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Exploring Downstream Effects of Racial Trauma Leading to PTSD: A Review of Systemic Physiological and Neurobehavioral Alterations

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Exploring Downstream Effects of Racial Trauma Leading to PTSD: A Review of Systemic Physiological and Neurobehavioral Alterations

A Capstone in Neurobehavioral Science by Alexis Verwoert

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ABSTRACT

Post-traumatic Stress Disorder (PTSD) is a debilitating psychiatric disease that often follows acute or chronic exposure to extreme stress. Hallmarks of this disorder include stressors, intrusion symptoms, avoidance, negative alterations in cognition and mood, and alterations in arousal and reactivity (Bovin, 2015). Since it was first defined in the Diagnostic and Statistical Manual of Mental Disorders-III (DSM-III), the diagnostic criteria for PTSD have evolved to encompass stressors as they may occur repeatedly across the lifespan (Bovin, 2015). Physiological and behavioral consequences of this extreme stress manifest themselves as systemic alterations in form and function. As is true of many diagnosable conditions, PTSD is most prevalent in African Americans and an estimated one in 11 people will be diagnosed in their lifetime (Torres, 2020). More recent studies have elucidated that race-based traumatic stress elicits a chronic stress response that mimics that of PTSD (Carter, 2020). While this disorder causes primary changes to the brain, further downstream effects cause global corporal alterations and lead to maladaptive responses to future stressors.

Previous studies have demonstrated that the hippocampus, amygdala, and medial prefrontal cortex play important roles in PTSD. Studies have expanded on these findings and traced their effects downstream in other body systems including the immune system, central nervous system, digestive system, and endocrine system (Stam, 2007). Though there is a significant understanding of the psychological and physiological manifestations of PTSD, the interactions of the distal branching effects of this disorder on the body are less substantiated as they relate to racial trauma. Only more recently have researchers begun to examine their findings through an interdisciplinary lens as they apply to race.

This capstone project is a literature review of the chronic stress response, secondary and downstream effects of stress on the numerous body systems, and their integrated implications on health as they relate to race-based stress. The research collected explores the physiological and neurobehavioral alterations which take place secondary to general trauma exposure, the long-term consequences of chronic stress, and the role of race as a social determinant of health. Various medical and academic interventions will be discussed, as well as the secondary effects of racial trauma on people of color.

INTRODUCTION

Allostasis and the Individual Stress Response

Stress has many working definitions but is concisely defined as an emotional or physical stimulus that disrupts homeostasis and may result in pathology (Stam, 2007). This response is non-specific as it does not affect one particular system, but many parts of the global system due to their inherent connections. Levine and Ursin (2013) defined stress as a condition caused by a lack of information leading to uncertainty or unpredictability about obtaining positive outcomes or avoiding negative outcomes. Such stress may follow a potential threat to the vital interests of the organism in which physiological and behavioral mechanisms are activated to eliminate the source of the stress and restore homeostasis (Levine and Ursin, 2013). Stress is a normal and essential physiological response, but when these responses become unregulated or occur in excess, problems arise.

Systemic changes occur following exposure to stressors. Much like the immune system, the body as a whole changes following exposure to specific and controllable stressors. These stressors prime the system for more refined and efficient responses during a second encounter. The subsequent changes are less adaptive when the source of stress is not immediately controllable (Stam, 2007). As stated by Stam (2007), when the source of stress is not acutely constrained, the stress response triggers a reorganization of neuronal circuitry in the brain and an acquisition of novel strategies to address the threat. If the stress response is repeated or sustained for an extended period without removal of the stressor, or if the stressful experience is so overwhelming that it over-taxes the homeostatic mechanisms, this “allostatic overload” may precipitate disease (Stam, 2007).

Allostasis is defined as achieving stability through change, or the process by which the organism maintains homeostasis in the face of new challenges (Faber, 2020). Allostatic systems enable an organism to maintain its essential functions in balance (McEwen, 2017). To maintain an allostatic state, the brain overrides the local effects of homeostasis in individual organs to allow for optimal global functioning. Allostasis is ultimately beneficial in that it allows for adaptation over time. This adaptation is achieved via ongoing evaluation of internal and external demands which permit physiological adjustments in advance of stress via anticipatory arousal (Ganzel, 2010).

Per McEwen (2017), the body initiates an allostatic response under normal circumstances to cope with a stressor for the duration of its presence. Once the challenge has passed, the body turns this response off. Two primary mediators of this ever-adapting system are the sympathetic nervous system and the Hypothalamic-Pituitary-Adrenal (HPA) axis. The activation of these systems leads to the release of catecholamines and other accompanying hormones, such as cortisol, while the deactivation results in a regression to baseline hormonal concentrations. When the allostatic response is limited to the period of challenge, adaptive protection outweighs adverse consequences. However, with persistent stress over weeks, months, or even years, the body is exposed to elevated levels of stress hormones and allostatic overload, resulting in pathophysiological damage (McEwen, 2017). McEwen went on to define allostatic load as the physiological cost associated with long-term, global changes across the body systems to match internal functioning to external demand. With persistent stress, the body's delicate and overlapping regulatory systems are overtaxed. This wear and tear can create runaway

dysregulation and leave these regulatory mechanisms catastrophically out of alignment (McEwen, 2017).

Under normal physiological conditions, the system's response to stress is highly individualized but ultimately occurs within stable and predictable limits defined by homeostasis. The individuality of the stress response depends on an individual's appraisal process, coping style, and social milieu. Individuals will experience a variable stress response based upon their experience of uncertainty and their subsequent appraisal of their ability to restore homeostasis. The appraisal process may be influenced by a person's social environment, race, ethnicity, biological sex, gender identity, socioeconomic status, and various other intersecting social determinants of health. In an example offered by Stam (2007), PTSD is more prevalent in biological females not necessarily due to increased exposure to traumatic events, but likely due to differences in coping styles, socio-economic resources, and levels of autonomic and hormonal arousal. Stam (2007) went on to explain that the psychological factors involved in gender-based differences in PTSD occurrence also play a role in gender-independent differences in PTSD vulnerability (Stam, 2007).

Part of this stress response process depends on a person's locus of control. In a study conducted by Karstoft (2015), locus of control and coping style were found to be highly predictive of PTSD outcomes. Specifically, an internal locus of control was correlated with a decreased risk of PTSD symptoms, as was the employment of problem-focused coping strategies as opposed to emotion-focused coping (Karstoft, 2015). Inability to cope with stress may result in clinically significant consequences in functioning, such as post-traumatic stress. Per the Diagnostic and Statistical Manual of

Mental Disorders-5 (DSM-5), higher rates of PTSD have been reported among U.S. Latinx, African Americans, and American Indians compared to other ethnic demographics (Trauma- and stressor-related disorders, 2013).

The stress response is made increasingly discrete and unpredictable by the involvement of epigenetic factors, particularly those involved in the pathogenesis of PTSD. Dr. Madhulika Gupta, M.D. (2013) stated environmental factors such as early life stress can modify gene expression via DNA methylation and histone modification. DNA methylation has been found to reduce gene expression while early life stressors have been implicated in the increased methylation of glucocorticoid receptor expression. The glucocorticoid receptor plays a critical role in the negative feedback regulation of the HPA axis. On a systemic level, these epigenetic mechanisms have been associated with the mechanisms of fear learning and memory consolidation which play a central role in PTSD (Gupta, 2013).

BACKGROUND

Post-traumatic stress disorder (PTSD)

The American Psychiatric Association revised the diagnostic criteria of post-traumatic stress disorder (PTSD) in its fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5). Per this most recent definition, PTSD is defined as "the development of characteristic symptoms following exposure to one or more traumatic events." These characteristic symptoms include intrusion symptoms, avoidance, negative alterations in cognition and mood, and alterations in arousal and reactivity. Intrusion symptoms include involuntary memories, recurrent and distressing dreams, and realistic flashbacks about the traumatic event. These intrusion symptoms are accompanied by avoidance of internal and external stimuli associated with the event. PTSD is distinguished from other trauma- and stressor-related disorders based on both timing and symptom profile (Trauma- and stressor-related disorders, 2013).

Symptoms required for diagnosis of PTSD	
DSM-5 criteria	**Proposed ICD-11 criteria**
Intrusion symptoms	
Recurrent, involuntary, and intrusive distressing memories Recurrent distressing dreams (content and/or affect related) Dissociative reaction (acting or feeling as if the event is recurring) Intense or prolonged psychological distress to cues Noticeable physiological reactions to cues	Vivid intrusive memories, flashbacks, or nightmares, typically accompanied by strong and overwhelming emotions such as fear or horror, and strong physical sensations
Avoidance	
Avoidance or efforts to avoid distressing thoughts or feelings about or closely associated with the trauma Avoidance or efforts to avoid external reminders (people, places, conversations, activities, objects, situations)	Avoidance of thoughts and memories of the event or events Avoidance of activities, situations, or people reminiscent of the event or events
Negative alterations in cognitions and mood	
Inability to remember an important aspect (typically due to dissociative amnesia) Persistent and exaggerated negative beliefs or expectations about oneself, others, or the world (for example, "I am bad," "No one can be trusted," "The world is completely dangerous") Persistent, distorted cognitions about the cause or consequences that lead to self-blame or the blame of others Persistent negative emotional state (for example, fear, horror, anger, guilt, shame) Noticeably diminished interest or participation in important activities Feelings of detachment or estrangement from others Persistent inability to experience positive emotions (for example, happiness, satisfaction, love)	Not applicable
Alterations in arousal and reactivity	
Irritable behavior and angry outbursts (with little or no provocation) Reckless or self-destructive behavior Hypervigilance Exaggerated startle response Problems with concentration Sleep disturbance	Persistent perceptions of heightened current threat — for example, as indicated by hypervigilance or an enhanced startle reaction to stimuli such as unexpected noises
Additional criteria for complex PTSD	
Not applicable	Severe and pervasive problems in affect regulation Persistent beliefs about oneself as diminished, defeated, or worthless, accompanied by deep and pervasive feelings of shame, guilt, or failure related to the stressor Persistent difficulties in sustaining relationships and in feeling close to others

Table 1. Symptoms required for diagnosis of PTSD. Adapted from Bisson, 2015.

Per the DSM-5, an appropriate PTSD diagnosis may specify whether the individual's symptoms include intrusion symptoms such as depersonalization or derealization, or if these symptoms occurred with delayed expression. The functional consequences of this disorder include impaired functioning across social, interpersonal, developmental, educational, physical health, and occupational domains (Trauma- and stressor-related disorders, 2013). The manifestations of this disorder exist on a spectrum, but nearly ubiquitously affect diagnosed individuals daily.

The Chronic Stress Response

The hallmark psychological symptoms of PTSD are associated with various somatic symptoms and medical conditions affecting multiple body systems. Further, these primary physical effects of PTSD are accompanied by secondary alterations in experience and quality of life. With the experience of chronic stress and allostatic overload, an individual's health is markedly affected. As hormones such as cortisol circulate in the body, fat is mobilized as sugar for use in the fight-or-flight response. Unused sugar is re-stored as fat in the midsection, potentially damaging the vital organs located in the region. The elevation of blood pressure and heart rate which accompany this sympathetic response can, in excess, lead to hypertension or cardiomegaly (Greenberg, 2020). Beyond the diagnosable symptoms of PTSD, chronic stress causes significant alterations across multiple mechanisms and profoundly exaggerates systemic damage.

Following exposure to a traumatic event, individuals diagnosed with PTSD experience notable changes in neuroanatomy, as well as the CNS. Structural and functional changes in the brain have been linked with the chronic experience of stress and

the exacerbation of PTSD symptoms such as hypervigilance. Holmes (2018) identified structural and functional alterations in the prefrontal cortex (PFC), hippocampus, amygdala, and cerebellum. Structurally, individuals with PTSD presented with smaller cerebellar, hippocampal, and PFC volume. Functionally, connectivity in the cerebellum and PFC were negatively correlated with PTSD severity, while the amygdala demonstrated increased activity. Analyses in this study revealed weakened connectivity within nodes of the central executive network of the PFC, as well as between nodes of the default mode network in the PFC and cerebellum. These deficits in cerebellar connectivity manifested as symptoms such as intrusive thoughts and avoidance. These findings were consistent with well-established knowledge on PTSD pathophysiology and the growing support of the involvement of the cerebellum (Holmes, 2018).

As has been established, patients diagnosed with PTSD develop neuroanatomic changes in the hippocampus, amygdala, and cortex. Reduced volume and activity in the amygdala alter stress responses and the extinction of memories. Increased activity in the amygdala promotes hypervigilance and impairs discrimination of threats. Reduced prefrontal cortical volume dysregulates executive function, reduced anterior cingulate volume impairs the extinction of fear responses, and decreased medial prefrontal activation leads to uncertain effects. These modifications cause neural circuitry to reorganize in a form of non-associative learning to strengthen defensive reflexes to previously neutral stimuli (Sherin, 2011).

Per Bremner (2006), persons with PTSD show increased cortisol and norepinephrine responses to stress, consistent with the traumatic stress response. In the long-term, these neurochemical systems become dysregulated in an attempt to cope with

fear. As is summarized in the image below, corticotropin-releasing hormone (CRH), part of the HPA axis, is released from the hypothalamus in response to stress. This stimulates the release of adrenocorticotropic hormone (ACTH) from the pituitary and the subsequent release of cortisol from the adrenal gland. This eventually results in negative feedback on the HPA axis. While cortisol facilitates survival in instances of acute danger, it is accompanied by CRH which centrally mediates fear behaviors and triggers downstream neurochemical responses to stress. One of these downstream responses involves the noradrenergic system located in the locus coeruleus of the brain stem. This system is associated with increased alertness and hypervigilance. As this neural circuitry becomes reorganized over time, the brain's defensive reflexes against previously neutral stimuli are maladaptively strengthened. This learned hyperresponsivity has lasting effects on the rest of the body (Bremner, 2006).

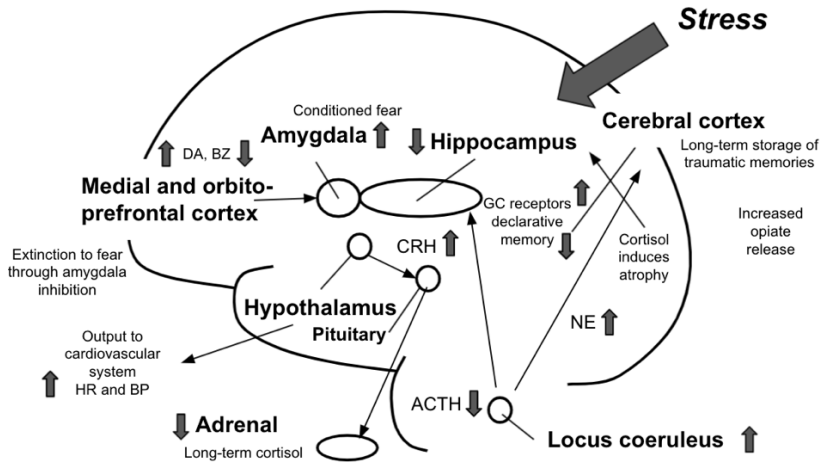

Figure 1. Lasting effects of trauma on the brain. Adapted from Bremner, 2006.

Neurobiological features of PTSD occur with abnormalities and further functional implications. The proinflammatory cytokines and stress hormones of the HPA axis do not exist in a vacuum, but are systemically impactful. Neuroendocrine shifts such as hypocortisolism and increased CRH drive abnormal stress processing and promote hippocampal atrophy, respectively. Parallel imbalances in the T3:T4 thyroid hormone ratio increase subjective anxiety (Sherin, 2011).

Variations in neurochemicals including catecholamines, serotonin, amino acids, and peptides each contribute to marked PTSD symptomatology. Increases in catecholamines such as dopamine and norepinephrine (NE) interfere with fear conditioning and increase arousal in the startle response. NE specifically increases pulse, blood pressure, and response to memories in individuals diagnosed with PTSD. Decreased serotonin levels in the many parts of the raphe nucleus disturb the dynamics between the amygdala and hippocampus, compromise anxiolytic effects, and increase vigilance. Decreases in amino acids such as GABA similarly increase anxiolytic effects, while increases in glutamate foster derealization and dissociation. Decreased plasma neuropeptide Y (NPY) leaves CRH and NE unopposed and upregulates the stress response. Increases in CSF β-endorphin levels foster numbing, stress-induced analgesia, and dissociation. These many changes stimulate the sympathetic nervous system (SNS) and trigger a potentially untimely fight-or-flight response. This hyperresponsivity of the CNS leads to altered processing of visceral and sensory stimuli (Sherin, 2011).

On a broader scale, traumatic stress in PTSD often co-occurs with global changes in immune function. These systemic changes include altered glucocorticoid sensitivity in immune cells, elevated proinflammatory cytokines, and shifts in immune cell distribution

(Neigh, 2016). Neigh (2016) described multiple candidate mechanisms of immune dysfunction in PTSD. While the dogmatic understanding of PTSD associates the disorder with elevated cortisol and catecholamine concentrations in response to stress, excessive activation of the HPA axis in this pathophysiology results in reduced circulating cortisol levels. In response to a sustained increase in CRH and this excess activation, CRH receptors are downregulated. This results in a reduction of downstream cortisol secretion and increased glucocorticoid sensitivity. These changes are linked to increased cytokine production and the hyper-inflammatory state comorbid with PTSD (Neigh, 2016).

The chronic low-grade inflammation consistent with PTSD is linked to numerous inflammatory biomarkers. The proinflammatory cytokines tumor necrosis factor α (TNF-α), interleukin-1β (IL-1β), and interleukin-6 (IL-6) have been implicated in the augmentation of PTSD symptoms in response to subsequent stressors. Cytokines additionally influence the HPA axis' signaling capabilities and impair cellular processes by stimulating oxidative stress. Studies have associated reactive oxygen species and proinflammatory cytokines with multiple sequelae of PTSD (Neigh, 2016).

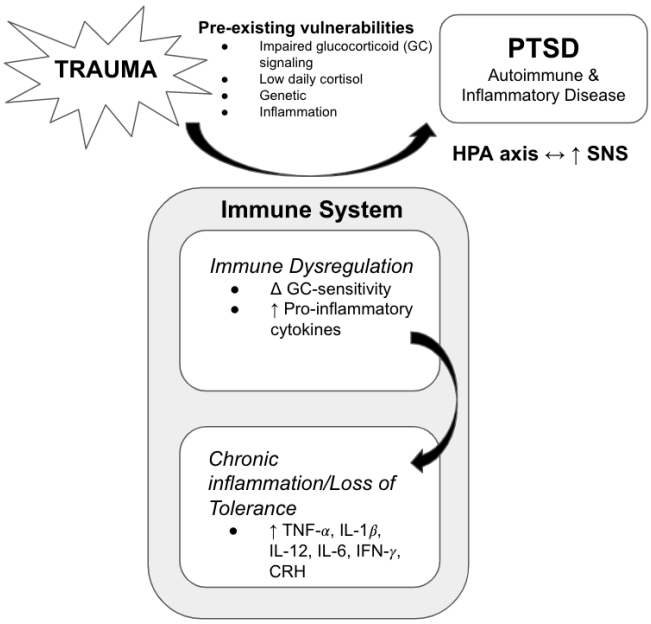

Figure 2. A psychoneuroimmunological model of PTSD. Adapted from Neigh, 2016.

While PTSD has been heavily correlated with several physical ailments, associations of this disorder with cardiovascular disease (CVD) are of particular concern. The cardiovascular consequences of psychological trauma were described by Arenson (2017) as occurring in both immediate and delayed fashions. Of note, individuals with PTSD are twice as likely to die from CVD compared to control groups. In the described participant group, each incremental increase in PTSD symptoms corresponded to a 20% increase in the risk of heart disease mortality (Arenson, 2017). Buckley (2001) observed that compared to non-trauma-exposed individuals, individuals carrying a PTSD diagnosis have higher resting heart rates and elevated blood pressures. These positive associations between PTSD and basal cardiovascular activity were consistent with established findings in this field of research (Buckley, 2001). Chronic cardiovascular

hyperresponsivity, and the catecholamine dysregulation discussed previously, could put individuals with PTSD at risk for further pathological changes.

In addition to CVD, PTSD has been established as a risk factor for both myocardial infarction (MI) and stroke (Remch, 2018). Remch (2008) went on to describe the negative role PTSD played in the individual healths of the study's participant pool. Weight gain, tobacco and alcohol use, hypertension, and hyperlipidemia are all comorbidities of PTSD. Therefore, the negative effects of these afflictions are inseparable from a patients' general condition and, in particular, their increased risk of cardiac incident (Remch, 2008).

At the intersection of the physical and emotional responses to stress is the gut. A system of bidirectional communication exists between the CNS and the digestive system called the gut-brain axis (GBA). Clapp (2017) expanded upon a growing field of research supporting the association of stress with gastrointestinal and extragastrointestinal diseases. Inversely, recent studies have also related dysbiosis and inflammation of the gut with mental illnesses such as anxiety and depression, which share prevalent symptomatology with PTSD. The resulting pathophysiology relating to this GBA dysfunction includes stress-induced mucosal immune alterations in intestinal permeability and impaired CNS ability to downregulate incoming visceral or somatic signals (Clapp, 2017).

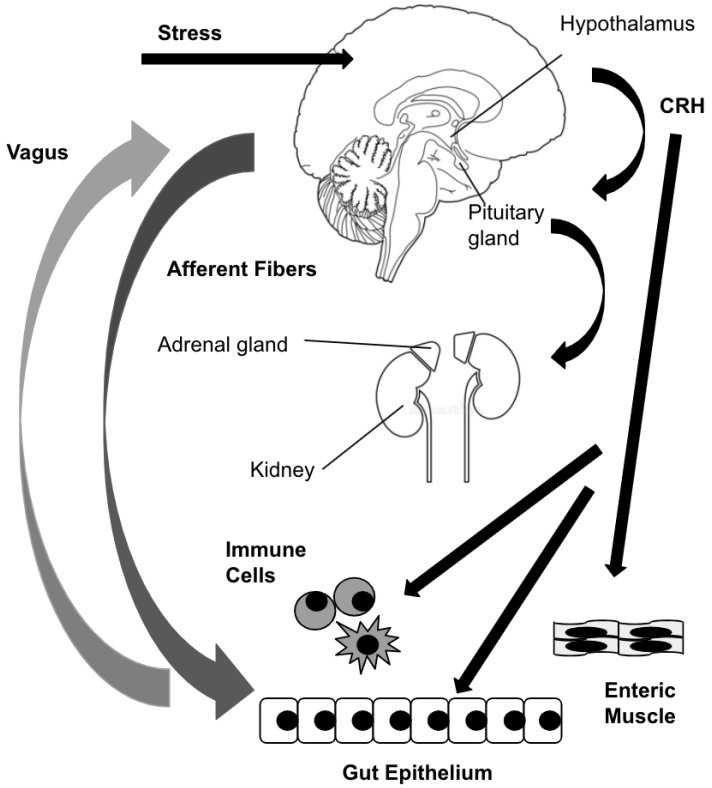

Figure 3. Gut-brain axis pathway diagram. Adapted from Clapp, 2017.

In a study conducted by Gradus (2017), PTSD was associated with diagnosed upper and lower GI disorders, as well as irritable bowel syndrome (IBS), gastroesophageal reflux disorder, and dyspepsia. The risk of GI disorder among PTSD patients was observed at 25% with an overall incidence rate 1.8 times higher than that of the general population. While the observed risk of malignancy varied with each specific disorder, compelling evidence was found associating PTSD with peptic ulcers, esophagitis, cholelithiasis, gastritis, and duodenitis. Additionally, these authors found evidence of effect modification by psychiatric PTSD comorbidities including depression and substance abuse (Gradus, 2017).

Unlike psychological comorbidities of trauma, which can develop in sequence or without a discernable pattern, physical post-traumatic symptomatology typically develops in parallel with the traumatic incident. Considerable physiological factors are common to both PTSD and chronic pain and are likely mutually reinforcing on a systemic level. The prevalence of PTSD is substantial in patients with chronic pain. Per Dr. Lorie DeCarvalho, Ph.D. (2019) of the U.S. Department of Veteran Affairs, 35% of current chronic pain patients also carry a diagnosis of PTSD compared to 3.5% of the general population. Inversely, the prevalence of chronic pain in individuals with PTSD is greater than in controls. Not only do individuals with PTSD experience higher rates of chronic pain, but this pain is quantified as increasingly intense when compared to control groups (DeCarvalho, 2019).

In addition to the increased incidence of chronic pain in individuals with PTSD, this affected population experiences various alterations in pain perception. Compared to control groups, individuals with PTSD displayed hyposensitivity to pain accompanied by hyper-reactivity to suprathreshold noxious stimuli. These features are likely attributable to alterations in emotional interpretation and sensory processing of painful stimuli (Defrin, 2008).

The inter-systemic effects of PTSD compound on one another and cause damage to the body over time. The compounding effects of these multiple pathologies accelerate the already grueling disease process of PTSD. These effects of chronic stress have secondary effects which place individuals at higher risk for comorbidities, alter their abilities to develop innate and adaptive immunity, shorten their telomeres, and place them at risk for serious illness, such as diabetes, heart disease, and COVID-19.

Racism at the Cellular Level

As stated by Kirkinis (2018), race as a social construct is well-established in the United States and has been used to categorize people into groups for centuries. This practice of grouping humans based on their eye shape, nose shape, hair texture, skin tone, etc. has resulted in *racial groupings*. These racial groupings exist in a social hierarchy that places Whites at the top and all people of color beneath that dominant group. As a result of this hierarchy, African Americans, Asians, Latinx, and Native Americans have and continue to endure systems of oppression as targets of variable forms of *racism*. Racism manifests as *racial discrimination* or the combination of ideological racial superiority with social structures associated with dominance and oppression. The subsequent normalization of these widespread inequities is called *structural racism* (Kirkinis, 2018).

Increasing evidence exists demonstrating the connection between racial discrimination and a variety of negative physical and mental health outcomes. Continuous with the definition of stress as "the inability to meet the demands of a situation," consequences exist for the experiential stress of racism or *racism-related stress*. When this race-based stress initiates a maladaptive psychological response, the resulting wounds to the self closely align with those observed in PTSD (Kirkinis, 2018).

A growing body of research exists which explores the impact of racist and discriminatory encounters on physical health. Empirical evidence supporting the validity of these studies consistently mirrors that of studies conducted in the field of chronic stress. Harrell et al. (2003) established that external stressors associated with acts of racism changed physiological functioning in affected individuals, ultimately contributing

to negative health outcomes. In this research, Harrell found that reactions to racism including anxiety and worry prompted rehearsed defensive reactions and aggressive responses as methods of coping and adaptation (Harrell, 2003). Examples of altered biological function in response to the physiological stress response include preterm or low birth weight deliveries, increased incidence of heart disease, and hypertension (Carter, 2007).

RESEARCH STRATEGIES

To ensure a high-quality review of literature, a comprehensive search was performed of peer-reviewed journals obtained from reputable databases. These databases included Google Scholar, NCBI, and PubMed Central. Further resources were obtained from the reference sections of each respective citation. All data and information discussed in this review were obtained by reading and reviewing this literature. Searches were refined based on article credibility and date of publishing.

DISCUSSION

Racial Disparities in Medicine

Independent of race's biological significance on an individual level, the societal and social consequences of racism are substantial and indisputable. Due to the continued dependence on race as a social stratifier in the United States, the resulting racial hierarchy has contributed to marked inequalities in power, opportunity, resources, and social status (Dominguez, 2008). These inequalities are experienced by individuals of affected populations at a molecular level. The weathering effects of living in a race-conscious society are greatest among people of color, especially African Americans (Geronimus, 2006). Research conducted in the realm of discrimination and racism suggests that the effects experienced by targeted individuals are consistent with those described in traumatic stress research (Carter, 2007). Pioneer to this field of study, Clark (1999) and Slavin (1991) asserted the perception of racism and racial hostility initiates stress reactions. The secondary and long-term effects of racial trauma manifest as systemic dysfunctions such as IBS and coronary artery disease (CAD), deterioration of birth outcomes, and emotional injury.

Studies cited by Carter (2007) suggest that acute exposure to racism is associated with increased cardiovascular activation (CVR), while past exposure to racism impacts current CVR in reaction to race-related stressors. This finding supports the increased prevalence of self-reported stress and physiological response following repeated or sequential racial encounters in people of color (Carter, 2007). In addition to this increased incidence of CAD, race has been identified as a precipitating factor in the lifetime occurrence of hypertension, myocardial infarction, and stroke. Hypertension is more

prevalent in African Americans than any other racial group and is known to increase the risk for CAD and stroke (American Heart Association, 2015). These cyclic comorbidities mimic those associated with cardiac dysfunctions in PTSD pathology.

The association between IBS and PTSD has been previously established in non-minority populations. Iorio (2014) investigated the same association in African American individuals, uncovering a significant relationship. This study produced results supporting an association between IBS and PTSD in people of color. African American individuals with IBS were twice as likely to suffer from PTSD, but this relationship is neither directional nor causal. Iorio went on to state that while the participants of this study carried histories of IBS before their subsequent PTSD diagnoses, it is impossible to determine which dysfunction preceded the other. IBS symptoms in this study's participants were often accompanied by bodily pain, inhibition of social functioning, and mental health deficits. Each of these additional symptoms negatively correlates to PTSD severity (Iorio, 2014).

The preceding physiological responses to racism and discrimination are moderated by variables of personality and cultural orientation (Harrell, 2003). Racism harms both physical and mental health. This impact, however, is not reflected in the theories of current psychological or psychiatric assessment models. Similar to the variability of the individual stress response, which depends upon appraisal and coping, researchers have asserted that stress outcomes relating to racial trauma vary based on racial group identity, frequency of discrimination, and severity of the traumatic event(s). The occurrence of such race-based incidents is positively associated with lower levels of physical health and higher levels of psychological distress (Carter, 2007). With these

observations in mind, emotional injury resulting from racial trauma should be assessed as a psychological symptom of racism with equal diagnostic significance to physical and mental health exam findings.

Race-based Medicine

Despite the failure of racially targeted drugs such as BiDil, a heart medication marketed toward African Americans, no clear guidelines exist on the use of race in medicine. Although sizable evidence exists disputing this notion, the belief that race is a reliable proxy for genetic difference has become insidiously embedded in medicine (Vyas, 2020). Subtle examples of this insidious insertion of race in medicine are observed in diagnostic algorithms and practice guidelines that adjust their outputs based on a patient's racial or ethnic identification. These algorithms are employed to guide clinical decision-making but, due to their consideration of race, actively perpetuate race-based health inequities. A significant danger of these race-adjusted algorithms is the potential swaying of clinical decision-making in favor of white patients (Vyas, 2020).

As stated by Vyas (2020), The American Heart Association's *Get with the Guidelines - Heart Failure Risk Score* predicts the risk of mortality in patients with acute heart failure. This race-corrected stratification tool creates inequities by raising the threshold for using clinical resources for black patients. By awarding three points to the risk scores of non-black patients, black patients are thereby regarded as lower risk. Similarly, equations used by cardiac surgeons to determine the risk of operative mortality and major complications quantify black patients as having up to 20% greater risk than white patients. When used preoperatively, this calculation may steer black patients away from such procedures (Vyas, 2020).

Similar race-considering algorithms and practices exist throughout medicine, namely in Urology, Pulmonology, Endocrinology, Oncology, Nephrology, and Obstetrics. In the case of obstetrics, the Vaginal Birth after Cesarean (VBAC) algorithm is used to estimate the probability of successful vaginal birth following a prior cesarean section and further counsel patients on their medical decisions. This algorithm accounts for race by subtracting from the projected success rate in patients identifying as black or Hispanic. The lower estimates of success produced from this algorithm may dissuade clinicians from recommending vaginal deliveries to people of color and have concretely resulted in higher rates of cesarean section in non-white women (Vyas, 2020). This dynamic actively exacerbates preexisting racial disparities in obstetrics and gynecology, including heightened maternal mortality and morbidity rates in black mothers (Vyas, 2019).

Further investigating the concept of obstetric racism, the Centers for Disease Control and Prevention (CDC) conducts national pregnancy-related mortality and morbidity surveillance to better understand the present differential reproductive outcomes in the United States. The Pregnancy Mortality Surveillance System (PMSS) defines a pregnancy-related death as one occurring while a woman is pregnant (or within 1 year of pregnancy) which is aggravated or otherwise caused by the pregnancy. Since the implementation of this surveillance, trends in pregnancy-related mortality ratios have revealed considerable disparities based on race and ethnicity (Centers for Disease Control and Prevention, 2020).

Per the CDC (2020), black mothers are three to four times more likely to die due to pregnancy-related causes than white women. Additionally, black women are more than

twice as likely to experience severe maternal morbidity. The causes of these pregnancy-related deaths vary to some degree, but the contributions of chronic health conditions such as hypertension, diabetes, and chronic heart disease have increased in recent years (Centers for Disease Control and Prevention, 2020). It is not by coincidence that the conditions which underlie a large number of pregnancy-related deaths are synonymous with both the conditions disproportionately affecting black and other minority populations, and those which co-occur with PTSD. This variable risk of complications based on race and ethnicity is likely precipitated by several factors including access to care, quality of care, prevalence of chronic disease, structural racism, and implicit biases (Hall, 2015).

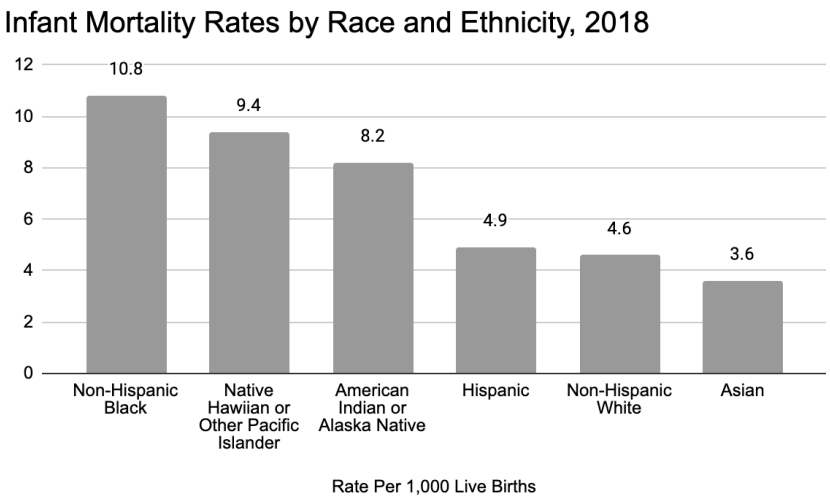

Figure 4. Graph of Infant Mortality Rates by Race and Ethnicity. Adapted from Centers for Disease Control and Prevention, 2020.

Unlearning Racial Bias

The National Academy of Medicine (NAM) has reported that racial and ethnic minorities receive lower-quality health care than their white counterparts, even when

income and education are taken into account. The NAM has additionally documented that despite the poverty in which people of color disproportionately live, this fact alone cannot account for their shortened lifespans when compared to white individuals. Perhaps even more disturbing is that black patients are more likely to receive "less desirable treatments." Khiara Bridges (2017), a professor of law and anthropology, offered examples such as higher rates of limb amputation and antipsychotic use, despite mounting evidence against them, as examples in her discussion of this second-rate treatment (Bridges, 2017).

With these facts in mind, scholars have begun to question the impact of racial biases, whether conscious or unconscious, in healthcare. While physicians' explicit racial bias is plausibly not to blame for these disparities, it is also unlikely that these inconsistencies in care have emerged as a result of physicians' intention to cause their patients harm. Physicians, despite their extensive medical educations, are not beyond exposure to society's negative racial discourse. Resulting unconscious negative attitudes about "othered" racial groups are likely to blame for the inferior care provided to patients of color. These individual biases, however, do not operate independently of the systems which, too, contribute to the compromised care afforded to these marginalized groups (Bridges, 2017).

Race, as a social construct, reflects the impact of polarized social experiences on health outcomes. However, race is often misrepresented in preclinical curricula as a biological category based on innate genetic differences. In this way, medical schools may potentially play a role in propagating physician bias. Fortunately, the use of race in medical education is an opportune area for improvement. Correction of this content

through the use of standardized language, appropriate contextualization, and evidence-based teachings will allow student physicians to unlearn race-based fallacies instilled by society (Amutah, 2021).

A step toward improving the uses of race and ethnicity in medicine includes careful interpretation of disease outcomes without assumption of race- or ethnicity-specific cause. Abandoning the use of race and ethnicity in medicine entirely is by no means a solution to this problem. Doing so would enable inequitable care systems to persist via a diversity-blind approach. Instead, the substitution of better disease predictors — such as genotypes, genetic ancestry, and biomarkers — could be used to address and eliminate inequities in research and medicine (Borrell, 2021).

Ultimately, the lived experience of racial trauma evokes pathophysiology in the body congruent to that of PTSD. The inclusion of racism in the trauma-focused care vernacular is imperative to standardize healthcare for people of all races and ethnicities. Intervention to mitigate the aforementioned inequities is both an urgent and necessary responsibility of all current and aspiring healthcare professionals. To this end, the maintenance of such racist tools and practices must be scrutinized and their improvement made a priority across all realms of medicine. Advocacy on behalf of the most marginalized individuals in our society will effect change to the benefit of those considered more centralized in this movement.

RECOMMENDATIONS FOR FUTURE STUDIES

Downstream disordered functioning following trauma exposure is well-established and verbosely supported, but discounts the ramifications of racism. A gap exists in the literature regarding the long-term effects of PTSD in non-white individuals, particularly concerning the role of racism as part of the disease process. Where research has been minimally and insufficiently conducted, it has focused primarily on the effects of racism in medicine and differential outcomes as they relate to African American patients. Because of these research gaps, it is impossible to assert a causal relationship exists between acts of race-based discrimination and observable physical and emotional symptoms consistent with the racial trauma response. This begs the question, how do these same effects impact other minority groups such as Hispanic/Latinx, Asian American Pacific Islander, and Native American individuals? Can the concept of traumatic discrimination rooted in racial trauma be applied to the discriminatory practices impacting individuals who identify as part of the LGBTQIAP+ community? Longitudinal studies focused on these minority groups could begin to fill this gap. Particular areas of interest include hypervigilance concerning the current political climate, PTSD across the lifespan in response to racial trauma, and the chronic stress sequelae related to COVID-19.

Progress in this field of study demands that the next edition of the DSM include racism as a potential stressor in PTSD. The current definition and criteria required for appropriate PTSD diagnosis neglect stressful events associated with the experience of racism and racial discrimination. Expansion of the conceptualization of trauma must account for non-life-threatening events which present a psychological threat and result in

emotional pain (Kirkinis, 2018). Without these evolved diagnostic criteria, affected populations will remain excluded from proper care and without appropriate therapeutic interventions.

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