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An Extension of the Perseverative Cognition Hypothesis to Posttraumatic Stress Disorder Symptomatology: Cardiovascular Recovery in Relation to Posttraumatic Stress Disorder Severity and Cognitive Appraisals of Stress

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Abstract

Beyond the construct of cardiovascular reactivity, the measurement of cardiovascular recovery from stress represents an important index of exaggerated physiological arousal and disease risk. Cardiovascular recovery in posttraumatic stress disorder (PTSD) has not received adequate attention. The present study examined whether cardiovascular recovery following an oral speaking stressor was associated with the severity of PTSD symptoms, and whether cognitive stress appraisals of threat and challenge were significantly associated with PTSD severity and recovery. The sample consisted of 50 trauma-exposed civilian women ranging from 19 to 49 years of age ($M = 30.9$, $SD = 7.8$). The PTSD severity indices were quantified based on structured interview. Cardiovascular recovery was assessed at two posttask time points as percentage return to baseline; the recovery measures consisted of impedance cardiography-derived cardiac output (CO) and total peripheral resistance (TPR), heart rate and blood pressure. Total PTSD severity was associated with less CO recovery, $r = -.39$, $p = .006$; this effect was similar across PTSD symptom categories, with significant correlations ranging from $r = -.30$ to $r = -.44$. However, only PTSD severity in the avoidance cluster was associated with less TPR recovery, $r = -.29$, $p = .047$. Total PTSD severity was associated with greater threat appraisal, $r = .30$, $p = .035$, and greater threat appraisal was associated with less CO recovery, $r = -.33$, $p = .019$. Results partially support the theory that greater PTSD severity and cognitive appraisals of threat contribute to less cardiovascular recovery when confronted with a stress-inducing situation.

Research has revealed an association between posttraumatic stress disorder (PTSD) and poor cardiovascular health (e.g., Kibler, Tursich, Ma, Malcolm, & Greenbarg, 2014; Xue et al., 2012). One proposed mechanism by which PTSD may confer cardiovascular disease (CVD) risk is through a prolonged pattern of greater cardiovascular reactivity to stress (Buckley, Holohan, Greif, Bedard, & Suvak, 2004; Dedert, Calhoun, Watkins, Sherwood, & Beckham, 2010). Research has consistently shown greater cardiovascular reactivity to trauma-related cues in PTSD (e.g., Orr, Pitman, Lasko, & Herz, 1993; Wolfe et al., 2000). These findings have been interpreted as conditioned learning of autonomic nervous system hyperarousal. However, studies that have utilized generic, nontrauma-related stress paradigms (e.g., anger

recall, mental arithmetic) to examine reactivity in PTSD have revealed mixed results; methodological features of study designs may have limited the effects in some of these studies. For example, one study used the period following task instruction and preceding the stress tasks (rather than a preinstruction period) as the prestressor baseline (Orr, Meyerhoff, Edwards, & Pitman, 1998); this method is confounded by anticipatory reactivity. A meta-analysis of the psychophysiological reactivity research in PTSD (Pole, 2007) indicated that PTSD is associated with less skin conductance habituation, greater facial electromyographic (EMG) reactivity during trauma cues, and greater heart rate (HR) reactivity during all types of stress prompts (trauma or nontrauma).

A disproportionately large segment of the research on PTSD and cardiovascular risk has involved retrospective examination of PTSD among older male combat veterans who already exhibit clinical symptoms of CVD at the time of the study. However, many civilians also suffer from PTSD that has resulted from the occurrence of threatening incidents such as sexual/physical assault or abuse and automobile accidents; studies have revealed high rates of lifetime exposure to potentially traumatic events among young women (e.g., McGruder-Johnson, Davidson, Gleaves, Stock, & Finch, 2000; Norris, Foster, & Weishaar, 2002). Women are more likely than men to develop chronic PTSD after trauma exposure (e.g., Davidson, 2000; Norris et al., 2002). The limited available evidence concerning PTSD-related CVD risk among women suggests that women may display similar levels of cardiovascular risk to that of men (e.g., Kubzansky, Koenen, Jones, & Eaton, 2009; Ouimette et al., 2004). Despite this evidence, a meta-analytic review that examined PTSD and psychophysiological reactivity revealed a very limited number of studies of civilian women (Pole, 2007); based on the limited evidence, there is no overall indication of gender differences in cardiovascular reactivity in PTSD. The greater prevalence of PTSD and elevated health risks for women, along with the underinclusion of women in psychophysiological studies of PTSD, highlight the need to better understand psychophysiological factors that contribute to the development of CVD in civilian women with PTSD.

Strengthening study designs (e.g., use of potent stressors, timing of physiological measures to maximize measurement of stressor effects, including women as participants) and expanding the framework for examining stress responses may assist in further elucidating the implications of cardiovascular responses to acute stress in PTSD. One way of expanding the framework for studying stress responses is by examining cardiovascular recovery from stress; cardiovascular recovery is generally defined as the amount of reduction in a stress response that takes place at a particular point after the stressor ends, as an individual's arousal moves from peak stress reactivity toward their baseline level. In addition to the assessment of cardiovascular stress reactivity, the measurement of cardiovascular recovery from stress is an important index of exaggerated cardiovascular arousal and disease risk (Brosschot, Pieper, & Thayer, 2005; Steptoe, Donald, O'Donnell, Marmot, & Deanfield, 2006; Steptoe & Marmot, 2005).

Brosschot, Gerin, and Thayer (2006) proposed the perseverative cognition hypothesis as a conceptual model for the associations of negative/perseverative cognitions with prolonged stress-related cardiovascular arousal, including the periods in advance of, during, and

following stressors (Brosschot et al., 2006). With an emphasis on prolonged cardiovascular stress responses, Brosschot and colleagues (2006) outlined the relevance to this model of delayed cardiovascular recovery. Although the model was not presented to characterize a particular mental or physical disorder, implications for CVD risk were outlined. The recovery period following a stressor has relevance in identifying maladaptive cardiovascular responses—it may even be a more representative index of the negative stress responses than reactivity, because delayed recovery is thought to reflect extended maintenance of cardiovascular stress reactions (Brosschot et al., 2005). Delayed blood pressure (BP) recovery (one of several potential measures of cardiovascular recovery) has been associated with greater carotid intima-media thickness (an indicator of atherosclerosis) among participants with low socioeconomic status (Steptoe et al., 2006). In another study, delayed poststress recovery of BP and total peripheral resistance (TPR; another measure of cardiovascular recovery) were associated with elevated resting BP measured 3 years after lab assessment of cardiovascular recovery (Steptoe & Marmot, 2005).

The conceptual framework of the current investigation is a potential extension of the perseverative cognition hypothesis (Brosschot et al., 2006) to individuals with PTSD symptomatology. Given that considerable research has identified patterns of negative cognitions/appraisals in PTSD (Clapp, Patton, & Beck, 2015; Dalgleish, Moradi, Taghavi, Neshat-Doost, & Yule, 2001; Olatunji, Armstrong, McHugo, & Zald, 2013), this model is applicable to PTSD. However, only a limited number of studies have addressed cardiovascular responses from the standpoint of recovery in PTSD (Beckham et al., 2002; Kibler & Lyons, 2004; Norte et al., 2013). In one study of PTSD and cardiovascular recovery among Vietnam combat veterans, higher PTSD symptom levels were associated with longer HR recovery following startle tones (Kibler & Lyons, 2004). Although controlling for reactivity levels did not statistically have a substantial impact on the association between HR recovery and PTSD, cognitive appraisal of ability to cope partially explained this association. The finding concerning cognitive appraisal is consistent with the theoretical view and empirical evidence that psychological factors such as appraisal, anger/hostility, expressive inhibition, and optimism/pessimism are associated with cardiovascular recovery from stress (Clapp et al., 2015; Olatunji et al., 2013; Salomon, Burgess, & Bosson, 2015; Verkuil, Brosschot, & Thayer, 2014). The appraisal of threat has been shown to be heightened in PTSD across a variety of situations (e.g., Dalgleish et al., 2001; Olatunji et al., 2013) and may be a particularly salient construct to examine with respect to delayed cardiovascular recovery.

Beckham et al. (2002) observed that systolic and diastolic BP recovery periods were longer following an anger recall task in veterans with PTSD compared with veterans without PTSD. In another study, Norte et al. (2013) examined cardiovascular recovery after exposure to reminders of a traumatic event in 19 male and female civilian PTSD patients from a university-based clinic, and 16 trauma-exposed controls. In both groups, mean HR increased significantly during exposure to personal trauma cues. However, the PTSD group sustained the HR increase during a 60-s postexposure, whereas the controls showed a decrease in HR to baseline levels during postexposure. The PTSD group also evidenced a significant decrease in HR variability during the exposure to trauma reminders, whereas no significant change occurred in controls; the reduction in HR variability in the PTSD group was

maintained during the postexposure period. A limitation of the Norte et al. (2013) study is that the brief 60-s recovery period did not appear to provide an adequate timeframe to evaluate the course of HR recovery in the PTSD group. Nonetheless, it is notable that the PTSD group did not recover as fast as the controls. In all, the findings from the limited number of studies on PTSD and cardiovascular recovery provide preliminary evidence for prolonged HR and BP recovery from stress in PTSD.

Additional research is needed to further evaluate the association between PTSD and cardiovascular recovery. To date, no study has incorporated impedance cardiography-derived measures of cardiovascular recovery in PTSD in order to index responses beyond HR and BP (i.e., cardiac output [CO] and TPR). Impedance cardiography is recommended as a reliable, noninvasive method for assessing the CO and TPR components of BP (Van de Water, Miller, Vogel, Mount, & Dalton, 2003; Yu, Nelesen, Ziegler, & Dimsdale, 2001). This approach provides more specific information about the physiological determinants of BP homeostasis. The impedance-derived CO measure is viewed as one of the best noninvasive estimates of cardiac activity, representing both HR and the quantity of blood being pumped per beat; the TPR measure reflects the activity of the peripheral vasculature, with respect to contraction (an increase in resistance), or dilation (Van De Water et al., 2003; Yu et al., 2001). Recommendations in the literature support the use of more sophisticated physiological measurement methods in order to develop a better understanding of the complex relationship between PTSD symptoms and cardiovascular risk (Pole, 2007). Including CO and TPR to better estimate cardiac and vascular responses will likely provide a more thorough understanding of this relationship.

Past studies have proposed various methods of assessing cardiovascular recovery. Compared with calculations that assess recovery as a difference score—subtracting baseline from the recovery value (Steptoe & Marmot, 2005)—assessing the amount of recovery or percent recovery from reactivity values has the advantage of accounting for the amount of reactivity, and not just the baseline value (Crowley et al., 2011). Taking into account the reactivity values ensures that the recovery index reflects the potential relationships to reactivity (Anderson, Linden, & Habra, 2006). Therefore, this approach was adopted for the present study, which was conducted to examine whether cardiovascular recovery following an oral speaking stressor (a general nontrauma-focused stress task) was associated with the severity of PTSD symptoms and cognitive stress appraisals. The distinction between PTSD severity (on a continuum) and a categorical diagnosis has implications for understanding the significance of PTSD symptom severity level for cardiovascular recovery. Studying cardiovascular recovery in PTSD using any paradigm is novel, given the limited research on this topic; to the extent that responses to the stress task are generalizable, longer (or delayed) cardiovascular recovery following this type of task may potentially estimate difficulties in recovery from general stressors in the environment for individuals with PTSD. To our knowledge, this is the first study to include the assessment of CO and TPR recovery in PTSD; therefore, use of these measures represents a strength with regard to novelty and expanding knowledge of recovery. Another relatively innovative strength of the present study is the thorough clinical assessment of PTSD using structured clinical interviews. Most studies that have associated cardiovascular risks with PTSD have relied on PTSD screening instruments or short participant- or researcherrated checklists. Given our finding of a

positive association between CO reactivity and PTSD severity (Malcolm et al., 2016), prior studies that have suggested delayed HR recovery in PTSD (Kibler & Lyons, 2004; Norte et al., 2013), hypothesized that greater PTSD severity would be associated with less/delayed cardiac recovery (HR and the cardiac component of BP [CO]). Analyses of PTSD severity in relation to vascular recovery (TPR) and BP recovery were approached as more exploratory. Likewise, exploratory analyses were conducted to determine if the relationships of PTSD severity with recovery varied across the three *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association [APA], 1994) clusters of PTSD symptoms. It was also hypothesized that greater PTSD severity would be associated with greater appraisal of threat and lower appraisal of challenge, and that these types of appraisals would be associated with less/delayed cardiac recovery. Exploratory analyses of the mediational role of appraisal in the PTSD/recovery associations were also conducted if each of the criteria for testing this model were met (i.e., significant associations among the three respective measures).

Method

Participants and Procedure

Civilian women aged 18 years or older were recruited via the posting of flyers, advertisements on scientific and public websites, and referrals and self-referral from a community mental health clinic. Inclusion criteria were (a) no history of major chronic illness, (b) premenopausal, (c) not currently taking any medications that could significantly influence physiological measures (e.g., beta-blockers, muscle relaxants), and (d) no Axis I psychiatric diagnoses (mental/emotional disorders as defined by the *DSM-IV* [APA, 1994]) aside from PTSD, unipolar depression, and/or substance abuse. Chronic illness was an exclusion criterion because it complicated our focus on factors that may increase risk for developing CVD. Menopause or postmenopausal status is known to affect cardiovascular stress responses, and is often exclusionary (or controlled in a systematic way) in studies involving reactivity/recovery; likewise exclusion of medications that could influence cardiovascular responses is common for stress reactivity/recovery research. Exclusion for most Axis I disorders was implemented in an effort to focus our findings on phenomena associated with PTSD symptoms. This study was approved by the Nova Southeastern University institutional review board.

The final sample for analysis consisted of 50 women (after 1 out of 51 participants was excluded from analysis due to missing recovery data). Participants' self-reported ethnicities were 50.0% Caucasian, 10.0% African American, 24.0% Hispanic White, 10.0% Caribbean Black, and 6.0% other. One-third of the sample reported annual family income less than \$20,000, another 36.7% reported in the \$20,000 to \$50,000 range, 20.0% reported income in the \$50,000 to \$100,000 range, and 10.0% reported income over \$100,000. With regard to education, 26.0% of participants reported achieving a high school degree or equivalent, 36.0% reported a bachelor's degree, 18.0% reported an associate's degree, 16.0% reported graduate or professional degrees, and 4.0% reported "other."

Although the study was open to participants both with and without trauma histories, all participants reported exposure to potentially traumatic events (see the Results section for

descriptive data on trauma exposure). Given that PTSD is often comorbid with depression and/or substance use, these symptoms were not exclusionary. Based on the clinical interview, 21 participants met the criteria for major depressive disorder based on lifetime depressive episode history (nine were participants with high PTSD symptoms), and six met criteria for a substance abuse disorder (one of whom had high PTSD symptoms). Data from quantitative measures of depressive/alcohol use symptoms (Beck Depression Inventory-II and Alcohol Use Disorders Identification Test) are included in Table 1.

Data collection took place during two separate assessment sessions—a structured diagnostic interview session and a laboratory assessment session. The interval between the interview and laboratory assessment was minimized (no more than 2 weeks) due to the possibility that symptom presentation could change over time. To control for menstrual cycle effects on the cardiovascular measures, participants completed the laboratory session during the follicular stage of the menstrual cycle (approximately days 5 to 9, depending on the participant's estimate of cycle length). When participants initially called to volunteer, the phone screening included a verbal history of typical monthly menstrual cycle length (in days) and the date the last menstrual cycle began. This information was used to anticipate the beginning of menses prospectively, which was verified by phone with the participant. Again at the laboratory visit, the information was confirmed with questions about what date the last menstrual period began, and how many days it had been since the previous menstrual period ended.

The CAPS and SCID were conducted in a university-based psychological services center by advanced clinical psychology doctoral students. Interviewers received extensive, focused training from the author prior to conducting the interviews. Each diagnostic interview was audiotaped, and an interviewer who did not conduct the original interview provided ratings of the participants' symptoms offline in order to calculate a correlation for inter-rater reliability of the CAPS PTSD assessments. The inter-rater reliability correlation was $r = .87$ for the CAPS.

For the cardiovascular reactivity protocol, participants were asked to refrain from caffeine and strenuous exercise for 3 hours prior to the beginning of the session, and smoking for 30 minutes prior to the beginning of the session. When the participant arrived at the laboratory, compliance with restrictions was assessed. Noncompliance with restrictions required rescheduling. This session was conducted early in the morning (approx. 8:00 a.m.) to minimize restriction hardship. Participants were provided with a standardized light breakfast snack (granola bar or cheese & cracker snack, and water, juice, or non-caffeinated soda) before the reactivity/recovery assessments, because hunger is known to affect cardiovascular parameters (Dworkin, 2007).

After participants were provided with a brief overview of the cardiovascular reactivity/recovery protocol, the experimenter attached the physiological measurement sensors for recording BP, the electrocardiogram (ECG), and the impedance cardiogram. The experimenter then attached the automated BP cuff (Tango; Suntech Med. Instr., Inc. Morrisville, NC) to the dominant arm. The psychophysiological assessment began with a 15 min prestressor rest period, after which the participant was presented with the stress task (evaluated speaking). After hearing instructions for the stress task, but before performing it,

the participant completed the Stress Appraisal Measure (SAM; Peacock & Wong, 1990) to rate the cognitive appraisals of the upcoming task. With regard to the speech task, instructions for the SAM were to “please respond according to how you view this situation right now.”

In the speech task, participants were given 3 min to formulate the speech mentally, and then the story was presented aloud during a subsequent 3-min period. The speech prompt used in the present study was introduced by Saab, Matthews, Stoney, and McDonald (1989) and has been utilized in numerous studies to effectively elicit cardiovascular arousal (e.g., Kline, Saab, & Llabre, 2005; Peckerman et al., 2000). Participants were instructed to prepare and present a speech about a make-believe situation (“pretend you are shopping at a department store in the mall. As you are leaving the store, a security guard approaches you and accuses you of stealing the belt that you are wearing and purchased there last week. The security guard goes to get the store manager.”). The speech was directed at the store manager, and the participant was meant to give their side of the story. Participants were also informed that their performance would be videotaped and rated by experts on the basis of poise, speaking style, and personal appearance. The participant was given five elements around which to organize the story. The prominent location of the camera was indicated prior to beginning the task. The participant was instructed when to begin the speech and when to stop, and provided with standard prompts to continue the speech (even if repetition of material was necessary), if the time had not elapsed. The speech task was followed by the posttask rest/recovery period. Upon completion of physiological measures, the recording equipment was removed. The experimenter then explained the overall purpose of the study and answered any remaining questions the participant might have had about the assessment.

Measures

Traumatic event exposure.—Prior to a structured interview for PTSD symptoms and other mental health symptoms, participants completed an assessment of prior exposure to potentially traumatic events using the Trauma Life Event Questionnaire (TLEQ; Kubany et al., 2000). The TLEQ is a 23-item self-report measure of 22 types of potentially traumatic events including robbery involving a weapon, other assault/violence, physical abuse, sexual assault/abuse, natural disasters, and being stalked (Kubany et al., 2000). For each event, respondents provide the number of times it occurred (*never to more than 5 times*) and whether fear, helplessness, or horror was present (*yes or no*). Some events include a question about presence of injury (*yes or no*), and for victimization questions, characteristics of the perpetrator (e.g., “stranger?,” to which the respondent is asked to answer *yes or no*). The last question asks respondents to identify the event that “causes you the most distress.” Respondents are also asked about their age upon first occurrence, date of last occurrence, and amount of distress caused (*no distress to extreme distress*). The TLEQ has yielded good reliability and good concurrent/predictive validity (Kubany et al., 2000).

Posttraumatic stress disorder.—The Clinician-Administered PTSD Scale for *DSM-IV* (CAPS) was used to assess PTSD symptoms. The CAPS is an international standard in diagnostic assessments for PTSD (Blake et al., 1995; Weathers, Ruscio, & Keane, 1999). The CAPS is designed to assess current and lifetime PTSD symptoms, and total CAPS

scores have been shown to have good inter-rater reliability (Cronbach's α s = .92 to .99), test-retest reliability (Cronbach's α s = .91 to .95), internal consistency (Cronbach's α s = .73 to .95), and convergent validity (Blake et al., 1995; Weathers, Ruscio, & Keane, 1999). To quantify the results of the CAPS assessment (as in the present study), the frequency and intensity of each symptom is rated, and the frequency/intensity scores are totaled for each symptom cluster and for total PTSD symptom severity. Five rationally derived severity score ranges were originally proposed for interpreting CAPS total severity: asymptomatic/few symptoms = 0 to 19, mild PTSD/subthreshold = 20 to 39, moderate PTSD = 40 to 59, severe PTSD = 60 to 79, and extreme PTSD = 80. In the present study, internal consistency for the symptom frequency and intensity ratings were observed to be good (Cronbach's α s = .73 for frequency and .80 for intensity).

Axis I disorders.—A diagnostic interview for other Axis I disorders utilized the Structured Clinical Interview for *DSM-IV* Axis I Disorders (SCID), research version, which is a gold standard for making major *DSM-IV* Axis I psychiatric diagnoses (Basco et al., 2000). Studies have shown high inter-rater reliabilities (kappa coefficients of agreement = .80 to .93) for diagnoses of major depression with the SCID (Zanarini, & Frankenburg, 2001) and demonstrated the superior validity of the SCID relative to standard unstructured interviews (Basco et al., 2000). Participants who endorsed any Axis I *DSM-IV* disorder aside from unipolar depression or substance abuse disorders (both of which often coexist with PTSD) on the SCID (or PTSD on the CAPS) were excluded from the study.

Depression.—The Beck Depression Inventory-II (BDI-II) is a 21-item self-report measure of affective, somatic, and cognitive symptoms associated with depression (Beck, Steer, Brown, & Gregory, 1996). Respondents indicate their level of distress on a 4-point scale, ranging from 0 (*none to minimal experience of a particular symptom*) to 3 (*highest level of severity for the symptom*). Total possible scores range from 0 to 63. Total scores are classified as minimal (0 to 13), mild (14 to 19), moderate (20 to 28), and severe (29 to 63). The BDI-II has been found to be a reliable and valid measure for numerous populations such as community samples of older and younger adults (Beck et al., 1996; Segal, Coolidge, Cahill, & O'Riley, 2008). In the present study, internal consistency for the total BDI score was high (Cronbach's α = .95).

Alcohol use.—The 10-item Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, de la Puente, & Grant, 1993) was included to assess alcohol use and binge drinking. The AUDIT was developed by the World Health Organization to identify persons whose alcohol consumption has become harmful to their health (Bradley, Boyd-Wickizer, Powell, & Burman, 1998). Each of the questions has a set of responses to choose from, and each response has a score ranging from 0 to 4; total scores of 8 or more are recommended as indicators of hazardous and harmful alcohol use, as well as possible alcohol dependence. In the present study, internal consistency for the total AUDIT score was good (Cronbach's α = .76).

Psychophysiological assessment.—The electrocardiogram (ECG) was placed in a standard three-lead configuration for determination of HR. An automated BP cuff (Tango;

Suntech Med. Instr., Inc. Morrisville, NC) was attached to the dominant arm for BP assessment. To produce the impedance cardiogram (which is used to assess CO and TPR), Z , dz/dt , and mean thoracic impedance (Z_o) were recorded using a standard full band tetrapolar configuration (Sherwood et al., 1990). Four current electrodes (Z_1 to Z_4) were placed, two above the chest at the neck and two below the chest. The Z_2 electrode was attached around the base of the neck, just superior to the suprasternal notch of the thorax. Exactly 3 cm separated Z_1 from Z_2 . The Z_3 electrode was placed at the xiphoid process, and 5 cm separated Z_3 and Z_4 . The ECG and impedance signals were transferred to the Hutcheson Impedance Cardiograph (HIC-3000/T, Integrated BP V6.21 system; Microtronics Corp. Chapel Hill, NC) for internally controlled analog signal filtering/processing and digital signal processing, sampling at 1 kHz/s and stored in a personal computer for data scoring and reduction. Computer-displayed signals were monitored for signal quality during the recording session.

Physiological data sampling.—The ECG, dz/dt and Z_o signals were collected continuously during the last four minutes of the prestressor baseline, and throughout all of the subsequent testing periods. Mean arterial BP (MAP) is incorporated into the calculation of TPR, and BP measurement was initiated at the 11:00 minute, 12:30 minute, and 14:00 minute periods of the 15 minute baseline; 15 second and 1:45 minute point of the stressor period; and at the 3:15 and 6:15 minute marks of the posttask recovery period. The measures were initiated in this manner to allow ample time for cuff inflation and to appropriately reflect the physiological activity during the designated phase. The recovery periods (4th minute and 7th minute post-task) were selected to reflect the most variability in recovery. Although relatively little recovery took place in the first 1 to 2 minutes posttask, most participants showed some amount of recovery in the first 4 minutes of posttask sampling, and further recovery was noted by minute 7 (see data on percent recovered in Results section). Beyond minute 7, most of the participants experienced full recovery; therefore, use of any later time period would not reflect enough variability for correlational analysis.

Physiological data reduction.—The dz/dt signal and Z_o calibration signal were computer-stored during data collection for later conversion of the dz/dt to the unit of ohm/s and derivation of stroke volume (ml). Ensemble averages of the cardiac cycles in the converted impedance cardiogram permitted the derivation of CO and the calculation of TPR as $(MAP/CO) \cdot 80$ (dyne-s/cm⁵) for each 1-minute period. For each physiological variable, a single average was calculated for the pretask baseline and stressor periods by averaging the last three baseline readings and the two stress task readings. Reactivity scores were derived by subtracting the respective baseline value from the stress task value. Recovery data were calculated using percentage of recovery from the mean stress task level toward the baseline level using the recovery data collected during the 4-minute and 7-minute posttask epochs.

Cognitive appraisal.—Pretask cognitive appraisal assessments of the upcoming stress task (speech stressor) were conducted after task instructions using the Threat and Challenge subscales of the SAM. Each subscale consists of four items rated from 1 (*not at all*) to 5 (*extremely*). Example items from the Threat subscale include: “How threatening is this situation?” and “Is this going to have a negative impact on me?” Items from the Challenge

subscale include: “To what extent am I excited thinking about the outcome of this situation?” and “Is this going to have a positive impact on me?” Internal consistency analyses and factor analyses from three studies indicated adequate internal consistencies for the Threat subscale (Cronbach’s α s = .65, .73, and .75, respectively, in the three studies) and Challenge subscale (Cronbach’s α s = .66, .74, and .79, respectively across the three studies), and that the Threat and Challenge subscales represent distinct and meaningful independent constructs (Peacock & Wong, 1990). The choice to utilize pretask appraisal assessments (rather than posttask assessments) was based on (a) concerns that the task reactions would bias any appraisals made after the task, (b) avoidance of posttask assessments that might interfere with or interrupt accurate recovery assessment, and (c) prior research indicating pretask appraisals are associated with physiological recovery (e.g., Kibler & Lyons, 2004; Schlotz, Hammerfald, Ehlert, & Gaab, 2011).

Data Analysis

Pearson correlations of the PTSD severity scores with the recovery measures were conducted. Exploratory analyses using partial correlations were conducted as post hoc tests of the mediational role of appraisal in the PTSD/recovery associations if each of the criteria for testing this model were met (i.e., significant associations among the three respective measures). The IBM SPSS Version 24 statistical package was utilized. Although associations of age and race with recovery indices were assessed as potential covariates, no significant relationships were evident for these tests. To limit type 1 error, correlations of cognitive appraisals with recovery measures were only conducted when the recovery measure and PTSD severity were significantly correlated. Participants were excluded from analysis if the respective data were missing for any of the study variables. Of the 51 total participants, 1 was missing CO/TPR recovery data, which resulted in the final sample size of 50.

Results

Descriptive Data

With regard to trauma histories, the most distressing “index” events consisted of sexual assault/abuse (24.0%), physical assault/abuse/victim of domestic violence (18.0%), witnessing violence (12.0%), motor vehicle accident (4.0%), natural disaster (4.0%), sudden unexpected death of a loved one (20.0%), stalking (4.0%), abortion (4.0%), sexual harassment (2.0%), and other traumas (e.g., jailed/lost in foreign country) or life-threatening accidents requiring hospitalization (8.0%). This was a sample with high levels of trauma exposure protracted over an extended period—in terms of frequency, all participants reported exposure to more than one potentially traumatic event/incident (mean number of events: 13, $SD = 11$). Varying levels of PTSD symptomatology were evidenced, from no symptoms to high severity (see Table 1 for PTSD symptom severity and cognitive appraisal data). Of the participants with the highest PTSD severity, 14 evidenced full diagnostic criteria for PTSD (28% of sample), and 5 had significant subthreshold PTSD symptoms (10% of sample) that did not meet full criteria (4 symptoms with 1 reexperiencing).

The descriptive results for the cardiovascular values at baseline and reactivity (mean and SD), and recovery periods (percentage of recovery) are listed in Table 2. The reactivity results have been published elsewhere (Malcolm et al., 2016) and are listed here only to aid in the evaluation of recovery and the overall patterns of physiological activation across the phases of the study—the reactivity change scores were significant $p < .001$, with the exception of TPR, for which mean reactivity was not a significant change from baseline. Notably, there was a wide range of variability for TPR reactivity.

At the 4th minute posttask, about half of the participants (54%) exhibited full recovery to baseline for all or most physiological measures. By the 7th minute posttask, 75% of participants had fully recovered. Each of the two posttask recovery time points were analyzed to examine whether participants' percentage of recovery at the respective time point related to PTSD severity and cognitive appraisals.

Primary Analyses

Severity of total PTSD symptoms was associated with less CO recovery at the minute posttask, $r = -.39$, $p = .006$; results of the exploratory correlational analyses for each category of PTSD symptom cluster indicated that this significant effect was similar across the three PTSD symptom categories: $r = -.30$, $p = .042$ for reexperiencing; $r = -.32$, $p = .027$ for arousal; and $r = -.44$, $p = .002$ for avoidance. However, only greater PTSD severity in the avoidance category was associated with less TPR recovery at the 4th minute posttask, $r = -.29$, $p = .047$. The PTSD severity measures were not significantly correlated with BP or HR recovery at the 4th minute of recovery, or any of the physiological recovery measures at minute 7 of recovery.

As predicted, greater PTSD severity (total) was associated with greater appraisal of threat, $r = .30$, $p = .035$; PTSD severity was not significantly associated with appraisal of challenge. Cognitive appraisal of greater threat was associated with less CO recovery, $r = -.33$, $p = .019$, at the 4th minute posttask, but was not significantly related to TPR recovery. Challenge appraisals were not significantly associated with PTSD severity or CO/TPR recovery.

Exploratory post hoc analyses that controlled for threat appraisal in the associations between PTSD severity and CO recovery using partial correlations reduced the magnitude of the correlations to a small degree. After controlling for threat appraisal, CO recovery in the 4th minute posttask was only significantly associated with total PTSD severity, $r = -.35$, $p = .030$, and severity in the avoidance category, $r = -.38$, $p = .016$. The correlations of CO recovery with reexperiencing severity, $r = -.28$, and arousal severity, $r = -.27$, were no longer significant.

Discussion

Among women with no diagnosed medical illnesses, PTSD severity and greater perceived threat were associated with less cardiovascular recovery from stress in the present study. The present findings concerning cardiovascular recovery are consistent with prior research that has indicated delayed recovery may be evidenced among individuals with high levels of PTSD (Beckham et al., 2002; Kibler & Lyons, 2004; Norte et al., 2013). The present

investigation expanded on prior findings by providing information about cardiac and vascular indices derived from impedance-cardiography measurement. Given that significant effects were observed for CO and TPR recovery, and not for BP or HR, in the present study, it is possible that these two physiological components of hemodynamic functioning are especially sensitive to the changes that occur during recovery in PTSD.

The association between PTSD severity and cardiovascular recovery may be accounted for in part by ongoing stress-related negative cognitions. The perseverative cognition hypothesis, which outlined associations of negative/perseverative cognitions with prolonged stress-related cardiovascular arousal (including the relevance of delayed cardiovascular recovery to this model) is a starting point for understanding this relationship (Brosschot et al., 2006). When some individuals with PTSD experience perseverative cognition on a prolonged basis, it may have detrimental effects on their ability to recover physiologically, and ultimately what Brosschot et al. (2006) refer to as a “chronic pathogenic state.” Further research in this area should assist in understanding how additional trauma-related cognitions, such as blame, control, and guilt (e.g., Engelbrecht & Jobson, 2014; Nishith, Nixon, & Resick, 2005) may relate to cardiovascular recovery. Another explanation for the present findings is that physiological changes following the development of PTSD may alter responses to stressors—changes or adaptations to autonomic nervous system regulation of cardiac activity (Hughes, Dennis, & Beckham, 2007; Keary, Hughes, & Palmieri, 2009) may lead to delays in cardiovascular recovery.

The present results concerning PTSD and recovery were more consistent for CO than for TPR. This observation could be a function of a less pronounced TPR response during the speech task relative to the CO response; with less TPR responding, there would be less variability in recovery and this could affect the ability to detect correlations. With regard to the null findings for HR and BP recovery in the present study, this is in contrast with the few prior studies of recovery in PTSD (Beckham et al., 2002; Kibler & Lyons, 2004; Norte et al., 2013). The scarce data in this area is insufficient to determine whether these differences across studies could be explained by the use of trauma-related versus nontrauma-related stressors; however, it may be that certain stress tasks elicit stronger responses in specific measures. Differences in the physiological demands of the stressors, regardless of whether they are trauma-related, could account for differences in the cardiovascular measures involved in significant associations. The differences observed for the various recovery measures within the present study, and across studies, warrant further study. Further research is also needed to examine whether delayed recovery in some individuals with PTSD contributes to elevated cardiovascular disease risk.

The finding that significant associations between cardiovascular recovery, and PTSD severity and appraisal, were evident at the 4th minute, but not the 7th minute, of recovery was not surprising. Because the 4th minute was observed to produce more variability in recovery responses (fewer participants had “recovered”), it was expected that this heterogeneity of responses would enhance the detection of associations involving recovery.

The positive association observed between PTSD severity and threat appraisal in the present study was expected given that heightened perception of threat (among other disrupted

cognitions) is consistently observed in PTSD (e.g., Dalgleish et al., 2001; Olatunji et al., 2013). The cognitive appraisal of a stressor plays a significant role in the emotional response and long-term coping with stressful situations (Tomaka, Blascovich, Kibler, & Ernst, 1997). The present finding that threat appraisals were associated with delayed cardiovascular recovery is conceptually meaningful in understanding the maintenance of behavioral and physiological responses to stress in PTSD. The observation that threat, and not challenge, was more closely related to PTSD severity and recovery in the present study was attributed to the saliency of threat in PTSD. Additional research may expand further on the role of these specific cognitive appraisals in the cardiovascular recovery of individuals with PTSD.

Primary limitations of the present study include the limited scope/generalizability of cardiovascular reactivity/recovery tasks. Although the oral speaking stressor employed in this study is a commonly used stress probe (e.g., Kline, Saab, & Llabre, 2005; Peckerman et al., 2000), it is possible that this task may not have adequately reflected the participants' propensity toward reactivity and the typical patterns of recovery from stress in the natural environment. Other limitations relate to the appraisal assessment; the SAM is a validated instrument, but some of the reliability coefficients in the psychometric studies were low. However, aggregating across the reliability coefficients in those studies, the reliability is acceptable. Nonetheless, any shortfalls in reliability could limit the ability to accurately detect effects. Also, the present focus on threat and challenge is not an ideal fit with the cognitions originally proposed as part of the perseverative cognition hypothesis (i.e., worry and rumination). It may be that expanding the present approach, by utilizing a broader measure or set of measures that also better captures worry and rumination over time, would provide a more thorough understanding of the role of PTSD-related cognitions in cardiovascular recovery. Another potential limitation of the appraisal assessment is that it was only conducted in the pretask period; because recovery occurs posttask and is temporally distal to the pretask appraisal, some extension of the appraisal assessment (longitudinally) beyond the pretask period could potentially provide a more thorough index of ongoing appraisals. Gerin, Davidson, Chistenfeld, Goyal, and Schwartz (2006) outlined an alternative approach, which integrates participants' thoughts during the posttask recovery period (cued by an auditory tone), and then follows up on these thoughts with a more in-depth assessment following the recovery period. In utilizing an appraisal assessment after a stressor, one would need to consider the potential impact of the stressor and corresponding reactivity on the appraisals and the potential for appraisal assessments carried out during the recovery to influence recovery itself. An additional limitation of the present study is the difficulty associated with comorbid PTSD and depression—the occurrence of comorbid PTSD and MDD is high (Pietrzak, Goldstein, Southwick, & Grant, 2011). It is difficult to distinguish the contributions of PTSD versus depression when the two disorders are comorbid. Interestingly for the focus of the present investigation, most of the proposed mechanisms that may underlie the association between depression and CVD would also be considered mechanisms in the association between PTSD and CVD. This similarity is consistent with the conceptualization of both PTSD and depression as stress-related disorders, with physiological hyperarousal as a central component (Friedman, & McEwen, 2004). The exclusive focus on women for the present investigation was another limitation—had men also been included, this would have permitted a comparison of findings by gender.

Despite its limitations, the present study provided data with conceptual implications for understanding the maintenance of stress responses among individuals with PTSD; because posttraumatic stress may often intersect with daily environmental stressors to produce enduring stress, the recovery period following a stressor may have even more relevance than do reactivity measures in the identification of long-term cardiovascular dysfunction due to the potential to reflect prolonged stress reactions (Brosschot et al., 2005). Research with extended evaluation of psychophysiological parameters is likely to further elucidate the extent to which stress reactions are maintained in PTSD. The present study also provides much-needed data among women, as most mechanistic studies of the cardiovascular risks associated with PTSD have been conducted with male veterans.

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Table 1

Descriptive Data for Posttraumatic Stress Disorder (PTSD) and Cognitive Appraisal Variables

Variable	Score	
	Mean	SD
PTSD Total CAPS	25.9	30.1
PTSD CAPS Reexperiencing	7.3	8.7
PTSD CAPS Avoidance	10.3	13.7
PTSD CAPS Arousal	8.3	10.0
Threat Appraisal Rating	2.1	0.9
Challenge Appraisal Rating	2.4	1.0
BDI-II	15.3	12.6
AUDIT	3.8	3.3

Note. CAPS = Clinician Administered Posttraumatic Stress Disorder Scale; BDI-II: Beck Depression Inventory II; AUDIT = Alcohol Use Disorders Identification Test.

Table 2

Descriptive Data for Cardiovascular Variables at Each Phase of the Study

Variable	Score	
	Mean	SD
HR Baseline (bpm)	70.2	11.3
HR Reactivity (bpm)	78.8	9.9
HR Recovery (% at minute 4) ^a	75.0	35.7
SBP Baseline (mmHg)	107.0	11.9
SBP Reactivity (mmHg)	123.9	15.0
SBP Recovery (% at minute 4) ^a	52.5	36.6
DBP Baseline (mmHg)	68.7	8.0
DBP Reactivity (mmHg)	78.5	8.9
DBP Recovery (% at minute 4) ^a	62.3	36.5
CO Baseline (l/min)	5.7	1.6
CO Reactivity (l/min)	6.4	1.6
CO Recovery (% at minute 4) ^a	65.8	38.5
TPR Baseline (dyne-s/cm ⁵)	1225.8	347.1
TPR Reactivity (dyne-s/cm ⁵)	1246.4	354.9
TPR Recovery (% at minute 4) ^a	81.7	30.9

Note. HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure; CO = cardiac output; TPR = total peripheral resistance

^aRefers to the 4th minute of the posttask recovery period.