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PTSD SUBTYPES, DEPRESSIVE PRESENTATION, DISSOCIATION, AND COGNITIVE APPRAISALS AND THEIR IMPACT ON CARDIOVASCULAR REACTIVITY RESPONSES TO STRESS

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by

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ABSTRACT

Both posttraumatic stress disorder (PTSD) and major depressive disorder (MDD) are related to an increased risk for cardiovascular disease (CVD), with links to ischemic heart disease, myocardial infarction, cardiac mortality, and other cardiac conditions. Given that these disorders are associated with increased medical costs, disability, and employer burden, research has investigated which factors increase the likelihood that one will develop CVD. While one hypothesis suggests that individuals who exhibit exaggerated responses to stress are at greater risk of developing CVD, individuals with PTSD/depression have been shown to exhibit both exaggerated and blunted stress responses. The purpose of the present study was to examine whether differing PTSD symptomatology, depressive presentation, dissociation, or cognitive appraisals of stressful events can predict the nature of one's cardiovascular response to stress. It was hypothesized that: (1) PTSD arousal would be positively related to cardiac reactivity and PTSD avoidance negatively related to cardiac reactivity; (2) depressive activation would be positively related to cardiovascular reactivity and depressive withdrawal negatively

related to cardiovascular reactivity; (3) dissociative symptoms would be negatively related to cardiovascular reactivity; and (4) challenge appraisals would be related to greater cardiac reactivity and threat appraisals to greater peripheral resistance. Female participants (N = 57) were administered the Clinician-Administered PTSD Scale (CAPS) and Structured Clinical Interview for DSM-IV Axis I Disorders (SCID) by graduate research assistants. Participants subsequently completed a demographic questionnaire and the Traumatic Life Events Questionnaire (TLEQ), Beck Depression Inventory-II (BDI-II), Dissociative Experiences Scale II (DES II), and Stress Appraisal Measure (SAM) to assess traumatic life events, depression, dissociation, and cognitive appraisals, respectively. Participants' cardiovascular reactivity (HR, CO, SBP, DBP, and TPR) in response to two stress tasks–mental arithmetic and public speaking–were assessed via impedance cardiography and an automated blood pressure cuff. Multivariate regression revealed support for some hypotheses as significant relationships were found between reactivity and PTSD avoidance, PTSD arousal, and depressive withdrawal. No significant relationships were found between reactivity and dissociation, depressive activation, or threat appraisals. This study provides preliminary support for the idea that differing psychological presentations can predict the type of cardiovascular response one exhibits in response to stress.

Keywords: cardiovascular disease, cardiovascular reactivity, psychophysiology, PTSD, PTSD subtypes, depression, depressive subtypes, activation, withdrawal, dissociation, cognitive appraisals, challenge appraisals, threat appraisals

CHAPTER I

Statement of the Problem

Research has noted a strong association between cardiovascular disease (CVD) and at least two psychological disorders: posttraumatic stress disorder (PTSD) and major depressive disorder (MDD) (Baune, Adrian, Arolt, & Berger, 2006; Jordan, Miller-Archie, Cone, Morabia, & Stellman, 2011; Mitka, 2005; Vaccarino et al., 2013). Both of these disorders are associated with a wide range of negative symptoms that can lead to an increased use of medical or mental health services. Though the link between these disorders and CVD is well-established, research has yet to determine the mechanisms by which one leads to the development of the other. This is especially important as research has found that individuals diagnosed with PTSD or MDD (especially the severe cases) tend to incur increased medical/healthcare costs and employer burden (Birnbaum et al., 2010). Specifically, studies that have investigated the impact that mental health conditions have on days out of role or DOR (i.e., the amount of days during which an individual is incapable of working due to health problems) have found that depression and PTSD are associated with some of the strongest individual-level effects on DOR per year (Alonso et al., 2011; Benjet, Casanova, Borges, & Medina-Mora, 2013).

One factor that has been linked to cardiovascular disease and poorer health outcomes in general is high cardiovascular reactivity. Cardiovascular reactivity has been hypothesized to explain the relationship between these disorders and CVD; and research has noted that individuals who suffer from PTSD or depression tend to exhibit greater cardiovascular reactivity in lab settings (Beckham et al., 2002; Matthews, Nelesen & Dimsdale, 2005; Tucker et al., 2007; Wolfe et al., 2000). This may suggest that

individuals who suffer from trauma-related/mood disorders exhibit an exaggerated response to stressful laboratory tasks as a result of chronic stress that they have been exposed to in their environment.

Although the literature is replete with studies attempting to examine the relationship between PTSD, MDD and cardiovascular reactivity, there are still mixed findings regarding the direction of the relationship. For instance, Kibler and Ma (2004) conducted a meta-analysis of 11 studies examining the relationship of depression to cardiovascular reactivity. They found small to moderate effect sizes that indicated a positive association between cardiovascular reactivity (in response to stress) and depressive symptoms. While some research has noted a positive relationship between depression and cardiac reactivity, where depressive symptoms are linked to increases in cardiovascular reactivity (Kibler & Ma, 2004; Matthews et al., 2005; Thornton & Hallas, 1999), other research has found an inverse relationship, where greater depressive symptoms are linked to blunted or attenuated reactivity (Carroll, Phillips, Hunt, & Der, 2007; Phillips, 2011a; Phillips, 2011b; Salomon, Bylsma, White, Panaite, & Rottenberg, 2013; Salomon, Clift, Karlsdòttir, & Rottenberg, 2009; York et al., 2007). There may be various explanations as to why some people exhibit blunted rather than exaggerated reactivity in response to stress.

A couple of factors that may explain the relationship include the activation factor (associated with depression) or the cognitive appraisals that individuals make when they encounter a stressful event. Thus, the current paper will attempt to describe PTSD and MDD, the prevalence of these disorders, potential mechanistic pathways to explain how PTSD or MDD act as risk factors in the development of cardiovascular issues, discuss the

role of exaggerated or attenuated cardiovascular reactivity in explaining negative health outcomes as well as attempt to delineate what other variables may impact the relationship between PTSD, MDD, and CVD.

Cardiovascular Disease

Cardiovascular disease, a term that encompasses a wide variety of diseases affecting the heart and blood vessels, is the leading cause of death in the United States (Heidenreich, et al., 2011; Murphy, Xu, & Kochanek, 2010; National Heart, Lung, and Blood Institute (NHLBIa), 2014; Spindler & Pedersen, 2005) as well as worldwide (Lopez, Mathers, Ezzati, Jamison, & Murray, 2006; Mathers, Boerma, & Ma Fat, 2009). It is predicted that between 2010 and 2030, the costs associated with CVD will increase exponentially. Specifically, Heidenreich and colleagues (2011) forecast that total indirect medical expenditures (i.e., costs associated with a loss of productivity) from 2010-2030 will increase from $171.7 billion to $275.8 billion; and total direct medical expenditures associated with CVD will multiply threefold—from $272.5 billion to $818.1 billion.

Accordingly, research has focused on attempting to determine which factors facilitate the pathogenesis of cardiovascular disease in order to provide recommendations that will foster primary prevention. Traditionally, the literature has enumerated numerous physiological risk factors associated with cardiovascular disease. They include hypertension, obesity, dyslipidemia, smoking, poor nutrition, hyperlipidemia, diabetes mellitus, and sedentary lifestyle [Centers for Disease Control and Prevention (CDC), 2011; Fryar, Chen, & Li, 2012]. In reviewing the data on which factors are thought to be causally related to the development of CVD, research has noted that only half of the variance is accounted for by the traditional cardiac risk factors (Wielgosz & Nolan,

2000). Similarly, other studies have found the traditional risk factors to account for less than 50% of the variance of atherosclerotic plaque burden (Rundek et al., 2013). Nevertheless, only a few studies have focused on investigating the impact that psychosocial factors may have on the development of CVD.

As aforementioned, two conditions that have been found to both precede and follow CVD are PTSD and MDD. Though there is a general consensus that there is some relationship between both these disorders and development or worsening of cardiovascular issues, research has yet to determine the specific mechanistic pathways that lead from one to the other. Some of the more common proposed pathways include biopsychological (e.g., dysregulation of the HPA axis, impaired immune functioning) or neurochemical (e.g., greater cytokine release, higher lymphocyte activation) pathways.

Posttraumatic Stress Disorder

Posttraumatic stress disorder is a term used to describe a condition that develops after an individual has witnessed and/or experienced a traumatic event of one form or another (American Psychiatric Association [APA], 2000). Though the diagnosis of PTSD was originally entered into the Diagnostic and Statistical Manual (DSM) as a result of the high number of veterans seeking treatment, individuals of all ages can develop and be diagnosed with PTSD. In the past, the diagnostic criteria for PTSD included: (1) must be threatened or experience an actual serious injury or death, (2) exhibit feelings of horror, fear, or helplessness as a result of the event(s), (3) constantly re-experience the event, (4), persistently engage in avoidant behavior or display a numb response in multiple settings, (5) experience increased and persistent symptoms of arousal, and 6) display these symptoms for one month or more (APA, 2000). If these symptoms caused a marked,

negative change in functioning (whether in the social or occupational realm) then a diagnosis of PTSD was given. In contrast, the DSM 5 has removed the language stipulating that the individual must experience fear, horror, or helplessness as it was found to have no utility in predicting PTSD onset. Furthermore, while the symptoms associated with PTSD have previously been grouped into three distinct categories–re-experiencing of the symptoms, avoidance, and hyperarousal–DSM 5 proposes four categories: re-experiencing, avoidance, negative cognitions and mood, and arousal (APA, 2013). Given that the measures used in this study were based on the DSM-IV criteria, the three aforementioned categories will be discussed below.

Re-experiencing symptoms

If an individual is suffering from PTSD, then he will undoubtedly experience symptoms surrounding the traumatic event. Individuals may report dreaming (or having nightmares) often about the event. They may experience recurrent and invasive thoughts or memories about the event and may even feel as if they are re-experiencing the traumatic event in the form of dissociative flashback episodes, hallucinations, re-experiencing of the event when intoxicated or upon awakening, or even illusions. For instance, these symptoms are likely to occur around the anniversary of the traumatic event. Additionally, the individual may feel extreme psychological distress or exhibit increased physiological reactivity when subjected/exposed to cues (whether internal or external) that resemble or symbolize the traumatic event (APA, 2000).

Avoidance

One of the more common features associated with all anxiety disorders is avoidance. In the case of PTSD, affected individuals may attempt to avoid any person,

place, feeling, thought, or activity that even remotely reminds them of the event. Also, in addition to displaying restricted affect, individuals may report less interest in participating in certain activities. This withdrawal may lead the individual to become separated/estranged from others and may result in them feeling detached from others. Lastly, the individual may feel as if his future is shortened in that he may not expect to ever have children, get married, or live a normal life (APA, 2000).

Hyperarousal

As a result of their traumatic experience, individuals may experience heightened symptoms of arousal. Some of the more common symptoms may include insomnia (difficulty falling and staying asleep), attention/concentration difficulties, hypervigilant behavior, frequent outbursts of anger or irritability, or an exaggerated startle response (APA, 2000). One of the more common examples of hyperarousal involves veterans' reactions to certain events—from taking cover at the sound of a slamming door or swerving frantically on the road to avoid debris that reminds them of an IED.

Prevalence

Though the most recent iteration of the DSM lists PTSD under the "Trauma and Stressor Related Disorders" chapter (APA, 2013b), PTSD originally fell under the category of anxiety disorders; and at 28.8% of all disorders, Kessler and his colleagues (2005a) note that they are the most prevalent category of psychological disorders. One of the earliest estimates of the lifetime prevalence rate of PTSD was provided via the National Comorbidity Study (NCS), a study that attempted to investigate the prevalence of mental health disorders, among other things. The study, which was comprised of a sample of over 8,000 Americans, found an estimated lifetime prevalence rate of 7.8%;

and women were 2 times more likely than men to experience PTSD during their lifetime—10.4% versus 5%, respectively (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995).

Subsequently, the National Comorbidity Survey Replication (NCS-R) was conducted with a sample of approximately 9,282 Americans; and they estimated that the lifetime prevalence rate of PTSD was 6.8% (Kessler et al., 2005b), a decrease of 1%. More recently, Kilpatrick and colleagues (2013), assessed the prevalence of PTSD–according to DSM-IV and DSM-5 criteria–in a national sample of 2,953 U.S. adults. PTSD caseness was determined using either composite (i.e., symptom criteria met to a combination of event types) or same (i.e., all symptoms criteria met to the same event type) event definitions. When based on the DSM-5 criteria, they found lifetime prevalence rates of 9.4% and 8.3% for composite event PTSD and same event PTSD, respectively.

When reviewing the data on gender differences within the PTSD population, Kessler et al. (2005a) found that the lifetime prevalence rate of PTSD was 3.6% among men and 9.7% among women. A more recent study by Pietrzak, Goldstein, Southwick, & Grant (2011) estimates the lifetime prevalence of PTSD to be 4.1% among men and 8.6% among women. With regards to the 12-month prevalence rate of PTSD, it was estimated at 1.8% among men and 5.2% among women (National Comorbidity Survey, 2005). This is in line with a previous finding that women are more likely than men to suffer from PTSD (Michopoulos, Rothbaum, Grinstead, Rothbaum, & Ressler, 2015) even though men are more likely to experience potentially traumatic events (Pratchett, Pelcovitz, & Yehuda, 2010; Tolin & Foa, 2006). In one study in particular, women were one-third less

likely to report experiencing a traumatic event–trauma being categorized as a unitary category–than men (Tolin & Breslau, 2007).

The NCS-R also investigated the 12-month prevalence of psychological disorders. Again, anxiety disorders were the most prevalent category of disorders in a one-year period at 18.1% (Kessler, Chiu, Demler, & Walters, 2005b); and the estimated 12-month prevalence rate of PTSD was 3.5%, with 36.6%, 33.1%, and 30.2% of individuals falling within the serious, moderate, and mild severity level, respectively (Kessler et al., 2005b). In comparison, when based on the DSM-5 criteria, Kilpatrick et al. (2103) found past-12-months PTSD prevalence rates of 5.3%% and 4.7% for composite event PTSD and same event PTSD, respectively.

Relationship between PTSD and CVD

Numerous studies have found an association between CVD and PTSD; and more specifically, even after controlling for certain confounding factors, the link between PTSD and cardiovascular issues remain. Research has noted a positive association between PTSD and chronic ischemic heart disease (IHD; Kang, Bullman, & Taylor, 2006), coronary heart disease (CHD; Vaccarino et al., 2013), angina pectoris and nonfatal myocardial infarction (Kubzansky, Koenen, Spiro III, Vokonas, & Sparrow, 2007), cardiovascular disease (Dedert, Calhoun, Watkins, Sherwood, & Beckham, 2010; Sawchuk et al., 2005), myocardial ischemia (Turner, Neylan, Schiller, Li, & Cohen, 2013), incident heart failure (Roy, Foraker, Girton, & Mansfield, 2015), arterial disorders/peripheral arterial disease (Schnurr, Spiro III, & Paris, 2000; Spitzer et al., 2009), and atrioventricular conduction defects and infarctions (Boscarino & Chang, 1999).

In addition, Ahmadi et al. (2011) sought to determine whether or not PTSD was linked to atherosclerotic coronary artery disease in a sample of 637 veterans. After analyzing individuals' coronary artery calcium (CAC) scores, they found that veterans with PTSD had more CAC in comparison to their non-PTSD counterparts. Additionally, results of a linear regression revealed that PTSD was an independent predictor of both the extent and presence of atherosclerotic CAD (Ahmadi et al., 2011). Findings such as these has led research to try to determine the mechanistic pathways that clarify how PTSD leads to cardiovascular disease, and consequently, negative outcomes. Though there are numerous proposed hypotheses, only two will be described herein.

HPA Axis Dysregulation and Allostatic Load

One hypothesis that has been proposed to explain the link between PTSD and CVD involves the hypothalamic-pituitary-adrenal axis and one's allostatic load. The HPA Axis, refers to the interactions between the hypothalamus, pituitary gland and adrenal glands; and it is usually triggered in response to a stressor. During non-stressful times, the nervous and endocrine systems work in conjunction with one another to maintain the body at a balanced, or homeostatic, state. Negative and positive feedback loops are instrumental in ensuring that everything in the body functions normally. Similarly, allostasis is a term used to describe the way in which the body's physiological systems fluctuate in order to meet external force demands (Bedi & Ahora, 2007).

If an individual is constantly being stressed, the chronic activation of the HPA axis (and the consequent neuroendocrine or neural responses) may adversely affect the body's allostatic load over an extended period of time. Thus, Schneiderman, Ironson, and Siegel (2005) notes that research has proposed that in this way, increases in the body's

allostatic load is related to chronic disease. This theory is supported by research which has shown that an increase in allostatic load, specifically associated with PTSD, is linked to several diseases including cardiovascular disease, hypertension, and high cholesterol (McFarlane, 2010). The chain of events commences when an individual is exposed to a stressor, at which point the HPA axis gets activated (Guilliams & Edwards, 2010). The HPA Axis then sets off a chain of events: the secretion of two peptide hormones (corticotrophin-releasing factor and arginine vasopressin) which eventually results in the posterior pituitary gland producing adrenocorticotropin hormone, which produces hydrocortisone (i.e., cortisol).

Glucocorticoids (a class under which cortisol falls) are necessary hormones in the body in that they help to mediate the effect between chronic stress and pathological effects (Sapolsky, 1996) and modulate the physiological pathways (e.g., brain, immune, or metabolic functions) that are associated with stress (Cavalcanti-Ribeiro et al., 2012). However, an overabundance of glucocorticoid secretions (such as during traumatic or chronically stressful events), can have adverse effects. Specifically, while an increase in glucocorticoids are an integral part of the stress response and necessary to redistribute energy resources, chronic/long-term glucocorticoid elevations inhibits immune functioning, increases susceptibility to disease, and starves tissues of essential resources (Conrad, 2008). In line with these findings, research has found that an increase or sustained cortisol secretion can lead to physiological changes that may weaken or impair one's immune functioning (Randall, 2011). In the case of individuals with PTSD, Walczewska, Rutkowski, Wizner, Cwynar and Grodzicki (2011) propose that the adrenergic axis is constantly being stimulated.

Therefore, individuals with PTSD are more likely to develop chronic health problems as a result of disturbances in the HPA and sympathoadrenal axis (Bedi & Ahora, 2007). Chronic stress leads to an increase in the allostatic load and amount of glucocorticoid receptors, higher cortisol suppression due to dexamethasone, decreased basal cortisol levels, frequent activation of norepinephrine, and sensitized pituitary glands. This leads to endocrine and hormonal abnormalities which are associated with an increase in the level of catecholamines in the body.

Though the release of catecholamines such as norepinephrine and epinephrine during periods of stress is a normal function of the adrenal glands, chronic catecholamine elevations have been linked to the development of arteriosclerosis (Jansen & Emerson, 2012). Additionally, catecholamines can have effects on the heart, its vasculature, and platelet functioning (Bedi & Ahora, 2007). Since platelet functioning is adjusted via the increasing circulating catecholamine levels, catecholamines can lead to (1) an increased aggregation of platelets, (2) increased platelet secretion of numerous products (such as platelet activating factor which causes vasoconstriction) (Bedi & Ahora, 2007), and (3) a higher level of agonist actions on platelet functions (Anfossi & Trovati, 1996). These changes in platelet functioning may accordingly increase the likelihood of cardiovascular morbidity and mortality (Bedi & Ahora, 2007). The link between heart disease mortality and PTSD (Ahmadi et al., 2011), serious psychiatric/medical morbidity and PTSD (Supelana et al., 2016), as well as risk of fatal CHD and PTSD symptom level (Kubzansky et al., 2007) has been found by other studies.

Immune Functioning

Another hypothesis that has been proposed to explain the link between PTSD and

CVD revolves around the role of the immune system. In addition to the HPA axis, the sympathetic nervous system (SNS) helps to modulate immune functioning via various mechanisms—from promoting the release of cytokines or interleukins (IL) to activating the production of T-cells (Kibler, Tursich, Ma, Malcolm, & Greenbarg, 2014). Cytokines are small, soluble proteins released by cells in the body (Jaimes, Inokuma, McIntyre, & Mittar, 2011) that act as intercellular signals that enable cells to respond to infection or injury (Kibler, 2009). They may also have direct effects on the interaction of and communications with other cells (Zhang & An, 2007) as well as multiple effects on a single cell or varying effects on different cells (Coondoo, 2011). Some of the more common cytokines include interleukins, lymphokines, chemokines, and monokines (Zhang & An, 2007).

Pro-inflammatory cytokines are an integral part of the immune system; and they are created predominantly by activated white blood cells called macrophages which help to up-regulate inflammatory reactions in the body (Zhang & An, 2007). As the name suggests, they are responsible for promoting inflammation at the site of injury or infection, and are also released in greater volumes during periods of stress (Kibler, 2009). However, chronic inflammation has been proposed as the mechanism that explains the relationship between PTSD and CVD (von Känel et al., 2007; von Känel et al., 2010), with studies demonstrating that individuals with PTSD evidence higher levels of inflammation (Gill, Saligan, Woods, & Page, 2009; Sondergaard, Hansson, & Theorell, 2004).

For example, inflammation has been shown to play a major role during all of the stages of progression of atherosclerosis (Libby, Ridker, & Hansson, 2009) and

atherothrombosis (van Leuven et al., 2008; Willerson & Ridker, 2004). Athero-thrombosis, is a term used to describe a complex disease which develops as a result of inflammation, cholesterol deposits, and thrombus (i.e., blood clot) formation (Fuster, Moreno, Fayad, Corti, & Badimon, 2005; Tatsumi & Mackman, 2015); and approximately 80% of all sudden cardiac deaths (SCD) have coronary artery disease (e.g., atherothrombosis) as the underlying cause (Albert, Ma, Rifai, Stampfer & Ridker, 2002; Chugh et al., 2008).

Moreover, numerous studies have noted that in comparison to controls, subjects with PTSD exhibit higher C-reactive proteins (Spitzer et al., 2010), interleukin-6 (Gill, Vythilingham, & Page, 2008; Maes, Lin, Delmeire, Van Gastel, & Kennis, 1999; Rohleder, Joksimovic, Wolf, & Kirschbaum, 2004), and interleukin-1β (Gola et al., 2013; Spivak et al., 1997; Tucker et al., 2004), all of which are pro-inflammatory proteins or cytokines. Thus, it is believed that individuals with PTSD experience changes in their immune response that lead to chronic inflammation which can have deleterious effects on the heart and body. Though some research has not replicated the above-mentioned findings, Gola and colleagues (2013) note that the inconsistent findings may be a result of small sample sizes, variations in trauma experience (childhood versus adulthood), differences in the sample characteristics, length of time between trauma and start of study, and differences in PTSD symptom severity, among other things.

Major Depressive Disorder

Major depressive disorder (MDD) is a term used to describe the presence of persistent depressed mood or loss of interest or pleasure that is accompanied by numerous other symptoms. According to the APA (2013), one of the primary criteria for

this diagnosis is that an individual must experience at least 5 symptoms during a 2-week period, and these symptoms must represent a change in previous functioning. These symptoms may include (1) depressed mood (that is present most or almost every day), (2) a distinctly diminished interest or pleasure in most or even all activities, (3) a change in appetite as evidenced by weight gain or significant weight loss not associated with dieting, (4) hypersomnia or insomnia almost every day, (5) psychomotor retardation/agitation almost every day (that is noticeable by others), (6) loss of energy and fatigue, (7) feelings of excessive or inappropriate guilt or worthlessness, (8) indecisiveness or a diminished ability to concentrate or think, and (9) frequent thoughts of death and/or suicidal ideation, or a suicide attempt or plan (APA, 2000). Once an individual is determined to have MDD, then it is categorized as either a single episode or a recurrent one.

Prevalence

In terms of the lifetime prevalence of psychological disorders, Kessler and his colleagues (2005a) note that major depressive disorder is the most prevalent lifetime disorder; however, in terms of the most prevalent category of disorders, it comes in at third place (20.8%) following anxiety (28.8%) and impulse-control disorders (24.8%). In surveying a sample of 9,282 Americans, they found a lifetime prevalence rate of 16.6% in the general population. More recent studies have found lifetime prevalence rates of major depressive disorders and major depressive episodes in the U.S. to be 28% (Vandeleur et al., 2017) and 19.2% (Bromet et al., 2011), respectively.

With regards to the 12-month prevalence, in 2015, it was estimated that approximately 16.1 million Americans (above 18 years of age) had experienced at least

one major depressive episode in the past year (National Institute of Mental Health [NIMH], n.d.). Similarly to the prevalence of PTSD, the estimated 12-month prevalence of a major depressive episode was higher in women than in men—8.4% versus 5.2%, respectively. Moreover, the World Health Organization (WHO) notes that among mental and behavioral disorders, the heaviest burden of disability falls on major depression, where 8.3% of all U.S. years lived with disability (YLD) and 3.7% of all U.S. disability-adjusted life years (DALY) is accounted for by major depressive disorder (NIMH, n.d.). This was echoed by Murray (2013) which found that major depressive disorder was second on the list of the diseases with the largest number of YLDs in 2010.

Relationship between Depression and CVD

The relationship between CVD and depression has been supported by several studies which have found that people with cardiovascular disease are more likely to experience depression following the cardiac event (Mitka, 2005; Thombs et al., 2006; Zuidersma, Ormel, Conradi, de Jonge, 2012). However, the relationship between these two variables seems to be bidirectional. Specifically, seemingly healthy individuals with depression have an increased likelihood of experiencing heart problems or future incidence of cardiovascular disease (Mitka, 2005); and depressed individuals are two to three times more likely to develop coronary heart disease than their non-depressed counterparts (NHLBI, 2014b). Additionally, individuals who have no history of depression may develop depressive symptoms following a heart attack or other heart failure (Mitka, 2005), with some arguing that there is consistent evidence that depression is a consequence of coronary heart disease, among other things (Prince et al., 2007). Moreover, Khawaja, Westermeyer, Gajwani, and Feinstein (2009) note that both cross-

sectional and longitudinal studies suggest that there is bidirectional link between coronary artery disease and depression.

This has led some to suggest that depression should be thought of as a cardiac risk factor (along with PTSD and other more traditional cardiac risk factors), as research continues to find that patients with cardiac issues and depression have a worse prognosis and poorer outcomes (Carney et al., 2008; Hare, Toukhsati, Johansson, & Jaarsma, 2014; Lichtman et al., 2014; Parashar et al., 2006). This is made more salient by the fact that several studies have found that individuals with cardiac issues and depression have a higher risk of cardiovascular morbidity and/or mortality. A review of the literature revealed that depression or depressed affect was related to fatal CHD (Whang et al., 2009), post-AMI mortality (Malach & Imperato, 2004), fatal or non-fatal myocardial infarction (Saran, Puri, & Agarwal, 2012), coronary death (Wu & Kling, 2016), all-cause mortality and cardiac mortality (Meijer et al., 2011), and CHD and MI (Gan et al., 2014).

Biopsychological Links between Depression and CVD

Due to the preponderance of information on the physiological changes associated with depression, many theories have been proposed regarding the relationship between depression and heart disease. Some of the theories regarding the physiological mechanisms include: (a) alterations in platelet activity as platelets are responsible for thrombus formation; (b) inflammation, as an essential contributor to CAD pathogenesis is atherosclerosis, which is often preceded by inflammatory cytokines; (c) heart rate variability as decreases in HRV are linked to depression, CVD, mortality, and anxiety; and (d) endothelial dysfunction (Mavrides & Nemeroff, 2015; Saran et al., 2012). Some of the theories regarding social and behavioral mechanisms include: physical inactivity

and smoking status (e.g., individuals who are depressed may engage in maladaptive habits, such as smoking, which may help to foster coronary disease); anhedonia; somatic symptoms (e.g., fatigue and difficulty sleeping); and increased social vulnerabilities, such as low perceived social support (Ye, Denton, Wasson, & Davidson, 2013).

Reverse Causation and Depression

One study has attempted to use reverse causation and the dose-response hypothesis in order to show that depression is causally linked to coronary heart disease. A dose-response relationship is a term used to describe the association between the level of exposure (or dose) and the proportion of individuals who exhibit specific and significant biological changes in incidence or the degree of change (the response) (EPA, 2011). In this case, Brunner et al. (2014) were interested in determining whether or not depressive disorder would exhibit a dose-response relationship with CHD events over the course of 20 years. In reviewing studies regarding depression and CVD, Brunner and his colleagues noted that little attention had been given to the reverse causal relationship (i.e., depression of a vascular origin). Thus, they proposed that:

> With repeated exposure measurements over a long period of adult life, causal inference would be strengthened if (a) reverse causation was excluded as an explanation for the link between depressive disorder and vascular events, and (b) a dose–response effect was evident (Brunner et al., 2014, p. 341).

Brunner et al. (2014) utilized data from the Whitehall II study, a cohort cardiovascular study of 10,308 civil servants in London. Participants completed the General Health Questionnaire (GHQ-30), the Center for Epidemiologic Studies Depression Scale (CES-D), and underwent a clinical examination during 1985-1988; they

were then seen in the clinic quinquennially (1991-2009). Results revealed that over two decades, the frequency of depressive disorder caseness exhibited a dose-response relationship with CHD events (Brunner et al., 2014). Additionally, after analyzing the dose-response based on 10-year cycles of observation, they determined that their inference that there is a causal relationship between depression and CHD was supported.

Cardiovascular Reactivity

One hypothesis that has been proposed to explain how CVD develops in response to stress revolves around cardiovascular reactivity (CVR). The cardiovascular reactivity hypothesis posits that large or exaggerated cardiovascular reactions in response to stress can lead to poorer health outcomes, such as an increased risk of developing future heart disease (Hamer & Malan, 2010), hypertension (Chida & Steptoe, 2010) and even atherosclerosis (Carroll, Phillips, Der, Hunt, & Benzeval, 2011). In this sense, individuals who are constantly stressed are thought to also experience chronic stimulation of the autonomic nervous systems (Kibler, 2009); thus, it is hypothesized that individuals who exhibit high CVR in response to lab stressors are displaying reactions that are consistent with the way they normally respond to stress in the real world (Phillips, 2011a). Cardiovascular reactivity can be measured in numerous ways; but only three will be mentioned below: blood pressure, heart rate and impedance cardiography.

Blood Pressure

Blood pressure measurement is an important part of clinical practice as it can be used in multiple ways; from screening for hypertension, assessing cardiovascular risk, or to determine risk prior to conducting certain medical procedures (Jahangir & McPherson, 2013). Blood pressure measurements can be taken using direct and indirect methods. The

direct method uses an intra-arterial catheter to obtain the blood pressure reading (Jahangir & McPherson, 2013); however, due to the invasiveness of this method, the indirect method may be used instead. This may involve using a sphygmomanometer and manual cuff (the auscultatory method) or an automated blood pressure device which removes observer bias (Andersen, 2009).

Regardless of which method is used, the clinician can obtain both systolic and diastolic blood pressure readings. The systolic blood pressure (SBP) reading represents the maximum amount of pressure the heart exerts during heartbeats; whereas the diastolic blood pressure (DBP) reading represents the pressure in the arteries between heart beats (Sheps, 2014). High and low blood pressure readings can both be problematic; however, more attention tends to be given to the SBP as a higher number can be associated with cardiovascular disease in older adults (American Heart Association, 2014). Additionally, pulse pressure, which is represented by the difference between the systolic and diastolic blood pressure, is an important figure as high pulse pressure readings are associated with a greater risk of developing cardiovascular disease (Sheps, 2014).

Heart Rate

Another measure of cardiovascular reactivity is heart rate. As the name suggests, heart rate is a term used to describe the amount of times one's heart beats per minute (bpm). The average resting heart rate is about 60-80 beats per minute (Cook, Togni, Schaub, Wenaweser, & Hess, 2006). Several epidemiological studies have found that a high heart rate is associated with an increased risk of cardiovascular events as well as all-cause mortality; however, the strength of this relationship has been more pronounced in men than in women (Perret-Guillaume, Joly, & Benetos, 2009). Additionally, resting

heart rate has been identified as an independent risk factor for cardiovascular disease (Cook et al., 2006; Cooney et al., 2010; Fox et al., 2007; Hjalmarson, 2007) even after controlling for numerous risk factors and characteristics.

Impedance Cardiography

Impedance cardiography (ICG) is a non-invasive, cost-effective technique a method for monitoring hemodynamics (i.e., blood flow/circulation), including cardiac output and stroke volume (Parry & McFetridge-Durdle, 2006; Siedlecka, Siedlecki, & Bortkiewicz, 2015; Turner, 2000). It can also be used to assess cardiovascular function (DeMarzo, 2013) or to identify the presence of cardiovascular abnormalities that may serve as risk factors for CVD (Bour & Kellett, 2008; DeMarzo, 2013). The use of ICG is based on Ohm's law and presumes that when a current travels through a conductor, variations in impedance are directly proportional to changes in voltage (Sodolski & Kutarski, 2007). The ICG and electrocardiograph (ECG) electrodes are placed at the base of the neck and at the xiphoid sternal junction. When the strip emits an alternating electrical current (of high frequency but low-amplitude), the current passes through the chest. The changes in voltage are proportional to the changes in impedance between the receiving electrodes (Cybulski, Strasz, Niewiadomski, & Gasiorowska, 2012).

While the literature is replete with studies that have assessed cardiac parameters such as heart rate and blood pressure, fewer studies have assessed other cardiac parameters such as cardiac output (CO) and total peripheral resistance (TPR). This is especially salient given that both CO and TPR are important indices of one's underlying hemodynamic patterns (Hill, Sollers III, & Thayer, 2013). For example, Heldt (2006) states that CO and cardiac filling measurements are important for monitoring the

progression of disease and diagnosing cardiac conditions early. He further notes the clinical utility of CO as during blood loss CO drops precipitously–in comparison to monitored systolic arterial blood pressure which decreases by a nonspecific amount–alerting the clinician to impending hemodynamic emergencies.

Additionally, TPR plays an essential role in the maintenance of healthy as well as pathological blood pressure; and alterations in peripheral vasculature functioning have been associated with both the etiological and pathophysiological course of cardiovascular disease (Hill et al., 2013). In spite of their clinical significance, obtaining measurements of CO is difficult (Fazeli & Hahn, 2012) or invasive/restrictive in methodology (Haslam, Gordhandas, Ricciardi, Heldt, & Verghese, 2011); and obtaining a reliable estimate of TPR tends to be a costly and complex process that requires sophisticated instrumentation (Hill et al., 2013).

In addition to assessing heart rate and blood pressure, the present study used impedance cardiography, as it is a non-invasive, cost-effective technique for obtaining these abovementioned cardiac parameters. Although each of these cardiovascular variables–HR, SBP, DBP, CO, and TPR–individually provide meaningful information about one's cardiovascular patterns, assessing all of these cardiovascular parameters provides a more comprehensive picture of an individual's hemodynamic patterns and the relationship to the psychological variables in question (i.e., PTSD, depression, dissociation, and cognitive appraisals). This in turn can help tailor the nature of recommendations made to address the varying cardiovascular reactivity patterns in response to stress found in this study.

PTSD and Reactivity

The cardiovascular reactivity hypothesis is supported by numerous studies which have found that most individuals with PTSD exhibit elevated physiological reactivity in response to stressors. One study by Wolfe and colleagues (2000), evaluated psychophysiological reactivity in female veterans, 30 of which were military veterans and six of which were former service organization volunteers who had experienced war-zone trauma in Vietnam. Eight of the participants met criteria for current war-related PTSD (PTSD-C), eight met criteria for lifetime war-related PTSD (PTSD-L), and 20 of the participants did not meet criteria for PTSD at any time (PTSD-N). Participants' HR, SBP, DBP and skin conductance (SC) measurements were taken during a baseline, neutral stimuli (e.g., snow scenes accompanied by light classical music), and trauma-related stimuli (e.g., audiovisual material that included explicit scenes of caretaking, treatment, and death, that was representative of women's wartime experiences) phase. Participants also used a joystick to make subjective units of distress (SUDS) ratings during the stressors. Although the three groups did not differ significantly during the baseline condition, women with PTSD evidenced significantly greater physiological reactivity (i.e., SC and BP) under trauma-cued conditions than the PTSD-N group. Furthermore, individuals with current PTSD evidenced greater subjective distress than individuals in the other two groups (PTSD-L and PTSD-N).

Similarly, Beckham et al. (2002) investigated cardiovascular reactivity in 118 male Vietnam combat veterans, 62 with PTSD and 56 without PTSD. All of the participants were asked to recall and eventually relive a self-chosen anger memory (i.e., a time they felt so angry they felt like they would explode). Participants' HR, SBP and DBP were measured continuously during the session and involved a 10-minute initial

resting baseline as well as measurement of physiological responses during relived anger as well as recovery. They found that veterans with PTSD exhibited a greater DBP response, felt angry quicker than their non-PTSD counterparts, reported greater anxiety and anger, and evidenced a greater magnitude of DBP during the recovery period.

Another study by Tucker et al. (2007), examined autonomic reactivity in 60 directly exposed survivors of the Oklahoma City bombing and 60 community comparison subjects 6.5 to 7 years after the event. Physiological reactivity (i.e., HR, SBP, DBP, and mean arterial pressure) was assessed before, during, and after the participants participated in a semi-structured interview to elicit their memories of the event. Although the participants in the survivor group evidenced low PTSD symptoms/diagnoses (attributed to healing over time or resiliency), they still evidenced greater physiologic reactivity (i.e., HR, SBP, DBP, and mean arterial pressure) than the participants in the community comparison group.

While several studies assessed physiological reactivity during discrete periods of time prior to and following a stressor, Buckley, Holohan, Greif, Bedard, and Suvak (2004) assessed 24 hour levels of ambulatory HR and BP in 19 veterans with chronic PTSD and 17 veterans who never met criteria for PTSD. All of the participants wore an ambulatory monitor (for a full 24-hour period) that took measurements (e.g., HR, SBP, and DBP) every 20 minutes and every 120 minutes during specified waking and sleeping hours, respectively. While wearing the ambulatory monitors, participants were asked to keep an activity log recording information such as caffeine intake, medication intake, cigarette smoking, posture, stress ratings, etc. Participants with PTSD evidenced elevated basal HR and higher BP reactivity during times of affective distress, even after

accounting for behavioral and metabolic influences such as smoking status or posture

Furthermore, a meta-analysis by Pole (2007) investigated the psychophysiology of PTSD across four different types of studies comparing individuals with and without PTSD. It included: 17 standardized trauma cue studies (which assess elevated psychophysiological reactivity to trauma reminders); 22 idiographic trauma cue studies (which assess psychophysiological reactivity as participants recall their own personal traumatic event); 25 startle studies (which assess exaggerated startle responses common to individuals with PTSD); and 58 resting baseline studies (which assess persistence of elevated psychophysiology levels in the absence of stressful stimuli). Pole (2007) found significant weighted mean effects of PTSD for HR in standardized trauma cue studies; frontalis EMG, corrugator EMG, HR, and SC in idiographic trauma cue studies; eyeblink EMG, HR, and SC habituation slope in startle studies; and HR and SC in resting baseline studies. Moreover, even after applying the most conservative tests of statistical significance, PTSD was linked to aggregate indices of larger physiological responses to idiographic and standardized trauma cues, higher resting baseline arousal, and larger responses to startling sounds (Pole, 2007).

In the studies assessing the relationship between PTSD and psychophysiological reactivity, some of the more common stressors chosen have included trauma script driven imagery (Sack, Cillien, & Hopper, 2012; Sack, Hopper, & Lamprecht, 2004), script-driven recall (O'Kearney & Parry, 2014), videos of varying emotional valence (Hauschildt, Peters, Moritz, & Jelinek, 2011), imagining of threatening and neutral events (McTeague et al., 2010; McTeague & Lang, 2012), and startle sounds (Jovanovic et al., 2009). While many studies examining cardiovascular reactivity to stressors among the

PTSD population have found significant elevations in response to stress, Orr, Metzger, and Pitman (2002) have suggested that as many as one-third of PTSD patients do not evidence increased physiological reactions in response to trauma-related cues. Thus, this paper will attempt to identify factors which may influence the relationship between PTSD and cardiovascular reactivity. One factor that has been suggested to explain this difference is the role of dissociation.

Dissociation

According to Lanius and Hopper (2008), one of the most common features associated with PTSD is dissociation. It is a term used to describe the process whereby certain commonly integrated functions (e.g., identity, memory, consciousness, perception of the environment) become disrupted in response to traumatic events. Although dissociation may have a negative connotation, research has suggested that it may be adaptive in that it is a defensive measure that helps protect the individual by allowing him to escape a threatening situation and the internal distress/arousal he experiences (Schalinski, Schauer, & Elbert, 2015).

While some may use the terms dissociation and avoidance interchangeably, dissociative responses are distinct in that they are internal/cognitive whereas avoidant responses are behavioral. This is especially salient given that the DSM-IV criteria for PTSD appeared to conflate the two. Specifically, Criteria C combined persistent avoidance of stimuli (e.g., activities, places, or people) associated with the traumatic event [i.e., avoidance] and numbing (e.g., feeling of detachment/estrangement, inability to recall important aspect of trauma, efforts to avoid certain thoughts/feelings) of one's general responsiveness [i.e., dissociation] (APA, 2000). Although avoidance and

dissociation both involve some type of escape (behavioral and cognitive/mental, respectively), combining active/volitional behavioral avoidance and dissociative/shutting down reactions in the avoidance cluster may obscure the clinical picture.

First, when individuals are exposed to traumatic events (events that are deemed uncontrollable and have a high negative valence), their responses can range across several different areas–cognitive, affective, behavioral and physiological (Dalenberg & Carlson, 2012) –and are accompanied by different underlying effects in the body. For example, in examining the difference between individuals who exhibit a dissociative versus hyperaroused response to trauma, neurobiological studies have found that there are specific patterns of brain activation that differentiate between the two (Brand & Loewenstein, 2010).

Second, the numbing response and avoidance of stimuli associated with PTSD can be viewed as defensive responses in response to the stressor. For instance, Schalinski, Schauer, and Elbert (2015), note that the more an individual experiences various types of traumatic events the more likely they are to display a shutdown dissociation response as their primary mode of responding. Although a common response to dangerous/stressful events involves the fight-or-flight response [what may be considered a more behavioral response], a third response that is hypothesized to occur in humans is the freeze or immobility response [what may be considered a more dissociative response] where the individual may exhibit depersonalization, derealization, and gaps in one's memory or awareness (Dalenberg & Carlson, 2012). Additionally, shutdown dissociation – which involves a shutting down of one's cognitive, perceptual, and affective information processing capabilities (Schalinski, Moran, Schauer, & Elbert, 2014) – is thought to

comprise a progression of the defense cascade (the term used to describe a set of automatic defensive behaviors) as it is thought to enhance survival.

Although each of the abovementioned responses comprise a step in the defense cascade, Kozlowska, Walker, McLean, and Carrive (2015), note that each of these differing defensive reactions have a distinct neural pattern. Moreover, Roelofs (2017) stated that whereas freezing involves parasympathetically dominated heart rate deceleration, fight-or-flight responses involve sympathetically driven heart rate acceleration. Although the DSM-IV criteria for PTSD suggested that the symptoms (i.e., re-experiencing, numbing, avoidance, and hyperarousal) represented and were related to one underlying biopsychological construct (Verbeck, 2014), the constructs of behavioral avoidance and dissociative responses, in individuals with PTSD, are accompanied by vastly difference physiological, neurobiological, and/or cardiovascular responses.

While various studies have examined dissociation among clinical and non-clinical populations, there are still debates raging regarding the mechanisms by which trauma leads to dissociation (Barlow & Freyd, 2009) as well as whether dissociation should be considered as a taxon, state, or trait (DePrince & Freyd, 2007). Pierre Janet—the man who coined the term dissociation—believed that healthy individuals rarely experienced dissociation, an experience marked by a discontinuity in awareness (Greyson, 2000).

As opposed to Janet's view, some believe that dissociation is a variable that exists on a continuum (a dimensional view), rather than as a pathological state. Specifically, a dimensional view of dissociation proposes that dissociation can comprise lesser as well as more severe states. Thus in this view, an example of a lesser (or adaptive) state would involve an individual who forgets themselves while driving or gets lost in a book/movie

(Guez, Hertzanu-Lati, Lev-Wiesel, & Rabin, 2015) while a more serious (or pathological) state could involve an individual who reports phenomena such as derealization or out-of-body experiences (DePrince, Chu, & Visvanathan, 2006). In contrast to this view, a pathological view of dissociation states that individuals who truly experience dissociation evidence profound developmental differences in their meta-cognitive functions, integration of behavior, and acquisition of developmental competencies (Putnam, 1997), and experience loss of continuity in subjective experience, intrusions in awareness and behavior, inability to access information, and experiential disconnectedness (Cardeña & Carlson, 2011). Thus, experiences such as daydreaming or absorption would not meet that criteria.

One study attempted to test whether or not there was a difference between pathological dissociation and alterations in consciousness (Waller et al., 1996). After conducting three taxometric analyses with 228 normal controls and 228 Multiple Personality Disorder (now known as Dissociative Identity Disorder) cases, they found that markers of pathological dissociation (e.g., depersonalization, amnesia for dissociative states, identity alteration, etc.) measured a typological construct or latent class whereas markers of non-pathological dissociation (e.g., imaginative involvement, absorption, etc.) measured a dimensional construct. Thus, they concluded that consistent with Janet's view individuals either fall into the group who do experience chronic dissociation or the group who do not experience dissociation.

More recently, van der Hart et al. (2005) argued that in order for a person to have truly experienced dissociation, he/she must have evidenced some phenomena that included a structural dividedness of his/her personality. Thus, they argue that individuals

who classify experiences such as daydreaming, absorption, or imaginative involvement as dissociation are guilty of over-inclusiveness. In this sense, dissociation reflects an inhibition of the typical integration of punctuated or fragmentary awareness, whereby in stressful situations, higher order integration may not occur, or may actively be prevented from occurring, as this enables the individual to cope with the stressor (Seligman & Kirmayer, 2008). Thus, two forms of dissociation are commonly recognized: peritraumatic dissociation and persistent dissociation. The former describes the dissociation an individual experiences during the traumatic event while the latter refers to the dissociation experienced in the aftermath of the traumatic event (DePrince et al., 2006).

Furthermore, research has suggested that there may be two distinct subtypes within the PTSD diagnosis: the dissociative subtype and the reexperiencing/hyperaroused subtypes (Lanius et al., 2010). Whereas a common response to trauma involves dissociation, which dissipates over time, a distinct minority of individuals who undergo a traumatic experience exhibit dissociative symptoms for months following the traumatic event (Carlson, Dalenberg, & McDade-Montez, 2011; Lanius et al., 2012). This group is conceptualized as the dissociative subtype of PTSD, and is characterized by inhibited/blunted physiological and/or emotional responses to trauma-related stimuli (Lanius et al., 2010).

According to Brand and Loewenstein (2010), research examining the dissociative/hypoemotional and hyperaroused/hyperemotional subtypes note that these subgroups tend to differ in several ways. Specifically, individuals with the dissociative subtype are more likely to present with severe, chronic, childhood and adult trauma

histories. In response to traumatic triggers/narrative, they are more likely to exhibit dissociation, numbing, decreased heart rate/skin conductance, blunted/decreased autonomic arousal, a delay in cortisol release, and brain areas that activate and overcontrol one's emotional experience and alter one's perception of one's self. In contrast, individuals with the hyperaroused subtype are more likely to have experienced less cumulative trauma or experienced later-occurring trauma. In response to traumatic triggers or narratives, they are more likely to experience increased heart rate/skin conductance, increased autonomic arousal, rapid elevations in cortisol levels, and have brain areas that activate and undercontrol one's emotional experiences. Thus, individuals in the dissociative subtype have a tendency toward overmodulation of their emotions whereas individuals in the hyperaroused subtype have a tendency toward undermodulation.

Dissociative responses can occur during the course of other psychological/ medical disorders such as psychotic disorders, panic disorder, mood disorders, migraines, and temporal lobe epilepsy (Lanius et al., 2002). While dissociation may serve a function in that moment, Bremner and Brett (1997) note that those individuals who exhibit dissociative reactions to traumatic events tend to respond in like manner even when confronted with minor stressors. Moreover, several studies have shown that experiencing a dissociative response during or after a stressful/traumatic event can actually predict the likelihood of being diagnosed with PTSD in the future (Breh & Seidler, 2007; Bremner & Brett, 1997; Ozer, Best, Lipsey, & Weiss, 2003).

Even though many studies have found a positive relationship between PTSD and exaggerated reactivity in response to stress, other studies have found that some

individuals with PTSD evidence blunted or no reactivity (Cuthbert et al., 2003; McTeague & Lang, 2012). Moreover, they have found that individuals who experience multiple traumatic events are more likely to exhibit blunted reactivity than those who experience only a single traumatic event. One theory suggests that individuals with PTSD may fall into two categories: (1) a hyperarousal group or (2) a dissociative group (Lanius, Brand, Vermetten, Frewen, & Spiegel, 2012). Consequently, it is hypothesized that the varying expression of PTSD symptoms and features (dissociative versus hyperreactive) may help to predict which individuals are more likely to experience blunted versus exaggerated reactivity.

Dissociation and Reactivity

Several studies have investigated the relationship between dissociation and reactivity; and most of them have found a negative relationship between the two variables. For instance, Griffin, Resick, and Mechanic (1997) investigated physiological reactivity (heart rate and skin conductance) and peritraumatic dissociation in 85 female victims of recent rape. The subjects' scores on the Peritraumatic Dissociation Index were used to categorize them as having high (1 SD above the mean) or low (0.50 SD below the mean) levels of dissociation. They found that in the high dissociation group, the physiological measures of arousal were significantly lower than their self-reported ratings of distress. Moreover, in comparison to low dissociators, high dissociators exhibited significantly lower heart rate and a significantly lower number of skin conductance responses during the trauma and final baseline phase.

A study by Lanius et al. (2002) used functional magnetic resonance imaging (fMRI) to study dissociation to traumatic script driven imagery in PTSD patients (with a

history of sexual/physical abuse) and control subjects. They found that although individuals who relived their traumatic experience evidenced an increase in heart rate, individuals who experienced a dissociative response evidenced no significant increase in heart rate. Another study assessed the cardiovascular responses (i.e., SBP, DBP, and HR) of 100 undergraduate students following the September 11th terrorist attacks (von Bergen, 2010). While reviewing a distressing 9/11 associated event, participants were administered the Impact of Event Scale (IES) and the Spiritual Well-Being Scale (SWBS). Consistent with the researcher's hypothesis, a multiple regression analysis found that numbing/dissociation symptoms associated with 9/11 events were very likely to predict decreased SBP reactivity.

Yet another study by Sack, Cillien, and Hopper (2012) assessed the psycho-physiological reactions of 61 patients with a history of trauma exposure as they were exposed to script-driven trauma imagery. Participants were divided into 4 groups based on the mean splits of their dissociation and re-experiencing subscale scores. They found that during trauma scripts, individuals who fell within the high dissociation group (i.e., high re-experiencing and high dissociation) exhibited significantly lowered heart rate in comparison to those in the other groups (Sack et al., 2012).

Though several studies have linked peritraumatic dissociation to decreased autonomic nervous system functioning, a few studies have not replicated these findings. A study by Ladwig et al. (2002) investigated the relationship between peritraumatic dissociation and one's startle response in 103 participants who had experienced a life-threatening cardiac event. All participants were subjected to 15 loud tones (at varying intervals) while electromyogram (EMG) and skin conductance responses (SCR) were

measured. They found that in comparison to individuals with low or no dissociation individuals with high, clinically significant levels of dissociation exhibited a higher magnitude of EMG and SCR. Additionally, subjects who exhibited high dissociation and severe PTSD evidenced higher EMG amplitudes during the 15 trials (Ladwig et al., 2002).

Similarly, Nixon, Bryant, Moulds, Felmingham, and Mastrodomenico (2005) attempted to test the relationship between dissociation and suppressed autonomic reactivity in a sample of motor vehicular accident and nonsexual assault victims. Participants' heart rate and skin conductance were measured as they spoke about their traumatic experiences in the present tense. They found that in contrast to their low dissociation counterparts, participants with high dissociation tended to exhibit higher heart rates during the course of the experiment. Furthermore, they noted that skin conductance level was comparable across both groups (high vs/ low dissociation) and they found no evidence of a suppressed autonomic response in the high dissociation group (Nixon et al., 2005). Regardless, mixed findings such as these may be due to various factors such as inter- and intra-person variability in response to trauma or trauma-related cues (Lanius, Bluhm, Lanius, & Pain, 2006), the use of different stress paradigms, varying populations under study (e.g., acute trauma versus an actual PTSD diagnosis), or even the time period during which retrospectively assessed dissociative symptoms are correlated with stress responses (e.g., weeks versus years later) (Sack et al., 2012).

Depression and Reactivity

The findings regarding the relationship between depression and reactivity has also been supported by the literature. However, where studies differ are with regards to

the direction of the relationship between depression and reactivity. Some research has found that individuals who are suffering from depressive symptoms evidence greater physiological reactivity. For example, one study subjected myocardial infarct patients with depressive symptoms to laboratory challenge tests (e.g., the Stroop, mental arithmetic, verbal challenge, etc.) while recording their blood pressure and heart rate. In addition to depression being the best predictor of cardiovascular responses (post-acute myocardial infarction), they also found that depression scores predicted HR variance post episode (though not at later assessments) (Thornton & Hallas, 1999).

Similarly, another study assessed the presence of high versus low depressive symptoms in healthy subjects. Blood pressure, impedance cardiography, and electrocardiography recordings were used to measure subjects' reactivity in response to the mirror star tracing task. Results revealed that individuals with high depressive symptoms evidenced a marked increase in systemic vascular resistance (SVR) (Matthews et al., 2005). Furthermore, a study by Light et al. (1998) assessed cardiovascular reactions in response to stress in a high BDI (Depressive) group and low BDI (Control) group. They found that subjects in the depressive group had higher blood pressure levels (during the rest and stressor periods); reduced heart rate variability and shorter pre-ejection period (PEP) across the stressor and rest periods; and higher cardiac output (CO) during the speech task. These findings are consistent with a meta-analytic study that found small to moderate effect sizes relating depression to SBP, DBP, and HR reactivity (Kibler & Ma, 2004).

In contrast to the above-mentioned studies, a few studies have found that individuals with depression exhibit blunted/attenuated cardiovascular reactivity in

response to stress. For instance, depression or depressed mood was negatively associated with SBP (Carroll et al., 2007) and heart rate and SBP (Phillips, 2011a; Phillips 2011b). In another study, SBP, heart rate (HR), and CO reactivity were also found to be negatively associated with depression (Salomon et al., 2009). Furthermore, York and colleagues (2007) found that individuals with higher depressive symptom scores exhibited lower cardiovascular reactivity in response to acute psychological stress; and this finding remained robust even after controlling for numerous potential confound variables.

Yet another study suggests that attenuated cardiovascular reactivity may be related to one's mood state. Specifically, individuals with current MDD exhibited blunted reactivity and impaired recovery; however, those with remitted MDD exhibited reactivity/recovery rates similar to healthy controls (Salomon et al., 2013). One concept that may help to explain the mixed findings regarding the relationship between depressive symptoms and cardiovascular reactivity is activation and how it relates to depression. In order to discuss the factor of activation, one must first describe the current literature on the factor analytic structure of the BDI-II, one of the most commonly used measures of depressive symptomatology.

The Factor Structure of the BDI-II

Studies regarding depression are replete with references to the Beck Depression Inventory (BDI), a self-report questionnaire that assesses the presence and severity of depressive symptoms. Though the original BDI was completed in 1961, most research studies and clinicians utilize the revised version, the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996). Revisions of the BDI followed revisions of the DSM

(Bühler, Keller, & Läge, 2014), as a change in the diagnostic criteria accordingly led to changes in how depression was diagnosed and assessed. One issue that has continued to be a source of contention among research studies is the factor analytic structure of the BDI-II.

Historically, a two-factor model was proposed to target the underlying factor structure of the BDI-II. However, Bühler et al. (2014) and Ward (2006) have noted that two separate two-factor models have commonly been found: (1) a cognitive-affective factor and a somatic factor, and (2) a cognitive factor and a somatic-affective factor. In the former model, some of the affective items of the BDI (e.g., loss of interest, sadness, loss of pleasure, etc.) loaded higher onto the cognitive factor whereas in the latter model some of the affective items loaded higher onto the somatic factor (Bühler et al., 2014).

There is some research that coincides with the aforementioned models. Steer and Clark (1997) conducted an exploratory factor analysis (EFA) of the BDI-II on students and found a cognitive-affective and somatic factor model with minor differences from the Beck study. Likewise, Arnau, Meagher, Norris, and Branson (2001) conducted an exploratory factor analysis of the BDI-II on primary care medical patients and found a somatic-affective and cognitive factor model very similar to the one proposed by Beck.

Though several exploratory and confirmatory factor analytic studies have found similar results (Bedi, Koopman, & Thomas, 2001; Storch, Roberti, & Roth, 2004; Whisman, Perez, & Ramel, 2000), some research has suggested that these models may not be the best fit to model the symptom structure of the BDI-II. A few studies have proposed a complex structure model (Arnau et al., 2001; Brouwer, Meijer, & Zevalkink, 2013; Ward, 2006) such as the bifactor model—which posits that there may be a general

(or ‘G’) factor that all items load on and several other factors that a fragment of items load on (Bühler et al., 2014).

Though these complex structure models may provide better fit indices, what they and the aforementioned simple structure models have in common is that they neglect to account for a potential other factor that may encompass the behavioral responses associated with depression. Depression can be conceptualized as a behavioral response to stress (whether a psychological stressor, such as in the case of a divorce, or a physical stressor, such as in the case of developing cardiovascular disease). Consequently, it is not farfetched to believe that there may be some form of “behavioral” factor underlying the symptom structure of the BDI-II. Thus far, a review of the literature revealed only one study that has attempted to propose an alternative factor to account for these behavioral responses using nonmetric multidimensional scaling (NMDS) and confirmatory factor analysis. Bühler and colleagues (2014) proposed that one factor that has received little attention in the BDI-II factor analysis literature is that of activation.

The “Activation” Factor

Activation was proposed by Bühler, Keller, and Läge (2012) as a factor underlying the symptom structure of the BDI-II after using NMDS on the BDI-II data of 266 depressed patients. For example, in their proposed model, some of the items that were expected to load onto the “activation” factor included items 10 (crying), 11 (activation), 17 (irritability), 19 (concentration difficulties), and 20 (tiredness or fatigue). In this sense, symptoms such as concentration difficulties or tiredness would fall under decreased activation whereas symptoms such as crying, irritability, and agitation would fall under increased activation.

In order to further test their hypothesis, they conducted confirmatory factor analyses on two separate samples: the norming sample (N = 266) of the BDI-II (the German version) and a sample (N = 569) of patients from a psychosomatic disorders clinic who were administered the BDI-II and later diagnosed with a primary affective disorder. Though a subset of the items did load onto the cognitive and somatic factors, results of their analysis revealed that 6 items significantly loaded onto the activation factor. They were items 6 (punishment feelings), 9 (suicidal thoughts), 11 (agitation), 15 (loss of energy), 19 (concentration difficulties), and 20 (tiredness).

Depression and the Potential Role of Activation in Reactivity

Though Bühler et al. (2014) are probably the first to propose activation as a factor, the concept of activation as it relates to depression is not a new one. Specifically, some studies have suggested that depression may express itself differently (or entail different subtypes) depending on the antecedent causes of the depressive episode, the severity or chronicity of symptoms, and the level of activation or arousal present in the individual among other things. One study by Cohen (2008) conducted a confirmatory multidimensional scaling (MDS) of the BDI-II and found two dimensions: the disturbance domain dimension and the level of arousal dimension. The latter refers to how disturbances are expressed (i.e., regular, over-active, or under-active). In this sense, the depressed individual who exhibits over-active disturbances or behavioral responses may differ in clinically significant ways from the depressed individual who exhibits under-active disturbances or behavioral responses.

In line with this view, while working on the Hamilton Rating Scale for Depression, Hamilton (1960) identified two types of depression (agitated and retarded), a

concept which was later re-introduced by Klein and Davis in their discussion on the subtypes of serious depression (Shorter, 2007). Shorter (2007) proposed that there are two types of depression: melancholia (which accounts for the prototypical depressive symptoms) and non-melancholia (which includes depressive character, dysphoria, and anxiety). In comparison, Koukopoulos and Koukopoulos (1999) mentioned the concept of agitated depression (e.g., marked by restlessness, anxiety, and delusions) in their discussion on depression and its various presentations. Moreover, Sobin and Sackeim (1997) note that psychomotor symptoms have high discriminative validity in that they can help to distinguish between different subtypes of depression and can also help to predict which individuals will be more responsive to tricyclic antidepressants (TCA).

Assuming that depression can be further differentiated into specific subtypes, it stands to reason that individuals who present with varying levels of behavioral or psychomotor symptomatology may exhibit different cardiovascular reactions in response to stress. Though some studies have discussed the differences between varying depressive presentations, there is a dearth of research assessing how one's subtype can lead to different cardiovascular reactivity. For example, individuals who are experiencing the more vegetative form of depression may be more likely to endorse certain symptoms such as social withdrawal, loss of interest, anhedonia, etc. In contrast, those who are experiencing the more agitated form of depression may be more likely to endorse symptoms such as irritability, concentration difficulties, etc. Consequently, this paper will attempt to address this gap by studying whether the above-mentioned distinction may help to explain why some individuals who suffer from depression experience exaggerated reactivity and others attenuated reactivity in response to stress.

Though high cardiovascular reactivity in response to stress is associated with poorer health outcomes, the converse does not hold true. That is, low physiological (or cardiovascular) reactivity in response to stress is not necessarily associated with better health outcomes. This has led some to conclude that the association between reactivity and pathological states may be curvilinear (Lovallo, 2011) in that any reactivity below or above what is considered average may be indicative of poorer health (Salomon et al., 2013). Additionally, since reactivity tends to be positively linked to an up-regulation of immune processes (related to host defense) it is thought that blunted reactivity may also be linked to a down-regulation of immune processes (which would equate to a decrease in the body's ability to combat infectious diseases) (Carroll, Phillips & Lovallo, 2009). Though there may be various characteristics that influence or mediate the relationship between stress and exaggerated or attenuated cardiovascular reactivity, one factor that may explain the relationship is one's cognitive appraisal of the event/stressor.

Cognitive Appraisal

Cognitive appraisal is a term used to describe the way in which a person's interpretation of a situation or event influences the degree to which they perceive a situation as being stressful (Campbell, Johnson, & Zernicke, 2013). It is based on the presumption that in studying stress and coping styles, one must take into account the individual's perspective and his or her appraisal of the environment (Quigley, Barrett, & Weinstein, 2002). Lovallo (2005) states that since cardiovascular reactivity is inherently a biopsychological phenomenon, it is dependent on one's autonomic response systems as well as one's cognitive appraisals. Thus, in this sense cognitive appraisals may mediate the stress response as Gomes, Faria, and Gonçalves (2013) reiterate that one's experience

of stress or strain is dependent on the way in which the individual evaluates a situation and his/her ability to cope with the situation. Cognitive appraisals are usually classified into two distinct categories: primary appraisal and secondary appraisal.

Primary Appraisal

Primary appraisal describes the individual's evaluation of the situation and the potential for harm or threat (Velichkovsky, 2009). Primary appraisals can come in three types: (1) irrelevant, (2) benign-positive, and (3) stressful (Brannon & Feist, 2009). An irrelevant primary appraisal occurs when an individual evaluates an event and determines that it has no implications for the person's well-being and, accordingly, requires no action. In comparison, a benign-positive appraisal occurs when an individual evaluates an event as having a positive outcome (or implications for one's well-being).

In contrast, stress appraisal is a term used to describe situations that an individual deems to be stressful. It includes harm/loss, threat, and challenge appraisals. As the name suggests, harm/loss appraisals occur when an event happens and is followed by some form of loss (e.g., the loss of a loved one) or damage to the individual. In contrast, threat and challenge appraisals precede stressful events when something valuable is at risk (Duranova & Ohly, 2016). Threat appraisals occur when an individual anticipates future harms or losses with little, if any, chance of gain; conversely, challenge appraisals occur when an individual anticipates future gain as well as loss. The primary difference between threat and challenge appraisals is that the former focuses on harm and is usually characterized by anxiety, anger or fear whereas the latter focuses on the potential gains/growth and is usually characterized by exhilaration, eagerness, or excitement (Maier, Waldstein, & Synowski, 2003).

Secondary Appraisal

As opposed to primary appraisal, secondary appraisal relates to the individual's perception of his/her resources and whether or not he/she will be able to cope with the situation (Velichkovsky, 2009). It involves taking into account which coping strategies/techniques the individual has available to him, the probability that those coping strategies will bring about the necessary results, and the likelihood that the individual will be able use the chosen coping strategy effectively. In the case of the aforementioned threat and challenge appraisal, an individual is likely to appraise a situation as threatening if he does not feel he has the necessary resources to cope with the stressor, and challenging if he does feel he has the necessary resources to cope with the stressor.

Cognitive Appraisal and Reactivity

In terms of cognitive appraisal, appraising a situation as a threat is associated with the experience of negative emotions (Jones, Meijen, McCarthy, & Sheffield, 2009); this can then lead to physiological responses which may impact the onset and/or progression of disease (Feldman, Cohen, Hamrick, & Lepore, 2004). Moreover, even the anticipation of stress—such as when an individual is asked to prepare to give a public speech—leads to elevations in cardiovascular reactivity (Baggett, Saab, & Carver, 1996). Since threat and challenge appraisals are a form of anticipatory appraisal, it has long been speculated that cognitive appraisals may be linked to cardiovascular reactivity. This finding has been established by the literature.

For example, Tomaka and colleagues (1993) used active and passive stressors to assess the effects on subjects' cardiovascular reactivity. They found that in response to the active stressor, individuals in the challenge appraisal group evidenced significantly

higher cardiac reactivity (i.e., PEP, CO, and HR) and significantly lower vascular resistance (i.e., TPR) whereas threat appraisals were negatively linked to cardiac reactivity. In addition, individuals in the threat appraisals group evidenced less cardiac reactivity and greater vascular resistances.

Similarly, Tomaka and Blascovich (1994) subjected participants to a stressful laboratory task (serial 7s) while recording their cardiac, vascular, and electrodermal physiological responses. Participants were split into two groups: those who were high in just world beliefs (which are thought to be related to challenge appraisals) and those who were low in just world beliefs (which are thought to be related to threat appraisals). In line with previous findings, they found that individuals with high just world beliefs had increased cardiac reactivity (PEP, HR, and a marginal increase for CO), a greater decrease in peripheral vascular resistance (TPR), and fewer skin conductance responses in comparison to those with low just world beliefs.

Yet another study by Zanstra, Johnston, and Rasbash (2010) attempted to investigate the relationship between cognitive appraisals and hemodynamic response patterns (i.e., myocardial responding and vascular resistance) during a stressful task. Specifically, twenty-four male participants were asked to provide their appraisals of an upcoming stressor: presenting a paper in a class. While presenting the paper, the participants' physiological reactivity was recorded using a blood pressure monitor. Results revealed that pre-stressor appraisals were significantly associated with CO 30-minutes before and during the stressor. Additionally, while challenged participants evidenced higher CO, increased threat states were associated with an increased TPR. Furthermore, research has also assessed how appraisals relate to one's cortisol response.

One study by Schlotz, Hammerfald, Ehlert, & Gaab, (2011), sought to determine how cognitive appraisals and perceived stress reactivity affect one's cortisol response to stress. In addition to completing measures of cognitive appraisals and perceived stress reactivity, participants were subjected to the Trier Social Stress Test (TSST; comprised of a simulated job interview and a mental arithmetic task) in front of two people. Several saliva samples were taken (before, during, and after the stress tests) to assess cortisol levels. They found that both primary appraisals and perceived stress reactivity were positively associated to cortisol levels. Additionally, in some domains, perceived stress reactivity was linked to steeper cortisol levels in response to stress; and this effect was mediated by one's primary appraisal.

Another study by Gaab, Rohleder, Nater, & Ehlert, (2005), investigated the relationship between anticipatory/retrospective appraisal processes, HPA Axis responses to acute stress, general personality factors and the cortisol response. They developed a questionnaire (the Primary Appraisal Secondary Appraisal scale; PASA) to assess anticipatory cognitive appraisal processes and later administered the measure to 81 male participants. After being introduced to the TSST, and completing the PASA, participants completed a simulated job interview followed by a mental arithmetic task in front of an audience of two people. Saliva samples were taken prior, during, and after the stress test to assess salivary free cortisol levels. Gaab and colleagues (2005) found that in contrast to general personality factors and retrospective stress appraisals, anticipatory cognitive appraisals explained up to 35% of the variance of the salivary cortisol response.

Based on these studies, it is clear that one's cognitive appraisal of a stressful event can mediate the level of cardiovascular reactivity. This concept can also be applied to the

notion of the different types of depression. Specifically, individuals with the more vegetative type of depression (or those low on activation) may be more likely to appraise situations as being threatening, and would thus be expected to experience increased vascular resistance and modest cardiac reactivity in response to stress. Conversely, individuals with the more agitated type of depression (or those high on activation) may be more likely to appraise situations as being challenging, and would thus be expected to experience increased cardiac reactivity and decreased vascular resistance in response to stress.

Though several of the aforementioned studies have assessed cardiovascular reactivity in response to stress in a sample of individuals exhibiting PTSD or depressive symptoms, the present study differs in that it is one of the only studies that has utilized impedance cardiography to assess cardiovascular responses to stress. Additionally, though several studies have noted the mixed findings regarding the relationship between cardiovascular reactivity and the presence of certain symptoms (e.g., depression, PTSD, dissociation, etc.), little attention has focused on delineating those factors that may affect the likelihood that one will experience exaggerated versus blunted reactivity. The present study will attempt to clarify the nature of the relationship between these variables.

Purpose of the Study

The purpose of this study is to investigate the relationships of PTSD and depressive symptoms, dissociation, and cognitive appraisal with cardiovascular stress reactivity. This study will address the gap in the literature by testing how different PTSD or depressive subtypes/presentations may lead to different (i.e., blunted vs. exaggerated) cardiovascular reactions in response to stress; by assessing the relationship between

dissociation and cognitive appraisals to reactivity; and by providing more reliable measures of cardiovascular reactivity via impedance cardiography.

Several studies have used cutoff scores or median split to dichotomize their predictor variables prior to conducting any analyses. This practice is especially salient for two reasons. First, it is helpful in research and clinical practice to be able to label subjects as having or lacking an attribute/symptom (e.g., depressed versus non-depressed, dissociative versus non-dissociative). Second, results subsequent to these analyses can provide relevant information to specific groups regarding diagnosis and treatment (Altman & Royston, 2006), as well as how their symptoms (or the lack thereof), will impact them on specific outcome variables (in this case, cardiovascular reactivity).

The present study examined continuous scores for the predictor variables (i.e., the Clinician-Administered Posttraumatic Stress Interview for DSM-IV, CAPS; BDI-II; Stress Appraisal Measure, SAM; Dissociative Experiences Scale II, DES II) for a couple of reasons. First, while categorization (i.e., grouping) or dichotomization of continuous variables is thought to simplify statistical analysis and the interpretation and presentation of results, it is unnecessary for statistical analysis (Altman & Royston, 2006; Naggara, Raymond, Guilbert, Weill, & Altman, 2011; Royston, Altman, & Sauerbrei, 2006). Second, dichotomization is associated with several disadvantages including: (1) loss of information; (2) a reduction in statistical power (that is effectively equivalent to a loss of sample size; Dawson & Weiss, 2012); (3) uncertainty in defining the cutoff point; (4) an underestimate of the extent of variability in outcome between the groups; (5) spurious significant results when independent variables are correlated; and (6) increased Type 1 and Type 2 errors, especially in the case of median splits (Altman & Royston, 2006;

Fitzsimons, 2008; Naggara et al., 2011; Royston, Altman & Sauerbrei, 2006; Rucker, McShane, & Preacher, 2015).

Moreover, another important reason for examining continuous scores relates to the nature of the variables in question. Specifically, whereas individuals with depression or PTSD are occasionally lumped into one category based on their scores on psychological measures, doing so overlooks the fact that there is much variability when it comes to symptom severity and presentation. For example, there is clinical variability among the presence and severity of depressive symptom dimensions (e.g., affective symptoms, somatic symptoms; Dotson, 2017); there are variations in depressive episodes, patterns of severity and persistence of symptoms (Mehta, Mittal, & Swami, 2014); and several studies have noted that for individuals diagnosed with depression, there is significant symptom variability within individuals across time and across individuals (Fried & Nesse, 2014).

Similarly, Williamson, Porges, Lamb, and Porges (2015) note that for individuals with PTSD, a categorical designation, there is much variability in PTSD presentation; and PTSD is comprised of different degrees of severity of trauma-elicited disturbances of one's disposition or stress responses. According to Fried & Nesse (2014) the belief that all symptoms indicate the same underlying disease (e.g., depression) suggests that it is the number, rather than the nature, of symptoms that are important. However, given that the present study attempted to investigate how differing symptom presentation is related to cardiovascular reactivity, it was thought that analyzing continuous scores would provide a clearer clinical picture of the way these variables–symptom presentation and symptom severity–relate to reactivity.

Hypotheses

Hypothesis 1: It is hypothesized that there will be a positive relationship between PTSD arousal symptoms and cardiac reactivity, and a negative relationship between PTSD avoidance symptoms and cardiac reactivity.

Hypothesis 2: It is hypothesized that depressive activation will be positively related to cardiovascular reactivity and depressive withdrawal will be negatively related to cardiovascular reactivity.

Hypothesis 3: It is hypothesized that there will be a negative relationship between dissociative symptoms and cardiovascular reactivity.

Hypothesis 4: It is hypothesized that challenge appraisals will be related to greater cardiac reactivity, whereas threat appraisals will be related to greater peripheral resistance.

CHAPTER II

METHOD

A subset of data collected from a larger study referred to as the Women's Health and Stress Study were analyzed. A description of the methods and procedures that were utilized are described below.

Participants

Individuals with symptoms of PTSD, depression, and those with no psychiatric symptoms were screened for participation into the study via telephone. All participants (N = 58) were women aged 18 or older. Participants were recruited through posting an ad on public and scientific websites, via therapist referral, or through posting and distributing fliers at a health fair and community mental health clinic. The volunteers were screened for inclusion criteria which included the following: 1) pre-menopausal, 2) no history of major chronic illness 3) not currently taking medications which might significantly impact the physiological measures (e.g. beta-blockers, muscle relaxants, or cholesterol lowering agents), and 4) ability to read and speak English fluently.

Volunteers who were screened as prospective candidates for inclusion were invited to participate in session I, when the Traumatic Life Events Questionnaire (TLEQ), Clinician-Administered PTSD Scale (CAPS), and the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID) were administered. The TLEQ was used to assess exposure to very stressful events. Any volunteers who reported undergoing an extreme stress event on the TLEQ were subsequently administered the CAPS in order to determine PTSD symptoms. Furthermore, in order to assess the presence of any other Axis I DSM-IV disorders, the SCID was administered. All volunteers who met criteria for Axis I disorders besides PTSD or depression were excluded from participating in the

remainder of the study. The participants were also administered the Beck Depression Inventory-II (BDI-II), the Dissociative Experiences Scale (DES) and the Stress Appraisal Measure (SAM). Following the first session, participants came in for a second session that included being attached to the physiological assessments prior to undergoing the stress tasks. Participants received a $40 and $50 honorarium for the first and second session, respectively.

Measures

Assessment of Demographic Information and Psychological Distress

Self-Report Questionnaire: All participants were administered ***s***elf-report questionnaires in order to collect demographic information such as age, ethnicity, race, educational level, marital status, and family income.

Traumatic Life Events Questionnaire (TLEQ). The TLEQ is a 23-item self-report measure that assesses the frequency and severity of 22 types of potentially traumatic events with one "other extremely disturbing" events/experience category (Kubany, 2000; Norris & Hamblen, 2004, p. 69). They include exposure to warfare, physical abuse, natural disasters, sexual abuse, domestic violence, stalking, miscarriage, etc. During validation of the TLEQ, 5 studies were conducted and participants were administered the TLEQ at least twice. The degree to which participants responded consistently from administration 1 to administration 2 ranged from 63-96% during the second study, and 71-100% during the fifth study. The TLEQ evidenced good convergent validity after a 1-week delay with Kappa coefficients ranging from 0.40 and up for 13 of 16 items and .60 and up for 5 items, and was found to have good discriminant validity and positive predictive power. Moreover, when compared to the SCID, the TLEQ

produced significantly higher identification of traumatic events in women than in men which led to an increase in PTSD diagnoses identified in the sample (from 24% to 33%, respectively; Pierce, Burke, Stoller, Neufeld, & Brooner, 2009). Thus, the TLEQ has added utility in that Pierce and her colleagues (2009) suggest that it is beneficial to use multiple comprehensive traumatic event assessments [e.g., in addition to measures such as the SCID] to assess PTSD diagnoses and traumatic events in samples, especially women**.**

Clinician-Administered Posttraumatic Stress Interview for DSM-IV (CAPS). The CAPS, a structured diagnostic interview recognized as the gold standard in PTSD symptom assessment (Gray, Litz, Hsu, & Lombardo, 2004), assesses 17 core symptoms as well as 8 associated symptoms of the DSM-IV criteria for a diagnosis of PTSD (Blake, 1994). The CAPS evidenced high reliability coefficients of .92 to .99 for the frequency and intensity scores of the 3 symptom clusters (i.e., re-experiencing, numbing and avoidance, and hyperarousal) (Blake, 1994). High internal consistency was also found as Cronbach's α coefficients for the 3 symptom clusters ranged from .73 to .85. Moreover, the CAPS was found to have good predictive value in terms of accurately diagnosing PTSD based on the SCID PTSD module. A review of the CAPS revealed that based on the accumulated literature, the CAPS has excellent psychometric properties, with interrater reliability scores of .90 and above (for continuous CAPS scores) and internal consistency alphas in the .80 to .90 range (for the entire PTSD syndrome and three symptom clusters; Weathers, Keane, & Davidson, 2001). Yet another study reviewing the use of the CAPS in civilian populations revealed an internal consistency alpha of .97 (for all CAPS items) (Pupo et al., 2009). Additionally, when the CAPS scores were compared

to the SCID, they revealed validity coefficients of 90%, 95%, and 7.1% for sensitivity, specificity, and misclassification rate, respectively.

Structured Clinical Interview for DSM-IV Axis I Disorders (SCID), research version. The SCID-I is a semi-structured interview used to diagnose DSM-IV Axis I disorders. The standard research version of the SCID is separated into diagnostic modules and assesses for issues such as mood, anxiety, psychotic, substance use, eating, somatoform, and adjustment disorders with other optional modules available (Weiner & Craighead, 2010). While First, Spitzer, Williams, and Gibbons (1997) found inter-rater reliability coefficients of .75-.85, Lobbestael, Leurgans and Arntz (2011) subsequently reported inter-rater Kappa values from 0.61 to 0.83 (M = 0.71). In terms of validity, "best estimate" diagnoses have been compared to SCID diagnoses; and the SCID has continually been established as having superior validity over other standard clinical interviews administered at intake (Weiner & Craighead, 2010). The SCID-I was utilized in order to ensure that all participants met the inclusion and exclusion criteria.

Beck Depression Inventory, Second Edition (BDI-II). The BDI-II is a 21-item self-report measure of depressive symptomatology that consists of a multiple choice format (Beck, Steer, & Brown, 1996). It can be administered to adolescents or adults where scores of 0-13, 14-19, 20-28, and 29-64 indicate a minimal, mild, moderate, or severe level of depression, respectively. Beck and his colleagues (1996) found a high 1-week test-retest reliability coefficient of 0.93 in 26 of their outpatients. They also found that the BDI-II evidenced good convergent and discriminant validity as it was more positively correlated with the Hamilton Psychiatric Rating Scale for Depression (r = 0.71) than the Hamilton Rating Scale for Anxiety (r = 0.47). Moreover, a study of the construct

validity of the BDI-II found that it was more highly correlated with the depression rather than anxiety subscale of the SCL-90-R, a self-report measure of various psychopathology (Steer, Ball, Ranieri, & Beck, 1997). A more recent study reviewed several relevant articles to identify the psychometric properties of the BDI-II and determine the feasibility of utilizing it in diverse populations (Wang & Gorenstein, 2013). Results revealed high internal consistency (alpha coefficients ranging from 0.83 to 0.96), retest reliability (ranging from 0.73 to 0.96), convergent validity with other measures of depression (coefficients ranging from 0.66 to 0.86), and criterion based validity (good sensitivity and specificity to detect depression).

Dissociative Experiences Scale II (DES II). The DES II is a 28-item self-report measure that measures the frequency of dissociative experiences (Craparo, Faraci, Rotondo, & Gori, 2013) such as depersonalization, absorption, and derealization in normal and clinical populations (Bernstein & Putnam, 1986; Bernstein & Putnam, 1993). Participants circle a percentage number (e.g., 0%, 10%...100%) to identify the frequency with which they experience the dissociative symptoms; and an average score is then derived from the circled percentages. The DES has yielded high internal reliabilities with Cronbach's alphas of over 0.90 in several studies (Oh, Kim, & Kim, 2015; Zingrone & Alvarado, 2001). The DES score evidenced a high test-retest reliability coefficient of 0.84 in the original study (Bernstein & Putnam, 1986) and a moderate 4-week test-retest reliability of 0.69 in a subsequent study (Oh, Kim, & Kim, 2015). Additionally, the DES has demonstrated good convergent and discriminant validity and adequate test-retest and split-half reliability (Craparo et al., 2013), and appears to have good construct validity as scale scores and item scores were highly correlated.

Stress Appraisal Measure (SAM). The SAM is a self-report measure that assesses six dimensions of primary and secondary appraisals (Levy, Nicholls, & Polman, 2012). The three primary appraisal dimensions are threat, challenge, and centrality and the three secondary appraisal dimensions are controllable-by-self, controllable-by-others, and uncontrollable-by-anyone. The SAM also includes a scale that provides an index of overall perceived stressfulness. The SAM has evidenced high internal consistency on six scales with alphas ranging from 0.74 to 0.90 (Peacock & Wong, 1990). Though the uncontrollable-by-anyone scale had an alpha of 0.51, Peacock and Wong (1990) note that since ratings on this scale consistently fell at the low end of the scale this may have led to an attenuated internal consistency estimate. Similarly, one study found Cronbach's alpha coefficients ranging from 0.67 to 0.85 on the six dimensions of primary and secondary appraisals and stressfulness (Levy et al., 2012); and yet another found internal consistency coefficients (for threat, challenge, uncontrollable-by-anyone, controllable-by-self, and controllable-by-others) ranging from 0.68 to 0.84 for adults and 0.70 to 0.86 for university students (Durak & Senol-Durak, 2013). The SAM was found to have good convergent validity with scales assessing dysphoric mood and psychological symptoms (Peacock & Wong, 1990). The mean intercorrelation between the scales was 0.22 which suggests that the SAM's subscales are tapping into fairly independent dimensions of appraisal.

Physiological Assessment

Cardiovascular Reactivity using BP, ECG and Impedance Cardiography. An automated BP cuff connected to a personal computer was used to collect and record SBP and DBP measurements (mmHG) during stress tasks. In order to identify the onset of

electromechanical systole and measure HR in beats per minute (bpm), ECG electrodes were placed in a standard 3-lead configuration. Impedance cardiography is a non-invasive, cost-effective technique for monitoring thoracic fluid status, stroke volume, cardiac output, and hemodynamics (Lasater & von Rueden, 2003; Parry & McFetridge-Durdle, 2006; Turner, 2000). In order to record the cardiovascular reactivity measures, a standard full band tetrapolar configuration was utilized. To determine TPR in peripheral resistance units (mmHg/L/min) and CO in liters per min (l/min), thoracic impedance was gathered via the placement of four impedance cardiograph tape electrodes placed at the neck (Z_1, Z_2) and thorax (Z_3, Z_4). While Z_1 was placed superior to the suprasternal notch of the thorax, Z_2 was placed approximately 3 cm above it. Z_3 was placed at the xiphoid process, and Z_4 was placed approximately 5 cm below it. Signals obtained from the ECG and impedance cardiography electrodes were transmitted to a personal computer for data scoring and reduction.

Stress Tasks. Two separate stress tasks were used in this study: a mental arithmetic task and a speech preparation and delivery task. The mental arithmetic task was a 3-minute stress task that consisted of having participants perform serial subtractions by steps of 13 out loud. In addition, the participants were informed that their performance on this task would be videotaped and rated by experts.

For the speech task, participants were given instructions to develop and deliver a speech based on a hypothetical situation. Specifically, they were asked to imagine that a store's security guard accuses them of stealing a belt that they are currently wearing (but that they had purchased 3 weeks before) and consequently gets the store manager. They then had to pretend they were explaining their side of the story to the manager. This task

consisted of a 3-minute speech delivery preparation phase followed by a 3-minute speech delivery phase. While the speech delivery task has been used to elicit cardiac reactivity response, both the mental arithmetic and speech preparation tasks have been used to evoke cardiac and vascular reactivity.

Procedures

Participants who were screened positive, as possible candidates were invited to participate in the study. The first session (interview) and second session (laboratory assessment) were scheduled no more than 2 weeks apart in order to control for any potential changes in symptom presentation. Any participant who failed to attend session 2 after already completing session 1 was re-interviewed prior to the rescheduled laboratory session. All participants were asked to call in on the first day of their menstrual cycle in order to control for menstrual cycle effects on CVR. The first session was scheduled within the first week of the menstrual cycle, and the second session was scheduled after menstruation had stopped, during the follicular stage (based on the participants' estimated cycle length approximately days 4-10 of the menstrual cycle).

During session 1, consent was obtained from the participants and they were asked to complete the TLEQ. Both the CAPS and SCID were administered by graduate research assistants (i.e., advanced clinical psychology doctoral students). After scoring the CAPS and SCID, eligible participants were consequently invited to participate in session 2.

During session 2, consents were obtained from the participants and they were asked to complete demographic questionnaires (e.g., age, ethnicity, race, education, marital status, and family income) and health behavior information (e.g., alcohol use, smoking, and physical activity). An overview of the laboratory protocol was provided to

all of the participants. Participants' height, weight, waist and hip circumference measurements were taken. The sensors for the ECG and impedance cardiography and automated BP cuff were attached to the participant and connected to the impedance cardiography equipment described above. Cardiograph and ECG sampling was initiated and the signals were sampled at exactly 1 kHz per second and stored in a personal computer. Subsequently, the participants' gender, birth date, height/weight measurements, and the measurement of the distance between the Z_2 and Z_3 electrodes (while standing) were entered into the COP-WIN/HRV software program on the personal computer. Participants were instructed to sit quietly (without moving or talking but keeping their eyes open). Timing for the initial rest period was initiated; and after the beginning of the rest period BP measurements were taken at 11:00 minutes, 12:30 minutes and 14:00 minutes. The instructions for the speech stress task described above were provided; and during the speech preparation stage BP was sampled at 00:15 minutes and 01:45 minutes. At 3:00 minutes, instructions to begin delivering the speech out loud were given; and BP sampling was taken at 03:15 minutes and 4:45 minutes. The participant was told that the speech task was over at 06:00 minutes and was asked to sit quietly for another rest period. After the beginning of the rest period, blood pressure measurements were taken at 00:15, 02:15, 04:15, 07:15, 10:15, 12:15 and 14:00 minutes. Instructions for the arithmetic task described above were given at 15:00 minutes. Similarly, to the speech task, blood pressure sampling was done at 00:15 minutes and 1:45 minutes from the beginning of the math task. At 3:00 minutes, participants were informed that the task was complete and were asked to sit quietly for another rest period. Blood pressure sampling was taken in the exact same manner as the post-speech rest

period described above. In order to control for task order effects, the task order was counterbalanced. At 15:00 minutes, the participant was told that the study was over. They were subsequently debriefed, received answers to all of their questions, and the procedure to receive compensations was explained.

Statistical Analyses

Data Reduction

In order to derive average waveform ensembles for the ECG and impedance cardiography signals during the speech and math task periods, a computer scoring program (COP/WIN) was used. During the rest periods preceding the speech and math tasks, COP/WIN software was used to derive baseline scores by calculating the average of the final three values of the assessed cardiovascular variables. Specifically, after the initial rest period was initiated, BP measurements were taken at 11:00, 12:30, and 14:00 minutes. Cardiovascular reactivity values were derived by averaging the two measurements taken during the math (00:15 and 01:45 minutes), speech prep (00:15 and 01:45 minutes) and speech delivery (03:15 and 04:45 minutes) tasks. Cardiac output was calculated using the formula (HR × SV) / 1000 and TPR was calculated as MAP/CO × 80. The arithmetic change scores were calculated for all measures (average reactivity value - average baseline value) to provide an index of task-induced changes in HR, CO, SBP, DBP, and TPR.

Data Analyses

Demographic characteristics (age, ethnicity, education level, and family income) and baseline measurements (i.e., HR, CO, SBP, DBP, and TPR) for the participants were analyzed for significant relationships to the cardiovascular reactivity measures, and all

data were inspected for normality. Standardized and studentized residual scatterplots and graphs were reviewed to assess for issues with linearity, normality, and homoscedasticity. All assumptions were deemed to be met. Analyses were also used to assess for the presence of univariate or multivariate outliers. One case with a high DES total score was found to be a univariate outlier. A review of this case revealed that the score, though extreme, was a legitimate part of the sample. Thus, as suggested by Tabachnick and Fidell (2001), in order to reduce the impact of this outlier the DES variable (which was positively skewed) was successfully transformed logarithmically. One other case was identified as a multivariate outlier through Mahalanobis' and Cook's distance scores. This outlier was also deleted from the analyses as it accounts for less than 5% of the total sample (Tabachnick & Fidell, 2001).

To assess the relationships of PTSD and depressive symptoms with cardiovascular reactivity to the stress task, regression analyses were performed; hierarchical multiple regression analysis were used to examine main effects and interactions relating the predictor variables—symptoms of PTSD (as measured by the CAPS), symptoms of depression (as measured by the BDI-II), symptoms of dissociation (as measured by the DES), and cognitive appraisals (as measured by the SAM)—to task-induced change-scores for the dependent variables (i.e., the cardiovascular measures–HR, CO, SBP, DBP, and TPR).

In order to retain power, the control variables (i.e., age, race, family income, and educational level) were examined first in order to determine which were significantly related to cardiovascular reactivity. Subsequently, the first hierarchical step contained those variables that were found to be related to the outcome variable. The second step

contained the proposed predictors. These include the PTSD symptoms (i.e., arousal, avoidance); depressive symptoms (i.e., activation and withdrawal); dissociation; and cognitive appraisals (challenge versus threat appraisals).

Test of Hypothesis 1: *It is hypothesized that there will be a positive relationship between PTSD arousal and cardiac reactivity, and a negative relationship between PTSD avoidance and cardiac reactivity.*

Multivariate regression models were used to test the hypothesis that there would be a positive relationship between the PTSD arousal symptom cluster (CAPS Cluster D) and cardiac reactivity (i.e., HR, CO, SBP, DBP, TPR), and a negative relationship between the PTSD avoidance symptom cluster (CAPS Cluster C) and cardiac reactivity. Since running multiple tests can lead to an increased type 1 error rate, the variables were first analyzed using a multivariate multiple regression. If the omnibus test was significant, the individual dependent variables (i.e., HR, SBP, DBP, CO, TPR) were then examined. This procedure was also used for any remaining hypotheses that assessed the relationship between one of the predictor variables and all of the above mentioned cardiovascular dependent variables.

Test of Hypothesis 2: *It is hypothesized that there will be a positive relationship between depressive activation and cardiovascular reactivity, and a negative relationship between depressive withdrawal and cardiovascular reactivity.*

Multivariate regression models were used to test the hypothesis that there would be a positive relationship between depressive activation and cardiovascular reactivity, and a negative relationship between depressive withdrawal and cardiovascular reactivity. The depressive activation variable was composed of the six BDI-II items previously

described by Bühler and colleagues (2014) as loading onto the "activation" factor. They are items 6 (punishment feelings), 9 (suicidal thoughts), 11 (agitation), 15 (loss of energy), 19 (concentration difficulties) and 20 (tiredness). According to this model, activation and withdrawal exist on the same metric in that lack of activation equals withdrawal and vice versa. Accordingly, some of the items were reverse scored so that the items would fall in the same direction when scored (i.e., high scores equaled increased activation and low scores equaled decreased activation or withdrawal). In comparison, an exploratory approach was used to assess other BDI-II items that conceptually seemed to fit with withdrawal behaviors. Specifically, a depressive withdrawal variable was created from five BDI-II items–4 (loss of pleasure), 12 (loss of interest), 16 (increase in sleeping), 18 (decrease in appetite) and 21 (loss of interest in sex)–in order to determine whether this variable would be significantly related to cardiovascular reactivity.

Test of Hypothesis 3: *It is hypothesized that there will be a negative relationship between dissociative symptoms and cardiovascular reactivity.*

Multivariate regression was used to test the hypothesis that there would be a negative relationship between dissociative symptoms (as measured by the DES total score) and cardiovascular reactivity.

Test of Hypothesis 4: *It is hypothesized that challenge appraisals will be related to greater cardiac reactivity whereas threat appraisals will be related to greater peripheral resistance.*

Multivariate regression models were used to test the hypothesis that challenge appraisals would be related to greater cardiac reactivity and threat appraisals to greater

peripheral resistance reactivity. Both challenge and threat appraisals were assessed via the SAM.

CHAPTER III

RESULTS

Descriptive Statistics

The sample consisted of women (N = 57) ranging from 19 to 52 years of age (M ± SD age = 30.72 ± 7.94. Demographic information is presented in Appendix A. Participants were 50.9% non-Hispanic White/Caucasian, 22.8% Hispanic White, 1.8% Hispanic Black, 10.5% African American, 8.8% Caribbean Black, 1.8% Haitian, 1.8% Asian or Asian-American, and 1.8% Bi-racial. Three control variables–ethnicity, education, and income–were found to be significantly related to one of the cardiovascular reactivity measures, SBP during Speech Prep. Subsequently, these control variables were entered into the first hierarchical step that assessed the relationship between the predictor variables and that dependent variable (i.e., SBP during Speech Prep).

Study Findings

PTSD and Cardiovascular Reactivity

PTSD avoidance

A multivariate regression analysis demonstrated a statistically significant omnibus effect for the relationship between PTSD avoidance symptoms and the cardiovascular reactivity measures during the speech prep, Wilks' Lambda $F(5, 37) = 2.47$, $p = 0.050$, $\eta_p^2 = 0.25$ (See Table 1). Follow up analyses of the significant multivariate effect with examination of the univariate relationships revealed a significant negative relationship between PTSD avoidance and DBP reactivity during speech prep, $F(1, 41) = 4.87$, $p = 0.033$, $\eta_p^2 = 0.11$, and a significant negative relationship between PTSD avoidance and TPR reactivity during speech prep, $F(1, 41) = 7.69$, $p = 0.008$, $\eta_p^2 = 0.16$.

Table 1. *CAPS Cluster C and Cardiovascular Reactivity During Speech Preparation*

	PTSD Avoidance		
Variable	*B*	*SE B*	*t*
HR Speech Prep Change Score	0.05	0.08	0.71
CO Speech Prep Change Score	0.01	0.01	0.67
SBP Speech Prep Change Score	-0.08	0.08	-0.98
DBP Speech Prep Change Score	-0.13	0.06	-2.21*
TPR Speech Prep Change Score	-3.93	1.42	-2.77*

Note. * = $p < 0.05$, ** = $p < 0.01$. Abbreviations: HR = heart rate in beats per minute (bpm); CO = cardiac output measured in liters per minute (l/min); SBP = systolic blood pressure measured in millimeters of mercury (mmHg); DBP = diastolic blood pressure measured in millimeters of mercury (mmHg); TPR = total peripheral resistance measured in peripheral resistance units (mmHg/L/min).

Table 2. *CAPS Cluster C and Cardiovascular Reactivity During Speech Task*

	PTSD Avoidance		
Variable	*B*	*SE B*	*t*
HR Speech Task Change Score	-0.05	0.08	-0.59
CO Speech Task Change Score	0.02	0.01	3.24**
SBP Speech Task Change Score	-0.13	0.12	-1.10
DBP Speech Task Change Score	-0.13	0.08	-1.64
TPR Speech Task Change Score	-5.69	1.28	-4.43**

Note. * = $p < 0.05$, ** = $p < 0.01$. Abbreviations: HR = heart rate in beats per minute (bpm); CO = cardiac output measured in liters per minute (l/min); SBP = systolic blood pressure measured in millimeters of mercury (mmHg); DBP = diastolic blood pressure measured in millimeters of mercury (mmHg); TPR = total peripheral resistance measured in peripheral resistance units (mmHg/L/min).

A multivariate regression analysis revealed a statistically significant omnibus effect for the relationship between PTSD avoidance and the cardiovascular reactivity measures during the speech task, Wilks' Lambda $F(5, 37) = 3.96$, $p = 0.006$, $\eta_p^2 = 0.35$ (See Table 2). Follow up of the significant multivariate effect with examination of the univariate relationships revealed a significant positive relationship between PTSD avoidance and CO reactivity during the speech task, $F(1, 41) = 10.46$, $p = 0.002$, $\eta_p^2 =$

0.20, and a significant negative relationship between PTSD avoidance and TPR reactivity during the speech task, $F(1, 41) = 19.65$, $p < 0.001$, $\eta_p^2 = 0.32$. No significant relationships were found between PTSD avoidance and any of the cardiovascular reactivity measures during the math task.

PTSD Arousal

For the speech task, a multivariate regression analysis demonstrated a statistically significant omnibus effect for the relationship between PTSD arousal and the cardiovascular reactivity measures during the speech task, Wilks' Lambda $F(5, 37) = 3.45$, $p = 0.012$, $\eta_p^2 = 0.32$.

Table 3. *CAPS Cluster D and Cardiovascular Reactivity During Speech Task*

	PTSD Arousal		
Variable	*B*	*SE B*	*t*
HR Speech Task Change Score	-0.21	0.11	-1.83
CO Speech Task Change Score	0.02	0.01	2.25*
SBP Speech Task Change Score	-0.27	0.17	-1.59
DBP Speech Task Change Score	-0.25	0.11	-2.20*
TPR Speech Task Change Score	-7.40	1.92	-3.87**

Note. * = $p < 0.05$, ** = $p < 0.01$. Abbreviations: HR = heart rate in beats per minute (bpm); CO = cardiac output measured in liters per minute (l/min); SBP = systolic blood pressure measured in millimeters of mercury (mmHg); DBP = diastolic blood pressure measured in millimeters of mercury (mmHg); TPR = total peripheral resistance measured in peripheral resistance units (mmHg/L/min).

Follow up analyses of the significant multivariate effect with examination of the univariate relationships revealed a significant positive relationship between PTSD arousal and CO reactivity during speech task, $F(1, 41) = 5.08$, $p = 0.030$, $\eta_p^2 = 0.11$, a significant negative relationship between PTSD arousal and DBP reactivity during speech task, $F(1, 41) = 4.86$, $p = 0.033$, $\eta_p^2 = 0.11$, and a significant negative relationship between PTSD

arousal and TPR reactivity during speech task, $F(1, 41) = 14.94$, $p < 0.001$, $\eta_p^2 = 0.27$ (See Table 3). No significant relationship was found between PTSD arousal and any of the cardiovascular reactivity measures during the speech prep and math tasks.

Depression and Cardiovascular Reactivity

Depressive activation

No significant relationships were found between depressive activation and the cardiovascular reactivity measures during the speech prep, speech delivery, or math tasks.

Depressive withdrawal

A multivariate regression analysis revealed a statistically significant omnibus effect for the relationship between depressive withdrawal and the cardiovascular reactivity measures during the speech prep, Wilks' Lambda $F(5, 37) = 2.35$, $p = 0.060$, $\eta_p^2 = 0.35$.

Table 4. *BDI-II Withdrawal and cardiovascular reactivity measures during Speech Prep*

	Depressive Withdrawal		
Variable	*B*	*SE B*	*t*
HR Speech Prep Change Score	0.49	0.42	1.16
CO Speech Prep Change Score	0.04	0.04	1.20
SBP Speech Prep Change Score	0.52	0.47	1.11
DBP Speech Prep Change Score	-0.59	0.34	-1.73
TPR Speech Prep Change Score	-15.97	7.51	-2.13*

Note. * = p < 0.05, ** = p < 0.01. Abbreviations: HR = heart rate in beats per minute (bpm); CO = cardiac output measured in liters per minute (l/min); SBP = systolic blood pressure measured in millimeters of mercury (mmHg); DBP = diastolic blood pressure measured in millimeters of mercury (mmHg); TPR = total peripheral resistance measured in peripheral resistance units (mmHg/L/min).

Follow up analyses of the significant multivariate effect with examination of the univariate relationships revealed a significant negative relationship between depressive

withdrawal and TPR reactivity during speech prep, $F(1, 41) = 4.53$, $p = 0.039$, $\eta_p^2 = 0.10$ (See Table 4).

A multivariate regression analysis revealed a statistically significant omnibus effect for the relationship between depressive withdrawal and the cardiovascular reactivity measures during the speech task, Wilks' Lambda $F(5, 37) = 3.35$, $p = 0.014$, $\eta_p^2 = 0.31$. Follow up analyses of the significant multivariate effect with examination of the univariate relationships revealed a significant negative relationship between depressive withdrawal and DBP reactivity during speech task, $F(1, 41) = 4.70$, $p = 0.036$, $\eta_p^2 = 0.10$ (See Table 5)

Table 5. *BDI-II Withdrawal and cardiovascular reactivity measures during Speech Task*

	Depressive Withdrawal		
Variable	*B*	*SE B*	*t*
HR Speech Task Change Score	-0.32	0.45	-0.70
CO Speech Task Change Score	-0.05	0.43	-1.19
SBP Speech Task Change Score	0.47	0.64	0.74
DBP Speech Task Change Score	-0.88	0.41	-2.17*
TPR Speech Task Change Score	-9.62	7.43	-1.29

Note. * = $p < 0.05$, ** = $p < 0.01$. Abbreviations: HR = heart rate in beats per minute (bpm); CO = cardiac output measured in liters per minute (l/min); SBP = systolic blood pressure measured in millimeters of mercury (mmHg); DBP = diastolic blood pressure measured in millimeters of mercury (mmHg); TPR = total peripheral resistance measured in peripheral resistance units (mmHg/L/min).

No significant relationship was found between depressive withdrawal and the cardiovascular reactivity measures math tasks.

Dissociation and Cardiovascular Reactivity

None of the relationships were found to be significant between dissociative symptoms and cardiovascular reactivity during the speech prep, speech task, and math task phases.

Cognitive Appraisals and Cardiovascular Reactivity

Challenge appraisals

No significant relationships were found between challenge appraisals and the cardiovascular reactivity measures during the speech prep, speech task or math task.

Threat Appraisals

No significant relationships were found between threat appraisals and the cardiovascular reactivity measures during the speech prep, speech task or math task.

CHAPTER IV

DISCUSSION

The present study sought to investigate the relationship(s) between psychological variables (i.e., PTSD, depressive activation and withdrawal, dissociation, and cognitive appraisal) and cardiovascular stress reactivity. In addition to utilizing non-invasive measures of cardiovascular reactivity that permit more in-depth measures of cardiovascular response beyond HR and BP (e.g., impedance cardiography), this study attempted to address a gap in the literature by testing the hypotheses that symptom type/category presentation (e.g., PTSD hyperarousal versus PTSD avoidance, depressive activation versus depressive withdrawal) or psychological variables (e.g., cognitive appraisals, dissociation) can be used to predict cardiovascular reactivity. The present approach also provided the advantage of examining cardiac versus vascular responses to stress.

Findings Relative to Previous Research

As hypothesized, there were significant relationships between both the PTSD arousal and PTSD avoidance subtypes and cardiovascular reactivity measures. While numerous studies have shown links between PTSD and measures of cardiovascular reactivity (Beckham et al., 2002; D'Andrea et al., 2013; McTeague et al., 2010; Metzger et al., 1999; O'Kearney & Parry, 2014; Paulus, Argo, Egge, 2013; Wolfe et al., 2000), this study is one of the first to provide limited evidence of a link between the subtypes, PTSD arousal and PTSD avoidance, and cardiovascular reactivity. PTSD arousal was positively related to CO, and was negatively related to DBP and TPR during the speech task. PTSD avoidance was also negatively related to DBP during speech prep and TPR

during speech prep and speech task.

The PTSD avoidance subtype was thought to represent those individuals who are more likely to evidence an avoidant/dissociative reaction in response to stress (such as those exposed to chronic or multiple trauma episodes). Thus, it was thought that individuals who exhibit PTSD avoidance would be more likely to exhibit blunted reactivity, as has been found in studies looking at the relationship between PTSD symptoms, trauma exposure(s), and reactivity (D'Andrea, Pole, DePierro, Freed, & Wallace, 2013; McTeague et al., 2010). However, this was not the case. Specifically, there was a similarity between the two proposed subtypes and their relationship to cardiovascular reactivity in that both subtypes were linked to increased cardiac reactivity and decreased vascular reactivity.

With regards to the PTSD avoidance subtype, one potential explanation regarding the cardiovascular reactivity pattern is that in the same sense that dissociation (a cognitive process) is distinctly different from avoidance (a behavioral process), PTSD avoidance may be distinctly different from PTSD dissociation. Specifically, any increase in mental activity, even during quiet tasks, results in a cardiovascular change (Hjemdahl, 2007). Given the nature of avoidance (as a behavioral rather than cognitive construct), the act of consciously making an effort to consistently avoid stimuli (i.e., either persons, places, or things) that trigger memories of the traumatic event may necessitate a heightened cardiovascular response. Thus, individuals who present with the PTSD avoidant subtype may not necessarily exhibit blunted reactivity during stressful tasks.

Additionally, another explanation for the similar cardiovascular response patterns of the two PTSD subtypes may relate to research regarding the typical patterns of

reactivity associated with each of the stressors used in this study. Laboratory stressors can be broken down into two specific categories: passive/inhibitory tasks or active coping tasks. In passive tasks (e.g., receiving an electric shock, cold pressor, or watching a disturbing film), participants are passive recipients to the provided stimulus/stressor, have little control over the situation and are not too involved with the task (Malhotra, 2009). This is in contrast to active tasks (e.g., Stroop test, public speaking, and mental arithmetic) which provide participants with a little more control and involvement/task engagement. According to Zanstra and Johnston (2011), both active and passive coping tasks generate differing hemodynamic response patterns. Specifically, while passive coping tasks tend to be linked to alpha (α) adrenergic responses, active coping tasks tend to be linked to beta (β) adrenergic responses (Gramer, 2006).

The α-adrenergic response pattern is characterized by a decrease in arterial compliance and an increase in diastolic blood pressure and total peripheral resistance; in contrast, the β-adrenergic response pattern is characterized by increases in cardiac reactivity (e.g., systolic blood pressure, heart rate, and cardiac output; Uys, 2012) and smaller or no effects on vascular resistance and diastolic blood pressure (Gramer, 2006). Results of the study found that both PTSD avoidance and PTSD arousal were linked to increases in cardiac reactivity (i.e., CO) and decreases in vascular reactivity (e.g., DBP, TPR) during the public speaking tasks. Thus, consistent with the literature, the speech task (an active coping task) evoked β-adrenergic response patterns irrespective of the PTSD subtypes under study.

Moreover, the results regarding increased cardiac reactivity in response to the public speaking stress task is consistent with other research which has shown that public

speaking produces a stable pattern of cardiac activation (Al'Absi et al., 1997). Specifically, while speech delivery tasks are often used to elicit cardiac reactivity responses, mental arithmetic and speech preparation tasks are frequently used to evoke cardiac and vascular reactivity. While Uys (2012) notes that researchers have argued that rather than the response depending on the stressor, an individual is more likely to exhibit a dominant adrenergic type response in general, the results of this study do not appear to support this assertion. Instead, it would seem that stimulus-response specificity–the process whereby specific stimuli lead to unique patterns of response (Arena & Schwartz, 2003) influenced the relationship between the above-mentioned variables. Thus, the ability to elicit a different cardiovascular response from the two PTSD subtypes may be dependent on the nature of the psychological stressor used.

Another potential explanation may relate to the previous findings regarding baseline measurements in individuals with PTSD, and the potential role of the sympathetic and parasympathetic nervous systems. In a meta-analysis by Buckley and Kaloupek (2001), results revealed that in comparison to individuals without PTSD (both trauma-exposed and non-trauma exposed), those with PTSD evidenced higher resting HR and BP. Similarly, Pole's (2007) meta-analytic study found significant weighted mean effects of PTSD for both HR and skin conductance. In addition, while Blanchard (1990) found basal elevations in HR and BP among individuals with PTSD relative to control groups, Keane et al. (1998) found elevations in HR and skin conductance in participants with current PTSD. Though some may attribute the basal elevations in individuals with PTSD to anticipatory anxiety, studies that have used 24-hour ambulatory monitoring have also found higher physiological arousal (i.e., HR and/or DBP) in individuals with PTSD

(Buckley et al., 2004; Muraoka, Carlson, & Chemtob, 1998).

Given this finding, this would seem to suggest that individuals with PTSD may evidence some dysfunction in their autonomic nervous system (ANS) that leads to chronically elevated physiological arousal. Specifically, the ANS plays an important role in several bodily functions including the maintenance of heart rate, blood pressure, and initiation/maintenance of sleep, among other things (Silver, Markoff, Naghi, Silver, & May, 2016); and it also helps to maintain homeostasis in the body. The ANS is further divided into two divisions, the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). Whereas the SNS takes a predominant role in fight-or-flight reactions and functions to prepare the individual for strenuous activity, the PNS takes the predominant role of calming down the individual, conserving and storing energy, and regulating basic body functions (McCorry, 2007). In individuals with elevated basal measurements, as in the case of some individuals with PTSD, this would seem to suggest some suppression of their PNS or reduced parasympathetic control that leads to chronically elevated basal measurements.

Specifically, individuals with PTSD by nature of their disorder, have been exposed to one or several traumatic events that have lasting psychological as well as physiological impacts on their body. Given the finding of chronically elevated basal measurements within this population, then this would appear to be indicative of an overabundance of SNS activity and a suppression of PNS activity. In PTSD, one's traumatic experience(s), and the chronicity of triggers/stressors (whether characteristics of the environment or one's intrusive cognitions/memories), often leads one to frequently experience hypervigilance. This hypervigilance may be accompanied by a chronic stress

response (e.g., activation of the SNS) which would result in one constantly experiencing the fight-or-flight response. Thus, Williamson and colleagues (2015) note that individuals with PTSD experience changes in their autonomic states (from fight-or-flight, withdrawal, immobilization, to dissociation) without an intervening calm state; and although this response is adaptive in life-threatening situations, in the context of day to day life it can contribute to the development of other negative health outcomes. This could then lead to suppression of the PNS, and increase the likelihood that one will experience elevated basal measurements which would in turn affect the relationship between PTSD symptoms and cardiovascular reactivity.

The current study also attempted to identify how different depressive presentation can affect cardiovascular reactivity in response to stress. Most of the studies assessing the relationship between depression and cardiovascular reactivity have focused on overall depression (e.g., total score on the BDI-II), depressive severity (e.g., high versus low depression), or depressive mood states (e.g., current versus remitted depression). However, one factor that is often overlooked is the clinical variability among the presence/severity of varying symptom dimensions of depression (for example, affective versus somatic symptoms; Dotson, 2017). Although studies of the BDI-II have often proposed somatic-affective and cognitive factors, a more recent study suggested that another factor, activation, may underlie the BDI-II's factor structure (Bühler et al., 2014). Thus, this study proposed that individuals who present with more agitated (i.e., more activation) versus vegetative (i.e., withdrawal) symptoms may represent specific depressive subtypes that may evidence distinct patterns of cardiovascular reactions in response to stress.

In contrast to our hypothesis, no significant relationships were found between depressive activation, comprised of the items described by Bühler and his colleagues (2014), and cardiovascular reactivity. One possible reason for the lack of the association may relate to the activation construct overall. Specifically, in the study by Bühler et al. (2014) the items that comprised the activation factor (i.e., 6-punishment feelings, 9-suicide, 11-agitation, 15-loss of energy, 19-concentration difficulties, 20-tiredness) related to varying levels of arousal/activation. Given the behavioral aspect of activation, it may be that individuals who present as high on depressive activation may present as more hemodynamically similar to individuals with remitted depression.

For example, a study by Salomon and colleagues (2013) sought to investigate whether blunted cardiovascular reactivity associated with depression was mood-state dependent, trait-like, or can be observed in vulnerable individuals without depression. They subjected participants–50 with MDD, 25 with remitted major depression (RMD), and 45 healthy controls–to a speech stressor and forehead cold pressor task while assessing their cardiovascular reactivity. Individuals in the RMD group evidenced BDI-II scores falling in the minimal or mild depression range (i.e., 0-19). Results revealed that while individuals with current MDD exhibited blunted reactivity, individuals in the RMD group exhibited similar reactivity patterns with the healthy controls in the group.

Another possible explanation relates to the factor loadings noted in the G3F model proposed by Buhler et al. (2014). Specifically, a review of the items comprising the activation factor (i.e., items 6-punishment feelings, 9-suicidal thoughts, 11-agitation, 15-loss of energy, 19-concentration difficulties and 20-tiredness) revealed that each of these items loaded significantly onto the activation factor in addition to two other factors

in the study (e.g., the general, cognitive, or somatic factors), a concept known as crossloading. According to Costello and Osborne (2005), crossloading is defined as an item loading at 0.32 or higher on two factors; and Yong and Pearce (2013) note that it is preferable that there are few item crossloadings so that each factor proposed in the model represents a distinct cluster of interrelated variables. The items comprising the activation factor evidenced multiple crossloadings with the above-mentioned factors; thus, this may suggest a diffuse pattern (or indistinct underlying structure) which may have affected the likelihood of detecting a relationship between depressive activation and cardiovascular reactivity.

Though it was hypothesized that depressive withdrawal would be negatively related to cardiovascular reactivity, results revealed that depressive withdrawal was negatively related solely to vascular reactivity. Specifically, depressive withdrawal was negatively related to TPR during the speech prep task and negatively related to DBP during the speech task. It is especially interesting to note that no significant relationships were found between depressive withdrawal and cardiac reactivity in spite of the traditional relationship between speech tasks and cardiac reactivity. As mentioned previously, public speaking tasks/active coping stressors are often associated with a β-adrenergic response characterized by increases in cardiac reactivity and smaller or no effects on vascular resistance and diastolic blood pressure. However, in contrast to this traditional relationship, individuals who were high on depressive withdrawal evidenced no significant cardiac reactivity.

One potential explanation relates to the factors that affect cardiovascular reactions in the body. Specifically, whereas the SNS prepares an individual for activity/fight-or-

flight reactions (e.g., by increasing heart rate, blood pressure, etc.), the PNS helps to calm down the individual (e.g., by decreasing heart rate, blood pressure, etc.). Moreover, the PNS is also responsible for vasodilation, the process whereby blood vessels widen, leading to a decrease in blood pressure and TPR. While several studies have found a link between depression and increased sympathetic activity or decreased parasympathetic activity (Frasure-Smith, Lesperance, & Talajic, 2000; Jarrett et al., 2003; Shi et al., 2014; Veith et al., 1994; Wang et al., 2013), the finding of a negative relationship between depressive withdrawal and reactivity, runs counter to this and would seem to suggest increased parasympathetic activity. This is consistent with a review by Plant and Stephenson (2009) regarding different types of depression. One depression type reviewed was dysthymic depression, a condition characterized by chronic, persistent, low levels of depression/depressed mood; and they noted that dysthymic depression is thought to be associated with decreased adrenal gland and sympathetic nervous system activity and increased parasympathetic activity. Given the nature of dysthymic depression and its symptom presentation (e.g., chronically depressed mood, loss of pleasure, loss of interest, decreased appetite,), individuals who score high on depressive withdrawal may share some behavioral/ underlying similarities with this depressive type and may evidence a similar hemodynamic response pattern.

Moreover, another study by Shinba (2014) sought to investigate autonomic activity and reactivity in depression by using heart-rate variability (HRV) and HR measurements. They found that some patients evidenced high parasympathetic activity; specifically, patients with depression evidenced changes in their HRV (during the rest condition) which were regarded as increments in their baseline arousal level as the

patients tried to stay relaxed. Accordingly, they suggested that a subset of patients with depression are likely to exhibit underarousal, which further indicates that there is variability among depression with regards to arousal state. While in a healthy individual the body can switch between sympathetic and parasympathetic states in response to stress, it may be that individuals high on depressive withdrawal (or vegetative) symptoms may evidence some dysfunction in their autonomic nervous system which makes this switch problematic. Thus, individuals who present with more withdrawal may subsequently evidence increased parasympathetic activity on a more regular basis.

Further support for the significance of the relationship between depressive withdrawal and cardiovascular reactivity was found in another study by Stewart and colleagues (2007). They sought to investigate the relative importance of anxiety symptoms, depressive symptoms, and hostility/anger in predicting 3-year progression of subclinical atherosclerosis (as measured via the assessment of carotid intima-media thickness; IMT). Although measures of hostility, anger experience/expression, and anxiety symptoms were found to be unrelated to IMT change, mild to moderate depressive symptoms (as measured by the BDI-II) were linked to greater 3-year change in carotid IMT.

More specifically, post-hoc analyses revealed that it was the somatic-vegetative subscale score, rather than the cognitive-affective subscale score, of the BDI-II which was predictive of IMT change. A review of the items comprising the somatic-vegetative subscale score revealed that it included all five of the items that comprised the proposed withdrawal variable in this study (i.e., items 4, 12, 16, 18, and 21) and a few other items (i.e., items 10-11, 15, 17, 19-20). In their study, the somatic-vegetative score explained

the same amount of variance as did the total score on the BDI-II, suggesting that this symptom cluster was responsible for the relationship between IMT change and the BDI-II total score. Thus, this may also help to explain the way in which depression, and more specifically depressive withdrawal/vegetative symptoms, can confer an increased risk for cardiovascular disease.

Several studies have assessed the relationship between depressive sympto-matology and cardiovascular reactivity (Betensky & Contrada, 2010; Light et al., 1998; Phillips, 2011b; Salomon et al., 2009; Salomon et al., 2013); and in these studies, no distinction was made between the proposed depressive subtypes. Specifically, the presence of depression or a mood disorder was assessed by using diagnostic interviews such as the SCID-I (Salomon et al., 2009) or total or established cutoff scores on self-report measures such as the BDI-II, HADS, HRSD, and CES-D (Betensky & Contrada, 2010; Light, Kothandapani, & Allen, 1998; Matthews et al., 2005; Phillips, 2011; Salomon et al., 2013). In contrast to the aforementioned studies, which often categorized participants as presenting with high versus low depression (i.e., via median split or cutoff scores), this study is the first to specifically look at two facets/types of depressive presentation–depressive activation and depressive withdrawal–and how they can affect one's cardiovascular response to stress.

This study provides some preliminary support for the relevance of the depressive withdrawal subtype and its potential impact on cardiovascular reactivity. While the present study did not find a link between depressive activation and cardiovascular reactivity, future studies should attempt to study this area of research as it may have implications for other studies. For example, one study that may lend credence to this

perspective focused on investigating the independent and interactive effects of depressive and aggressive traits on HR, SBP, and DBP (Betensky & Contrada, 2010). They found that only depressed participants with high verbal aggression–not low verbal aggression–evidenced increased SBP and DBP reactivity during a stressful speaking task. Although there could be several explanations to support this finding perhaps this presentation–depressive with high verbal aggression–may be indicative of or similar to the depressive activation subtype. This could accordingly help to explain why only individuals who presented with depressive and high, rather than low, verbal aggression evidenced high SBP and DBP.

In contrast to previous studies which have shown a relationship between dissociation and cardiovascular reactivity, no statistically significant relationships were found between dissociation and cardiovascular reactivity in this study. This is consistent with other studies that have found comparable levels of reactivity between high and low dissociators (Kaufman et al., 2002; Nixon et al., 2005) or no association between dissociation and physiological reactivity (Halligan, Michael, Wilhelm, Clark, & Ehlers, 2006). One factor that may explain this finding relates to the nature of the DES which was used to measure dissociation in this sample.

Several types of dissociation have been mentioned in the literature (e.g., peritraumatic dissociation, persistent dissociation, somatoform dissociation, etc.). The DES quantifies the frequency of dissociative phenomena experienced in daily life (Giesbrecht, Lynn, Lilienfeld, & Merckelbach, 2008) and assesses a trait form of dissociation (Holmes, Oakley, Stuart, & Brewin, 2006). In contrast, other studies that have found a significant relationship between dissociation and reactivity (Griffin et al.,

1997; Ladwig et al., 2002; Pole et al., 2005; Sledjeski & Delahanty, 2012) have often used measures (e.g., the PDEQ) that specifically assess for peritraumatic dissociation, a state form of dissociation that occurs during or after a traumatic event.

Second, most of the above-mentioned studies have used trauma-specific (or trauma-related) imagery, cues, or recall to stress their participants prior to assessing their levels of dissociation. While speech and mental arithmetic tasks are frequently successfully used in studies to elicit a cardiovascular stress response, Reinders et al. (2014) note that the use of personal trauma scripts often results in individuals reporting symptoms of dissociation and exhibiting blunted reactivity.

Third, the relationship between dissociation and cardiovascular reactivity may be impacted by the general distribution of dissociation among individuals with PTSD and the nature of the dissociative subtype. Although dissociation is a symptom that is commonly associated with PTSD/trauma, research has shown that only a distinct subset of individuals with PTSD also/concurrently exhibit dissociative symptoms (Lanius et al., 2014). Putnam and colleagues (1996) found that among individuals with PTSD, mean dissociation scores were carried by a small proportion of individuals who exhibited high dissociation, rather than dissociation being evenly distributed among the entire group. This was later corroborated by Waelde, Silvern and Fairbank (2005) who found evidence for a discontinuous distribution of DES scores, with distinct cases at the tail end of the distribution that exhibited high levels of PTSD.

Furthermore, Wolf and colleagues (2012) used latent profile analysis to examine the evidence for a dissociative subtype of PTSD in a sample of 492 individuals (i.e., veterans and their intimate partners) who had a history of trauma. They found that in

addition to a low PTSD severity subgroup and a high PTSD severity subgroup, there was a third dissociative subgroup–12% of the sample–who evidenced high scores on depersonalization and derealization. These findings were replicated by Steuwe, Lanius, and Frewen (2012) and Stein et al. (2013) who found that 25% of their civilian sample and 14% of their cross-cultural sample, respectively, could be classified as comprising a dissociative subgroup based on their high scores on depersonalization and derealization.

While some of this study's participants endorsed items on the DES, most of the participants did not endorse significantly elevated symptoms (especially on items assessing depersonalization or derealization), and accordingly, would not appear to comprise this dissociative subgroup. Given that individuals who fall into the dissociative subgroup differ significantly from their counterparts with PTSD who do not evidence these symptoms, they may be more likely to exhibit exaggerated or blunted cardio-vascular reactivity patterns in response to stress. That is, with regards to influences on cardiovascular reactivity, there is most likely a distinct difference between individuals who exhibit normal (low level) dissociation (i.e., symptoms such as daydreaming, absorption, etc.) and those who exhibit pathological (high level) dissociation (e.g., symptoms such as depersonalization, derealization, etc.). Future studies should attempt to determine whether or not cardiovascular reactivity is more likely to occur in the latter, dissociative subgroup.

While several studies have found a link between cognitive appraisals and cardiovascular reactivity (Maier et al., 2003; Quigley et al., 2002; Schneider, 2004; Tomaka & Blascovich, 1994; Zanstra et al., 2009), no statistically significant relationships were found between appraisals and cardiovascular reactivity in this study.

This finding was similar to other studies which have not found significant relationships between cognitive appraisals and cardiovascular reactivity patterns (Hartley, Ginsburg, & Heffner, 1999; Kelsey et al., 2000; Malhotra, 2009; Meijen, Jones, Sheffield, & McCarthy, 2014).

The lack of a relationship between cognitive appraisals and cardiovascular reactivity may be explained in a couple of ways. First, although studies appear to use the terms challenge/threat appraisals and challenge/threat states interchangeably, it is possible that these two characteristics may be differentiated by the way in which they are assessed or evoked, respectively, and may have differential effects on cardiovascular reactivity. For example, in this study, participants provided their cognitive appraisals of the stress tasks using the SAM. In this sense, it was expected that the participants' automatic and unconscious appraisal of the stressors, as being challenging or threatening, would impact their self-report of their own personal experience and would consequently affect their cardiovascular reactivity response patterns. In contrast, challenge and threat states are frequently manipulated via task instructions (Feinberg & Aiello, 2010; Moore, Vine, Wilson, & Freeman, 2012) and lead the individual to enter into a challenge or threat state. Thus, while Blascovich and Mendes (2000) note that individuals generally arrive at a threat or challenge state without being aware of the process, it is probable that the use of verbal manipulations affects the automaticity of the process, and may also impact the type of cardiovascular response one experiences, if any.

Additionally, as previously mentioned, cognitive appraisals, describe the process whereby an individual appraises an event and the importance it holds for that individual's well-being. One's cognitive appraisal of any given situation entails a primarily

unconscious process; and Monroe & Kelley (1997) note that it is difficult to measure cognitive appraisals as it is a process that is constantly evolving internally. In this particular study, as participants were presented with the stress tasks, they most likely appraised the task– rather unconsciously–as being challenging or threatening for them which would then affect their cardiovascular reaction to the stress tasks. Given the rather automatic nature of the cognitive appraisal process, Malhotra (2009) states that asking individuals to self-report their appraisals may not entirely tap into the unconscious appraisal process. Accordingly, he notes that some research has suggested that the assessment of one's cognitive appraisals would probably be better ascertained via the use of observer ratings.

Limitations of the Study

In terms of the limitations of the present study, given that the sample was comprised solely of women this affects the generalizability of the findings. This is especially relevant given that it is well documented that sex differences exist in cardiovascular reactivity to stress (Whited & Larkin, 2009). Future studies should attempt to assess these variables in men in order to determine if the findings are replicated in this population as well. In addition, one case was removed prior to the analyses as it was identified as a multivariate outlier. A review of this case revealed that this participant differed significantly from other participants on several physiological responses (e.g., elevated HR and CO and decreased TPR) during the stress tasks. Thus, the results of this study would not generalize well to an individual who presents with a similar presentation (Tabachnick & Fidell, 2001). Moreover, the statistically significant results linking psychological factors to cardiovascular reactivity in this study occurred during the speech

prep or speech task phases of the study. While numerous studies have successfully used both public speaking and mental arithmetic tasks as laboratory stressors, it would appear that in the present study the math stressor may not have been deemed stressful enough and accordingly did not elicit much of a cardiovascular response.

Furthermore, research has previously discussed the possibility that cardiovascular responses to laboratory stressors (e.g., mental arithmetic, mirror tracing, cold pressor, etc.) may not always generalize to everyday settings (Waldstein, Neumann, Burns, & Maier, 1998). Thus, suggestions have been made that laboratory stressors that are more comparable to events one would encounter in daily life may increase the generalizability of one's findings (Turner et al., 1994). Future studies should attempt to use other stressors, whether social (e.g., public speaking) or trauma-relevant (e.g., trauma imagery, scripts, and recall), in order to evoke a cardiovascular stress response as the latter have been shown to elicit greater cardiovascular reactivity, especially in individuals with PTSD (Wolfe et al., 2000). This is especially salient as several studies have shown that individuals with PTSD may evidence altered psychophysiological responses when presented with non-trauma related situations (Kibler, 2009).

Implications for Practice and Future Research

Despite its limitations, this study is distinct in that it has provided preliminary support for the theory that different PTSD (avoidant versus arousal) or depressive (activation versus withdrawal) subtypes may lead to different cardiovascular reactions in response to stress. This is especially salient given the multitude of research which has found a link between both PTSD and depression and future incidences of cardiovascular disease/conditions. Although PTSD was initially considered to be a normative–though

extreme–response to traumatic/stressful situations (Sherin & Nemeroff, 2011), Kumar and Noel (2007) note that acute mental stress serves as a trigger for major autonomic cardiovascular responses as well as acute cardiac events. Specifically, PTSD is contingent upon psychosocial stressors (Bonne, Grillon, Vythilingham, Neumeister, & Charney, 2004); and psychosocial stress is an integral precipitant of morbidity (Tawakol et al., 2017). While one's stress response is indicative of the body's systems-level response to address threats to homeostasis (Carroll, Ginty, Whittaker, Lovallo, & 2017), individuals with PTSD, or even depression, can become sensitized to negative events in their environment and may consistently evince heightened or attenuated cardiovascular reactions in response to stress. Thus, changes in one's autonomic nervous system, in response to acute or chronic psychosocial stressors, can lead to over or under responding. This is especially salient given that rigidity of response reflects inflexibility and inability to adapt (Salmon et al., 2013), which negatively impacts the body's ability to address and/or recover from future stressors.

Additionally, while no significant relationships were found between depressive activation and cardiovascular reactivity, dissociation and cardiovascular reactivity or challenge/threat appraisals and cardiac reactivity, this study has suggested several potential factors that may impact the presence of a relationship between these variables. They include the hemodynamic presentation of individuals high on depressive activation; factor analytic crossloading of the BDI-II items associated with activation; nature of the traits assessed by the measures administered during the study; the use of trauma-relevant stimuli to stress participants; the distribution of dissociation among individuals with PTSD; differences between challenge/threat states and challenge/threat appraisals; and

the way in which appraisals are determined (i.e., via self-report or observer ratings). Given the bulk of studies which have shown some links between depression, dissociation, cognitive appraisals, and cardiovascular reactivity, future studies should attempt to further delineate factors that may influence the nature of the relationships between these variables and their implications for one's psychological and physiological health.

Research regarding the link between PTSD and depression and cardiovascular reactivity is important for several reasons. First, in accordance with the exaggerated reactivity hypothesis, Malhotra (2009) states that elevated cardiovascular reactions in response to stress are thought to contribute to the pathogenesis of cardiovascular disease (e.g., by damaging one's blood vessels which leads to inflammatory responses in the body). Thus, psychophysiological studies such as the present study allow for a better understanding of the ways in which stress affects the body, especially in susceptible populations (such as those with PTSD or depression).

Moreover, whereas PTSD has been linked to subsequent cardiovascular disease, a bidirectional relationship has been found between depression and cardiovascular disease, where seemingly healthy individuals with depression have an increased likelihood of experiencing cardiovascular disease (Hales, 2016; Mitka, 2005); and depressed individuals evidence an increased likelihood of developing coronary heart disease (NHLBI, 2014b). Given these findings, it was important to determine specific factors associated with depressive diagnoses that may negatively influence one's cardiovascular reaction in response to stress. While the literature was replete with studies that linked overall depression (or depressive severity) to cardiovascular reactivity, the present study adds to the literature by suggesting specific subtypes which may affect one's

cardiovascular reaction in response to stress. As more studies attempt to investigate these factors further it is thought that clinical recommendations could be made that are tailored to individuals' unique symptom presentations.

Future studies should attempt to replicate this study's findings as they may have far reaching implications for theory development and clinical practice. This is especially relevant as both PTSD and depression have frequently been linked to the etiology and progression of cardiovascular diseases. Furthermore, a deeper understanding of the relationships between these psychological variables and cardiovascular reactivity can help to foster primary prevention by understanding how these variables influence one another or facilitate the development of other diseases/conditions. Thus, further research and longitudinal studies can provide information that will help add to the knowledge base regarding preventative practices.

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APPENDIX A

Table 1. *Demographic Characteristics*

Characteristics	Participants (N = 57)
Ethnicity	
White or Caucasian/Non-Hispanic	29 (50.9)
African American	6 (10.5)
Hispanic/White or Caucasian	13 (22.8)
Hispanic Black	1 (1.8)
Asian or Asian-American	1 (1.8)
Caribbean Black	5 (8.8)
Haitian	1 (1.8)
Bi-racial	1 (1.8)
Marital Status	
Single, Never Married	37 (64.9)
Divorced or Separated	6 (10.5)
Married/Domestic Partner	14 (24.6)
Education	
High School Diploma or Equivalent	16 (28.1)
Associate's Degree (Junior College)	12 (21.1)
Bachelor's Degree	20 (35.1)
Master's Degree	7 (12.3)
Professional Degree (MD, JD, DDS, etc.)	2 (3.5)
Income	
$0-$5,000	5 (8.8)
$5,001-$10,000	5 (8.8)
$10,001-$15,000	3 (5.3)
$15,001-$20,000	6 (10.5)
$20,001-$30,000	5 (8.8)
$30,001-$40,000	7 (12.3)
$40,001-$50,000	8 (14.0)
$50,001-$75,000	8 (14.0)
$75,001-$100,000	5 (8.8)
$100,001 or more	5 (8.8)