) found that obese and overweight individuals have a higher risk of developing CVD, experiencing a cardiovascular event, and mortality compared to those with normal BMI. Further, the risk increases with age (Khan et al., 2018). In this case, BMI serves as a proxy measure of health status provide a slice of information of health status. BMI that falls into overweight and obese categories

could indicate poor nutrition intake (e.g., junk food), or a sedentary lifestyle that has been linked to impact aspects of cardiovascular health such as blood pressure or the development of plaque in the blood vessel.

Initial evidence showed association between BMI and sleep disorder and quality. One study discovered that those who are overweight or obese have less hours of sleep than those with normal BMI (Vorona et al., 2005). Interestingly, this relationship is reversed in the extreme obese category, where the number of sleeping hours is dramatically increased (Vorona et al., 2005). Further, a study reported that individuals with BMI larger than 32 were at higher risk for a sleep disorder, specifically obstructive sleep apnea (Morris et al., 2008).

Proposed Analytical Model and Hypotheses

Based on the conceptual model described in the previous section, an analytical model with key variables that represent the concepts is proposed (see Figure 2). This analytical model will be used to answer the broader question: How does PTSD influence physiological response and disease among active military servicemembers? To answer this question, the following hypotheses will be addressed.

Hypothesis 1: Higher physiological stress response is associated with higher pre-disease biomarkers prior to PTSD diagnosis.

1A.1 Higher systolic BP is associated with a higher leukocyte count prior to PTSD diagnosis.

1A.2 Higher systolic BP is associated with a higher platelet count prior to PTSD diagnosis.

1A.3 Higher systolic BP is associated with a higher N/L ratio prior to PTSD diagnosis.

1B.1 Higher diastolic BP is associated with a higher leukocyte count prior to PTSD diagnosis.

1B.2 Higher systolic BP is associated with a higher platelet count prior to PTSD diagnosis.

1B.2 Higher diastolic BP is associated with a higher N/L ratio prior to PTSD diagnosis.

1C.1 Higher pulse rate is associated with a higher leukocyte count prior to PTSD diagnosis.

1C.2 Higher pulse rate is associated with a higher platelet count prior to PTSD diagnosis.

1C.3 Higher pulse rate is associated with a higher N/L ratio prior to PTSD diagnosis.

Hypothesis 2: Higher pre-disease biomarkers is associated with a higher risk of being diagnosed with PTSD.

2A. Higher leukocyte count is associated with a higher risk of being diagnosed with PTSD.

2B. Higher platelet count is associated with a higher risk of being diagnosed with PTSD.

2C. Higher N/L ratio is associated with a higher risk of being diagnosed with PTSD.

Hypothesis 3: Diagnosis of PTSD is associated with higher physiological responses.

3A. Diagnosis of PTSD is associated with higher systolic blood pressure.

3B. Diagnosis of PTSD is associated with higher diastolic blood pressure.

3C. Diagnosis of PTSD is be associated with higher pulse rate.

Hypothesis 4: Diagnosis of PTSD is associated with higher pre-disease biomarkers.

4A. Diagnosis of PTSD is associated with higher leukocyte counts.

4B. Diagnosis of PTSD is associated with higher platelet counts.

4C. Diagnosis of PTSD is be associated with higher N/L ratio.

Hypothesis 5: PTSD diagnosis is associated with a higher risk of pathology.

5A. PTSD diagnosis is associated with a higher risk of cardiovascular disease.

5B. PTSD diagnosis is associated with a higher risk of sleep disorder.

Hypothesis 6: Higher physiological responses is associated with higher pre-disease biomarkers.

6A. Higher blood pressure systolic is associated with higher pre-disease biomarkers.

6A.1 Higher blood pressure systolic is associated with higher leukocyte count.

6A.2 Higher blood pressure systolic is associated with higher platelet count.

6A.3 Higher blood pressure systolic is associated with higher N/L ratio

6B. Higher blood pressure diastolic is associated with higher pre-disease biomarkers.

6B.1 Higher blood pressure diastolic is associated with higher leukocyte count.

6B.2 Higher blood pressure diastolic is associated with higher platelet count.

6B.3 Higher blood pressure diastolic is associated with higher N/L ratio

6C. Higher pulse rate is associated with higher pre-disease biomarkers.

6B.1 Higher pulse rate is associated with higher leukocyte count.

6B.2 Higher pulse rate is associated with higher platelet count.

6B.3 Higher pulse rate is associated with higher N/L ratio

Hypothesis 7: Higher physiological responses is associated with higher risk for pathology.

7A. Higher systolic BP is associated with higher risk for pathology.

7A.1 Higher systolic BP is associated with higher risk for CVD.

7A.2 Higher systolic BP is associated with higher risk for sleep disorder.

7B. Higher diastolic BP is associated with higher risk for pathology.

7B.1 Higher diastolic BP is associated with higher risk for CVD.

7B.2 Higher diastolic BP is associated with higher risk for sleep disorder.

7C. Higher pulse rate is associated with higher risk for pathology.

7C.1 Higher pulse rate is associated with higher risk for CVD.

7C.2 Higher pulse rate is associated with higher risk for sleep disorder.

Hypothesis 8: Higher level of pre-disease biomarkers is associated with higher risk for pathology.

8A. Higher leukocytes count is associated with higher risk for pathology.

8A.1 Higher leukocytes count is associated higher risk for CVD.

8A.2 Higher leukocytes count is associated higher risk for sleep disorder.

8B. Higher platelet count is associated with higher risk for pathology.

8B.1 Higher platelet count is associated with higher risk for CVD.

8B.2 Higher platelet count is associated with higher risk for sleep disorder.

8C. Higher N/L ratio is associated with higher risk for pathology.

8C.1 Higher N/L ratio is associated with higher risk for CVD.

8C.2 Higher N/L ratio is associated with higher risk for sleep disorder.

Hypothesis 9: The diagnosis of cardiovascular disease is associated with the diagnosis of sleep disorder.

Hypothesis 10: Physiological responses mediate the association between PTSD and pre-disease biomarkers following PTSD diagnosis.

10A. Systolic BP mediates the relationship between PTSD and pre-disease biomarkers.

10A.1 Systolic BP mediates the relationship between PTSD and leukocyte count.

10A.2 Systolic BP mediates the relationship between PTSD and platelet count.

10A.3 Systolic BP mediates the relationship between PTSD and N/L ratio.

10B. Diastolic BP mediates the relationship between PTSD and pre-disease biomarkers.

10B.1 Diastolic BP mediates the relationship between PTSD and leukocyte count.

10B.2 Diastolic BP mediates the relationship between PTSD and platelet count.

10B.3 Diastolic BP mediates the relationship between PTSD and N/L ratio.

10C. Pulse rate mediates the relationship between PTSD and pre-disease biomarkers.

10C.1 Pulse rate mediates the relationship between PTSD and leukocyte count.

10C.2 Pulse rate mediates the relationship between PTSD and platelet count.

10C.3 Pulse rate mediates the relationship between PTSD and N/L ratio.

Hypothesis 11: Pre-disease biomarkers mediate the relationship between physiological responses and pathology.

11A. Leukocyte count mediates the relationship between physiological responses and pathology.

11A.1 Leukocyte count mediates the relationship between systolic BP and CVD.

11A.2 Leukocyte count mediates the relationship between systolic BP and sleep disorder.

11A.3 Leukocyte count mediates the relationship between diastolic BP and CVD.

11A.4 Leukocyte count mediates the relationship between diastolic BP and sleep disorder.

11A.5 Leukocyte count mediates the relationship between pulse rate and CVD.

11A.6 Leukocyte count mediates the relationship between pulse and sleep disorder.

11B. Platelet count mediate the relationship between physiological responses and pathology.

11B.1 Platelet count mediates the relationship between systolic BP and CVD.

11B.2 Platelet count mediates the relationship between systolic BP and sleep disorder.

11B.3 Platelet count mediates the relationship between diastolic BP and CVD.

11B.4 Platelet count mediates the relationship between diastolic BP and sleep disorder.

11B.5 Platelet count mediates the relationship between pulse rate and CVD.

11B.6 Platelet count mediates the relationship between pulse rate and sleep disorder.

11C. N/L ratio mediates the relationship between physiological responses and pathology.

11C.1 N/L ratio mediates the relationship between systolic BP and CVD.

11C.2 N/L ratio mediates the relationship between systolic BP and sleep disorder.

11C.3 N/L ratio mediates the relationship between diastolic BP and CVD.

11C.4 N/L ratio mediates the relationship between diastolic BP and sleep disorder.

11C.5 N/L ratio mediates the relationship between pulse rate and CVD.

11C.6 N/L ratio mediates the relationship between pulse rate and sleep disorder.

Hypothesis 12: Physiological responses and pre-disease markers mediate the relationship between PTSD and pathology.

12A. Systolic BP and leukocyte count mediate the relationship between PTSD and pathology.

12A.1 Systolic BP and leukocyte count mediate the relationship between PTSD and CVD.

12A.2 Systolic BP and leukocyte mediate the relationship between PTSD and sleep disorder.

12B. Systolic BP and platelet count mediate the relationship between PTSD and pathology.

12B.1 Systolic BP and platelet count mediate the relationship between PTSD and CVD.

12B.2 Systolic BP and platelet count mediate the relationship between PTSD and sleep disorder.

12C. Systolic BP and N/L ratio mediate the relationship between PTSD and pathology.

12C.1 Systolic BP and N/L ratio mediate the relationship between PTSD and CVD.

12C.2 Systolic BP and N/L ratio mediate the relationship between PTSD and sleep disorder.

12D. Diastolic BP and leukocyte count mediate the relationship between PTSD and pathology.

12D.1 Diastolic BP and leukocyte count mediate the relationship between PTSD and CVD.

12D.2 Diastolic BP and leukocyte count mediate the relationship between PTSD and sleep disorder.

12E. Diastolic BP and platelet count mediate the relationship between PTSD and pathology.

12E.1 Diastolic BP and platelet count mediate the relationship between PTSD and CVD.

12E.2 Diastolic BP and platelet count mediate the relationship between PTSD and sleep disorder.

12F. Diastolic BP and N/L ratio mediate the relationship between PTSD and pathology.

12F.1 Diastolic BP and N/L ratio mediate the relationship between PTSD and CVD.

12F.2 Diastolic BP and N/L ratio mediate the relationship between PTSD and sleep disorder.

12G. Pulse rate and leukocyte count mediate the relationship between PTSD and pathology.

12G.1 Pulse rate and leukocyte count mediate the relationship between PTSD and CVD.

12G.2 Pulse rate and leukocyte count mediate the relationship between PTSD and sleep disorder.

12H. Pulse rate and platelet count mediate the relationship between PTSD and pathology.

12H.1 Pulse rate and platelet count mediate the relationship between PTSD and CVD.

12H.2 Pulse rate and platelet count mediate the relationship between PTSD and sleep disorder.

12I. Pulse rate and N/L ratio mediate the relationship between PTSD and pathology.

12I.1 Pulse rate and N/L ratio mediate the relationship between PTSD and CVD.

12I.2 Pulse rate and N/L ratio mediate the relationship between PTSD and sleep disorder.

Hypothesis 13: Sex, race age, and BMI moderate the proposed model

13A. Sex moderate relationships in the proposed model.

13B. Race moderate the relationships in the proposed model.

13C. Age moderate the relationships in the proposed model.

13D. BMI moderates the relationships in the proposed model.

CHAPTER III

METHODOLOGY

Description of the Data

Data analytics for this study utilized a secondary dataset from CERNER Health Facts® (Cerner-HF). Cerner-HF is a HIPAA compliant dataset based on patients' electronic health records (EHR) managed by the Center for Health System Innovation (CHSI) at Oklahoma State University. CERNER-HF data includes 63 million patients and 380 million observations since 2000 and is donated by more than 600 CERNER hospitals and clinics across the United States (CHSI, 2019).

EHR has been reported to have many potential benefits for patients, providers, and overall quality of healthcare services. The integrated health data allows transparency and accessibility for the patients. This allows for patients to be educated and involved in the decision-making process relevant to their health. The consistency of treatment across healthcare providers could be subjective especially if the information relevant to the prior treatment is not easily available. This absence of key information could affect the quality of care. The accessibility of EHR could also help ease the transfer process betweenproviders. EHR also provides a platform to monitor the quality of care, which could be beneficial for all parties. For providers, this may mean that they could use this tool to maintain excellent services for the patients and for the administrators and it could

serve as a resource for understanding the quality care provided within their healthcare office.

Beyond improving the quality of healthcare services, EHR could also serve the purpose of advancing our understanding of health. her, as an integrated data registry, allows extensive and longitudinal data to be collected without additional cost, which would be an excellent tool for research. EHR data could be beneficial for health trend monitoring process and in turn, the findings could be used for program and policy development. Therefore, the program and policy will be more targeted in the area where it is needed. EHR has been utilized in some studies in the fields of medical science, public health, and epidemiology. However, EHR is a novel data type to also be used in the social sciences research. In the context of the current study, EHR provided a unique opportunity to understand the connection between mental health and physical health diagnosis.

Participants

The sample was obtained through data mining from the full data set of EHR of 63 million patients. The sole inclusion criterion in the current study was military insurance (previously known as CHAMPUS). If the individual utilized this military insurance coverage at least once with the health service they obtained from CERNER hospitals or clinics, then they were eligible to be included in the sample. In addition to military personnel, any spouse and/or dependent of military personnel are eligible for the health insurance coverage therefore, it is important to note that military personnel, spousesand/or dependents were potentially included in the current study. After applying the inclusion criteria and narrowing down the visits to a maximum of three for each

patient. In the final data included for the analysis, there were approximately 280,000 encounters from 18,253 patients ranging in age between 0 and 90 years. Thirty-four percent of patients are male, and thirty-three percent are female. However, thirty-two percent of participants did not report their gender. Forty-nine percent of the sample were White Caucasian and fourteen percent were non-White. Thirty-six percent did not report their race. Of total sample, 12,042 patients did not have a PTSD diagnosis and 6,211 (34.03%) had been diagnosed with PTSD.

Procedure

Access to CERNER Health Facts® requires a procedural request through the CHSI at Oklahoma State University. Due to the size of the full CERNER-HF dataset, researchers are required to submit criteria to create sample of the data. For the current study, the inclusion criterion is patients who utilized military insurance. The data included various aspects of EHR such as diagnoses, lab procedures, medication and treatment, clinical events, and demographics of patients. For the purpose of the current study, only data on diagnosis, laboratory procedures and demographic information of patients were included in the final dataset.

Additional data management and cleaning was conducted using STATA 16. To establish temporal precedence in the data, PTSD had to be diagnosed prior to any cardiovascular, or sleep disorder, otherwise the patients were dropped from the data. Then, researcher identified a time point before and after the patients' PTSD diagnosis. Hence, each participant had a maximum of three data points. Afterwards, additional screening for outliers was conducted using descriptive statistic and any outliers found were eliminated. After data cleaning and management steps were completed, data

analysis was carried out. Further description on the data analysis process is provided in the analysis section.

Measures

Sociodemographic Measures

Information on age, sex, and race were obtained from the data. Age was coded into one of the age groups; 0-17 years coded as 1, 18-49 years coded as 2, and 50 years and older coded as 3. Sex was coded 0 = male, and 1 = female. Race was coded 0 = white, and 1 = non-white.

PTSD

In the current study, disruption in functioning resulting from a psychological trauma was measured by a clinical diagnosis of Posttraumatic Stress Disorder (PTSD). The diagnosis was based on ICD 9-CM or ICD 10-CM. The PTSD code for ICD 9-CM 309.81 and for ICD-10-CM F43.10 for PTSD unspecified, F43.11for PTSD acute and F43.12 for PTSD chronic. Diagnoses of Posttraumatic Stress Disorder were conducted by health professionals. The presence of psychological trauma was transformed into dummy-code variable (0 = no PTSD, 1 = has PTSD diagnosis) based on the diagnosis code variable in the original dataset.

Physiological Stress Response

Physiological stress was assessed through vital signs. Vital signs were measured on each visit. For purposes of this study, information of physiological stress was obtained from the visit prior to PTSD diagnosis and after the diagnosis listed. Vital signs based on objective clinical measures of systolic and diastolic blood pressure (BP) and pulse rate serve as the primary assessment indicators of physiological stress.

Blood Pressure (BP). BP contains two measurements: systolic and diastolic. Both systolic and diastolic were measured in millimeters of mercury (mmHg). Systolic BP refers to the pressure of the blood against the wall of the heart while the heart pumps blood to the rest of the body. Diastolic BP refers to the pressure of blood against the wall while blood enters the heart.

Pulse Rates. Pulse rates refers to the speed of the contraction of the heart in a minute. It is also known as heart rate. It is measured in beats per minute (BPM unit). In the current study, pulse was measured during each visit to the CERNER hospitals/clinic. AHA specified normal heart rates are between 60-100 BPM.

Pre-Disease Biomarkers

Information on pre-disease stage were obtained from the laboratory test result relevant to inflammation. Inflammation is an immune system reaction toward any threat including stress or pathogen. In the proposed model, the pre-disease stage was indicated by inflammation measured through immune system reactions including leukocyte, platelet, and neutrophils/lymphocytes ratio. Leukocyte or white blood cell count were used as indicators of immune system reaction. Platelet is a blood cell component that plays important role on clotting. Leukocyte and platelet were measured in thousand per microliter ($10^3/\mu l$). Neutrophils is a type of white blood cell that has a specific role in restoring damage tissues and reducing infection. On the other hand, lymphocytes are a different type of leukocyte that plays role in destroying invaders including virus and bacteria. The ratio of neutrophils and lymphocytes indicates systemic inflammation. Information on pre-disease biomarkers is only limited to some participants who were required to have the test done due to their condition.

Pathology

In the current study, the main outcome of interest was the diagnosis of diseases. The diagnosis process was conducted by health professionals and listed in the dataset as ICD 9-CM and 10 codes as well as the disease name. Diagnosis was only included if the date of diagnosis occured after the date of diagnosis for PTSD. Further, there are two types of disease groups that were included in the analysis: cardiovascular disease (CVD), and sleep disorder. Below is further description of each disease.

Cardiovascular Disease (CVD). CVD is defined as various type of diseases that are located in the heart and vascular system. An example of a CVD is myocardial infarction, which listed as 410 – 414 in ICD 9-CM and I21 – I23, I29 in ICD 10. For the purpose of the current study, the absence of CVD was recoded as 0, or 1 if there was evidence of one or more CVD diagnosis. Additionally, any congenital heart disease was also excluded from analysis.

Sleep Disorder. Sleep disorders are various conditions that affect the quality of sleep. Insomnia and nightmare disorder are examples of sleep disorders. Night terror is listed with 307.46 in ICD-9 CM F51.4 in ICD 10. Based on the information on EHR, the presence of any sleep disorder was coded as 1 whereas 0 represented no diagnosis of a sleep disorder.

Analytical Procedure

In the current study, the data analysis process began with data cleaning and management. After data cleaning and management were completed, descriptive analysis was conducted followed by the path analysis and multiple groups analysis. Further description of each step is provided below.

Data cleaning and management were conducted prior to analyzing the proposed model using STATA 16 software. Initial descriptive statistics analysis including frequencies, means, and standard deviation were conducted to obtain basic information of the data. Based on the descriptive statistics, the researcher identified any missing data, outliers, and recoded variables. The normal range of each variable listed in the literature review were used to determine whether or not to drop the data. Further, some variables were also recoded as described in the variable section. In addition to descriptive analysis, correlation between variables were also examined to evaluate relationships between variables in the proposed model.

After data cleaning and management process were completed, the analysis was then proceeded using MPLUS 8. Path analyses were utilized to evaluate hypothesis 1 through 9. Indirect effects stated in hypotheses 10, 11 and 12 were also included in this analysis. MLR estimation was utilized. Further, due to the amount of missing data in the current study, Monte Carlo estimation were also utilized. Global fit statistics were not available for the current model. Path analysis is a structural equation method of analysis used to evaluate the fit of a proposed model with the data (Kline, 2006).

There are several recommended steps involved in conducting structural equation modeling (SEM) including model specification, identification, evaluation of the model fit, interpretation of estimates, consideration of equivalent or near-equivalent models, and reporting results (Kline, 2011). In the current study, a model has been specified and identified based on comprehensive literature reviews of the variables. To evaluate hypothesis 13, the researcher utilized a mixture modeling to evaluate the path analysis for

multiple group comparison. MLR and Monte Carlo estimations were also utilized for this model.

Rescaling of variables

Given the significant findings but smaller parameter yielded from the proposed model, rescaling was conducted for interpretability. The rescaled variables included systolic and diastolic BP, pulse rate, leukocyte count, platelet count, and N/L ratio both in pre-PTSD, and post-PTSD models. For systolic and diastolic BP, the variables were rescaled by multiplying them to 100. Similarly, pulse rate was also rescaled to 100. Leukocyte count and N/L ratio were rescaled to 10 times the original and platelet count was multiplied and rescaled to 1000 times the original.

Delimitations

The data for the current study has some potential limitations that are explained in this section. First, the data was collected and entered by a third party, which leavespotential for the method of data collection not being consistent across participants. Unfortunately, CHSI was unable to provide the questionnaire used to collect data. Further, the data were based on objective measures from EHR. The potential downside of objective measures, specifically in the area of the current study, is that we were unable to capture the subjective aspect of patients' experience. Another potential limitation is that there was no available identification of whether the patient was an active military servicemember or military dependent. Thus, it was not possible to evaluate the difference in impact for active military members who may have been directly involved in combat and military dependents who experienced secondary stress.

CHAPTER IV

RESULTS

Associations between physical responses, pre-disease biomarkers, PTSD, and pathology before and after a traumatic experience emerged from the proposed model using structural equational modeling, specifically path analysis and multiple group path analysis (see Figure 2). The physiological responses and pre-disease biomarkers were evaluated prior to a PTSD diagnosis. Physiological responses included three different measures: systolic and diastolic BP and pulse rate. Pre-disease biomarkers included leukocyte count, platelet count, and neutrophil to lymphocyte ratio (N/L ratio). Further, physiological responses, pre-disease biomarkers, and pathology were also examined in the aftermath of a PTSD diagnosis. Sleep disorder and cardiovascular disease were specific pathologies that were included in the proposed model. Beyond the direct paths between aforementioned variables, indirect path associations within the proposed model were also evaluated.

Full Model

The full model is presented in Figure 3 and included 13,314 individuals. In the pre-PTSD model, the analysis indicated both positive and negative relationships between the physiological responses and pre-disease biomarkers. A significant path between pulse rate and leukocyte count ($\beta = 0.293$, $p = 0.004$) was found, which means that for a ten

beat per minute increase in pulse rate, the leukocyte counts increased by 293 leukocyte count per microliter. A positive relationship was also found between systolic BP and platelet count ($\beta = 0.055$, $p = 0.005$). A ten mmHg boost in systolic BP was associated with an elevation of 550 platelet count per microliter. These findings offered some support for the connection between physiological responses and pre-disease biomarkers as stated in the first hypothesis. On the contrary, evidence of the opposite relationship was also noted; an increase in diastolic BP by 10 mmHg was significantly associated with a lowering of 0.343 in N/L ratio ($\beta = -0.343$, $p = 0.026$). Further, when the leukocyte count increased by a thousand count per microliter, the risk for a diagnosis of PTSD was augmented by 2.28 ($\beta = 0.228$, $p = 0.007$), which supported the second hypothesis that proposed connection between pre-disease biomarker and the risk of being diagnosed with PTSD. The pre-PTSD model provided some insights into the physiological profiles associated with the risk of being diagnosed with PTSD.

Further, the model also evaluated various physiological impacts of PTSD. PTSD was found to impact physiological responses, pre-disease biomarkers, and pathology. In fact, having a PTSD diagnosis altered physiological responses differently, as it was noted to reduce the systolic BP by 3.4 mmHg ($\beta = 0.034$, $p < 0.001$), and elevated pulse rate by 2.9 bpm ($\beta = 0.029$, $p < 0.001$). Individuals who have PTSD diagnosis were found to have 1.04 unit lower in N/L ratio ($\beta = -0.104$, $p = 0.027$). Being diagnosed with PTSD was also associated with 0.093 higher likelihood of being diagnosed with cardiovascular disease ($\beta = 0.093$, $p < 0.001$) and 0.121 higher risk for sleep disorder ($\beta = 0.121$, $p < 0.001$). These findings provided some support for hypothesis 3, 4, and 5.

The model also yielded evidence of relationships between physiological responses, pre-disease biomarkers, and pathology. In the current model, when the pulse rate was faster by 10 bpm, the leukocyte count also increased by 330 per microliter ($\beta = 0.330$, $p < 0.001$), 5000 platelet count per microliter ($\beta = 0.050$, $p = 0.001$), and 0.656 in N/L ratio ($\beta = 0.656$, $p < 0.001$). In addition, higher systolic BP by 10 mmHg was also found to predict 4900 count per microliter elevation of platelet count ($\beta = 0.049$, $p = 0.001$). These significant paths supported the sixth hypothesis positing an association between physiological responses and pre-disease biomarkers. However, two significant paths suggested different effects. A ten mmHg increase in systolic BP was associated with 86 leukocyte count per microliter decrease ($\beta = -0.086$, $p < 0.035$) and 0.757 N/L ratio lower ($\beta = -0.757$, $p < 0.001$). Further, both physiological responses and pre-disease biomarkers were also examined to predict pathology. Ten bpm slower pulse rate projected significantly 0.005 higher risk for CVD ($\beta = -0.059$, $p = 0.006$) and marginal risk for sleep disorder ($\beta = -0.033$, $p = 0.054$). Lower diastolic BP by ten mmHg also predicted 0.004 higher risk for being diagnosed with CVD ($\beta = -0.040$, $p = 0.006$). Additionally, two pre-disease biomarkers significantly predicted CVD. When platelet count lowered by 1000 count per microliter, it reflected higher risk of 0.248 for CVD ($\beta = -0.248$, $p < 0.001$). A ten unit increase in N/L ratio suggested 0.026 increase of CVD risk ($\beta = 0.026$, $p < 0.001$). However, only a thousand decrease in platelet count was associated with 0.077 of increase risk for sleep disorder ($\beta = -0.077$, $p = 0.030$). The current findings of positive relationships of both physiological responses and pre-disease biomarkers with the risk of being diagnosed with disease suggests support for hypothesis 7 and 8. Moreover, there was also evidence of comorbidity between CVD and sleep disorder ($\beta =$

0.009, $p < 0.001$), which provided support for hypothesis 9. Taken together, these findings indicate connections between all three variables which can offer insights into the mechanism linking PTSD and pathology.

Beyond the examination of direct effects, the current study also evaluated indirect path associations in the aftermath of PTSD. First, evidence of significant indirect paths between physiological responses and pathology via pre-disease biomarkers were noted. A total indirect effect that connects systolic BP to the risk of being diagnosed with CVD was found to be significant ($\beta = -0.033$, $p < 0.001$), specifically through platelet count ($\beta = -0.012$, $p = 0.002$), and N/L ratio ($\beta = -0.020$, $p < 0.001$). Two specific indirect paths between pulse rate and CVD mediated by platelet count ($\beta = -0.013$, $p = 0.001$) and N/L ratio ($\beta = 0.017$, $p < 0.001$) were also found to be significant. Another total indirect effect between systolic BP and sleep disorder was also found ($\beta = -0.010$, $p = 0.030$). This path was significantly mediated by platelet count ($\beta = -0.004$, $p = 0.044$). A significant specific indirect effect between pulse and sleep disorder was also found, specifically through platelet count ($\beta = -0.004$, $p = 0.042$). The indirect model also yielded significant total and specific indirect effects between PTSD and pre-disease biomarker mediated by physiological responses. Two total indirect effects connected PTSD and leukocyte count ($\beta = 0.012$, $p < 0.001$) and N/L ratio ($\beta = 0.047$, $p < 0.001$). Both of the total indirect effects were mediated by diastolic BP ($\beta_{leukocyte} = 0.003$, $p = 0.047$; $\beta_{N/Lratio} = 0.026$, $p < 0.001$) and pulse rate ($\beta_{leukocyte} = 0.010$, $p = 0.003$; $\beta_{N/Lratio} = 0.019$, $p = 0.003$). Two additional specific indirect effects were also found that connected PTSD and platelet mediated by diastolic BP ($\beta = -0.002$, $p = 0.003$) and pulse rate ($\beta = 0.001$, $p = 0.009$). Moreover, six specific indirect effects were also discovered in the model. The connection

between PTSD and CVD was mediated by pulse (β = -0.002, p = 0.047) and N/L ratio (β = -0.003, p = 0.022). The same indirect path was also mediated by diastolic BP and platelet (β = 0.000, p = 0.006), pulse rate and platelet count (β = 0.000, p < 0.006), diastolic BP and N/L ratio (β = 0.001, p = 0.002), as well as pulse rate and N/L ratio (β = 0.001, p = 0.011). Based on the evaluation of indirect model, there was some evidence of hypothesis 10, 11, and 12. Overall, findings from direct effects and indirect effects suggested some mechanism that connects PTSD and pathology.

Multiple Group Analysis

Multiple groups analysis was also conducted to compare the paths and evaluate potential moderation effects as stated in hypothesis 12. In the current study, multiple group analysis was conducted to compare the full model with sex, race, age, and body mass index (BMI). Further details on the findings of these analyses are provided below.

Sex

A multiple group analysis was conducted to compare the proposed model for the full model (see Figure 3), males (see Figure 4) and female participants (see Figure 5). Direct effect results for the full model are displayed in Table 1, males in Table 3 and females in Table 5. Additionally, indirect effect results for the full model were listed in Table 2, males in Table 4 and females in Table 6. The final sample for this model included 9,479 individuals.

In the pre-PTSD model, findings indicated that physiological responses were partly associated with pre-disease biomarkers. The positive relationships were noted among male participants, specifically between faster pulse rate and increased leukocyte count (β = 0.509, p = 0.001), platelet count (β = 0.046, p = 0.019), and N/L ratio (β =

0.506, $p = 0.010$). Higher systolic BP was also linked to increased platelet count (β = 0.074, $p = 0.003$). However, there was limited connection between physiological responses and pre-disease markers among female participants. Only faster pulse rate predicted elevated leukocyte count in this sample ($\beta = 0.223$, $p = 0.046$). In this case, findings from male study participants yielded more similarities to the full model than the female participants. Further, there was not any significant path found in male and female models. Only a marginal effect of higher platelet count associated with PTSD diagnosis in the male sample ($\beta = 0.592$, $p = 0.064$), and higher N/L ratio with PTSD diagnosis in the female sample ($\beta = 0.122$, $p = 0.058$) were noted. Comparisons of relationships between pre-disease biomarkers as predictors of the risk for being diagnosed with PTSD suggest that findings in the male and female samples deviated from the full model. The evidence from multiple group analysis of sex indicated some support for hypothesis 13A that stated sex moderated the relationships in the pre-PTSD model.

Following PTSD diagnosis, both male and female participants showed different physical responses. Among males, being diagnosed with PTSD was associated with lower systolic BP ($\beta = -0.044$, $p < 0.001$), faster pulse rate ($\beta = 0.060$, $p < 0.001$), lower N/L ratio ($\beta = -0.211$, $p = 0.026$), higher likelihood of being diagnosed with CVD (β = 0.109, $p < 0.001$) and sleep disorder ($\beta = 0.133$, $p < 0.001$). In comparison, females had a significantly lower systolic BP ($\beta = -0.052$, $p < 0.001$), elevated platelet count (β = 0.014, $p = 0.003$), decreased N/L ratio ($\beta = -0.145$, $p = 0.008$) and enhanced the likelihood of being diagnosed with CVD ($\beta = 0.127$, $p < 0.001$) and sleep disorder (β = 0.127, $p < 0.001$). The impact of PTSD on male participants and the full model showed more similarity to both male and female participants. The evidence of PTSD impact

provided minimal support to the differential impact of physiological responses, pre-disease biomarkers, and pathology among males and females as stated in hypothesis 13A.

Results from the current study also revealed associations between physiological response and pre-disease biomarkers in the aftermath of trauma. Among males, faster pulse rate was found to be associated with higher leukocyte count ($\beta = 0.207$, $p < 0.001$) and N/L ratio ($\beta = 0.875$, $p = 0.002$). In the same model, increased systolic BP predicted higher platelet count ($\beta = 0.057$, $p < 0.001$), but lower N/L ratio ($\beta = -0.998$, $p < 0.001$). Similarly, pulse rate was also associated with higher leukocyte count ($\beta = 0.524$, $p = .001$), platelet count ($\beta = 0.101$, $p = 0.008$) and N/L ratio ($\beta = 0.468$, $p = 0.002$) among females in the sample.

Further, associations between physiological responses and pre-disease biomarkers with pathology were also evaluated. Lower diastolic BP ($\beta = -0.154$, $p = 0.005$) and pulse rate ($\beta = -0.269$, $p = 0.003$) were found to significantly predicted CVD diagnosis among male study participants. Only slower pulse rate marginally predicted higher risk of being diagnosed with a sleep disorder ($\beta = -0.130$, $p = 0.068$) in the same sample. In comparison, higher diastolic BP was the only significant predictor of pathology, specifically sleep disorder ($\beta = 0.091$, $p < 0.001$) among female study participants. Moreover, relationships between pre-disease biomarkers and pathology were also identified. Lower platelet count ($\beta = -0.468$, $p < 0.001$) and higher N/L ratio ($\beta = 0.146$, $p < 0.001$) were predictive of CVD diagnosis for males. In contrast, pre-disease biomarkers had no direct association with pathology among women. In this case, findings pertinent to physiological responses and pre-disease biomarkers as precursors for

pathology from the full model shared more similar paths with male than among female participants.

Indirect effects were also evaluated relative to sex. First, there was some evidence that supported an indirect relationship between physiological responses and pathology through pre-disease biomarkers. Systolic BP was indirectly related to risk of CVD among male participants (β = -0.171, $p < 0.001$) through platelet count (β = -0.027, p = 0.002) and N/L ratio (β = - 0.146, $p < 0.001$). Pulse rate also indirectly predicted the risk of being diagnosed with CVD among male study participants (β = 0.018, p = 0.009). The specific indirect effect through N/L ratio only had a marginal effect (β = 0.128, p = 0.003). Systolic BP was also indirectly related to the risk of being diagnosed with sleep disorder (β = -0.048, p = 0.023), only marginally mediated by N/L ratio (β = -0.042, p = 0.051). There were no significant total and specific indirect effects found between physiological responses and pathology in female participants. The findings from male study participants indicated more similarities to the full model compared to female study participants.

PTSD also indirectly associated with pre-disease biomarkers through physiological responses. In male study participants, being diagnosed with PTSD was linked to all three pre-disease biomarkers; leukocyte count (β = 0.017, $p < 0.001$) significantly mediated by pulse rate (β = 0.012, p = 0.003), N/L ratio (β = 0.094, $p <$ 0.001) through diastolic BP (β = 0.044, p = 0.001) and pulse rate (β = 0.052, p = 0.006). Additionally, a specific indirect effect was also noted for PTSD and platelet count mediated by diastolic BP (β = -0.002, p = 0.001). In comparison, PTSD indirectly associated with leukocyte (β = 0.016, p = 0.029), and N/L ratio (β = 0.032, p = 0.030) in

female study participants. There was only a marginal specific indirect effect identified that connected PTSD and N/L ratio through diastolic BP (β = 0.022, p = 0.051)

The examination of indirect model also yielded relationships between PTSD and pathology. PTSD showed significant indirect association to CVD (β = -0.034, p = 0.022). The link was associated through several mediators including pulse rate (β = -0.016, p = 0.008), N/L ratio (β = -0.031, p = 0.018), diastolic BP and platelet count (β = 0.001, p = 0.008), diastolic BP and N/L ratio (β = 0.006, p = 0.001), as well as pulse rate and N/L ratio (β = 0.008, p = 0.009). There were no significant total or specific indirect effects linking PTSD and sleep disorder for male study participants. On the other hand, only a total indirect effect was found to be significant between PTSD and CVD (β = -0.003, p = 0.048). Overall, the indirect model of male participants yielded more similarities to the full model than female participants. These findings suggest that the proposed indirect associations between physiological responses and pathology better fit male study participants. It is possible that females have different indirect mechanisms linking PTSD and pathology.

Race

Multiple group path analyses were also conducted to compare the full model and racial groups (see Figures 6 and 7). In the current study, the racial group was separated into two, a sample of white and non-white individuals. The direct effects for the white sample were reported in Table 7 and the non-white sample in Table 9. The indirect effects for the white sample are listed in Table 8 and the non-white sample in Table 10. The sample for this analysis included 9,090 individuals.

There was some evidence of moderation in relationships between physiological stress response and pre-disease biomarkers in the pre-PTSD model, providing support to hypothesis 13B. Faster pulse rate was associated with elevated platelet count ($\beta = 0.345$, $p = 0.006$) among white study participants. A similar trend also applied to systolic BP and platelet count ($\beta = 0.051$, $p = 0.018$). In comparison, non-white study participants who maintained lower systolic BP ($\beta = -0.436$, $p = 0.012$) and higher pulse rate ($\beta = 0.362$, $p = 0.048$) reported higher N/L ratio. The findings from white study participants yielded more similarities with the full model. Evidence of an association between pre-disease biomarkers and the risk for a PTSD diagnosis also existed in the model. Higher leukocyte count was found to be associated with higher likelihood of receiving PTSD diagnosis among both white ($\beta = 0.222$, $p = 0.025$) and non-white ($\beta = 0.379$, $p < 0.001$) study participants. This finding supports the second hypothesis that stated elevated pre-disease biomarker will mark higher risk to being diagnosed with PTSD. Additionally, non-white study participants with lower N/L ratio also reported higher risk for PTSD ($\beta = -0.214$, $p = 0.003$). Findings on lower pre-disease markers were exclusive to the full model and non-white study participants, which may imply blunted stress response due to pre-existing allostatic loads.

In both groups, PTSD influenced the physiological response, pre-disease biomarkers, and pathology in the aftermath of trauma. White study participants with PTSD diagnosis had lower systolic BP ($\beta = -0.039$, $p < 0.001$), higher pulse rate ($\beta = 0.028$, $p = 0.004$), and marginally lower N/L ratio ($\beta = -0.117$, $p = 0.050$). On the other hand, non-white study participants with PTSD only had lower systolic BP ($\beta = -0.029$, $p = 0.017$). No connection between PTSD and pre-disease biomarkers was found in the

non-white study participants. PTSD also significantly enhanced the likelihood of being diagnosed with CVD ($\beta_{CVD\ white}$ = 0.088, $p < 0.001$; $\beta_{\text{CVD non-white}}$ = 0.065, p < 0.001), and sleep disorder ($\beta_{sleep\ white}$ = 0.125, $p < 0.001$; $\beta_{sleep\ \text{non-white}}$ = 0.130, $p < 0.001$) for both groups. This evidence conveys more similarities for PTSD impacts between findings from the full model and white study participants. White study participants appeared to experience more alterations following PTSD diagnosis, which offers support to the previous findings that non-white individuals have higher pre-existing allostatic load. Despite the different impacts on physiological responses and pre-disease biomarkers, those with a PTSD diagnosis from the full model, white and non-white study participants were consistently found to have elevated risk for disease pathology. The findings further suggested higher risk for pathology for non-white individuals. Taken together, these findings may offer insights into various mechanism linking PTSD to pathology, yet it yielded similar final adverse outcomes.

The model was evaluated by race to identify potential mechanisms of pathology development. Following the PTSD diagnosis, white study participants who maintained faster pulse rate also had higher leukocyte count (β = 0.307, p = 0.001), platelet counts (β = 0.040, p = 0.006), and N/L ratio (β = 0.659, p = 0.001). In the same group, those with higher systolic BP reported higher platelet count (β = 0.044, p = 0.002). Non-white study participants with higher pulse rate also reported elevation in leukocyte count (β = 0.413, $p < 0.001$), and N/L ratio (β = 0.510, p = 0.001). Increases in diastolic BP also associated with higher N/L ratio (β = 0.740, p = 0.046). Interestingly, elevated systolic BP was associated with decreased N/L ratio in both groups (β_{white} = -0.725, $p < 0.001$; $\beta_{non\text{-}white}$ = -1.015, p = 0.023). Results from white study participants indicate closer similarities with

the full model suggesting race had a moderating effect on the relationship between physiological responses and pre-disease biomarkers in the aftermath of a PTSD diagnosis. The model also indicated evidence of significant paths that connect physiological responses and pathology for both groups.

White study participants with lower diastolic BP (β = -0.070, p = 0.005) and pulse rate (β = -0.090, p = 0.015) were at elevated risk for being diagnosed with CVD. In comparison, findings from non-white study participants indicated lower pulse rate significantly associated with sleep disorder (β = -0.117, p = 0.025). In this case, results on the relationship between physiological responses and pathology from non-white individuals illustrated similar paths with the full model. These findings also indicated that race moderated the relationship between the two. Moreover, evidence also provided support for pre-disease biomarkers as a precursor to disease pathology. Lower platelet count was significantly associated with the higher likelihood of being diagnosed with CVD (β = -0.465, $p < 0.001$) for white participants. The opposite effect was found for N/L ratio which was associated with higher risk of CVD (β = 0.041, p = 0.005). Additionally, lower platelet counts significantly predicted sleep disorder (β = -0.141, p = 0.020). On the contrary, there was no evidence of pre-disease biomarkers as predictors for pathology in non-white study participants. The paths that connected pre-disease biomarkers and pathology in the full model displayed more similarities with white study participants. It further suggested mechanisms of pathology development may be moderated by race as each group may experience different challenges relevant to their psychological trauma.

Indirect effects were also evaluated to compare the white and non-white study participants relevant to physiological functions following PTSD diagnosis. Results yielded indirect relationship between physiological responses and pathology limited to white study participants. Systolic BP significantly predicted CVD (β = -0.051, $p < 0.001$), specifically through platelet (β = -0.020, p = 0.006), and N/L ratio (β = -0.029, p = 0.008). Systolic BP also indirectly associated with sleep disorder (β = -0.017, p = 0.022) mediated by platelet (β = -0.006, p = 0.046). Additionally, some specific indirect effects also supported this notion. Pulse rate was associated with CVD through platelet (β = -0.019, p = 0.006) and N/L ratio (β = 0.027, p = 0.014). There was no significant total or specific indirect effect between physiological responses and pathology in the sample of non-white study participants. It appears that the indirect associations between physiological responses and pathology found in the white study participants shared commonalities with the full model. The absence of indirect effect in non-white individuals suggested potentially distinct mechanism compared to the white individuals and the full model.

Result on indirect effects also illustrated the relationship between PTSD and pre-disease biomarkers through physiological responses in both white and non-white participant groups. Among white study participants, a total indirect effect between PTSD and leukocyte was noted (β = 0.011, p = 0.006). The indirect path was significantly mediated by pulse rate (β = 0.009, p = 0.020). There were also additionally two specific indirect paths identified; PTSD and platelet count through diastolic BP (β = -0.002, p = 0.005) and pulse rate (β = 0.001, p = 0.031). In non-white study participants, being diagnosed with PTSD was indirectly associated with leukocyte count (β = 0.014, p =

0.047*),* without any significant specific indirect path. Results yielded from both white study participant groups displayed more similarities to the full model. These findings may suggest that indirect connections between PTSD and pre-disease biomarkers are moderated by race.

Further, in the sample of white study participants, PTSD was found to be associated with pathology through physiological responses and pre-disease biomarkers. PTSD was found to be indirectly associated to CVD by diastolic BP and platelet (β = 0.001, p = 0.011*),* pulse rate and platelet count (β = -0.001, p = 0.031*),* as well as diastolic BP and N/L ratio (β = 0.001, p = 0.015*).* In comparison, only a marginal specific indirect effect was found between PTSD and sleep disorder mediated by diastolic BP and platelet count (β = 0.000, p = 0.058*).* There were not any total and specific indirect effects found in non-white study participants. Both findings from white and non-white study participants displayed deviation from the full model. This may suggest unique indirect mechanism between PTSD and pathology relevant to experiences unique to each racial group. Overall, the findings from race multiple group path analysis provided some support for the proposed model. White study participants displayed more similar paths to the full model. When examining non-white study participants, one may need to be cautious as different challenges associated with the traumatic experience could alter physiological responses and outcomes.

Age

Comparison between age groups were also explored. Age was separated in three groups: 0-17 years of age (children), 18-49 years (adults), and 50 years of age and older (older adults). The sample included 18,242 individuals. The direct and indirect findings

from child study participants listed in Tables 11 and 12, respectively. Table 13 showed direct effects and Table 14 displayed indirect effects from adult study participants. Tables 15 and 16 indicated direct and indirect effects from older adult study participants. Additionally, Figures 8, 9 and 10 displayed path models for the three age groups.

The connection between physiological responses and pre-disease biomarkers in the pre-PTSD model indicated some significant findings. In the child sample, it was noted that increased systolic BP was related to lower leukocyte count ($\beta = -1.155$, $p < 0.001$). When children experienced increased pulse rate, it was followed with decreased platelet count ($\beta = -0.114$, $p = 0.015$). Evidence for associations between physiological responses and pre-disease biomarkers were also found in adult participants. Faster pulse rate was associated with elevated leukocyte count ($\beta = 0.310$, $p = 0.004$). Increased systolic BP was also found to be associated with elevated platelet count ($\beta = 0.066$, $p = 0.005$). Additionally, higher diastolic BP was associated with a decrease of N/L ratio ($\beta = -0.550$, $p = 0.015$). There was only a significant path between physiological responses and pre-disease biomarkers in older adult participants; higher systolic BP was related to higher platelet count ($\beta = 0.066$, $p = 0.032$). In the pre-PTSD model, evidence also provided support that pre-disease biomarkers predicted the risk of PTSD limited to adult and older adult groups. Elevated leukocyte count ($\beta = 0.487$, $p < 0.001$) and lower N/L ratio ($\beta = -0.396$, $p = 0.009$) indicated enhanced risk of being diagnosed with PTSD in the adult sample. In comparison, elevated N/L ratio was associated with the risk of PTSD in the older adult sample ($\beta = 0.515$, $p < 0.001$). Results discovered more similar paths between the full model and the adult group in the pre-PTSD model. Taken together, documented

evidence may provide insights into the role of age in understanding the physiological profile relevant to risk for PTSD.

PTSD effect on physiological responses, pre-disease biomarkers, and pathology seemed to be exclusive to selected groups. Children who were diagnosed with PTSD had significantly lower systolic BP ($\beta = -0.040$, $p = 0.001$) and pulse rate ($\beta = -0.171$, $p < 0.001$). They also experienced an increase in diastolic BP ($\beta = 0.030$, $p = 0.002$). In comparison, PTSD increased pulse in adults ($\beta = 0.026$, $p = 0.025$) and reduced systolic BP in older adult participants ($\beta = -0.061$, $p < 0.001$). The impact of PTSD on pre-disease biomarkers was only noted in older adult participants. Specifically, PTSD was associated with lowered platelet count ($\beta = -0.011$, $p = 0.038$). Further, there was no evidence for the connection between PTSD and pathology in the child sample. However, enhanced risk of pathology was noted among adult and older adult study participants. PTSD was linked to CVD ($\beta_{adult} = 0.029$, $p < 0.001$; $\beta_{olderadult} = 0.194$, $p < 0.001$) and sleep disorder ($\beta_{adult} = 0.107$, $p < 0.001$; $\beta_{olderadult} = 0.138$, $p < 0.001$) in both adult and older adult study participants. Overall, the findings from adult and older adult participants revealed more common paths with the full model. Evidence indicated the role of age in moderating the impacts of PTSD.

The proposed model further investigated the potential role of age moderating the relationships between physiological responses, pre-disease biomarkers, and pathology. The result yielded significant connections between physiological responses and pre-disease biomarkers in the post-PTSD model exclusive to adult and older adult groups. In adult sample, faster pulse rate was linked to elevated leukocyte count ($\beta = 0.236$, $p < 0.001$), platelet count ($\beta = 0.029$, $p = 0.006$), and N/L ratio ($\beta = 0.223$, $p = 0.002$). Higher

systolic BP also predicted an increase in platelet count ($\beta = 0.034$, $p = 0.043$) and had an opposite effect in N/L ratio ($\beta = -0.249$, $p = 0.017$). In comparison, faster pulse rate was related to lower platelet count ($\beta = -0.080$, $p = 0.049$), whereas higher systolic BP linked to lower N/L ratio ($\beta = -1.807$, $p < 0.001$) in the older adult group. Compared to the full model, findings from the samples of adult participants indicate more similar paths of physiological responses and pre-disease biomarkers. This may suggest potential maturation and aging effects to physiological response and pre-disease biomarkers that resulted in different responses following PTSD.

The model also examined relationships between physiological responses and pre-disease biomarkers as precursors for pathology, exclusive to the older adult group. There was not any significant path indicated in children and adult study participants. It was noted that lower systolic BP ($\beta = -0.615$, $p < 0.001$) and pulse rate ($\beta = -0.694$, $p < 0.001$) related to an enhanced risk for CVD in older adult study participants. Lower systolic BP ($\beta = -0.114$, $p = 0.002$), pulse rate ($\beta = -0.108$, $p = 0.001$) and higher diastolic BP ($\beta = 0.090$, $p = 0.008$) were found as precursors for sleep disorder in the same sample. Relevant to pre-disease biomarkers as precursors of pathology, it was noted that elevated leukocyte count ($\beta_{CVD} = 0.245$, $p = 0.000$; $\beta_{sleep} = 0.057$, $p = 0.002$), lower platelet count ($\beta_{CVD} = -1.471$, $p < 0.001$; $\beta_{Sleep} = -0.406$, $p < 0.001$) and N/L ratio ($\beta_{CVD} = -0.189$, $p < 0.001$; $\beta_{Sleep} = -0.018$, $p = 0.012$) linked to the risk for both CVD and sleep disorder in the older adult sample. Comparison of findings from all three groups suggested that the older adults resembled most similar paths to the full model.

The indirect effects were also examined across all three age groups; children, adult and older adult group. The connection between physiological responses and

pathology was limited to older adult study participants. Systolic BP was indirectly linked to CVD (β = 0.318, $p < 0.001$), significantly mediated by leukocyte count (β = -0.028, $p = 0.044$) and N/L ratio (β = 0.342, $p < 0.001$). Additionally, four total indirect effects were also noted; diastolic BP to CVD (β = 0.186, $p = 0.012$), pulse rate to CVD (β = 0.228, $p = 0.004$), diastolic BP to sleep disorder (β = -0.026, $p = 0.014$), and pulse rate to sleep disorder (β = 0.050, $p < 0.001$). There was not any specific indirect effects associated with these significant paths. Further, indirect associations between PTSD and pre-disease biomarkers were evident in adult and older adult participants. There was only one total indirect effect between PTSD and N/L ratio (β = 0.010, $p = 0.014$) in adult participants. On the other hand, PTSD diagnosis predicted leukocyte count (β = 0.013, $p = 0.021$) as well as PTSD and N/L ratio (β = 0.104, $p = 0.013$) mediated by diastolic BP (β = 0.111, $p = 0.003$) in older adult participants. Finally, indirect relationships between PTSD and pathology were only evident in older adult groups. There were four specific indirect effects noted; PTSD and CVD mediated by diastolic BP (β = 0.038, $p < 0.001$), PTSD and CVD mediated by diastolic NL and N/L ratio (β = -0.021, $p = 0.001$), PTSD and sleep disorder mediated by diastolic BP (β = 0.007, $p = 0.005$), and PTSD and sleep disorder mediated by diastolic BP and N/L ratio (β = -0.002, $p = 0.031$). The evaluation of indirect findings indicated most similarities between the full model and older adult participants, which may suggest distinct physiological processes associated with different aging state.

Body Mass Index (BMI)

Multiple group path analysis also explored the role of BMI in influencing the relationship between psychological trauma, physiological responses, pre-disease

biomarkers, and pathology. The sample size for this analysis was 2,155. BMI were categorized into two groups: lower and higher BMI. Lower BMI group included those with BMI between 0-25. The higher BMI group included individuals with BMI of 25 and larger. Direct effects for lower BMI group were listed in Table 17, and Table 19 for higher BMI group. Indirect effects were showed in Tables 18 and 20 for lower and higher BMI groups, respectively. Additionally, Figures 11 and 12 displayed the path models for both groups. Further description of each model is provided below.

In pre-PTSD model, there was evidence of connection between physiological responses and pre-disease biomarkers specific to participants exclusive to higher BMI. Those with faster pulse rate were found to have higher leukocyte count ($\beta = 0.271$, $p = 0.020$). Increases in systolic BP also predicted higher platelet count ($\beta = 0.069$, $p = 0.005$). Similar patterns were also found in the connection between pre-disease biomarkers and the risk of being diagnosed with PTSD as there was no significant path found in the lower BMI group. However, decreases in N/L ratio indicated higher risk in being diagnosed with PTSD ($\beta = -0.191$, $p < 0.001$) noted in the higher BMI group. Findings in the higher BMI group indicate more similar paths to the full model compared to the lower BMI group. These findings indicate different physiological mechanisms between lower and higher BMI groups, which support potential moderation effects of BMI.

Further, the post-PTSD model indicated some physical health impacts. PTSD did not appear to affect physiological responses and pre-disease biomarkers in individuals with lower BMI. However, in this group there was evidence of relationship between PTSD and pathology, specifically sleep disorder ($\beta = 0.109$, $p < 0.001$). The analysis also revealed individuals with higher BMI and diagnosed with PTSD had lower systolic BP (β

= -0.031, p = 0.036) and higher risk of being diagnosed with sleep disorder (β = 0.037, p = 0.048). There was no connection between PTSD and pre-disease biomarkers in this group. The impacts of PTSD showed minimal support in the full model. The only consistent evidence throughout the full model for lower and higher BMI was the association between PTSD and pathology, which may suggest similar outcomes but through distinct processes.

The model also revealed some relationships between physiological responses and pre-disease biomarkers. In the lower BMI group, a positive association was noted between pulse rate and leukocyte count (β = 0.374, p = 0.021). Higher systolic BP (β = 0.094, p = 0.024) and pulse rate (β = 0.073, p = 0.025) were associated with elevated platelet count in the same group. Lower systolic BP was found to be associated with higher N/L ratio (β = -2.277, p = 0.031). On the contrary, a negative relationship was found between systolic BP and platelet count (β = 0.085, p = 0.002) in the higher BMI group. Higher systolic BP (β = -0.871, p = 0.049) also linked to lower N/L ratio in the same group. Both low and high BMI groups shared some similar paths with the full model, yet there were some distinct paths as well, possibly representing different physiological processes.

Investigations were extended to the relationship between physiological responses and pre-disease biomarkers as precursors for pathology. Individuals with lower BMI and elevated systolic BP were shown to have higher risk of being diagnosed with CVD (β = 0.158, p = 0.045). In comparison, elevated diastolic BP was the only physiological response precursor that predicted the risk of being diagnosed with sleep disorder (β = 0.150, p = 0.024) in the sample of participants with higher BMI. Additionally, pre-

disease biomarkers also related to the risk of pathology. In the sample of individuals with lower BMI, elevated N/L ratio was associated with higher risk for CVD diagnosis (β = 0.040, p = 0.001). Depleted platelet count was linked to sleep disorder diagnosis (β = -0.251, p = 0.012) in the same sample. In the higher BMI group, higher platelet predicted CVD (β = -0.815, p < 0.001), whereas lower platelet count was linked to sleep disorder (β = -0.396, p = 0.006). Taken together, these findings suggest some similarities between each group and the full model. Nevertheless, distinct paths were also noted across the two groups, implying potential moderation effects of BMI in physiological functions.

The currents study also evaluated the indirect effects in a post-PTSD model. There was no significant total and specific indirect effect in the lower BMI group. On the other hand, there was a few significant total indirect effects in the higher BMI group. Systolic BP was indirectly associated with the diagnosis of CVD (β = -0.0110, p = 0.011). A specific path indicated that the association was only significant through platelet count (β = -0.069, p = 0.019). A specific path that connected systolic BP and sleep disorder through platelet (β = -0.034, p = 0.036) was also noted. The findings from the evaluation of the indirect model suggested minimal moderation effects of BMI. BMI may influence different physiological functions.

CHAPTER V

DISCUSSION

The current study focused on the interplay between physiological functions and relevant risks for PTSD and pathology in the context of military families. To date, research has documented adverse physical health consequences and potential spillover effects related to PTSD in the military population (Paulus et al., 2013; Lindqvist et al., 2014; Beristianos et al., 2016; Williams et al., 2015; Allen et al., 2018; Lester et al., 2010). However, there are limited studies exploring the underlying connection between the two. The current section provides summary of the findings and further explore the explanation behind them.

In the pre-PTSD model, as expected, a faster pulse rate and higher leukocyte count was linked to an enhanced risk of being diagnosed with PTSD. Alterations in physiological responses and pre-disease biomarkers support the concept of allostatic mechanism responses to environmental demands (McEwen, 1998). Abboud and Singh (2017) provide an example of the connection between physiological responses and pre-disease biomarkers. In a case of hypertension, high blood pressure may damage the blood vessel wall, sending signals to start the immune response (Abboud & Singh, 2017). Therefore, the combination of both physiological responses and pre-disease biomarkers

provide insights into the physiological profile and mechanisms that can be used to assess the risk for PTSD.

To date, several studies have explored the physiological profile relevant to the risk for PTSD. Sumner and colleagues (2019) highlighted a potential bidirectional relationship between PTSD and inflammation and suggested the idea of certain physiological measures as an alternative to assess risks for PTSD. Longstanding evidence of increased inflammation in the aftermath of trauma has been documented (Sumner et al., 2019; Lindqvist et al., 2017). On the other hand, Sumner and colleagues (2019) discussed the potential role of existing inflammation in altering one's response to a threat which then could increase the subsequent risk of developing PTSD (Sumner et al., 2019). Pre-existing inflammation can influence amygdala functions that are essentials for memory and learning which are key components for PTSD development. A study discovered greater amygdala responses relevant to those with higher inflammation (Muscatell et al., 2015). Overactivity in the amygdala has been linked to various problems including emotional regulations which can enhance the risk for PTSD, as it limits one’s ability to cope with trauma. These findings suggest that individuals with a higher level of inflammation may also be at a higher risk for developing PTSD following psychological trauma. Physiological profiles can be used to identify individuals who are at higher risks for PTSD and physical health problems.

The current study also documents mixed impacts of PTSD on different physiological aspects and pathology. Being diagnosed with PTSD has mixed effects on physiological responses. These findings are partially consistent with Buckley and colleagues (2008) who documented evidence of higher blood pressure and pulse rate

among those with PTSD diagnoses. Interestingly, individuals with PTSD diagnoses reported lower pre-disease biomarkers. A previous study indicated the opposite impact of PTSD on inflammatory responses marked by a higher count of leukocytes and platelet counts (Lindqvist et al., 2017). However, different studies suggest a blunted stress response following early trauma exposure (Lovallo et al., 2012). As the stress response system overloads, the ability to identify threats decrease. Thus, the lower pre-disease biomarkers responses may indicate an overloaded system. PTSD was directly linked to enhanced risks for pathology which is consistent with results in previous research. A recent study highlighted elevated incident risks for cardiovascular disease in aging veterans with PTSD (Beristianos et al., 2016). Similarly, PTSD is also associated with several sleep disorders (Williams et al., 2015). Finally, there was evidence of comorbidity between cardiovascular disease and sleep disorders. One article documented evidence of comorbidity between insomnia and cardiovascular disease, potentially due to dysregulation of the HPA, which affects inflammation (Javaheri & Redline, 2017).

Despite accumulated studies documenting alteration in physical functions following PTSD, there are limited studies exploring the underlying connection between various physiological alterations following PTSD. In the current study, the evidence provided supports for all three proposed direct and indirect paths that connect physiological functions, pre-disease biomarkers, and pathology illuminate insights into the mechanism between PTSD and pathology.

Multiple group analysis

The current study also investigated multiple group analyses based on several demographic characteristics in addition to the full model and how they influence

physiological functions. The characteristics include sex, race, age, and BMI. Further discussion of each model is provided below.

Sex

A comparison of male and female study participants provided support for sex as a moderator for the relationships between PTSD and physical functions. There were more similarities between male study participants and the full model then between female study participants and the full model, which may suggest that PTSD impacts physiological functions of female study participants distinctively. Thus, the current model provides the best insights limited to male study participants. Further investigations of the underlying mechanism for females are needed.

A higher prevalence of PTSD in females compared to their male counterparts was found (Breslau, 2009; Olff, 2017), which supports the notion of sex difference in PTSD. Further, different studies also documented different symptoms presented by males and females. Males reported more impulsivity following PTSD diagnoses (Pineless et al., 2017). On the other hand, females displayed self-blame as a form of cognitive symptoms (Pineless et al., 2017). Another study reported that numbing symptoms are more prevalent among male active-duty military, while female experienced avoidance, numb, and hyperarousal, but not hypervigilance (Hourani et al., 2015). The differential risk and presentation raise the question of the need for a distinct diagnosis due to the heterogeneity of presentation of PTSD across sex groups.

The difference in prevalence and symptom presentation may be explained by different factors that influence outcomes following trauma, including factors that shape the traumatic experience as well as risk or protective factors. The mechanisms of stress

response system have been found to work differently between males and females. Animal studies have documented evidence of differential responses to stress. Sexual dimorphism was indicated in fear learning, as male and female rats displayed different behaviors (Pooley et al., 2018). Male rats displayed startle responses following a stressor, meanwhile female rats did not display similar responses (Pooley et al., 2018). These results raise the question of whether males and females perceive stress differently. Something that is perceived as a threat for males may not be perceived as a threat for females.

Race

The investigation also investigated the relationship between the full model, white, and non-white study participants. Results support differential responses and risks relevant to PTSD and physiological functions. White study participants shared more similar paths with the full model. Therefore, the mechanisms that connects PTSD and physiological function remain elusive. Exploration of different physiological functions are needed to understand the extent of damages relevant to PTSD in non-white study participants.

Race serves as a moderator for relationships between stress and health. Accumulated evidence indicates worse health outcomes among non-white individuals. Duru and colleagues (2012) found that non-white individuals have a higher level of stress that is associated with an elevated risk of mortality. Further, a study by Ferguson and colleagues (2013) detected lowered inflammation in African American individuals, suggesting a potential blunted stress response. Race serves as a proxy beyond group membership. It also provides insights into potential socioeconomic status such as additional stressors unique to a specific race group. This evidence provides support for

Cumulative Advantage/Disadvantage Theory. This theory explains the systemic cumulative gains/losses associated with certain characteristics (e.g., race). In other words, being non-white individuals are associated with certain benefits or challenges. In this case, experiencing racism or poverty may attribute additional stressors following psychological trauma, which then could contribute to a higher risk for PTSD.

Data on race unfolds information beyond affiliation to certain groups or cultures. It further provides information on challenges as well as resources that are unique to a group. Comparisons of race discover different relationships between PTSD and physiological functions. The absence of significant paths in non-white individuals especially elucidates deviation from the proposed model, which maintains elevated risks for non-white individuals as intervention remains unprecedented. Further exploration of different physiological functions are needed to gain insight on the mechanisms that fit non-white individuals.

Age

Comparisons of age groups indicated the effect of moderation, suggesting differential risks for PTSD and physical health problems. Findings on children provided minimal support to the proposed model. On the other hand, older adult study participants displayed most shared paths with the full model which also implies most vulnerability were from the effects of PTSD. These findings raise questions on different mechanisms for children and adults.

Differential risks across age groups may be explained by select biological factors, such as the maturation of the stress response system. The limbic system serves as the central processing for stress, as it appraises the situation prior to sending out a signal to

start a chain of reactions (Canteras et al., 2020). In children, this area is not fully developed yet. Therefore, they may perceive a threat differently, which then influences responses produced by this particular age group that may differ from older individuals. In comparison, older adults were reported to have distinct cortisol production patterns as well as limbic system structure (Ennis et al., 2019). This finding suggests that the aging process intervenes with the alteration in stress response system activity and structure.

Further, the accumulation of stress also has implications on the functions of the stress response system. Crimmins and colleagues (2003) indicated that the allostatic load accumulation did not become significant prior to the age of twenty and exponentially worsened as people continued to age up to sixty, where it stabilized. Exposure to stress in children appears to link to allostasis but not allostatic load. On the other hand, older adults have an accumulation of allostasis that may create allostatic load or overload. Due to their higher allostatic load or overload, worse health outcomes are expected out of this group. These results provide further support for the allostatic load model and its contribution to pathology development (McEwen, 1998).

Body Mass Index (BMI)

BMI has been documented as a proxy of health status as it is highly correlated with different health outcomes, including morbidity and mortality (Abdelaal et al., 2017), and quality of life (Korhonen et al, 2014). In the current study, BMI was investigated as a moderator of psychological trauma and physical function relationships, however, little evidence about this was available.

As the current model fails to capture the physiological alterations following PTSD relevant to BMI, existing studies provide evidence of the impacts of BMI on different

physiological functions. Wester and colleagues (2014) observed a higher level of cortisol among obese individuals compared to their non-obese counterparts. A prolonged elevated level of cortisol means a high-level activity of the stress response system which can increase the risk for various mental health symptoms including depression (Santen et al., 2011). Interestingly, a study on PTSD indicated that opposite patterns of low cortisol levels were linked to a higher vulnerability for this specific mental health diagnosis (Mouthaan et al., 2014). This raises a question on whether cortisol level provides a protective mechanism for PTSD among those with higher BMI. Additionally, Choi and colleagues (2013) also observed elevated C-reactive proteins, a marker of inflammation, among individuals with high BMI. These findings provide insights that BMI may moderate PTSD impacts on physiological functions beyond the current model.

In addition to potentially serving as a mediator between PTSD and physiological responses, evidence also suggests change in BMI as a trauma response (Kubzansky et al., 2014). Similar finding was also noted by Smith and colleagues (2015). Moreover, LeardMann and colleagues (2015) observed both increase and decrease of BMI following PTSD. This finding suggests potential bidirectional impacts of PTSD on BMI. The alterations in behavioral responses, including eating and physical inactivity as a form of coping with the symptoms of PTSD. Therefore, these types of behaviors are relevant and need to be considered in future diagnosis process of PTSD.

Model comparisons

Throughout the entire model, heterogeneity of the physiological responses and pre-disease biomarkers were noted in pre-and post-PTSD models. Previous literature has documented mixed findings in this area. Several studies noted accelerated responses such

as elevated leukocyte levels and increased heart rate (Buckley et al., 2004) and elevated inflammatory responses (Lindqvist et al., 2017). However, d'Andrea and colleagues (2013) noted mixed findings. In addition to acceleration in heart rate and skin conductance responses, they also observed deceleration on the same measures. Further examination of the sub-samples indicated that those who experienced moderate exposure to trauma and exhibited moderate symptoms of PTSD reported acceleration responses. On the contrary, those with extreme trauma exposure and PTSD symptoms displayed decelerated responses, which suggest blunted stress responses (d'Andrea et al., 2013). Deriving from this finding, individuals with higher exposure to trauma may also have higher allostatic load accumulation. Incorporating information relevant to the traumatic experience and pre-existing allostatic load can provide better insight into the expected outcome. This also highlighted the importance of not overlooking different responses to PTSD as decelerated responses still tell a story relevant to the psychological trauma. Despite the various paths between the variables, evidence of the relationships between PTSD and pathology were observed across models. This finding confirms evidence from previous works (Beristianos et al., 2016). The homogeneity of pathology outcomes suggests equifinality of PTSD. PTSD may affect physiological functions unique to each individual. However, once the alterations reach a certain threshold, they may contribute to pathology development. Explorations of PTSD effects across different physiological functions are important to advance the understanding of diverse pathology development mechanisms.

Findings from the current study also suggest that the data support the full model. However, comparisons of sub-samples suggested otherwise. This implies that although

the current proposed model provides insights on a mechanism that connects PTSD, physiological functions, and pathology, the model is not inclusive for all individuals. In the case of race analysis, for example, most participants were white. Thus, it may provide limited insights into the mechanism in non-white individuals. Further explorations of various mechanisms are needed to develop inclusive and comprehensive physiological profiles relevant to PTSD.

CHAPTER VI

CONCLUSION

Overall, the current study identifies physiological profiles of individuals who are at a higher risk for PTSD and for higher impacts following PTSD. Pulse rate and leukocyte counts are relevant measures for the risk of PTSD, while pulse rate and N/L ratios following PTSD diagnoses serve as precursors for pathology development. Evidence of pathology as adverse health outcomes are also established. The results also provide insights into a mechanism that connects PTSD and pathology. However, it is worth mentioning that the model comparisons indicate limitations in capturing mechanisms of PTSD and physiological functions in select groups. Nevertheless, these results can be utilized as a monitoring framework. Finally, based on multiple group analyses, females, non-white, and older adults were at higher risk for adverse physical health impacts following PTSD diagnoses.

Implications

The current study advances our understanding of the relationship between mental and physical health. Understanding the interconnection between the two should be used to encourage movement between research in physical and mental health services into less binary areas as the two are highly interconnected. Advancement in the science of PTSD can also be further used to design evidence-based prevention and intervention efforts to

minimize the adverse health outcomes from PTSD. Mental health screening is the first step needed to be taken to identify stressors, symptoms, risks or protective factors, and allostatic load. This information can then be utilized to develop a profile relevant to risks for mental disorders or physical health issues. Routine monitoring should be implemented for those with high-risk physiological profiles. The physiological profile can also be used to design a personalized treatment plan that can potentially have higher effectiveness, as it addresses the specific problems and needs of patients.

Limitations

The nature of the secondary data utilized for the current study contributes to several limitations. First, the current data focuses on objective physiological measures. Although it provides insights into physical health, it fails to capture the subjective components of trauma experience. Secondly, the data was collected from EHR, which means that data is only available when participants visited the CERNER-associated clinics. It inadequately encapsulates information in-between visits or when participants visit healthcare facilities outside the system. Additionally, there were relevant concerns about the consistency of the data collection process as it was done by different health professionals. Further, the identification for military status came from the type of insurance utilized to access healthcare. Unfortunately, it is impossible to identify those with active military status, veterans, or spouses due to HIPPA. Finally, psychological trauma was identified using ICD code for PTSD diagnosis. Limited information on healthcare professionals who provided diagnoses raised questions on the accuracy of the diagnoses.

Future directions

Aside from the current limitations, this study contributes to the advancement of knowledge relevant to the interplay between PTSD and physiological functions. Examinations of different physical functions (e.g., cortisol levels) and pathology (e.g., autoimmune disorder) may expand our understanding of mechanisms between PTSD and physical health. Future studies will also need to include primary data collection so researchers can identify the military status (e.g., active, veteran, spouse). This will allow us to understand the magnitude of the impacts of trauma for each group. Additionally, an assessment of subjective experiences, including the type of stressor participants are facing, and other psychosocial resources, will enrich our understanding of psychological trauma in the context of military families.

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APPENDICES

APPENDIX A

TABLES

Table 1

Direct effects for full model

Predictors	Outcomes	β	P-value
Systolic BP Pre	Leukocyte Pre	-0.006	0.936
Diastolic BP Pre		-0.106	0.128
Pulse Pre		0.293	0.004*
Systolic BP Pre	Platelet Pre	0.055	0.005*
Diastolic BP Pre		-0.005	0.796
Pulse Pre		0.028	0.220
Systolic BP Pre	N/L ratio Post	-0.761	0.079
Diastolic BP Pre		-0.343	0.026*
Pulse Pre		0.315	0.138
Leukocyte Pre	PTSD	0.228	0.007*
Platelet Pre		-0.338	0.279
N/L ratio Pre		-0.143	0.064
PTSD	Systolic BP Post	-0.034	0.000*
	Diastolic BP Post	0.008	0.115
	Pulse Post	0.029	0.000*
Systolic BP Post	Leukocyte Post	-0.086	0.035*
Diastolic BP Post		-0.056	0.103
Pulse BP Post		0.330	0.000*
PTSD		0.003	0.832
Systolic BP Post	Platelet Post	0.049	0.001*
Diastolic BP Post		0.008	0.413
Pulse Post		0.050	0.001*
PTSD		-0.002	0.578
Systolic BP Post	N/L ratio	-0.757	0.000*
Diastolic BP Post		0.0221	0.122
Pulse Post		0.656	0.000*
PTSD		-0.104	0.027*
Leukocyte Post	CVD	0.008	0.330
Platelet Post		-0.248	0.000*
N/L ratio Post		0.026	0.000*
PTSD		0.093	0.000*
Systolic BP Post		-0.001	0.964
Diastolic BP Post		-0.040	0.006*
Pulse Post		-0.059	0.006*
Leukocyte Post	Sleep disorder	0.002	0.842
Platelet Post		-0.077	0.030*

N/L ratio	0.008	0.150
PTSD	0.121	0.000*
Systolic BP Post	-0.030	0.076
Diastolic BP Post	0.025	0.118
Pulse Post	-0.033	0.054
CVD WITH SLEEP	0.009	0.000*

Table 2

Indirect effects for Full Model

	Predictors	Outcomes	β	p-value
Total indirect effect	Systolic BP post	CVD	-0.033	0.000*
Specific indirect	Systolic BP post-leukocyte post-CVD		-0.001	0.375
	Systolic BP post-platelet post-CVD		-0.012	0.002*
	Systolic BP post-N/L ratio post-CVD		-0.020	0.000*
Total indirect effect	Diastolic BP post	CVD	0.003	0.492
Specific indirect	Diastolic BP post-leukocyte post-CVD		0.000	0.396
	Diastolic BP post-platelet post-CVD		-0.002	0.426
	Diastolic BP post-N/L post-CVD		0.006	0.159
Total indirect effect	Pulse post	CVD	0.007	0.287
Specific indirect	Pulse post-Leukocyte post-CVD		0.003	0.332
	Pulse post- platelet post-CVD		-0.013	0.001*
	Pulse post-N/L post-CVD		0.017	0.000*
Total indirect effect	PTSD	CVD	-0.003	0.096
	PTSD-systolic BP post-CVD		0.000	0.241
	PTSD-diastolic BP post-CVD		0.000	0.964
	PTSD-pulse post-CVD		-0.002	0.047*
	PTSD-leukocyte post-CVD		0.000	0.839
	PTSD-platelet post-CVD		0.000	0.586
	PTSD-N/L post-CVD		-0.003	0.022*
	PTSD-systolic BP post-leukocyte post-CVD		0.000	0.460
	PTSD- diastolic BP post-leukocyte post-CVD		0.000	0.380
	PTSD-pulse post-leukocyte post-CVD		0.000	0.346
	PTSD-systolic BP post-platelet post-CVD		0.000	0.487
	PTSD-diastolic BP-platelet post-CVD		0.000	0.006*
	PTSD-pulse post-platelet post-CVD		0.000	0.006*
	PTSD-systolic BP post-N/L post-CVD		0.000	0.343
	PTSD-diastolic BP post-N/L post-CVD		0.001	0.002*
	PTSD-pulse post-N/L post-CVD		0.001	0.011*
Total indirect effect	Systolic BP post	Sleep disorder	-0.010	0.030*
Specific indirect	Systolic BP post-leukocyte post-sleep disorder		0.000	0.841
	Systolic BP post-platelet post-sleep disorder		-0.004	0.044*
	Systolic BP post-N/L post-sleep disorder		-0.006	0.132
Total effect	Diastolic BP post	Sleep disorder	0.001	0.506
Specific indirect	Diastolic BP post-leukocyte post-sleep disorder		0.000	0.843
	Diastolic BP post-platelet post-sleep disorder		-0.001	0.465
	Diastolic BP post-N/L post-sleep disorder		0.002	0.183
Total effect	Pulse post	Sleep disorder	0.002	0.732
Specific indirect	Pulse post-leukocyte post-sleep disorder		0.001	0.842
	Pulse post-platelet post-sleep disorder		-0.004	0.042*

	Pulse post-N/L post-sleep disorder		0.005	0.175
Total effect	PTSD	Sleep disorder	0.000	0.976
	PTSD-systolic BP-sleep disorder		0.000	0.373
	PTSD-diastolic BP-sleep disorder		0.001	0.084
	PTSD-pulse post-sleep disorder		-0.001	0.104
	PTSD-leukocyte post-sleep disorder		0.000	0.879
	PTSD-platelet post-sleep disorder		0.000	0.583
	PTSD-N/L post-sleep disorder		-0.001	0.186
	PTSD-systolic BP post-platelet post-sleep disorder		0.000	0.845
	PTSD-Diastolic BP post-leukocyte post-sleep disorder		0.000	0.841
	PTSD-pulse post-leukocyte post-sleep disorder		0.000	0.842
	PTSD-Systolic BP post-platelet post-sleep disorder		0.000	0.515
	PTSD-Diastolic BP post-platelet post-sleep disorder		0.000	0.055
	PTSD-pulse post-platelet post-sleep disorder		0.000	0.068
	PTSD-systolic BP post-N/L post-sleep disorder		0.000	0.351
	PTSD-diastolic BP post-N/L post-sleep disorder		0.000	0.145
	PTSD-pulse post-N/L post-sleep disorder		0.000	0.202
Total indirect effect	PTSD	Leukocyte post	0.012	0.000*
	PTSD-systolic BP post-leukocyte post		0.000	0.277
	PTSD-diastolic BP post-leukocyte post		0.003	0.047*
	PTSD-pulse post-leukocyte post		0.010	0.003*
Total indirect effect	PTSD	N/L post	0.047	0.000*
	PTSD-systolic blood pressure-N/L post		0.002	0.326
	PTSD-diastolic blood pressure-N/L post		0.026	0.000*
	PTSD-pulse post-N/L post		0.019	0.003*
Total indirect	PTSD	Platelet post	0.000	0.853
	PTSD-Systolic BP-Platelet post		0.000	0.479
	PTSD-Diastolic BP-platelet post		-0.002	0.003*
	PTSD-pulse post-platelet post		0.001	0.009*

Table 3

Direct effects for male study participants

Predictors	Outcomes	β	P-value
Systolic BP pre	Leukocyte Pre	0.220	0.082
Diastolic BP pre		-0.148	0.126
Pulse Pre		0.509	0.001*
Systolic BP pre	Platelet Pre	0.074	0.003*
Diastolic BP pre		0.003	0.889
Pulse Pre		0.046	0.019*
Systolic BP pre	N/L ratio Pre	-0.122	0.556
Diastolic BP pre		-0.096	0.644
Pulse Pre		0.506	0.010*
Leukocyte Pre	PTSD	0.042	0.658
Platelet Pre		0.592	0.064
N/L ratio Pre		0.148	0.396
PTSD	Systolic BP Post	-0.044	0.000*
PTSD	Diastolic BP Post	-0.006	0.555
PTSD	Pulse Post	0.060	0.000*
Systolic BP post	Leukocyte Post	-0.091	0.060
Diastolic BP post		-0.077	0.121
Pulse post		0.207	0.000*
PTSD		0.005	0.180
Systolic BP post	Platelet Post	0.057	0.000*
Diastolic BP post		0.006	0.544
Pulse post		0.016	0.170
PTSD		0.005	0.180
Systolic BP post	N/L ratio	-0.998	0.000*
Diastolic BP post		0.253	0.242
Pulse post		0.875	0.002*
PTSD		-0.211	0.026*
Leukocyte post	CVD	-0.011	0.728
Platelet post		-0.468	0.000*
N/L ratio post		0.146	0.000*
PTSD		0.109	0.000*
Systolic BP post		-0.019	0.785
Diastolic BP post		-0.154	0.005*
Pulse post		-0.269	0.003*
Leukocyte post	Sleep disorder	-0.014	0.603
Platelet post		-0.116	0.375
N/L ratio post		0.043	0.060
PTSD		0.133	0.000*
Systolic BP post		-0.044	0.385
Diastolic BP Post		-0.022	0.509
Pulse post		-0.130	0.068
CVD WITH Sleep		0.007	0.000*

Table 4

Indirect effects for male study participants

	Predictor	Outcome	β	p-value
Total indirect effect	Systolic BP	CVD	-0.171	0.000*
Specific effect	Systolic BP post-leukocyte post-CVD		0.001	0.732
	Systolic BP post-platelet post-CVD		-0.027	0.002*
	Systolic BP post-N/L post-CVD		-0.146	0.000*
Total indirect effect	Diastolic BP post	CVD	0.035	0.257
Specific effect	Diastolic BP post-leukocyte post-CVD		0.001	0.735
	Diastolic BP post-platelet post-CVD		-0.003	0.547
	Diastolic BP post-N/L post-CVD		0.037	0.217
Total indirect effect	Pulse post	CVD	0.118	0.009*
Specific effect	Pulse post-leukocyte post-CVD		-0.002	0.728
	Pulse post-platelet post-CVD		-0.008	0.146
	Pulse post-N/L post-CVD		0.128	0.003*
Total indirect effect	PTSD	CVD	-0.034	0.022*
Specific effect	PTSD-systolic BP post-CVD		0.001	0.538
	PTSD-diastolic BP post-CVD		0.001	0.786
	PTSD-pulse post-CVD		-0.016	0.008*
	PTSD-leukocyte post-CVD		0.000	0.732
	PTSD-platelet post-CVD		-0.003	0.219
	PTSD-N/L post-CVD		-0.031	0.018*
	PTSD-systolic BP post-leukocyte post-CVD		0.000	0.769
	PTSD-diastolic BP post-leukocyte post-CVD		0.000	0.733
	PTSD-pulse post-leukocyte post-CVD		0.000	0.728
	PTSD-systolic BP post-platelet post-CVD		0.000	0.662
	PTSD-diastolic BP post-platelet post-CVD		0.001	0.008*
	PTSD-pulse post-platelet post-CVD		0.000	0.157
	PTSD-systolic BP post_N/L post-CVD		0.000	0.596
	PTSD-diastolic BP post-N/L post-CVD		0.006	0.001*
	PTSD-pulse post-N/L post-CVD		0.008	0.009*
Total indirect effect	Systolic BP post	Sleep disorder	-0.048	0.023*
Specific effect	Systolic BP post-leukocyte post-sleep disorder		-0.042	0.051
	Systolic BP post-platelet post-sleep disorder		0.001	0.624
	Systolic BP post-N/L post-sleep disorder		-0.007	0.368
Total indirect effect	Diastolic BP post	Sleep disorder	0.011	0.223
Specific effect	Diastolic BP post-leukocyte post-sleep disorder		0.001	0.621
	Diastolic BP post-platelet post-sleep disorder		-0.001	0.623
	Diastolic BP post-N/L post-sleep disorder		0.011	0.206
Total indirect effect	Pulse post	Sleep disorder	0.033	0.171
Specific effect	Pulse post-leukocyte post-sleep disorder		-0.003	0.605

	Pulse post-platelet post-sleep disorder		-0.002	0.416
	Pulse post-N/L post-sleep disorder		0.037	0.119
Total indirect effect	PTSD	Sleep disorder	-0.012	0.120
Specific effect	PTSD-systolic BP post-sleep disorder		0.000	0.660
	PTSD-diastolic BP post-sleep disorder		0.002	0.389
	PTSD-pulse post-sleep disorder		-0.008	0.072
	PTSD-leukocyte post-sleep disorder		0.000	0.620
	PTSD-platelet post-sleep disorder		-0.001	0.484
	PTSD-N/L post-sleep disorder		-0.009	0.122
	PTSD-systolic BP post_leukocyte post-sleep disorder		0.000	0.707
	PTSD-diastolic BP post_leukocyte post-sleep disorder		0.000	0.625
	PTSD-pulse post-leukocyte post-sleep disorder		0.000	0.606
	PTSD-systolic BP post-platelet post-sleep disorder		0.000	0.697
	PTSD-diastolic BP post-platelet post-sleep disorder		0.000	0.375
	PTSD- pulse post-platelet post-sleep disorder		0.000	0.420
	PTSD-systolic BP post-N/L post-sleep disorder		0.000	0.596
	PTSD-diastolic BP post-N/L post-sleep disorder		0.002	0.066
	PTSD-pulse post-N/L post-sleep disorder		0.002	0.133
Total indirect effect	PTSD	Leukocyte post	0.017	0.000*
Specific effect	PTSD-systolic BP post-leukocyte post		0.000	0.585
	PTSD-diastolic BP post-leukocyte post		0.004	0.076
	PTSD-pulse post-leukocyte post		0.012	0.003*
Total indirect effect	PTSD	N/L post	0.094	0.000*
Specific effect	PTSD-systolic BP post-N/L post		-0.002	0.600
	PTSD-diastolic BP post-N/L post		0.044	0.001*
	PTSD-pulse post-N/L post		0.052	0.006*
Total indirect effect	PTSD	Platelet post	-0.002	0.113
Specific effect	PTSD-systolic BP post-platelet post		0.000	0.660
	PTSD-diastolic BP post-platelet post		-0.002	0.001*
	PTSD-pulse post-platelet post		0.001	0.181

Table 5

Direct effects for female study participants

Predictors	Outcomes	β	p-value
Systolic BP pre	Leukocyte Pre	-0.159	0.077
Diastolic BP pre		-0.103	0.282
Pulse Pre		0.223	0.046*
Systolic BP pre	Platelet Pre	0.033	0.226
Diastolic BP pre		0.025	0.399
Pulse Pre		0.004	0.894
Systolic BP pre	N/L ratio Pre	-1.154	0.130
Diastolic BP pre		-0.318	0.181
Pulse Pre		0.468	0.242
Leukocyte Pre	PTSD	-0.233	0.173
Platelet Pre		0.810	0.154
N/L ratio Pre		0.122	0.058
PTSD	Systolic BP Post	-0.052	0.000*
PTSD	Diastolic BP Post	-0.004	0.652
PTSD	Pulse Post	0.020	0.105
Systolic BP post	Leukocyte Post	-0.106	0.096
Diastolic BP post		-0.029	0.510
Pulse post		0.524	0.001*
PTSD		-0.029	0.159
Systolic BP post	Platelet Post	0.045	0.152
Diastolic BP post		0.021	0.324
Pulse post		0.101	0.008*
PTSD		0.014	0.003*
Systolic BP post	N/L ratio	-0.428	0.043*
Diastolic BP post		0.063	0.590
Pulse post		0.468	0.002*
PTSD		-0.145	0.008*
Leukocyte post	CVD	-0.001	0.932
Platelet post		-0.060	0.377
N/L ratio post		0.002	0.541
PTSD		0.127	0.000*
Systolic BP post		0.040	0.116
Diastolic BP post		-0.007	0.688
Pulse post		-0.020	0.201
Leukocyte post	Sleep disorder	0.005	0.607
Platelet post		-0.066	0.149
N/L ratio post		0.009	0.107
PTSD		0.127	0.000*
Systolic BP post		-0.035	0.167
Diastolic BP post		0.091	0.000*
Pulse post		-0.026	0.164
CVD WITH Sleep		0.019	0.000*

Table 6

Indirect effects for female study participants

	Predictor	Outcome	β	P-value
Total indirect effect	Systolic BP post	CVD	-0.003	0.400
Specific indirect	Systolic BP post-leukocyte post-CVD		-0.001	0.551
	Systolic BP post-platelet post-CVD		0.000	0.932
	Systolic BP post-N/L post-CVD		-0.003	0.431
Total indirect effect	Diastolic BP post	CVD	-0.001	0.569
Specific indirect	Diastolic BP post-leukocyte post-CVD		0.000	0.934
	Diastolic BP post-platelet post-CVD		-0.001	0.509
	Diastolic BP post-N/L post-CVD		0.000	0.737
Total indirect effect	Pulse	CVD	-0.006	0.464
Specific indirect	Pulse-leukocyte post-CVD		0.000	0.932
	Pulse post-platelet post-CVD		-0.006	0.295
	Pulse post-N/L post-CVD		0.001	0.548
Total indirect effect	PTSD	CVD	-0.003	0.048*
Specific indirect	PTSD-systolic BP post-CVD		0.000	0.757
	PTSD-diastolic BP post-CVD		-0.002	0.122
	PTSD-pulse post-CVD		0.000	0.387
	PTSD-leukocyte post-CVD		0.000	0.933
	PTSD-platelet post-CVD		-0.001	0.365
	PTSD-N/L post-CVD		0.000	0.544
	PTSD-systolic BP post-leukocyte post-CVD		0.000	0.935
	PTSD-diastolic BP post-leukocyte post-CVD		0.000	0.932
	PTSD-pulse post-leukocyte post-CVD		0.000	0.932
	PTSD-systolic BP post-platelet post-CVD		0.000	0.738
	PTSD-diastolic BP post-platelet post-CVD		0.000	0.435
	PTSD-pulse post-platelet post-CVD		0.000	0.360
	PTSD-systolic BP post-N/L post-CVD		0.000	0.786
	PTSD-diastolic BP post-N/L post-CVD		0.000	0.552
	PTSD-pulse post-N/L post-CVD		0.000	0.576
Total indirect effect	Systolic BP post	Sleep disorder	-0.007	0.064
Specific indirect	Systolic BP post-leukocyte post-sleep disorder		-0.001	0.613
	Systolic BP post-platelet post-sleep disorder		-0.003	0.234
	Systolic BP post-N/L post-sleep disorder		-0.004	0.143
Total indirect effect	Diastolic BP post	Sleep disorder	-0.001	0.641
Specific indirect	Diastolic BP post-leukocyte post-sleep disorder		0.000	0.678
	Diastolic BP post-platelet post-sleep disorder		0.001	0.601
	Diastolic BP post-N/L post-CVD		-0.001	0.458
Total indirect effect	Pulse post	Sleep disorder	0.000	0.987
Specific indirect	Pulse post-leukocyte post-sleep disorder		0.003	0.617
	Pulse post-platelet post-sleep disorder		-0.007	0.167
	Pulse post-N/L post-sleep disorder		0.004	0.153
Total indirect	PTSD	Sleep	-0.001	0.603

effect		disorder		
Specific indirect	PTSD-systolic BP post-sleep disorder		0.000	0.643
	PTSD-diastolic bp post-sleep disorder		0.002	0.177
	PTSD-pulse post-sleep disorder		-0.001	0.337
	PTSD-leukocyte post-sleep disorder		0.000	0.645
	PTSD-platelet post-sleep disorder		-0.001	0.205
	PTSD-N/L post-sleep disorder		-0.001	0.085
	PTSD-systolic BP post-leukocyte post-sleep disorder		0.000	0.765
	PTSD-diastolic BP post-leukocyte post-sleep disorder		0.000	0.614
	PTSD-pulse post-leukocyte post-sleep disorder		0.000	0.633
	PTSD-systolic Bp post-platelet post-sleep disorder		0.000	0.731
	PTSD-diastolic BP post-platelet post-sleep disorder		0.000	0.244
	PTSD-pulse post-platelet post-sleep disorder		0.000	0.298
	PTSD-systolic BP post-N/L post-sleep disorder		0.000	0.729
	PTSD-diastolic BP post-N/L post-sleep disorder		0.000	0.154
	PTSD-pulse post-N/L post-sleep disorder		0.000	0.297
Total indirect effect	PTSD	Leukocyte post	0.016	0.029*
Specific indirect	PTSD-systolic BP post-leukocyte post		0.000	0.724
	PTSD-diastolic BP post-leukocyte post		0.006	0.105
	PTSD-pulse post-leukocyte post		0.011	0.144
Total indirect effect	PTSD	N/L post	0.032	0.030*
Specific indirect	PTSD-systolic BP post-N/L post		0.000	0.725
	PTSD-diastolic BP post-N/L post		0.022	0.051
	PTSD-pulse post-N/L post		0.010	0.157
Total indirect effect	PTSD	Platelet post	0.000	0.853
Specific indirect	PTSD-systolic BP post-platelet post		0.000	0.718
	PTSD-diastolic BP post-platelet post		-0.002	0.162
	PTSD-pulse post-platelet post		0.002	0.156

Table 7

Direct effects for white study participants

Predictors	Outcomes	β	p-value
Systolic BP Pre	Leukocyte Pre	0.046	0.628
Diastolic BP Pre		-0.140	0.085
Pulse BP Pre		0.345	0.006*
Systolic BP Pre	Platelet Pre	0.051	0.018*
Diastolic BP Pre		-0.014	0.467
Pulse BP Pre		0.027	0.226
Systolic BP Pre	N/L ratio Pre	-0.873	0.123
Diastolic BP Pre		-0.401	0.050
Pulse BP Pre		0.376	0.214
Leukocyte Pre	PTSD	0.222	0.025*
Platelet Pre		-0.548	0.245
N/L ratio Pre		-0.135	0.147
PTSD	Systolic BP Post	-0.039	0.000*
PTSD	Diastolic BP Post	0.010	0.109
PTSD	Pulse Post	0.028	0.004*
Systolic BP post	Leukocyte Post	-0.082	0.062
Diastolic BP post		-0.069	0.068
Pulse post		0.307	0.001*
PTSD		0.009	0.600
Systolic BP post	Platelet post	0.044	0.002*
Diastolic BP post		0.017	0.087
Pulse post		0.040	0.006*
PTSD		-0.003	0.351
Systolic BP post	N/L ratio post	-0.725	0.000*
Diastolic BP post		0.122	0.442
Pulse post		0.659	0.001*
PTSD		-0.117	0.050
Leukocyte post	CVD	0.015	0.297
Platelet post		-0.465	0.000*
N/L ratio post		0.041	0.005*
PTSD		0.088	0.000*
Systolic BP post		-0.017	0.621
Diastolic BP post		-0.079	0.005*
Pulse post		-0.090	0.015*
Leukocyte post	Sleep disorder	-0.001	0.963
Platelet post		-0.141	0.020*
N/L ratio post		0.016	0.112
PTSD		0.125	0.000*
Systolic BP post		-0.052	0.071
Diastolic BP post		0.040	0.177
Pulse post		-0.030	0.244
CVD WITH Sleep		0.011	0.000*

Table 8

Indirect effects for white study participants

	Predictor	Outcome	β	P-value
Total indirect effect	Systolic BP post	CVD	-0.051	0.000*
Specific effect	Systolic BP post-leukocyte post-CVD		-0.001	0.362
	Systolic BP post-platelet post-CVD		-0.020	0.006*
	Systolic BP post-N/L post-CVD		-0.029	0.008*
Total indirect effect	Diastolic BP post	CVD	-0.004	0.649
Specific effect	Diastolic BP post-leukocyte post-CVD		-0.001	0.352
	Diastolic BP post-platelet post-CVD		-0.008	0.088
	Diastolic BP post-N/L post-CVD		0.005	0.466
Total indirect effect	Pulse post	CVD	0.013	0.362
Specific effect	Pulse post-leukocyte post-CVD		0.005	0.311
	Pulse post-platelet post-CVD		-0.019	0.006*
	Pulse post-N/L post-CVD		0.027	0.014*
Total indirect effect	PTSD	CVD	-0.004	0.326
Specific effect	PTSD-systolic BP post-CVD		-0.001	0.139
	PTSD-diastolic BP post-CVD		0.001	0.622
	PTSD-pulse-CVD		-0.003	0.096
	PTSD-leukocyte post-CVD		0.000	0.650
	PTSD-platelet post-CVD		0.001	0.352
	PTSD-N/L post-CVD		-0.005	0.063
	PTSD-systolic BP post-leukocyte post-CVD		0.000	0.401
	PTSD-diastolic BP post-leukocyte post-CVD		0.000	0.367
	PTSD-pulse post-leukocyte post-CVD		0.000	0.332
	PTSD-systolic BP post-platelet post-CVD		0.00	0.202
	PTSD-diastolic BP post-platelet post-CVD		0.001	0.011*
	PTSD-pulse post-platelet post-CVD		-0.001	0.031*
	PTSD-systolic BP post-N/L post-CVD		0.000	0.511
	PTSD-diastolic BP post-N/L post-CVD		0.001	0.015*
	PTSD-pulse post-N/L post-CVD		0.001	0.056
Total indirect effect	Systolic BP post	Sleep disorder	-0.017	0.022*
Specific effect	Systolic BP-leukocyte post-sleep disorder		0.000	0.963
	Systolic Bp post-platelet post-sleep disorder		-0.006	0.046*
	Systolic BP post-N/L post-sleep disorder		-0.011	0.096
Total indirect effect	Diastolic BP post	Sleep disorder	0.000	0.898
Specific effect	Diastolic BP post-leukocyte post-sleep disorder		0.000	0.963
	Diastolic BP post-platelet post-sleep disorder		-0.002	0.179
	Diastolic BP post-N/L post-sleep disorder		0.002	0.429
Total indirect effect	Pulse post	Sleep disorder	0.004	0.606
Specific effect	Pulse post-leukocyte post-sleep disorder		0.000	0.963
	Pulse post-platelet post-sleep disorder		-0.006	0.056
	Pulse post-N/L post-sleep disorder		0.010	0.142

Total indirect effect	PTSD	Sleep disorder	0.000	0.621
Specific effect	PTSD-systolic BP post-Sleep disorder		0.000	0.347
	PTSD-diastolic BP post-Sleep disorder		0.002	0.078
	PTSD-pulse-Sleep disorder		-0.001	0.285
	PTSD-leukocyte post-Sleep disorder		0.000	0.964
	PTSD-platelet post-Sleep disorder		0.000	0.372
	PTSD-N/L post-Sleep disorder		-0.002	0.123
	PTSD-systolic BP post-leukocyte post-Sleep disorder		0.000	0.963
	PTSD-diastolic BP post-leukocyte post-Sleep disorder		0.000	0.963
	PTSD-pulse post-leukocyte post-Sleep disorder		0.000	0.963
	PTSD-systolic BP post-platelet post-Sleep disorder		0.000	0.272
	PTSD-diastolic BP post-platelet post-Sleep disorder		0.000	0.058
	PTSD-pulse post-platelet post-Sleep disorder		0.000	0.096
	PTSD-systolic BP post-N/L post-Sleep disorder		0.000	0.482
	PTSD-diastolic BP post-N/L post-Sleep disorder		0.000	0.110
	PTSD-pulse post-N/L post-Sleep disorder		0.000	0.188
Total indirect effect	PTSD	Leukocyte post	0.011	0.006*
Specific effect	PTSD-systolic BP post-leukocyte		-0.001	0.188
	PTSD-diastolic BP post-leukocyte post		0.003	0.075
	PTSD- pulse post-leukocyte post		0.009	0.020*
Total indirect effect	PTSD	N/L post	0.105	0.383
Specific effect	PTSD-systolic BP post-N/L post		0.099	0.261
	PTSD-diastolic BP post-N/L post		0.024	0.689
	PTSD-pulse post-N/L post		-0.018	0.761
Total indirect effect	PTSD	Platelet post	0.000	0.638
Specific effect	PTSD-systolic BP post-platelet post		0.000	0.203
	PTSD-diastolic BP post-platelet post		-0.002	0.005*
	PTSD-pulse post-platelet post		0.001	0.031*

Table 9

Direct effects for non-white study participants

Predictors	Outcomes	β	p-value
Systolic BP Pre	Leukocyte Pre	-0.145	0.301
Diastolic BP Pre		0.060	0.674
Pulse BP Pre		0.279	0.076
Systolic BP Pre	Platelet Pre	0.086	0.119
Diastolic BP Pre		0.048	0.269
Pulse BP Pre		0.056	0.352
Systolic BP Pre	N/L ratio Pre	-0.436	0.012*
Diastolic BP Pre		-0.080	0.597
Pulse BP Pre		0.362	0.048*
Leukocyte Pre	PTSD	0.379	0.000*
Platelet Pre		-0.172	0.539
N/L ratio Pre		-0.214	0.003*
PTSD	Systolic BP Post	-0.029	0.017*
PTSD	Diastolic BP Post	-0.008	0.677
PTSD	Pulse Post	0.028	0.075
Systolic BP post	Leukocyte Post	-0.100	0.380
Diastolic BP post		-0.001	0.993
Pulse post		0.413	0.000*
PTSD		-0.040	0.126
Systolic BP post	Platelet post	0.097	0.233
Diastolic BP post		-0.076	0.343
Pulse post		0.139	0.074
PTSD		0.003	0.608
Systolic BP post	N/L ratio post	-1.015	0.023*
Diastolic BP post		0.740	0.046*
Pulse post		0.510	0.001*
PTSD		-0.080	0.281
Leukocyte post	CVD	-0.015	0.324
Platelet post		-0.014	0.911
N/L ratio post		0.015	0.191
PTSD		0.065	0.000*
Systolic BP post		0.079	0.067
Diastolic BP post		0.010	0.741
Pulse post		-0.043	0.095
Leukocyte post	Sleep disorder	0.022	0.331
Platelet post		0.040	0.705
N/L ratio post		-0.008	0.214
PTSD		0.130	0.000*
Systolic BP post		-0.045	0.397
Diastolic BP post		0.058	0.124
Pulse post		-0.117	0.025*
CVD WITH Sleep		0.012	0.002*

Table 10

Indirect effects for non-white study participants

	Predictor	Outcome	β	P-value
Total indirect effect	Systolic BP post	CVD	-0.015	0.461
Specific indirect	Systolic BP post-leukocyte post-CVD		0.001	0.533
	Systolic BP post-platelet post-CVD		-0.001	0.905
	Systolic BP post-N/L post-CVD		-0.015	0.366
Total indirect effect	Diastolic BP post	CVD	0.012	0.438
Specific indirect	Diastolic BP post-leukocyte post-CVD		0.000	0.993
	Diastolic BP post-platelet post-CVD		0.001	0.903
	Diastolic BP post-N/L post-CVD		0.011	0.395
Total indirect effect	Pulse post	CVD	0.000	0.982
Specific indirect	Pulse post-leukocyte post-CVD		-0.006	0.325
	Pulse-platelet post-CVD		-0.002	0.907
	Pulse post-N/L post-CVD		0.008	0.308
Total indirect effect	PTSD	CVD	-0.004	0.089
Specific indirect	PTSD-systolic BP post-CVD		0.000	0.739
	PTSD-diastolic BP post-CVD		-0.002	0.138
	PTSD-pulse post-CVD		-0.001	0.241
	PTSD-leukocyte post-CVD		0.001	0.400
	PTSD-platelet post-CVD		0.000	0.913
	PTSD-N/L post-CVD		-0.001	0.255
	PTSD-systolic BP post-leukocyte post-CVD		0.000	0.993
	PTSD-diastolic BP post-leukocyte post-CVD		0.000	0.555
	PTSD-pulse post-leukocyte post-CVD		0.000	0.386
	PTSD-systolic BP post-platelet post-CVD		0.000	0.905
	PTSD-diastolic BP post-platelet post-CVD		0.000	0.906
	PTSD-pulse post-platelet post-CVD		0.000	0.906
	PTSD-systolic BP post-N/L post-CVD		0.000	0.706
	PTSD-diastolic post-N/L post-CVD		0.000	0.397
	PTSD-pulse post-N/L post-CVD		0.000	0.381
Total indirect effect	Systolic BP post	Sleep disorder	0.010	0.517
Specific indirect	Systolic BP post-leukocyte post-sleep disorder		-0.002	0.518
	Systolic BP post-platelet-sleep disorder		0.004	0.749
	Systolic BP post-N/L post-sleep disorder		0.008	0.354
Total indirect effect	Diastolic BP post	Sleep disorder	-0.009	0.479
Specific indirect	Diastolic BP post-leukocyte post-sleep disorder		0.000	0.993
	Diastolic BP post-platelet post-sleep disorder		-0.003	0.761
	Diastolic BP post-N/L post-sleep disorder		-0.006	0.387
Total indirect effect	Pulse post	Sleep disorder	0.010	0.638
Specific indirect	Pulse post-leukocyte post-sleep disorder		0.009	0.356
	Pulse post-platelet post-sleep disorder		0.006	0.738
	Pulse post-N/L post-sleep disorder		-0.004	0.277
Total indirect effect	PTSD	Sleep disorder	-0.002	0.469
Specific indirect	PTSD-systolic BP post-sleep disorder		0.000	0.647
	PTSD-diastolic BP post-sleep disorder		0.001	0.436
	PTSD-pulse post-sleep disorder		-0.003	0.175
	PTSD-leukocyte post-sleep disorder		-0.001	0.429
	PTSD-platelet post-sleep disorder		0.000	0.753

	PTSD-N/L post-sleep disorder		0.001	0.295
	PTSD-systolic BP post-leukocyte post-sleep disorder		0.000	0.993
	PTSD-diastolic BP post-leukocyte post-sleep disorder		0.000	0.535
	PTSD-pulse post-leukocyte post-sleep disorder		0.000	0.424
	PTSD-systolic BP post-platelet post-sleep disorder		0.000	0.816
	PTSD-diastolic BP post-platelet post-sleep disorder		0.000	0.748
	PTSD-pulse post-platelet post-sleep disorder		0.000	0.747
	PTSD-systolic BP post-N/L post-sleep disorder		0.000	0.704
	PTSD-diastolic post-N/L post-sleep disorder		0.000	0.393
	PTSD-pulse post-N/L post-sleep disorder		0.000	0.353
Total indirect effect	PTSD	Leukocyte post	0.014	0.047*
Specific indirect	PTSD-systolic BP post-leukocyte post		0.000	0.993
	PTSD-diastolic BP post-leukocyte count		0.003	0.420
	PTSD-pulse post-leukocyte post		0.011	0.106
Total indirect effect	PTSD	N/L post	0.038	0.089
Specific indirect	PTSD-systolic BP post-N/L post		-0.006	0.683
	PTSD-diastolic BP post-N/L post		0.029	0.098
	PTSD-pulse post-N/L post		0.014	0.117
Total indirect effect	PTSD	Platelet post	0.002	0.606
Specific indirect	PTSD-systolic BP post-platelet post		0.001	0.720
	PTSD-diastolic BP post-platelet post		-0.003	0.265
	PTSD-pulse post-platelet post		0.004	0.242

Table 11

Direct effects for age group 0-17 (children)

Predictor	Outcome	β	p-value
Systolic BP pre	Leukocyte pre	-1.155	0.000*
Diastolic BP pre		0.412	0.248
Pulse BP pre		-0.477	0.088
Systolic BP pre	Platelet pre	0.007	0.945
Diastolic BP pre		0.139	0.316
Pulse BP pre		-0.114	0.015*
Systolic BP pre	N/L pre	-0.208	0.388
Diastolic BP pre		0.380	0.283
Pulse BP pre		0.052	0.750
Leukocyte pre	PTSD	-0.035	0.213
Platelet pre		0.257	0.071
N/L pre		-0.011	0.485
PTSD	Systolic BP pre	-0.040	0.001*
	Diastolic BP pre	0.030	0.002*
	Pulse BP pre	-0.171	0.000*
Systolic BP post	Leukocyte post	-0.390	0.055
Diastolic BP post		0.103	0.700
Pulse BP post		0.277	0.137
PTSD		-0.041	0.329
Systolic BP post	Platelet post	-0.079	0.209
Diastolic BP post		0.070	0.168
Pulse BP post		0.045	0.225
PTSD		-0.010	0.316
Systolic BP post	N/L post	-0.035	0.678
Diastolic BP post		-0.044	0.699
Pulse BP post		0.172	0.100
PTSD		0.005	0.933
Leukocyte post	CVD	0.000	0.451
Platelet post		0.000	0.515
N/L post		0.000	0.353
PTSD		0.000	0.328
Systolic BP post		0.000	0.510
Diastolic BP post		-0.001	0.328
Pulse post		0.000	0.506
Leukocyte post	Sleep disorder	0.004	0.284
Platelet post		0.012	0.407
N/L post		-0.001	0.060
PTSD		0.042	0.000*
Systolic BP post		0.008	0.117
Diastolic BP post		-0.001	0.816
Pulse post		-0.005	0.368
CVD WITH Sleep disorder		0.032	0.000*

Table 12

Indirect effects for age group 0-17 years (children)

	Predictor	Outcome	β	p-value
Total indirect effect	Systolic BP post	CVD	0.000	0.478
Specific indirect effect	Systolic Bp post-leukocyte post-CVD		0.000	0.714
	Systolic BP post-Plat post-CVD		0.000	0.497
	Systolic BP post-N/L-CVD		0.000	0.584
Total indirect effects	Diastolic BP post	CVD	0.000	0.593
Specific indirect effect	Diastolic BP post-Leuko post-CVD		0.000	0.732
	Diastolic BP post- Plat post-CVD		0.000	0.744
	Diastolic BP Pot-N/L- CVD		0.000	0.566
Total indirect effect	Pulse post	CVD	0.000	0.502
Specific indirect effect	Pulse post-leukocyte post-CVD		0.000	0.514
	Pulse post-platelet post-CVD		0.000	0.563
	Pulse post-N/L post-CVD		0.000	0.454
Total indirect effect	PTSD	CVD	0.000	0.467
Specific indirect effect	PTSD-sys BP post-CVD		0.000	0.347
	PTSD-diastolic BP post-CVD		0.000	0.493
	PTSD-pulse post-CVD		0.000	0.505
	PTSD-leukocyte post-CVD		0.000	0.555
	PTSD-platelet post-PTSD		0.000	0.598
	PTSD-N/L post-PTSD		0.000	0.935
	PTSD-systolic BP post-leuko post-CVD		0.000	0.743
	PTSD-diastolic BP post-leuko post-CVD		0.000	0.504
	PTSD-pulse post-leuko post-CVD		0.000	0.514
	PTSD-systolic BP post-plat post-CVD		0.000	0.572
	PTSD-diastolic BP post-plat post-CVD		0.000	0.586
	PTSD-pulse post-plat post-CVD		0.000	0.564
	PTSD-systolic BP post-N/L post-CVD		0.000	0.731
	PTSD-diastolic BP post-N/L post-CVD		0.000	0.717
	PTSD-pulse post-N/L post-CVD		0.000	0.453
Total indirect effect	Systolic BP post	Sleep disorder	-0.003	0.349
Specific indirect effect	Systolic BP post-leuko post-sleep disorder		-0.002	0.378
	Systolic BP post-plat post-sleep disorder		-0.001	0.520
	Ssytolic BP post-N/L post-sleep disorder		0.000	0.696
Total indirect effect	Diastolic BP post	Sleep disorder	0.001	0.538
	Diastolic BP post-leuko post-sleep disorder		0.000	0.731
	Diastolic BP post-plat post-sleep disorder		0.001	0.494
	Diastolic BP post-N/L post-sleep		0.000	0.719

	disorder			
Total indirect effect	Pulse post	Sleep disorder	0.002	0.391
Specific indirect effect	Pulse post-leuko post-sleep disorder		0.001	0.408
	Pulse post-plat post-sleep disorder		0.001	0.489
	Pulse post-N/L post-sleep disorder		0.000	0.294
Total indirect effect	PTSD	Sleep disorder	0.001	0.567
Specific indirect effect	PTSD-sys BP post-sleep disorder		0.000	0.818
	PTSD-diastolic BP post-sleep disorder		0.000	0.131
	PTSD-pulse post-sleep disorder		0.001	0.367
	PTSD-leukocyte post-sleep disorder		0.000	0.478
	PTSD-platelet post-sleep disorder		0.000	0.543
	PTSD-N/L post-sleep disorder		0.000	0.934
	PTSD-systolic BP post-leuko post-sleep disorder		0.000	0.731
	PTSD-diastolic BP post-leuko post-sleep disorder		0.000	0.391
	PTSD-pulse post-leuko post-sleep disorder		0.000	0.408
	PTSD-systolic BP post-plat post-sleep disorder		0.000	0.502
	PTSD-diastolic BP post-plat post-sleep disorder		0.000	0.524
	PTSD-pulse post-plat post-sleep disorder		0.000	0.490
	PTSD-systolic BP post-N/L post-sleep disorder		0.000	0.717
	PTSD-diastolic BP post-N/L post-sleep disorder		0.000	0.700
	PTSD-pulse post-N/L post-sleep disorder		0.000	0.292
Total indirect effect	PTSD	Leukocyte post	-0.060	0.053
Specific indirect effect	PTSD-systolic BP post-leukocyte post		0.003	0.700
	PTSD-diastolic BP post-leukocyte post		-0.016	0.092
	PTSD-pulse post-leukocyte post		-0.047	0.139
Total indirect effect	PTSD	Platelet post	-0.009	0.139
	PTSD-systolic BP post-platelet post		0.002	0.197
	PTSD-diastolic BP post-platelet post		-0.003	0.230
	PTSD-pulse post-platelet post		-0.008	0.229
Total indirect effect	PTSD	N/L post	-0.032	0.090
Specific indirect effect	PTSD-systolic BP post-N/L post		-0.001	0.698
	PTSD-diastolic BP post-N/L post		-0.001	0.682
	PTSD-pulse post-N/L post		-0.029	0.098

Table 13

Direct effects for age group 18-49 years (adult)

Predictor	Outcome	β	p-value
Systolic BP pre	Leukocyte pre	-0.069	0.507
Diastolic BP pre		-0.091	0.436
Pulse BP pre		0.310	0.004*
Systolic BP pre	Platelet pre	0.066	0.005*
Diastolic BP pre		0.007	0.797
Pulse BP pre		-0.007	0.758
Systolic BP pre	N/L pre	-1.502	0.187
Diastolic BP pre		-0.550	0.015*
Pulse BP pre		0.791	0.077
Leukocyte pre	PTSD	0.487	0.000*
Platelet pre		-1.375	0.096
N/L pre		-0.396	0.009*
PTSD	Systolic BP pre	-0.011	0.091
	Diastolic BP pre	0.012	0.132
	Pulse BP pre	0.026	0.025*
Systolic BP post	Leukocyte post	-0.027	0.632
Diastolic BP post		-0.072	0.187
Pulse BP post		0.236	0.000*
PTSD		-0.024	0.138
Systolic BP post	Platelet post	0.034	0.043*
Diastolic BP post		0.007	0.649
Pulse BP post		0.029	0.006*
PTSD		0.002	0.649
Systolic BP post	N/L post	-0.249	0.017*
Diastolic BP post		0.007	0.161
Pulse BP post		0.223	0.002*
PTSD		-0.043	0.238
Leukocyte post	CVD	-0.005	0.237
Platelet post		-0.015	0.416
N/L post		-0.001	0.257
PTSD		0.029	0.000*
Systolic BP post		-0.011	0.281
Diastolic BP post		-0.002	0.790
Pulse post		0.006	0.364
Leukocyte post	Sleep disorder	-0.010	0.197
Platelet post		0.014	0.731
N/L post		0.000	0.933
PTSD		0.107	0.000*
Systolic BP post		-0.027	0.112
Diastolic BP post		0.013	0.338
Pulse post		-0.020	0.108
CVD WITH Sleep disorder		0.005	0.000*

Table 14

Indirect effects for age group 18-49 years (adult)

	Predictor	Outcome	β	P-value
Total indirect effect	Systolic BP post	CVD	0.000	0.847
Specific indirect effect	Systolic Bp post-leukocyte post-CVD		0.000	0.636
	Systolic BP post-Plat post-CVD		-0.001	0.445
	Systolic BP post-N/L-CVD		0.000	0.312
Total indirect effects	Diastolic BP post	CVD	0.000	0.427
Specific indirect effect	Diastolic BP post-Leuko post-CVD		0.000	0.692
	Diastolic BP post- Plat post-CVD		0.000	0.334
	Diastolic BP Pot-N/L- CVD		0.000	0.385
Total indirect effect	Pulse post	CVD	-0.002	0.145
Specific indirect effect	Pulse post-leukocyte post-CVD		-0.001	0.252
	Pulse post-platelet post-CVD		0.000	0.425
	Pulse post-N/L post-CVD		0.000	0.284
Total indirect effect	PTSD	CVD	0.003	0.216
Specific indirect effect	PTSD-sys BP post-CVD		0.000	0.793
	PTSD-diastolic BP post-CVD		0.001	0.372
	PTSD-pulse post-CVD		0.000	0.424
	PTSD-leukocyte post-CVD		0.000	0.356
	PTSD-platelet post-PTSD		0.000	0.697
	PTSD-N/L post-PTSD		0.001	0.395
	PTSD-systolic BP post-leuko post-CVD		0.000	0.480
	PTSD-diastolic BP post-leuko post-CVD		0.000	0.653
	PTSD-pulse post-leuko post-CVD		0.000	0.313
	PTSD-systolic BP post-plat post-CVD		0.000	0.703
	PTSD-diastolic BP post-plat post-CVD		0.000	0.489
	PTSD-pulse post-plat post-CVD		0.000	0.451
	PTSD-systolic BP post-N/L post-CVD		0.000	0.419
	PTSD-diastolic BP post-N/L post-CVD		0.000	0.396
	PTSD-pulse post-N/L post-CVD		0.000	0.331
Total indirect effect	Systolic BP post	Sleep disorder	0.001	0.609
Specific indirect effect	Systolic BP post-leuko post-sleep disorder		0.000	0.647
	Systolic BP post-plat post-sleep disorder		0.000	0.742
	Systolic BP post-N/L post-sleep disorder		0.000	0.933
Total indirect effect	Diastolic BP post	Sleep disorder	0.001	0.312
	Diastolic BP post-leuko post-sleep disorder		0.001	0.364
	Diastolic BP post-plat post-sleep disorder		0.000	0.769
	Diastolic BP post-N/L post-sleep		0.000	0.934

	disorder			
Total indirect effect	Pulse post	Sleep disorder	-0.002	0.418
Specific indirect effect	Pulse post-leuko post-sleep disorder		-0.002	0.226
	Pulse post-plat post-sleep disorder		0.000	0.738
	Pulse post-N/L post-sleep disorder		0.000	0.933
Total indirect effect	PTSD	Sleep disorder	0.000	0.800
Specific indirect effect	PTSD-sys BP post-sleep disorder		0.000	0.450
	PTSD-diastolic BP post-sleep disorder		0.000	0.235
	PTSD-pulse post-sleep disorder		-0.001	0.253
	PTSD-leukocyte post-sleep disorder		0.000	0.314
	PTSD-platelet post-sleep disorder		0.000	0.768
	PTSD-N/L post-sleep disorder		0.000	0.933
	PTSD-systolic BP post-leuko post-sleep disorder		0.000	0.435
	PTSD-diastolic BP post-leuko post-sleep disorder		0.000	0.622
	PTSD-pulse post-leuko post-sleep disorder		0.000	0.294
	PTSD-systolic BP post-plat post-sleep disorder		0.000	0.773
	PTSD-diastolic BP post-plat post-sleep disorder		0.000	0.746
	PTSD-pulse post-plat post-sleep disorder		0.000	0.740
	PTSD-systolic BP post-N/L post-sleep disorder		0.000	0.934
	PTSD-diastolic BP post-N/L post-sleep disorder		0.000	0.933
	PTSD-pulse post-N/L post-sleep disorder		0.000	0.933
Total indirect effect	PTSD	Leukocyte post	0.005	0.109
Specific indirect effect	PTSD-systolic BP post-leukocyte post		-0.001	0.322
	PTSD-diastolic BP post-leukocyte post		0.000	0.648
	PTSD-pulse post-leukocyte post		0.006	0.059
Total indirect effect	PTSD	Platelet post	0.000	0.416
	PTSD-systolic BP post-platelet post		0.000	0.664
	PTSD-diastolic BP post-platelet post		0.000	0.193
	PTSD-pulse post-platelet post		0.001	0.077
Total indirect effect	PTSD	N/L post	0.010	0.014*
Specific indirect effect	PTSD-systolic BP post-N/L post		0.002	0.319
	PTSD-diastolic BP post-N/L post		0.003	0.174
	PTSD-pulse post-N/L post		0.006	0.082

Table 15

Direct effects for age group older than 50 (older adult)

Predictor	Outcome	β	p-value
Systolic BP pre	Leukocyte pre	0.034	0.732
Diastolic BP pre		-0.099	0.206
Pulse BP pre		0.199	0.291
Systolic BP pre	Platelet pre	0.066	0.032*
Diastolic BP pre		-0.030	0.232
Pulse BP pre		0.038	0.462
Systolic BP pre	N/L pre	-0.089	0.566
Diastolic BP pre		0.030	0.874
Pulse BP pre		0.378	0.084
Leukocyte pre	PTSD	-0.176	0.104
Platelet pre		-0.648	0.156
N/L pre		0.515	0.000*
PTSD	Systolic BP post	-0.061	0.000*
	Diastolic BP post	0.003	0.779
	Pulse BP post	0.029	0.053
Systolic BP post	Leukocyte post	-0.114	0.051
Diastolic BP post		-0.031	0.464
Pulse BP post		0.223	0.097
PTSD		0.026	0.236
Systolic BP post	Platelet post	-0.003	0.928
Diastolic BP post		0.028	0.119
Pulse BP post		-0.080	0.049*
PTSD		-0.011	0.038*
Systolic BP post	N/L post	-1.807	0.000*
Diastolic BP post		0.722	0.078
Pulse BP post		-0.301	0.558
PTSD		0.056	0.683
Leukocyte post	CVD	0.245	0.000*
Platelet post		-1.471	0.000*
N/L post		-0.189	0.000*
PTSD		0.194	0.000*
Systolic BP post		-0.615	0.000*
Diastolic BP post		0.203	0.081
Pulse post		-0.694	0.000*
Leukocyte post	Sleep disorder	0.057	0.002*
Platelet post		-0.406	0.000*
N/L post		-0.018	0.012*
PTSD		0.138	0.000*
Systolic BP post		-0.114	0.002*
Diastolic BP post		0.090	0.008*
Pulse post		-0.108	0.001*
CVD WITH Sleep disorder		0.001	0.040

Table 16

Indirect effects for age group older than 50 (older adult)

	Predictor	Outcome	β	p-value
Total indirect effect	Systolic BP post	CVD	0.318	0.000*
Specific indirect effect	Systolic Bp post-leukocyte post-CVD		-0.028	0.044*
	Systolic BP post-Plat post-CVD		0.004	0.928
	Systolic BP post-N/L-CVD		0.342	0.000*
Total indirect effects	Diastolic BP post	CVD	0.186	0.012*
Specific indirect effect	Diastolic BP post-Leuko post-CVD		-0.008	0.483
	Diastolic BP post- Plat post-CVD		-0.041	0.156
	Diastolic BP Pot-N/L- CVD		-0.136	0.051
Total indirect effect	Pulse post	CVD	0.228	0.004*
Specific indirect effect	Pulse post-leukocyte post-CVD		0.054	0.180
	Pulse post-platelet post-CVD		0.117	0.121
	Pulse post-N/L post-CVD		0.057	0.571
Total indirect effect	PTSD	CVD	0.017	0.624
Specific indirect effect	PTSD-sys BP post-CVD		0.001	0.781
	PTSD-diastolic BP post-CVD		0.038	0.000*
	PTSD-pulse post-CVD		-0.020	0.056
	PTSD-leukocyte post-CVD		0.006	0.241
	PTSD-platelet post-PTSD		0.016	0.073
	PTSD-N/L post-PTSD		-0.011	0.687
	PTSD-systolic BP post-leuko post-CVD		0.000	0.783
	PTSD-diastolic BP post-leuko post-CVD		0.002	0.052
	PTSD-pulse post-leuko post-CVD		0.002	0.282
	PTSD-systolic BP post-plat post-CVD		0.000	0.773
	PTSD-diastolic BP post-plat post-CVD		0.000	0.928
	PTSD-pulse post-plat post-CVD		0.003	0.202
	PTSD-systolic BP post-N/L post-CVD		0.000	0.787
	PTSD-diastolic BP post-N/L post-CVD		-0.021	0.001*
	PTSD-pulse post-N/L post-CVD		0.002	0.596
Total indirect effect	Systolic BP post	Sleep disorder	0.028	0.089
Specific indirect effect	Systolic BP post-leuko post-sleep disorder		-0.006	0.062
	Systolic BP post-plat post-sleep disorder		0.001	0.928
	Systolic BP post-N/L post-sleep disorder		0.033	0.019*
Total indirect effect	Diastolic BP post	Sleep disorder	-0.026	0.014*
	Diastolic BP post-leuko post-sleep disorder		-0.002	0.480
	Diastolic BP post-plat post-sleep disorder		-0.011	0.148

	Diastolic BP post-N/L post-sleep disorder		-0.013	0.120
Total indirect effect	Pulse post	Sleep disorder	0.050	0.000*
Specific indirect effect	Pulse post-leuko post-sleep disorder		0.013	0.180
	Pulse post-plat post-sleep disorder		0.032	0.087
	Pulse post-N/L post-sleep disorder		0.006	0.599
Total indirect effect	PTSD	Sleep disorder	0.009	0.091
Specific indirect effect	PTSD-sys BP post-sleep disorder		0.000	0.785
	PTSD-diastolic BP post-sleep disorder		0.007	0.005*
	PTSD-pulse post-sleep disorder		-0.003	0.071
	PTSD-leukocyte post-sleep disorder		0.001	0.261
	PTSD-platelet post-sleep disorder		0.005	0.060
	PTSD-N/L post-sleep disorder		-0.001	0.691
	PTSD-systolic BP post-leuko post-sleep disorder		0.000	0.783
	PTSD-diastolic BP post-leuko post-sleep disorder		0.000	0.072
	PTSD-pulse post-leuko post-sleep disorder		0.002	0.286
	PTSD-systolic BP post-plat post-sleep disorder		0.000	0.772
	PTSD-diastolic BP post-plat post-sleep disorder		0.000	0.928
	PTSD-pulse post-plat post-sleep disorder		0.001	0.176
	PTSD-systolic BP post-N/L post-sleep disorder		0.000	0.788
	PTSD-diastolic BP post-N/L post-sleep disorder		-0.002	0.031*
	PTSD-pulse post-N/L post-sleep disorder		0.000	0.621
Total indirect effect	PTSD	Leukocyte post	0.013	0.021*
Specific indirect effect	PTSD-systolic BP post-leukocyte post		0.000	0.782
	PTSD-diastolic BP post-leukocyte post		0.007	0.060
	PTSD-pulse post-leukocyte post		0.006	0.221
Total indirect effect	PTSD	Platelet post	-0.002	0.336
	PTSD-systolic BP post-platelet post		0.000	0.772
	PTSD-diastolic BP post-platelet post		0.000	0.928
	PTSD-pulse post-platelet post		-0.002	0.143
Total indirect effect	PTSD	N/L post	0.104	0.013*
Specific indirect effect	PTSD-systolic BP post-N/L post		0.002	0.787
	PTSD-diastolic BP post-N/L post		0.111	0.003*
	PTSD-pulse post-N/L post		-0.009	0.586

Table 17

Direct effects for lower BMI group

Predictor	Outcome	β	p-value
Systolic BP pre	Leukocyte pre	-0.211	0.082
Diastolic BP pre		-0.080	0.383
Pulse BP pre		0.167	0.380
Systolic BP pre	Platelet pre	0.041	0.334
Diastolic BP pre		0.010	0.817
Pulse BP pre		-0.007	0.884
Systolic BP pre	N/L pre	-2.324	0.057
Diastolic BP pre		-0.794	0.180
Pulse BP pre		1.428	0.075
Leukocyte pre	PTSD	0.084	0.494
Platelet pre		-0.113	0.774
N/L pre		-0.046	0.102
PTSD	Systolic BP post	-0.022	0.361
	Diastolic BP post	0.049	0.100
	Pulse BP post	-0.003	0.937
Systolic BP post	Leukocyte post	-0.152	0.392
Diastolic BP post		-0.196	0.270
Pulse BP post		0.374	0.021*
PTSD		-0.007	0.907
Systolic BP post	Platelet post	0.094	0.024*
Diastolic BP post		-0.039	0.306
Pulse BP post		0.073	0.025*
PTSD		0.011	0.422
Systolic BP post	N/L post	-2.277	0.031*
Diastolic BP post		2.104	0.094
Pulse BP post		0.770	0.052
PTSD		-0.010	0.968
Leukocyte post	CVD	0.014	0.645
Platelet post		-0.161	0.300
N/L post		0.040	0.001*
PTSD		0.055	0.081
Systolic BP post		0.158	0.045*
Diastolic BP post		-0.011	0.895
Pulse post		0.000	1.000
Leukocyte post	Sleep disorder	-0.018	0.377
Platelet post		-0.251	0.012*
N/L post		0.003	0.681
PTSD		0.109	0.000*
Systolic BP post		0.022	0.604
Diastolic BP post		0.006	0.888
Pulse post		0.019	0.447
CVD WITH Sleep disorder		0.009	0.175

Table 18

Indirect effects for lower BMI group

	Predictor	Outcome	β	p-value
Total effect	Systolic BP post	CVD	-0.107	0.079
	Systolic BP post-leukocyte post-CVD		-0.002	0.709
	Systoli BP post-platelet post-CVD		-0.015	0.383
	Systolic BP post-N/L post-CVD		-0.090	0.078
	Diastolic BP post	CVD	0.087	0.138
	Diastolic BP post-leukocyte post-CVD		-0.003	0.631
	Diastolic BP post-platelet post-CVD		0.006	0.459
	Diastolic BP post-N/L post-CVD		0.083	0.132
	Pulse post	CVD	0.024	0.348
	Pulse post-leukocyte post-CVD		0.005	0.645
	Pulse-platelet post-CVD		-0.012	0.315
	Pulse post-N/L post-CVD		0.030	0.096
Total indirect effect	PTSD	CVD	0.000	0.985
	PTSD-systolic BP post-CVD		-0.001	0.895
	PTSD-diastolic BP post-CVD		-0.004	0.433
	PTSD-pulse post-CVD		0.000	1.000
	PTSD-leukocyte post-CVD		0.000	0.904
	PTSD-platelet post-CVD		-0.002	0.442
	PTSD-N/L post-CVD		0.000	0.968
	PTSD-systolic BP post-leukocyte post-CVD		0.000	0.628
	PTSD-diastolic BP post-leukocyte post-CVD		0.000	0.741
	PTSD-pulse post-leukocyte post-CVD		0.000	0.938
	PTSD-systolic BP post-platelet post-CVD		0.000	0.509
	PTSD-diastolic BP post-platelet post-CVD		0.000	0.562
	PTSD-pulse post-platelet post-CVD		0.000	0.937
	PTSD-systolic BP post-N/L post-CVD		0.004	0.284
	PTSD-diastolic post-N/L post-CVD		0.002	0.438
	PTSD-pulse post-N/L post-CVD		0.000	0.937
Total indirect effect	Systolic BP post	Sleep disorder	-0.027	0.270
	Systolic BP post-leukocyte post-sleep disorder		0.003	0.463
	Systolic BP post-platelet-sleep disorder		-0.024	0.134
	Systolic BP post-N/L post-sleep disorder		-0.006	0.674
Total indirect effect	Diastolic BP post	Sleep disorder	0.019	0.341
	Diastolic BP post-leukocyte post-sleep disorder		0.003	0.535
	Diastolic BP post-platelet post-sleep disorder		0.010	0.387
	Diastolic BP post-N/L post-sleep disorder		0.006	0.681
Total indirect effect	Pulse post	Sleep disorder	-0.023	0.128
	Pulse post-leukocyte post-sleep disorder		-0.007	0.354
	Pulse post-platelet post-sleep disorder		-0.018	0.065
	Pulse post-N/L post-sleep disorder		0.002	0.671

Total indirect effect	PTSD	Sleep disorder	-0.001	0.789
	PTSD-systolic BP post-Sleep disorder		0.000	0.889
	PTSD-diastolic BP post-Sleep disorder		0.000	0.657
	PTSD-pulse post-Sleep disorder		0.000	0.938
	PTSD-leukocyte post-Sleep disorder		0.000	0.907
	PTSD-platelet post-Sleep disorder		-0.003	0.448
	PTSD-N/L post-Sleep disorder		0.000	0.967
	PTSD-systolic BP post-leukocyte post-Sleep disorder		0.000	0.549
	PTSD-diastolic BP post-leukocyte post-Sleep disorder		0.000	0.595
	PTSD-pulse post-leukocyte post-Sleep disorder		0.000	0.937
	PTSD-systolic BP post-platelet post-Sleep disorder		0.000	0.468
	PTSD-diastolic BP post-platelet post-Sleep disorder		0.001	0.467
	PTSD-pulse post-platelet post-Sleep disorder		0.000	0.937
	PTSD-systolic BP post-N/L post-Sleep disorder		0.000	0.707
	PTSD-diastolic post-N/L post-Sleep disorder		0.000	0.709
	PTSD-pulse post-N/L post-Sleep disorder		0.000	0.937
Total indirect effects	PTSD	Leukocyte post	-0.007	0.679
	PTSD-systolic BP post-leukocyte post		-0.010	0.320
	PTSD-diastolic BP post-leukocyte count		0.003	0.567
	PTSD-pulse post-leukocyte post		-0.001	0.937
Total indirect effects	PTSD	N/L post	0.153	0.173
	PTSD-systolic BP post-N/L post		0.104	0.270
	PTSD-diastolic BP post-N/L post		0.051	0.412
	PTSD-pulse post-N/L post		-0.002	0.937
Total indirect effect	PTSD	Platelet post	-0.004	0.332
	PTSD-systolic BP post-platelet post		-0.002	0.408
	PTSD-diastolic BP post-platelet post		-0.002	0.423
	PTSD-pulse post-platelet post		0.000	0.938

Table 19

Direct effects for higher BMI group

Predictor	Outcome	β	P-value
Systolic BP pre	Leukocyte pre	0.088	0.496
Diastolic BP pre		-0.117	0.285
Pulse BP pre		0.271	0.020*
Systolic BP pre	Platelet pre	0.069	0.005*
Diastolic BP pre		-0.019	0.351
Pulse BP pre		0.000	0.990
Systolic BP pre	N/L pre	-0.275	0.179
Diastolic BP pre		-0.383	0.055
Pulse BP pre		-0.130	0.441
Leukocyte pre	PTSD	0.054	0.447
Platelet pre		-0.606	0.153
N/L pre		-0.191	0.000*
PTSD	Systolic BP post	-0.031	0.036*
	Diastolic BP post	0.025	0.089
	Pulse BP post	-0.009	0.760
Systolic BP post	Leukocyte post	0.023	0.827
Diastolic BP post		0.004	0.973
Pulse BP post		0.023	0.881
PTSD		0.013	0.736
Systolic BP post	Platelet post	0.085	0.002*
Diastolic BP post		0.004	0.481
Pulse BP post		0.023	0.881
PTSD		0.013	0.736
Systolic BP post	N/L post	-0.871	0.049*
Diastolic BP post		-0.154	0.656
Pulse BP post		2.048	0.089
PTSD		-0.175	0.109
Leukocyte post	CVD	-0.026	0.578
Platelet post		-0.815	0.000*
N/L post		0.046	0.338
PTSD		0.021	0.354
Systolic BP post		0.059	0.492
Diastolic BP post		-0.011	0.884
Pulse post		-0.177	0.194
Leukocyte post	Sleep disorder	-0.012	0.687
Platelet post		-0.396	0.006*
N/L post		0.028	0.388
PTSD		0.037	0.048*
Systolic BP post		0.004	0.947
Diastolic BP post		0.150	0.024*
Pulse post		-0.096	0.296
CVD WITH Sleep disorder		0.008	0.002*

Table 20

Indirect effects for higher BMI group

	Predictor	Outcome	β	P-value
Total effect	Systolic BP post	CVD	-0.110	0.040*
	Systolic BP post-leukocyte post-CVD		-0.001	0.851
	Systoli BP post-platelet post-CVD		-0.069	0.019*
	Systolic BP post-N/L post-CVD		-0.040	0.358
Total indirect effect	Diastolic BP post	CVD	-0.041	0.145
	Diastolic BP post-leukocyte post-CVD		0.000	0.973
	Diastolic BP post-platelet post-CVD		-0.034	0.097
	Diastolic BP post-N/L post-CVD		-0.007	0.719
Total indirect effect	Pulse post	CVD	0.095	0.510
	Pulse post-leukocyte post-CVD		-0.001	0.875
	Pulse-platelet post-CVD		0.001	0.942
	Pulse post-N/L post-CVD		0.094	0.485
Total indirect effect	PTSD	CVD	0.002	0.892
	PTSD-systolic BP post-CVD		0.000	0.884
	PTSD-diastolic BP post-CVD		-0.002	0.513
	PTSD-pulse post-CVD		0.002	0.779
	PTSD-leukocyte post-CVD		0.000	0.765
	PTSD-platelet post-CVD		0.009	0.206
	PTSD-N/L post-CVD		-0.008	0.332
	PTSD-systolic BP post-leukocyte post-CVD		0.000	0.973
	PTSD-diastolic BP post-leukocyte post-CVD		0.000	0.852
	PTSD-pulse post-leukocyte post-CVD		0.000	0.868
	PTSD-systolic BP post-platelet post-CVD		-0.001	0.238
	PTSD-diastolic BP post-platelet post-CVD		0.002	0.134
	PTSD-pulse post-platelet post-CVD		0.000	0.946
	PTSD-systolic BP post-N/L post-CVD		0.000	0.727
	PTSD-diastolic post-N/L post-CVD		0.001	0.387
	PTSD-pulse post-N/L post-CVD		-0.001	0.809
Total indirect effect	Systolic BP post	Sleep disorder	-0.058	0.101
	Systolic BP post-leukocyte post-sleep disorder		0.000	0.849
	Systolic BP post-platelet-sleep disorder		-0.034	0.036*
	Systolic BP post-N/L post-sleep disorder		-0.024	0.421
Total indirect effect	Diastolic BP post	Sleep disorder	-0.021	0.205
	Diastolic BP post-leukocyte post-sleep disorder		0.000	0.974
	Diastolic BP post-platelet post-sleep disorder		-0.016	0.149
	Diastolic BP post-N/L post-sleep disorder		-0.004	0.727
Total indirect effect	Pulse post	Sleep disorder	0.057	0.534
	Pulse post-leukocyte post-sleep disorder		0.000	0.882
	Pulse post-platelet post-sleep disorder		0.001	0.942
	Pulse post-N/L post-sleep disorder		0.056	0.518
Total indirect effect	PTSD	Sleep	0.005	0.528

		disorder		
	PTSD-systolic BP post-Sleep disorder		0.004	0.159
	PTSD-diastolic BP post-Sleep disorder		0.000	0.947
	PTSD-pulse post-Sleep disorder		0.001	0.786
	PTSD-leukocyte post-Sleep disorder		0.000	0.806
	PTSD-platelet post-Sleep disorder		0.004	0.222
	PTSD-N/L post-Sleep disorder		-0.005	0.377
	PTSD-systolic BP post-leukocyte post-Sleep disorder		0.000	0.974
	PTSD-diastolic BP post-leukocyte post-Sleep disorder		0.000	0.850
	PTSD-pulse post-leukocyte post-Sleep disorder		0.000	0.877
	PTSD-systolic BP post-platelet post-Sleep disorder		0.000	0.279
	PTSD-diastolic BP post-platelet post-Sleep disorder		0.000	0.150
	PTSD-pulse post-platelet post-Sleep disorder		0.000	0.946
	PTSD-systolic BP post-N/L post-Sleep disorder		0.000	0.735
	PTSD-diastolic post-N/L post-Sleep disorder		0.001	0.446
	PTSD-pulse post-N/L post-Sleep disorder		0.000	0.811
Total indirect effect	PTSD	Leukocyte post	-0.001	0.880
	PTSD-systolic BP post-leukocyte post		0.000	0.973
	PTSD-diastolic BP post-leukocyte count		-0.001	0.828
	PTSD-pulse post-leukocyte post		0.000	0.870
Total indirect effects	PTSD	N/L post	0.005	0.939
	PTSD-systolic BP post-N/L post		-0.004	0.670
	PTSD-diastolic BP post-N/L post		0.027	0.145
	PTSD-pulse post-N/L post		-0.018	0.774
Total indirect effect	PTSD	Platelet post	-0.002	0.427
	PTSD-systolic BP post-platelet post		0.001	0.223
	PTSD-diastolic BP post-platelet post		-0.003	0.098
	PTSD-pulse post-platelet post		0.000	0.946

APPENDIX B

FIGURES

Figure 1

Proposed Theoretical Model

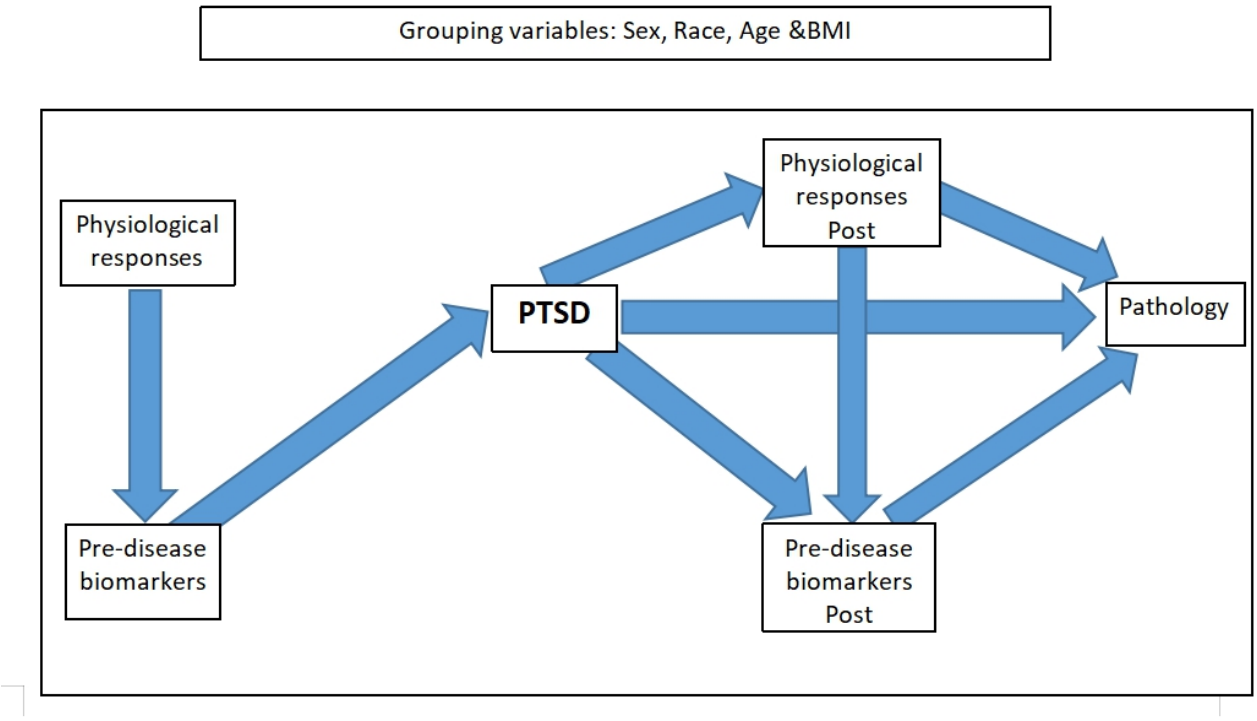
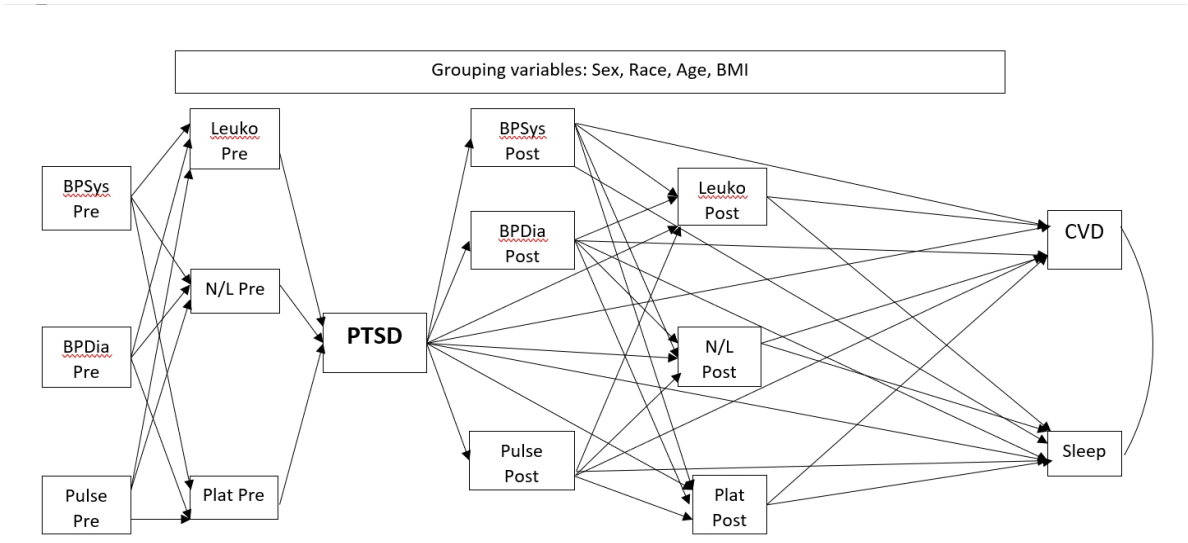


Figure 2

Proposed Analytical Model



Figures 3

Result for Full Model

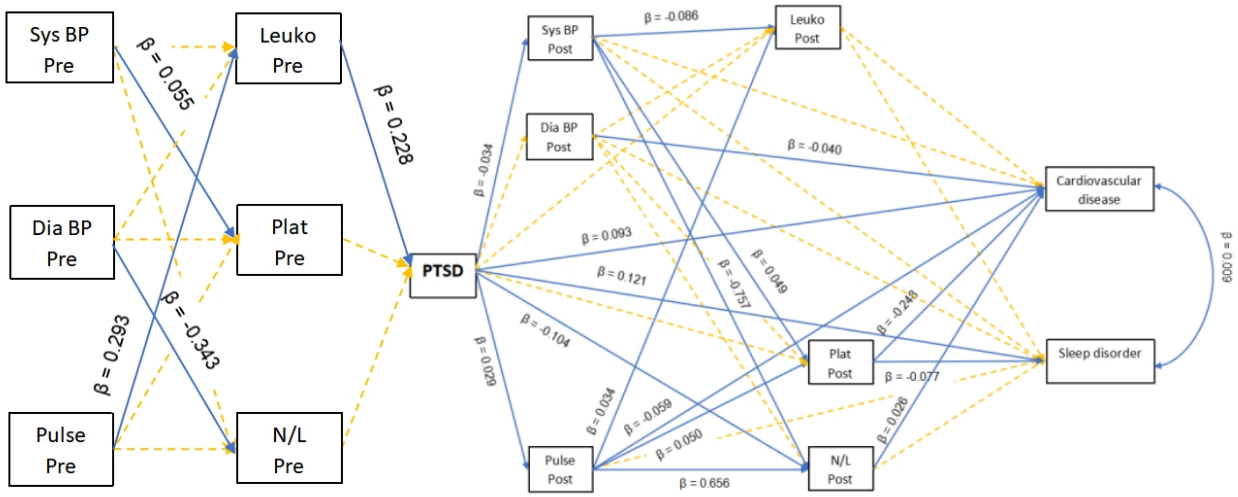


Figure 4

Path Model for Male Study Participants

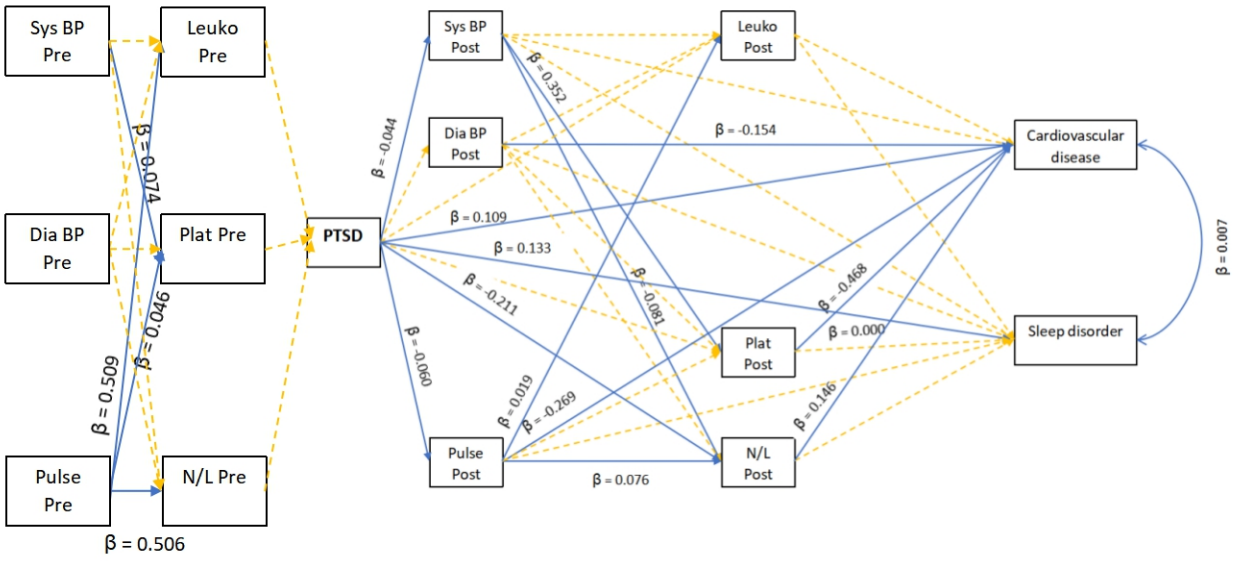


Figure 5

Path Model for Female Study Participants

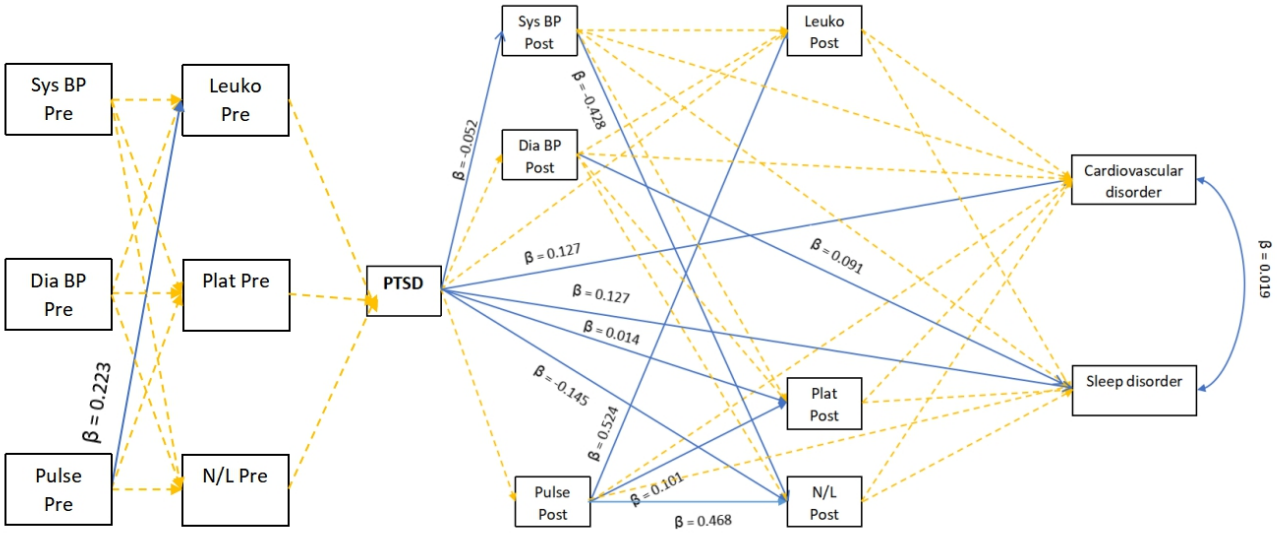


Figure 6

Path model for white study participants

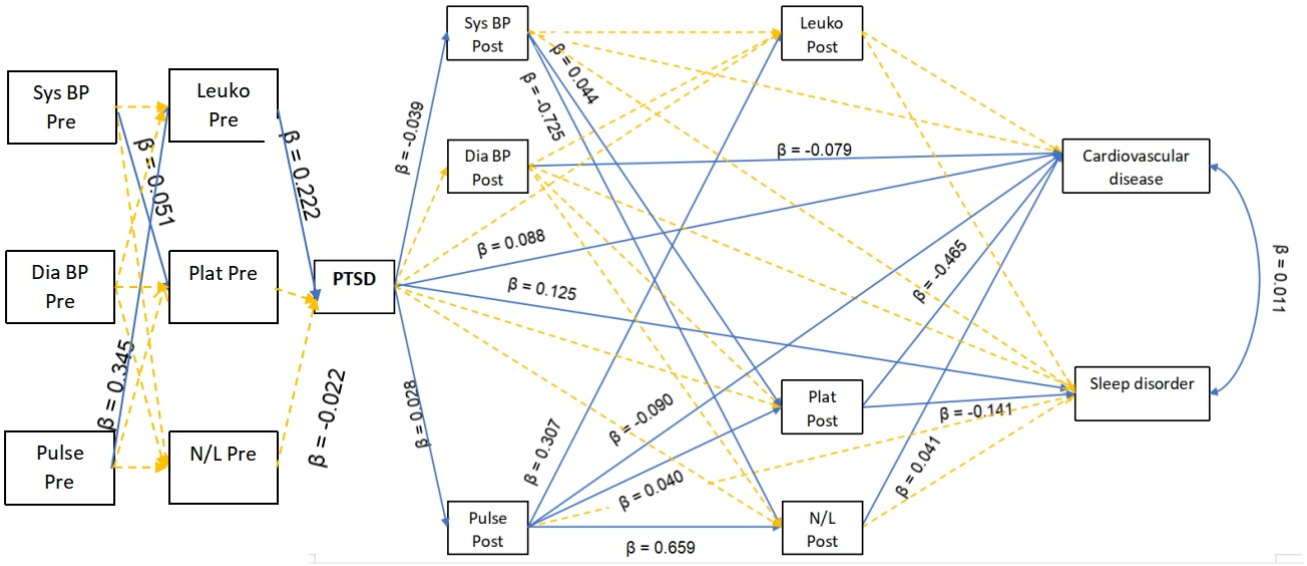


Figure 7

Path model for non-white study participants

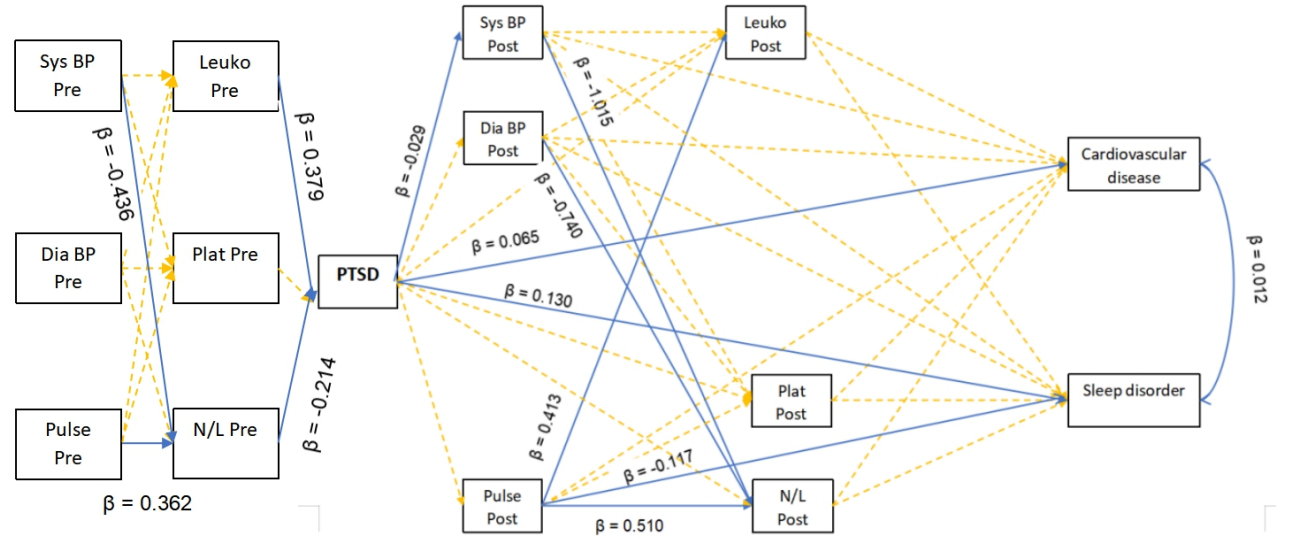


Figure 8

Path model for age group 0-17 years (children)

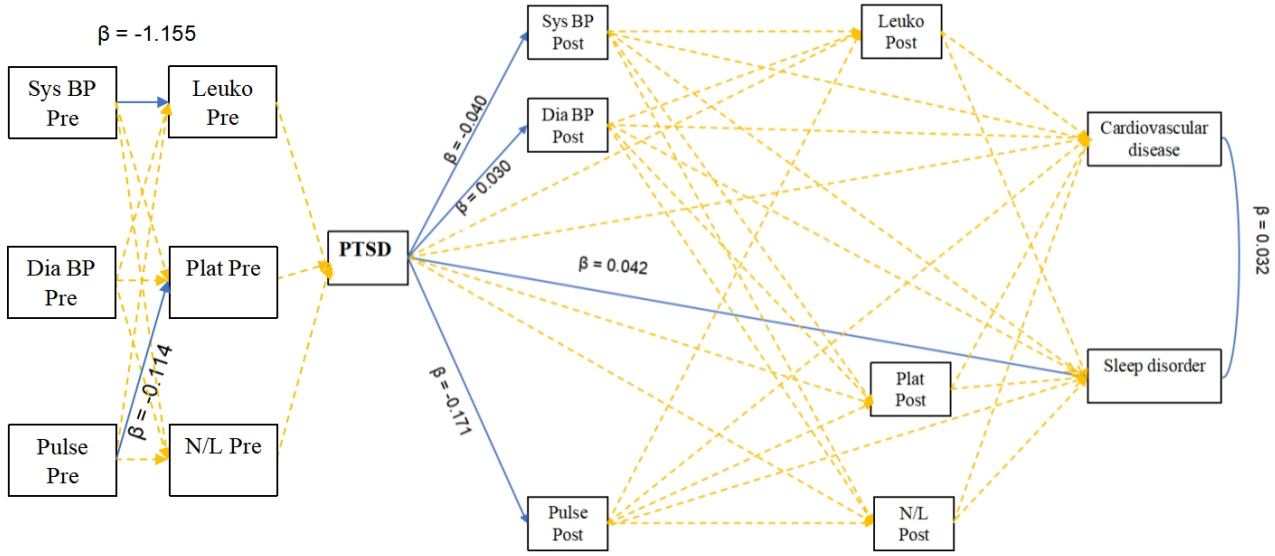


Figure 9

Path model for age group 18-49 years

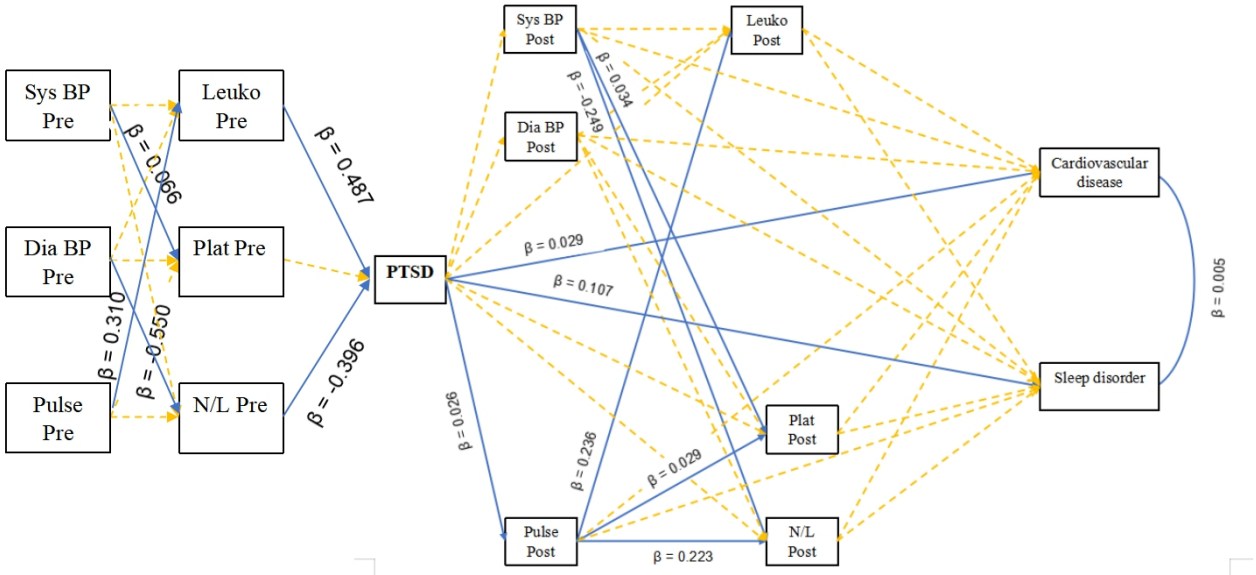


Figure 10

Path model for age group 50 years and older (older adult)

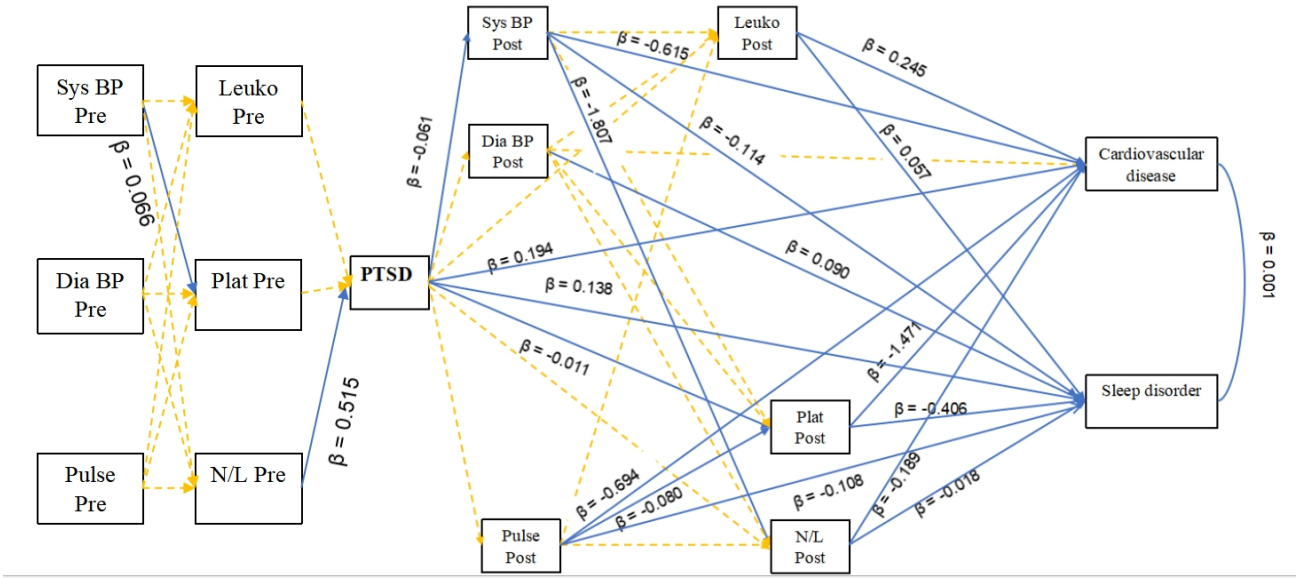


Figure 11

Path model for lower BMI group

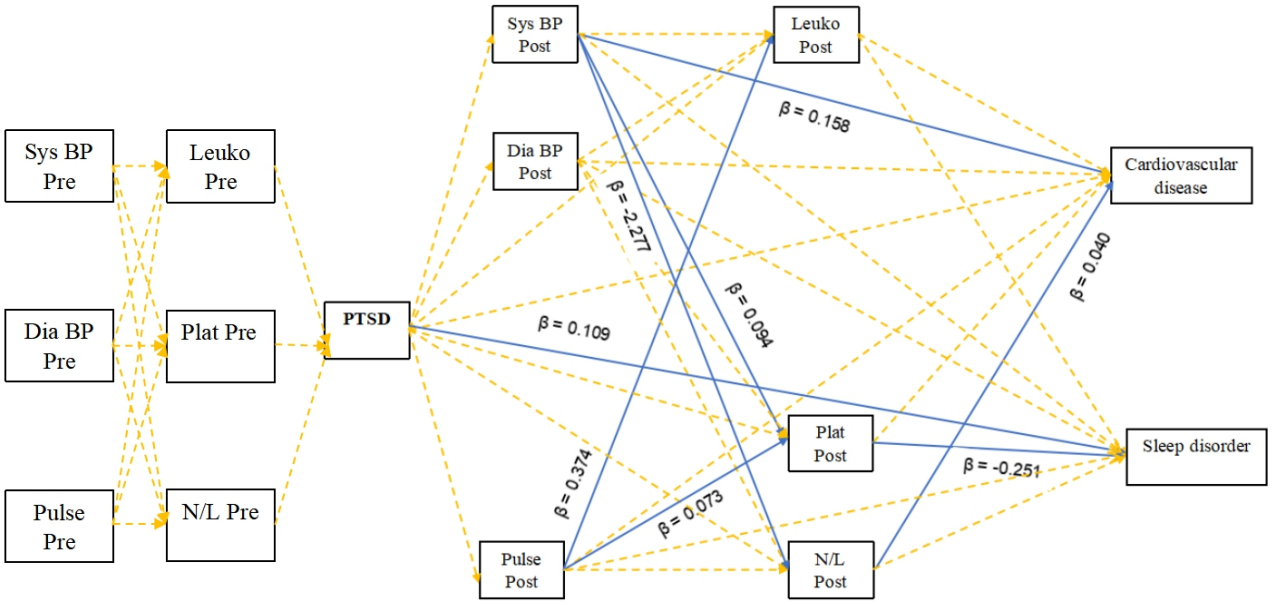
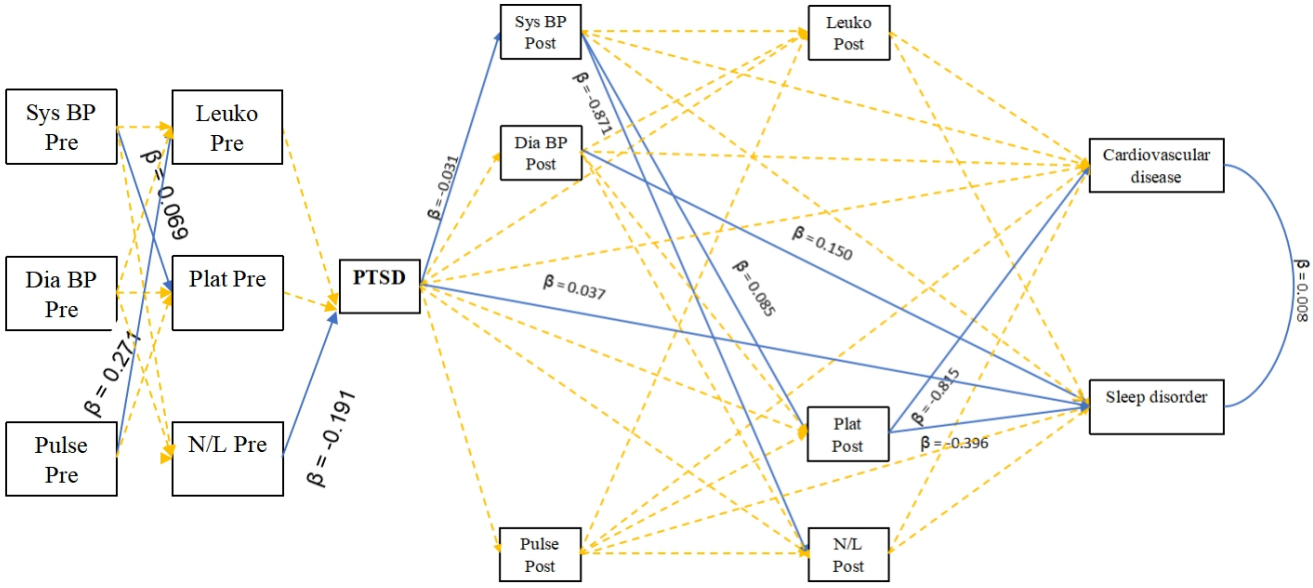


Figure 12

Path model for higher BMI group



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