FRONTAL CORTICAL ASYMMETRY, HIGH-FREQUENCY HEART RATE VARIABILITY, AND DISTRESS PSYCHOPATHOLOGY AMONG UNDERGRADUATES WHO EXPERIENCED TRAUMA

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ABSTRACT OF THE DISSERTATION

Frontal Cortical Asymmetry, High-Frequency Heart Rate Variability, and Distress Psychopathology Among Undergraduates Who Experienced Trauma

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Exposure to traumatic events can increase the likelihood of developing distress psychopathology (i.e., depression, anxiety disorders, and PTSD) and young adulthood represents a critical window during which these disorders often develop. Dysregulations in two processes might contribute to the development of distress psychopathology: emotion regulation (which can be assessed using high frequency heart rate variability [HF-HRV] via EKG) and avoidance motivation (which can be assessed using frontal cortical asymmetry via EEG). One-hundred eighteen (118) college students were recruited, baseline HF-HRV was assessed, and frontal cortical asymmetry was assessed before and after a personally relevant laboratory stressor. Affect in response to the stressor and depressive, anxiety, and PTSD symptoms were assessed using self-report measures. Contrary to our hypotheses, greater baseline HF-HRV predicted greater post-stressor negative affect, less left-hemisphere post-stressor neural activity was associated with fewer PTSD symptoms, and greater pre-stressor frontal cortical asymmetry was associated with greater distress psychopathology assessed as a higher-order latent factor.
Consistent with our hypotheses, there was a nonsignificant trend towards greater post-stressor frontal cortical asymmetry predicting fewer depressive symptoms and a nonsignificant trend towards less post-stressor left-sided frontal neural activity predicting greater anxiety symptoms. These findings raise questions regarding the roles of HF-HRV and frontal cortical asymmetry in distress psychopathology. We discuss the implications for clinical science research utilizing these psychophysiological tools, the benefits of multiple measures of physiology and psychopathology (including use of both symptom-specific measures and measures of high-order latent factors), and the limitations of the current study.
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Introduction

A traumatic event, as defined by the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), is any event that involves exposure to actual or threatened death, serious injury, or sexual violence (American Psychiatric Association [APA], 2013). The vast majority of individuals, about 90%, will experience at least one traumatic event in their lives (Kilpatrick et al., 2013), and about 8% will go on to develop post-traumatic stress disorder (PTSD; Kessler et al., 2012). In the general population, about 14% of individuals will experience major depressive disorder (MDD), and 33% of people will experience an anxiety disorder (e.g., generalized anxiety disorder; [GAD]) or related disorder at some point in their lives (Kessler et al., 2012). Trauma exposure, particularly early in life (Chu et al., 2013), can increase the likelihood of developing a depressive or anxiety disorder (Asselmann et al., 2018; Ghafoori et al., 2009; McLaughlin et al., 2010; Shaley et al., 1998).

The prevalence of trauma exposure among young adults and undergraduate students is similar to that of the general population (Overstreet et al., 2017), and trauma exposure is similarly associated with an increased risk for PTSD and depressive and anxiety disorders among these individuals (Amstadter et al., 2013; Overstreet et al., 2017; Perkonigg et al., 2000). However, the prevalence of mental illness is greater among young adults (i.e., individuals aged 18-25) than their older counterparts (Substance Abuse and Mental Health Services Administration [SAMHSA], 2020). Despite this, young adults are less likely to receive mental health treatment (SAMHSA, 2020). The majority of mental illnesses develop by the time of young adulthood (Kessler et al., 2007) and about 50% of college students meet diagnostic criteria for a mental disorder in the past
Further, the transition to college is often a period that is characterized by high stress levels that can contribute to vulnerability to psychopathology (Arnett, 2000; Pedrelli et al., 2015). Thus, one’s time in college might represent a critical developmental period during which the risk of mental illness onset is relatively high.

Despite the high rates of prevalence of these disorders among college students and the general population, available gold-standard treatments only lead to remission in about half of patients (Bradley et al., 2005; Dimidjian et al., 2006; Springer et al., 2018). Meanwhile, the advent of new neuroimaging techniques and the proliferation of clinical psychophysiological research have not produced novel or improved treatments as had been expected decades before (Etkin, 2019; Hyman, 2013; Kapur et al., 2012). This has led to a critical reevaluation of the current state of psychopathology research that has centered on two potential paradigm shifts: 1) a reconceptualization of psychopathology as existing along dimensions rather than discrete categories (Insel et al., 2010; Kotov et al., 2017), and 2) a focus on transdiagnostic neurobiological processes that might underlie those dimensions (Insel et al., 2010; Sanislow et al., 2010). These paradigm shifts are reflected in the novel frameworks that have emerged as potential alternatives to the DSM-5, namely the Hierarchical Taxonomy of Psychopathology (HiTOP), a data-driven, dimensional, and hierarchical classification system for mental disorders that is based on the latent structure of psychopathological symptoms (Kotov et al., 2017, 2021); and the Research Domain Criteria (RDoC), a research framework focused primarily on neurobiological mechanisms that might underlie maladaptation in multiple functional domains (Insel et al., 2010; Sanislow et al., 2010).
Notably, both HiTOP and RDoC eschew the syndrome-based categories of the DSM-5. This is due to the preponderance of evidence that has emerged indicating that psychopathology is dimensional in nature rather than categorical (e.g., Haslam et al., 2020), which suggests that existing DSM-5 categorical diagnoses thus have low ecological validity. For example, subthreshold psychopathology (i.e., the presence of pathologic symptoms of insufficient quantity, severity, or duration to meet criteria for a DSM-5 diagnosis) is more common than diagnosable psychopathology based on DSM-5 criteria (Angst et al., 1997; Carter et al., 2001; Haller et al., 2014). Subthreshold symptoms can develop into full syndrome disorders (e.g., Shankman et al., 2009) and, even when they do not, subthreshold symptoms can still cause significant distress and functional impairment (e.g., Haller et al., 2014); yet the DSM-5 categorical approach to psychopathology does not provide an adequate framework for their study and treatment. Further, evidence from factor analytic studies has suggested that disorders that are considered distinct by the DSM-5 are actually related to the same latent construct. For example, PTSD, GAD, and MDD load onto a single latent factor (Elhai et al., 2011; Krueger & Markon, 2006; Watson, 2005), labeled distress disorders or distress psychopathology, yet are classified in separate categories in the DSM-5 (APA, 2013). The HiTOP framework incorporates such latent factors into its nosology (Kotov et al., 2017).

Research also suggests that the neurobiological mechanisms that underlie psychopathology are not unique to any specific DSM-5 diagnosis (e.g., Goodkind et al., 2015). Neural circuits related to dysregulations in affective functioning, in particular, appear to be involved in a multitude of forms of psychopathology (Berboth & Morawetz,
The RDoC framework proposes that developing a better understanding of the neurobiological mechanisms that underlie the various spectra of psychopathology-related cognitive, affective, or behavioral dysfunctions would more effectively lead to improved assessment and treatment tools (Insel et al., 2010; Sanislow et al., 2010). This concurs with the proposition of the HiTOP framework, whose authors also suggest that research that considers the underlying latent structure of psychopathology, along with its dimensional nature, might facilitate the discovery of neurobiological mechanisms (Latzman et al., 2020; Perkins et al., 2020) and the development of more effective treatments (Kotov et al., 2021).

Consistent with these more recent approaches to the study of psychopathology, Beauchaine and Zisner (2017) propose that motivation and emotion regulation are two constructs with measurable biological correlates that are critical to understanding the etiology of psychopathology from a dimensional and latent variable perspective. Motivation involves an organism’s action or preparation to either approach a target in the environment or avoid it, and is inextricably associated with distinct affective experiences (Gray, 1987). Emotion regulation involves a top-down modulation of affective experiences that may be instrumental in goal-attainment (Gross, 1998, 2010). Distinct, yet interrelated, neural systems underlie both processes, and dysfunctions in both processes, as well as their neural correlates, are found across a wide swath of psychopathology (Beauchaine & Zisner, 2017). Indeed, the affective changes that occur following the experience of a traumatic event (Brewin & Holmes, 2003; Foa & Kozak, 1986; Lokshina et al., 2021; Yehuda & LeDoux, 2007) suggest that traumatic stress is
associated with dysfunction in both motivational and emotion regulatory processes and might lead to the development of distress psychopathology.

**Motivation**

An organism’s ability to appraise a stimulus and determine whether to approach or avoid it is thought to be a critical evolutionary adaptation (Gray, 1987; McNaughton et al., 2016). Approach and avoidance motivational appear to be produced by distinct, yet interrelated neural systems (McNaughton et al., 2016), and in humans, each motivational direction may be inextricably associated with a specific affective state (Davidson & Irwin, 1999; Lang & Bradley, 2010; McNaughton et al., 2016). Thus, these affective states reflect part of an individual’s response to environmental conditions. For example, avoidance motivation is most commonly linked with negative affect, which can include emotional states such as fear, anxiety, or sadness, and involves behaviors relevant to avoidance of threat or withdrawal from social interaction (Elliot & Covington, 2001; McNaughton et al., 2016). The neural circuitry involved in approach and avoidance motivation includes subcortical regions such as the amygdala, ventral striatum, and nucleus accumbens, and cortical regions such as the prefrontal cortex and anterior cingulate cortex (Davidson & Irwin, 1999; Fernando et al., 2013; Lang & Bradley, 2010). Among the subcortical regions involved in approach and avoidance motivation, some play a role in both motivational directions (e.g., the amygdala, which is involved in identifying both aversive and appetitive stimuli in the environment), whereas others are specific to a particular motivational direction (e.g., the ventral striatum and nucleus accumbens which are involved in approach of appetitive stimuli). Among the cortical regions involved in approach and avoidance motivation, there appears to be lateralization
such that avoidance motivation is reflected in greater right-sided frontal lobe activity, and approach motivation is reflected in greater left-sided frontal lobe activity (Davidson & Irwin, 1999; Rutherford & Lindell, 2011) (in right-handed individuals). The function of this lateralization is thought to be to minimize conflict between the approach and avoidance systems (Davidson, 1998; Davidson & Irwin, 1999).

Distress psychopathology is often characterized by excess avoidance motivation (Arnaudova et al., 2017; Foa et al., 1989; Newman & Llera, 2011; Rottenberg, 2005; Trew, 2011), and in some cases insufficient approach motivation (Nawijn et al., 2015; Trew, 2011); and congruent dysregulation in the neural circuitry involved in approach and avoidance motivation is also found in distress psychopathology (Berboth & Morawetz, 2021; McTeague et al., 2017, 2020). This suggests that maladaptive functioning of the approach and avoidance neural systems might contribute to the onset and maintenance of distress symptoms.

**Frontal Cortical Asymmetry and Motivation**

Given that approach-avoidance motivation is reflected in lateralized brain activity (Rutherford & Lindell, 2011), the presence of an avoidant motivational direction can be indexed by measuring electrocortical activity in the frontal lobes via the electroencephalogram (EEG). Activity in the alpha band (8-12 Hz) provides an inverse reflection of neural activity at the cortical regions over which the electrode is placed (Cook et al., 1998; Davidson et al., 1990a), thus allowing for a comparison of cortical activity between the right and left frontal lobes, referred to as frontal cortical asymmetry (Allen et al., 2004). Relatively greater cortical activity in the left frontal lobe is associated with approach motivation, while relatively greater cortical activity in the right frontal
lobe is associated with avoidance motivation (Davidson et al., 1990b; Harmon-Jones, 2003; Harmon-Jones & Gable, 2018; Reznik & Allen, 2018). Although frontal alpha asymmetry was initially conceived of as a reflection of negative and positive affect (i.e., greater right-sided cortical activity associated with negative affective states) (Davidson, 1998; Davidson & Irwin, 1999), later research on the associations between frontal cortical asymmetry and anger has demonstrated that it more accurately reflects motivational direction. Anger is a negative affective state with an approach-based motivational direction (Carver & Harmon-Jones, 2009) and is associated with greater left frontal activity and greater frontal cortical asymmetry (Harmon-Jones, 2003, 2007).

Frontal cortical asymmetry has been the subject of clinical and affective science research for over 30 years, as detailed in numerous reviews (e.g., Coan & Allen, 2004; Harmon-Jones & Gable, 2018; Reznik & Allen, 2018). Many of these studies have focused on resting-state (i.e., not in response to a task) frontal cortical asymmetry as a correlate of trait-like motivational tendencies. For example, greater right-sided frontal cortical activity is associated with trait behavioral avoidance motivation (Shackman et al., 2009; Sutton & Davidson, 1997), while greater left-sided frontal cortical activity is associated with trait behavioral approach motivation (Amodio et al., 2008; Coan & Allen, 2003; Sutton & Davidson, 1997), as assessed by the behavioral inhibition/behavioral activation scales (Carver & White, 1994). Resting-state frontal cortical asymmetry is also associated with trait impulsivity (Gable et al., 2015; Santesso et al., 2008).

Critical to psychopathology, greater right-sided resting-state cortical activity is associated with trait negative affect (Tomarken et al., 1992), as well as a stronger self-reported emotional response to negative stimuli (Tomarken et al., 1990; Wheeler et al.,
Indeed, frontal cortical asymmetry first generated interest among researchers as a potential marker of depression following several studies that demonstrated relatively lower left-sided cortical activity is associated with depressive symptoms (Allen et al., 1993; Henriques & Davidson, 1990; Schaffer et al., 1983). Since then, several studies have replicated these results (Allen & Reznik, 2015) and extended them to anxiety disorders (Mathersul et al., 2008; Thibodeau et al., 2006) and PTSD (Meyer et al., 2015). However, the literature is somewhat mixed as some studies have failed to link resting-state frontal cortical asymmetry with psychopathology (e.g., Blackhart et al., 2006; Jesulola et al., 2015; McFarland et al., 2006).

Some researchers have suggested that differences in the literature might be due to the variability in cognitive processes that might occur during a resting-state EEG recording (Coan et al., 2006; Papousek & Schulter, 2004). Thus, assessment of frontal cortical asymmetry during or following an emotionally-evocative task, particularly one that has personal relevance to an individual (e.g., Gable & Harmon-Jones, 2008), might be a better indicator of the dysregulated motivational processes that might contribute to psychopathology (Coan et al., 2006). Notably, fewer studies have tested associations between frontal cortical asymmetry and affect during laboratory tasks meant to evoke approach or avoidance motivation compared to frontal cortical asymmetry during the resting state. However, the studies that have examined evoked motivational responses have found more consistent associations between greater left-sided frontal cortical activity and approach motivation and between greater right-sided activity and avoidance motivation (Harmon-Jones & Gable, 2018; Reznik & Allen, 2018). Such studies have made use of affective images (Harmon-Jones, 2007; Schöne et al., 2016), film clips
(Lopez-Duran et al., 2012; Papousek et al., 2014), audio clips (Papousek et al., 2011), and monetary incentives (Miller & Tomarken, 2001). Accordingly, less frontal cortical asymmetry (i.e., comparatively less left versus right frontal activity) during motivationally-evocative tasks is associated with greater depressive (Allen & Reznik, 2015), greater anxiety (e.g., Meyer et al., 2018), and greater PTSD (e.g., Meyer et al., 2018; Rabe et al., 2006) symptoms.

Given this pattern of results, it has been suggested that frontal cortical asymmetry might tap into the neural systems that mediate approach-avoidance responses to emotionally salient situations or stimuli (Coan & Allen, 2004; Davidson, 1993; Reznik & Allen, 2018). Further support for this is found in the results of several experiments that have used neurofeedback to alter frontal cortical asymmetry directly and have found consequent changes in motivational direction consistent with the left (approach) and right (avoidance) lateralization described in much of the literature (Allen et al., 2001; Harmon-Jones et al., 2008; Mennella et al., 2017; Quaedflieg et al., 2016).

In the context of stress, then, frontal cortical asymmetry might be indicative of an individual’s motivational action tendency when confronted with a specific stressor. Recent life stress is associated with greater right-sided cortical activity during the resting state (Lewis et al., 2007) as is a stronger acute physiological stress response to a laboratory stressor (Ma et al., 2021). Further, relatively greater right-sided cortical activity during a laboratory stressor is associated with greater attention to negative stimuli (Papousek et al., 2014; Pérez-Edgar et al., 2013) and subsequent negative affect (Papousek et al., 2014). However, the role of frontal cortical asymmetry in the stress response remains unclear, and notably, no studies to our knowledge, have tested changes
in frontal cortical asymmetry following a self-relevant stressor (e.g., recall of a negative life event).

**Emotion Regulation**

Emotion regulation refers to any process that attempts to alter emotion, including the type and intensity of emotional feelings, its expression, and when and where it occurs (Gross, 1998, 2010). Emotion regulation most often involves attempts to reduce the intensity of negative affective experiences (Gross, 2010). Given that much psychopathology is characterized by dysregulated affective experiences, the role of dysfunction in emotion-regulatory ability has received increased attention from researchers and clinicians over the past two decades (Gross & Jazaieri, 2014; Tamir, 2011). Indeed, dysfunctional emotion regulation has been proposed as a transdiagnostic process that might contribute to a wide variety of mental disorders (Cludius et al., 2020; Sloan et al., 2017). A meta-analysis found that ineffective emotion regulation is associated with numerous mental disorders, particularly internalizing disorders (Aldao et al., 2010). Further, neuroimaging studies have indicated that emotion regulation involves the modulation of activity in subcortical regions by the prefrontal cortex (Ochsner & Gross, 2005; Ochsner et al., 2012), and distress disorders are characterized by aberrant functioning within these circuits (Disner et al., 2011; Etkin & Wager, 2007; Hayes et al., 2012; Patel et al., 2012). Transdiagnostic psychotherapeutic treatments aimed at improving emotion-regulatory ability have been found to produce reliable reductions in distress disorder symptoms (Carlucci et al., 2021; Renna et al., 2017; Sakiris & Berle, 2019). Thus, these lines of evidence suggest that while approach and avoidance motivation might represent an emotion-generative process in response to a stimulus,
emotion regulation might represent a top-down regulatory process influencing those emotions, which can reduce maladaptive avoidance-related negative affect and associated distress symptoms.

**Heart Rate Variability and Emotion Regulation**

Heart rate variability (HRV), or the fluctuations in the time intervals between heart beats, has long been of interest to clinicians and researchers as a potential cardiovascular index of autonomic nervous system activity (Berntson et al., 1997). In particular, resting high-frequency heart rate variability (HF-HRV), which occurs in a frequency band between 0.15 and 0.40 Hz, has been found to reflect parasympathetic nervous system (PNS) activity, for example, in studies involving pharmacological blockade, such that higher power within that band reflects greater parasympathetic activity (Berntson et al., 1997; Shaffer & Ginsberg, 2017). Prefrontal cortical regions influence autonomic nervous system function via inhibition of subcortical regions, such as the amygdala, which project onto portions of the brainstem responsible for autonomic function, such as the medulla (Thayer et al., 2009, 2012). Thus, greater prefrontal activity might be indirectly reflected in greater activity in the PNS, which, in turn, may be a marker for numerous self-regulatory processes, including executive functioning, effortful control, and emotion regulation (Appelhans & Luecken, 2006; Porges, 1995, 2007; Thayer & Lane, 2000), leading to the proposition that HF-HRV might serve as an index of those processes.

Further, a meta-analysis has found that greater resting (i.e., when an individual is not engaged in a specific task) HRV is associated with more effective self-regulation, including successful emotion regulation, across a variety of tasks, contexts, and
populations (Holzman & Bridgett, 2017). Thus, baseline HF-HRV has been conceptualized as a marker for an individual’s general, trait-like capacity for self-regulation (Porges, 2007; Thayer & Lane, 2000). In the context of stress, having a trait-like propensity towards higher HRV at baseline increases the likelihood of an individual having an adaptive response to acute stress (Porges, 1995, 2007).

A possible association between HF-HRV and emotion regulation has led to a number of studies testing baseline HF-HRV as a marker of dysregulated emotional functioning across mental disorders (Beauchaine, 2015). Individuals with depression have lower baseline HF-HRV compared to individuals without depression (Kemp et al., 2010; Koch et al., 2019; Rottenberg, 2007) and greater severity of depressive symptoms is associated with lower HF-HRV (Kemp et al., 2010). Individuals with a variety of anxiety disorders have also been found to have lower baseline HF-HRV (Chalmers et al., 2014; Wang et al., 2023), even when controlling for psychiatric comorbidities (Chalmers et al., 2014). Similarly, individuals with PTSD have lower HF-HRV compared to individuals without PTSD (Schneider & Schwerdtfeger, 2020), and greater PTSD symptom severity is associated with lower baseline HF-HRV (Campbell et al., 2019). Thus, researchers have proposed that lower baseline HF-HRV might be a transdiagnostic correlate of emotion dysregulation across distress disorders (Beauchaine, 2015; Beauchaine & Thayer, 2015).

Notably, and contrary to the frontal cortical asymmetry literature, the literature on HF-HRV in response to a task or stressor has been more mixed than that of baseline HF-HRV (Hamilton & Alloy, 2016; Schneider & Schwerdtfeger, 2020). Results have ranged from suggesting that psychopathology is associated with increases in PNS activity (e.g.,
Liang et al., 2015) during stress, with excessive decreases in PNS activity (e.g., Dennis et al., 2016; Keary et al., 2009; Shinba et al., 2008) during stress, to being unrelated to PNS activity during stress (e.g., Beauchaine et al., 2019; Cyranowski et al., 2011; Ehrenthal et al., 2010; Hammel et al., 2011).

**Frontal Cortical Asymmetry and Autonomic Nervous System Activity**

The literature reviewed above suggests that heightened avoidance motivation and diminished emotion regulation ability, which can be indexed by frontal cortical asymmetry and HF-HRV, respectively, might be involved in the development and maintenance of distress psychopathology and might characterize maladaptive responses to stress associated with distress psychopathology. However, there is little research that has simultaneously examined both frontal cortical asymmetry and autonomic nervous system functioning. A few studies have found that greater right frontal cortical activity during stress is associated with concurrent heat rate (Ma et al., 2021; Papousek et al., 2014; Zhang et al., 2018). However, given that heart rate reflects both sympathetic and parasympathetic influences (Berntson et al., 1997; Shaffer & Ginsberg, 2017), these studies are not able to clarify associations between frontal cortical asymmetry and PNS activity. Only two studies, to our knowledge, assessed the relationship between frontal cortical asymmetry and heart rate variability. One small-sample study (Brouwer et al., 2011), found no relationship between the root mean square of successive differences (a metric of heart rate variability largely reflecting PNS activity) (Shaffer & Ginsberg, 2017) during stress and frontal cortical asymmetry during stress. The second study, found no association between HF-HRV and frontal cortical asymmetry at rest among women with depression, although both physiological measures were independently associated
with depression (Chang et al., 2012). Thus, the nature of the relationship between PNS activity and frontal cortical asymmetry, and the extent to which these measures reflect distinct or overlapping processes that contribute to distress psychopathology, remain unclear.

**The Current Study**

The purpose of this study was to examine baseline emotion regulation capacity (indexed by HF-HRV), motivational direction during a stressor (indexed by change in frontal cortical asymmetry) and change in self-reported negative affect during a stressor, as correlates of distress disorder symptoms among undergraduates who had experienced trauma. Frontal cortical asymmetry is thought to reflect approach and avoidance processes involved in generating emotional responses, while HF-HRV is thought to reflect the capacity to regulate those responses. However, it remains unclear whether frontal cortical asymmetry and HF-HRV represent distinct processes that independently participate in negative affective states.

There were several specific aims. First, we tested whether distress psychopathology (PTSD, anxiety, and depressive symptoms assessed both independently and as a latent factor) was associated with change in affect following a personally relevant stressor. We predicted that psychopathology symptoms would be associated with greater increases in negative affect, and greater reductions in positive affect, following the stressor. Second, we tested whether baseline HF-HRV was associated with change in affect following the stressor and with distress psychopathology. We predicted that greater baseline HF-HRV would be associated with smaller increases in negative affect following the stressor and with fewer psychopathology symptoms. Third, we tested
whether frontal cortical asymmetry, and electrocortical activity measured at specific electrode sites (both indexed by alpha power), changed following a personally relevant stressor. We predicted that there would be reductions in frontal cortical asymmetry; electrocortical activity was expected to show reductions in left frontal regions and increases in right frontal regions. Fourth, we tested whether distress psychopathology was associated with changes in frontal cortical asymmetry and changes reflecting neural activity at specific electrode sites. We predicted that greater psychopathology would be associated with smaller changes in frontal alpha asymmetry after the stressor, greater reductions in neural activity in left frontal areas, and greater increases in neural activity in right frontal areas. Finally, we tested the association between baseline HF-HRV and frontal cortical asymmetry. We predicted that greater baseline HF-HRV would be associated with greater frontal cortical asymmetry following the stressor.

Methods

Participants

Participants were young-adult, undergraduate students who had experienced trauma. Participants were recruited from an undergraduate psychology department subject pool at a Northeastern university. All individuals in the potential participant pool were prescreened and those who met inclusion criteria were invited to participate in the study. Inclusion criteria required that participants: a) be between the ages of 18-25, b) reported experiencing at least one DSM-5 criterion A traumatic event at some point in their lives, c) were not currently taking psychotropic or cardiovascular medications, and d) were right-handed.
One-hundred fifty-six (156) individuals participated in the study, of whom 38 were excluded from analyses based on responses to study questionnaires administered during the lab session (see below): 27 for not reporting having experienced a criterion A trauma, 10 for reporting they had taken a psychotropic medication, and 1 for reporting having taken a cardiovascular medication. Thus, $n = 118$ participants remained.

Seventeen (17) participants had poor or no EKG data, thus the finale sample size for HRV analyses was $n = 101$. Sixteen (16) participants were excluded from analyses involving EEG data due to being left-handed, and an additional 24 participants had poor or no EEG data. Thus, the final sample size for EEG analyses was $n = 78$. Participant demographics are reported in Table 1.

**Measures**

*Life Events Checklist for DSM-5 (LEC-5).* The LEC-5 (Weathers et al., 2013a) was used to assess whether participants experienced a DSM-5 criterion a traumatic event that would qualify them for a potential PTSD diagnosis (e.g., physical or sexual assault, serious accident, natural disaster, etc.). The LEC-5 is a 17-item, self-report measure that provides a list of potential traumatic events and asks participants to indicate whether they experienced the event, witnessed it, learned about it happening to someone else, or were exposed to the event as part of their job. The LEC-5 has been shown to have reliability and validity in assessing likely trauma-exposure according to DSM-5 criteria (Weathers et al., 2013a).

*Beck Anxiety Inventory (BAI).* The BAI (Beck et al., 1988) was used to assess anxiety symptom severity. The BAI is a 21-item, self-report measure in which participants indicate the extent to which they were bothered by anxiety symptoms over
the past month. The BAI employs a Likert-type scale that ranges from 0 (“not at all”) to 3 (“severely”). Total scores range from 0 to 63 with a score of 22 or higher indicating greater than mild anxiety. The BAI has been shown to have reliability and validity in assessing anxiety symptoms (Beck et al., 1988).

*Center for Epidemiological Studies Depression Scale (CESD)*. The CES-D (Radloff, 1977) was used to assess depressive symptoms. The CESD is a 20-item, self-report measure in which participants indicate the extent to which they experienced symptoms of depression in the past week. The CES-D employs a Likert-type scale that ranges from 0 (“rarely or none of the time”) to 3 (“most or all of the time”). Total scores range from 0 to 60, and a score of 16 or higher indicates greater than mild depressive symptoms. The CES-D has been shown to have reliability and validity in assessing depressive symptoms (Radloff, 1977).

*PTSD Checklist for the DSM-5 (PCL-5)*. The PCL-5 (Weathers et al., 2013b) was used to assess symptoms of PTSD. The PCL-5 is a 20-item, self-report measure in which participants indicate the extent to which they were bothered by PTSD symptoms over the past month. The PCL-5 employs a Likert-type scale that ranges from 0 (“not at all”) to 4 (“extremely”). Total scores range from 0 to 80, and a score of 33 or higher indicates a likely PTSD diagnosis. The PCL-5 has been shown to have reliability and validity in assessing PTSD symptoms (Weathers et al., 2013b).

*Positive and Negative Affect Schedule (PANAS)*. The PANAS (Watson et al., 1988) was used to assess momentary negative affect before and after the stress task. The PANAS is a 20-item, self-report measure in which participants indicate the extent to which they feel certain emotions. The PANAS employs a Likert-type scale that ranges
from 1 (“very slightly or not at all”) to 5 (“extremely”). The PANAS is divided into negative affect and positive affect subscales, wherein total scores for each subscale range from 10 to 50. The PANAS has been shown to have reliability and validity in assessing positive and negative affective experiences (Watson et al., 1988). See Appendix I for copies of all self-report measures used in the current study.

**EEG Recording.** Electroencephalograph (EEG) data were collected using a Neuroscan Synamps 2 (Compudemics Neuroscan, Herdon, VA) amplifier and a cap (Electro-Cap International, Eaton, OH) with 19 tin electrodes located at scalp sites based on the International 10/20 System (AFz, Fz, F3, F4, F7, F8, FCz, Cz, C3, C4, T3, T4, T7, T8, Pz, P3, P4, O7, O8) and 4 additional electrodes for the left and right mastoids (M1 and M2) and above and below the left eye (VEOG). The ground electrode was AFz. The mastoid and VEOG sites were cleaned and exfoliated. Then an electrode cap of the appropriate size was placed on the participant’s scalp and electrode gel was applied to each electrode site. Impedances were measured in Neuroscan software and were kept at or below 5 kΩ. Data were collected at a sampling rate of 1000 Hz. The left mastoid (M1) served as the online reference.

**EEG Data Processing.** Data processing followed established procedures used for frontal cortical asymmetry (Allen et al., 2004). Data were re-referenced offline to the average of the left and right (M2) mastoids. A band-pass filter was applied at 0.1-100 Hz, with a 60-Hz notch filter. Ocular artifacts were reduced using an established regression-based procedure (Gratton et al., 1983) in the Neuroscan software. Data segments with remaining artifacts were rejected using the moving window algorithmic detection tool in EEGLAB (Delorme & Makeig, 2004). The window width was set at 200 ms, the window
step was set at 50 ms, and the rejection threshold was set at $\pm 100 \mu\text{V}$. Each one-min segment of EEG data was divided into 1.5-sec epochs with 50% overlap. A fast Fourier transform (FFT) was used to calculate power spectra and total power in the alpha frequency band (8-13 Hz) was obtained for each data segment. Power values were log-transformed and averaged across the eight data segments. Frontal cortical asymmetry scores were calculated as the natural log of alpha power on the right hemisphere minus the natural log of alpha power on the left hemisphere, using midfrontal (F4-F3) and lateral frontal (F8-F7) electrodes.

**EKG Recording.** Electrocardiogram (EKG) data were collected using a Biopac MP100 system (Biopac Systems, Inc., Goleta, CA) and the Biopac Acqknowledge software. Electrode sites were cleaned and exfoliated prior to electrode placement. Silver/Silver-chloride electrodes were placed on the right and left wrists and the ground electrode was placed on the right ankle. Data were collected at 1000 Hz.

**EKG Data Processing.** Data processing was completed using Mindware software (Mindware Technologies Ltd., Gahanna, Ohio) and followed guidelines described by Berntson et al. (1997). Data were divided into 1-min segments. The minimum heart rate was set at 40 beats per minute (BPM) and the maximum was set at 200 BPM. Baseline and muscle noise filters were applied to reduce noise in the 0.25 Hz to 0.40 Hz range. Continuous R-R intervals were identified using the R-peak detection algorithm. Both the IBI minimum/maximum and the Minimum Artifact Deviation and Maximum Expected Deviation (MAD/MED) algorithms were used to detect artifacts. The high-frequency band was set at 0.120 Hz to 0.4 Hz. Data were visually inspected and errors in R-wave markings were corrected. Only epochs that contained at least 30 sec of contiguous EKG
data were included in high-frequency band power calculations. The number of manual R-peak estimations based on visual inspection was kept below 10% of all R-peaks for each participant.

**Procedure**

Participants were invited for participation based on results of a prescreen asking about potential trauma-exposure. Participants were instructed to refrain from smoking, drinking, drug use, or caffeine consumption for 2 hours prior to arriving to the lab. After arriving to the lab, they provided informed consent and the study questionnaires were administered. Following completion of the questionnaires, the EEG and EKG instruments were attached to the participants. Participants then completed self-report measures of state affect. Then simultaneous EEG and EKG recordings were taken for an 8-minute baseline period. During this recording, and subsequent resting-state recordings, participants sat at rest and the experimenter instructed participants to either open or close their eyes for 1-minute intervals, such that there were four 1-minute intervals of data recorded while participants had their eyes opened and closed. While participants’ eyes were open, they were instructed to look at a fixation cross on a computer screen to minimize eye-movement artifact in the EEG. The EKG data, but not the EEG data, during this baseline period are included in the analyses of the current study.

Following the baseline recording, participants completed an emotional image task designed to assess event related potentials in the EEG. This task involved passively viewing a series of negative, neutral, and positive images, presented in random order, and took approximately 10 minutes to complete. Only EEG data were collected during this task. These data are not included in the current analyses and are reported on elsewhere.
Following the emotional image task, self-reported state affect data were collected and pre-stressor, resting EEG and EKG data were recorded for 8 minutes, with participants receiving the same instructions as during the baseline period. Next, participants completed a stressful speaking task which involved 2 min of thinking about “the most stressful event,” that the participant experienced, and 3 min of describing the event aloud. Participants were instructed to “try to think about the most stressful thing that has ever happened to you. Concentrate on the associated thoughts, feelings, and bodily sensations, as well as anything you saw, smelled, or heard. Think about responding to the following questions: How did you feel? What triggered that feeling? What did you see? How did your body respond? What did you do? Try to recreate the whole experience in your mind.” These instructions are based on research that suggests that elicitation of sensory details during autobiographical memory recall can evoke stronger emotional responses (Lang, 1979). The participants were alone in the room during both portions of the stress task, while research assistants were able to observe and verify compliance with instructions from an adjoining room through a two-way mirror. Following the task, self-reported state affect data were collected and a final, post-stressor 8-min recording of EEG and EKG data was completed. Finally, participants were debriefed and given course credit for participating. Due to technical issues, the pre-stressor EKG recordings for most of the participants in this study were compromised and cannot be used. Thus, we only include the baseline EKG data in the current analyses.

Statistical Analyses

Univariate outliers (defined as scores that fall outside of plus or minus two interquartile ranges from the median) were be identified and winsorized prior to analyses.
Given that biased results might arise from using multiple imputation to impute missing physiological data from self-report data, when the aim of analyses is to test whether relationships between physiological and self-report data exist in the first place (Peters et al., 2012; Richter et al., 2019), we did not use multiple imputation to account for missing physiological data. We used pairwise deletion to remove cases that are missing data from analyses involving the missing variables, while maintaining power for analyses in which all variables are available. Alpha power values for individual electrode sites were natural log transformed according to convention (J. J. B. Allen et al., 2004).

Paired samples t-tests were used to test whether there was a change in self-reported negative and positive affect following the stressor, as well as whether there was a change in frontal cortical asymmetry and alpha power at specific electrode sites. Multiple regression analyses were performed to test whether individual self-reported symptom scores predicted change in affect and predicted baseline HF-HRV, and whether baseline HF-HRV predicted change in affect. Multiple regression analyses were also used to test whether frontal cortical asymmetry predicted change in affect, and whether individual symptom scores predicted change in frontal cortical asymmetry and alpha power at specific electrode sites. Finally, multiple regression analyses were used to test whether baseline HF-HRV predicted change in frontal cortical asymmetry. Structural equation modeling was used to detect a distress psychopathology latent factor from the BAI, CES-D, and PCL-5 observed variables and to test frontal cortical asymmetry and HF-HRV as predictors of the distress psychopathology latent factor in separate models.
Results

Participant Characteristics

Clinical characteristics of the sample are displayed in Table 2. The mean score on the PCL-5 was 20.51 ($SD = 15.42$), and 23.7% of participants had a PCL-5 score of 33 or above, suggesting a likely PTSD diagnosis. Participants, on average, reported personally experiencing 2.84 lifetime traumatic events ($SD = 1.71$). The most frequently reported traumatic events were natural disaster (59.3%); physical assault (49.2%); transportation accident (44.1%); other unwanted sexual experience (34.7%); serious accident at home, work, or recreational activity (20.5%); and sexual assault (17.8%). The mean score on the CES-D was 19.08 ($SD = 10.26$), and 53.4% of participants had a CES-D score of 16 or above, suggesting a likely depressive disorder diagnosis. The mean score on the BAI was 12.87 ($SD = 9.67$), suggesting that, on average, the current sample had low anxiety symptoms, and 22% of participants had a BAI score of 21 or above, indicating anxiety symptoms that are moderate or higher in severity.

We performed independent samples $t$-tests to test whether men and women differed on baseline high-frequency heart rate variability (HF-HRV), mid frontal cortical asymmetry, and lateral frontal cortical asymmetry. There was no difference between men ($M = 6.601, SD = 1.085$) and women ($M = 6.380, SD = 1.141$) on HF-HRV, $t (99) = 0.984, p = 0.328$. There were no differences between men ($M = 0.035, SD = 0.113$) and women ($M = 0.013, SD = 0.109$) on pre-stressor mid frontal cortical asymmetry, $t (77) = 0.806, p = 0.423$, nor between men ($M = 0.027, SD = 0.096$) and women ($M = 0.041, SD = 0.110$) on post-stressor mid frontal cortical asymmetry, $t (76) = -0.773, p = 0.442$. Likewise, there were no difference between men ($M = 0.025, SD = 0.209$) and women ($M
= 0.055, $SD = 0.173$) on post-stressor lateral frontal cortical asymmetry, $t (77) = -0.069, p = 0.945$, nor between men ($M = -0.011, SD = 0.161$) and women ($M = -0.007, SD = 0.198$) on post-stressor lateral frontal cortical asymmetry, $t (76) = -0.087, p = 0.931$.

**Effect of the Stressor on State Affect**

We performed paired-samples $t$-tests to test the effects of the stressor on participants’ self-reported state affect (Figure 1). Participants reported lower positive affect following the stressor ($M = 18.84, SD = 7.12$) compared to before the stressor ($M = 21.53, SD = 7.88$), $t (103) = -4.65, p < 0.001, d = 0.456$. Participants also reported higher negative affect following the stressor ($M = 19.49, SD = 7.27$) compared to before the stressor ($M = 15.86, SD = 5.33$), $t (103) = 6.43, p < 0.001, d = 0.630$. The effect of the stressor on state affect is depicted in Figure 1.

**Symptom Scores as Predictors of Change in Affect**

We performed multiple regression analyses to test whether individual symptom scores predicted change in affect pre- to post-stressor. The self-reported affect rating obtained following the stressor was the outcome, and the corresponding pre-stressor self-reported affect score was included as a predictor. The model predicting post-stressor negative affect was significant, $F (4,103) = 17.585, p < 0.001, R^2 = 0.415$. Pre-stressor negative affect predicted post-stressor negative affect, $\beta = 0.601, t = 7.512, p < 0.001, sr^2 = 0.333$. PTSD symptoms did not predict post-stressor negative affect, $\beta = 0.066, t = 0.699, p = 0.486, sr^2 = 0.003$; nor did anxiety symptoms, $\beta = -0.125, t = -1.2626, p = 0.210, sr^2 = -0.009$. Depressive symptoms were a marginally non-significant predictor of post-stressor negative affect, $\beta = 0.185, t = 1.825, p = 0.071, sr^2 = 0.020$. 
The model predicting post-stressor positive affect also was significant, $F(4, 103) = 24.788, p < 0.001, R^2 = 0.500$. Pre-stressor positive affect predicted post-stressor positive affect, $\beta = 0.694, t = 9.707, p < 0.001, sr^2 = 0.476$. PTSD symptoms did not predict post-stressor positive affect, $\beta = 0.061, t = 0.712, p = 0.478, sr^2 = 0.003$; nor did anxiety symptoms, $\beta = 0.093, t = -1.020, p = 0.310, sr^2 = -0.072$. Depressive symptoms were a marginally non-significant predictor of post-stressor positive affect, $\beta = -0.172, t = -1.836, p = 0.069, sr^2 = -0.005$.

**Baseline High-Frequency Heart Rate Variability as a Predictor of Change in Affect**

We performed a similar set of multiple regression analyses to test whether baseline high-frequency heart rate variability (HF-HRV) predicted change in affect pre- to post-stressor. Self-reported affect following the stressor was the outcome, and the corresponding pre-stressor self-reported affect score was included as a predictor. The model predicting post-stressor negative affect was significant, $F(2,92) = 36.927, p < 0.001, R^2 = 0.439$. Pre-stressor negative affect predicted post-stressor negative affect, $\beta = 0.658, t = 8.410, p < 0.001, sr^2 = 0.432$, and higher baseline HF-HRV predicted greater post-stressor negative affect, $\beta = 0.172, t = 2.200, p = 0.030, sr^2 = 0.030$.

The model predicting post-stressor positive affect also was significant, $F(2,92) = 43.410, p < 0.001, R^2 = 0.491$. Pre-stressor positive affect predicted post-stressor positive affect, $\beta = 0.702, t = 9.316, p < 0.001, sr^2 = 0.491$; however, HF-HRV did not predict post-stressor positive affect, $\beta = -0.031, t = -0.407, p = 0.685, sr^2 = -0.001$.

**Symptom Scores as Predictors of Baseline High-Frequency Heart Rate Variability**

We performed multiple regression analyses to test whether individual symptom scores predicted HF-HRV. PTSD symptoms, anxiety symptoms, and depressive
symptoms were each entered as predictors, and HF-HRV was entered as the outcome. The model was not significant, $F (3, 100) = 0.788, p = 0.503, R^2 = 0.024$. Neither PTSD symptoms, $\beta = 0.072, t = 0.587, p = 0.559, sr^2 = 0.003$; anxiety symptoms, $\beta = -0.116, t = -0.902, p = 0.369, sr^2 = 0.011$; nor depressive symptoms, $\beta = 0.147, t = 1.067, p = 0.289, sr^2 = 0.008$, predicted baseline HF-HRV.

**Baseline High-frequency Heart Rate Variability as a Predictor of Distress Psychopathology**

We tested a structural equation model wherein baseline HF-HRV predicted a distress psychopathology latent factor, with PTSD, depressive, and anxiety symptoms scores loading onto a single latent factor (Figure 3). Fit indices suggest that the model fit our data, $\chi^2 (df = 2) = 1.482, p = 0.477, CFI = 1.000$ (exact fit), TLI = 1.017 (exact fit), SRMR = 0.022 (acceptable fit), RMSEA < 0.001 (90% CI [< 0.001, 0.167]; exact fit). However, considering the low power these analyses achieved, these fit indices should be interpreted with caution. Further, distress psychopathology did not predict baseline HF-HRV, $\beta = 0.108, SE = 0.681, p = 0.983$. 
Figure 3. Structural equation model of high-frequency heart rate variability (HF-HRV) predicting distress psychopathology.

**Effect of the Stressor on Frontal Cortical Asymmetry**

We performed paired-samples $t$-tests to test the effects of the stressor on participants’ frontal cortical asymmetry (calculated as $\ln$[right sided alpha power] – $\ln$[left sided alpha power]) (Figure 2). There was no difference in frontal cortical asymmetry at midfrontal sites before the stressor ($M = 0.024, SD = 0.108$) compared to after the stressor ($M = 0.041, SD = 0.105$), $t(75) = -1.652, p = 0.103, d = 0.189$. There was also no difference in frontal cortical asymmetry at lateral frontal sites before the stressor ($M = 0.008, SD = 0.187$) compared to after the stressor ($M = -0.009, SD = 0.187$), $t(75) = 1.116, p = 0.268, d = 0.128$. 
Effect of the Stressor on Alpha Power at Specific Electrode Sites

We also performed paired-samples t-tests to test the effects of the stressor on neural activity (indexed by lower alpha power) at specific electrode sites. There was no difference in alpha power at the left midfrontal site before the stressor ($M = 1.040, SD = 0.587$) compared to after the stressor ($M = 1.117, SD = 0.552$), $t(75) = -1.622, p = 0.109, d = 0.186$. However, participants exhibited lower alpha power (i.e., more neural activity) at the right midfrontal region before the stressor ($M = 1.067, SD = 0.569$) compared to after the stressor ($M = 1.180, SD = 0.546$), $t(68) = -3.077, p = 0.003, d = 0.353$. There was no difference between alpha power at the left lateral frontal site before the stressor ($M = 0.779, SD = 0.644$) compared to after the stressor ($M = 0.825, SD = 0.586$), $t(75) = -1.033, p = 0.305, d = 0.118$. Participants exhibited marginally lower alpha power (i.e., a more neural activity) at the right lateral frontal site before the stressor ($M = 0.747, SD = 0.607$) compared to after the stressor ($M = 0.806, SD = 0.578$), $t(75) = -1.846, p = 0.069, d = 0.212$.

Symptom Scores as Predictors of Change in Frontal Cortical Asymmetry

We performed multiple regression analyses to test whether individual symptom scores predicted change in frontal cortical asymmetry pre- to post-stressor. Frontal asymmetry scores following the stressor were the outcomes, and the corresponding pre-stressor frontal cortical asymmetry scores were included as predictors. The model predicting post-stressor frontal cortical asymmetry at midfrontal sites was significant, $F(4,75) = 16.547, p < 0.001, R^2 = 0.482$. Pre-stressor frontal cortical asymmetry predicted post-stressor frontal cortical asymmetry, $\beta = 0.677, t = 7.796, p < 0.001, sr^2 = 0.444$. PTSD symptoms did not predict post-stressor frontal cortical asymmetry, $\beta = 0.131, t =$
1.220, \( p = 0.226, \sigma^2 = 0.011 \); nor did anxiety symptoms, \( \beta = -0.019, t = -0.178, p = 0.859, \sigma^2 < 0.001 \). However, the relationship between depressive symptoms and post-stressor frontal cortical asymmetry was marginally significant, \( \beta = -0.224, t = -1.888, p = 0.063, \sigma^2 = 0.026 \), such that a greater frontal cortical asymmetry post-stressor was associated with fewer depressive symptoms.

The model predicting post-stressor frontal cortical asymmetry at lateral frontal sites was significant, \( F (4,75) = 23.463, p < 0.001, R^2 = 0.569 \). Pre-stressor frontal cortical asymmetry predicted post-stressor frontal cortical asymmetry, \( \beta = 0.778, t = 9.535, p < 0.001, \sigma^2 = 0.552 \). PTSD symptoms did not predict post-stressor frontal cortical asymmetry, \( \beta = -0.065, t = -0.651, p = 0.517, \sigma^2 = 0.003 \); nor did anxiety symptoms, \( \beta = 0.017, t = 0.168, p = 0.667, \sigma^2 < 0.001 \); nor depressive symptoms, \( \beta = -0.066, t = -0.608, p = 0.545, \sigma^2 = 0.002 \).

**Symptom Scores as Predictors of Alpha Power at Specific Electrode Sites**

We performed multiple regression analyses to test whether individual symptom scores predicted change in alpha power at specific electrode sites before and after the stressor. Pre-stressor alpha power and PTSD, depressive, and anxiety symptoms were entered as predictors and post-stressor alpha power was entered as the outcome. The model predicting post-stressor alpha power at the left midfrontal electrode site was significant, \( F (4,75) = 24.724, p < 0.001, R^2 = 0.582 \), and pre-stressor alpha power predicted post-stressor alpha power, \( \beta = 0.749, t = 9.649, p < 0.001, \sigma^2 = 0.377 \). Greater post-stressor alpha power (i.e., less neural activity) was associated with fewer PTSD symptoms, \( \beta = -0.215, t = -2.243, p = 0.028, \sigma^2 = 0.030 \). The relationship between post-stressor alpha power and anxiety symptoms was marginally significant, \( \beta = 0.183, t = \)
1.850, $p = 0.068$, $sr^2 = 0.020$, such that greater post-stressor alpha power (i.e., less neural activity) was associated with more anxiety symptoms. There was no relationship between alpha power and depressive symptoms, $\beta = 0.015$, $t = -0.144$, $p = 0.886$, $sr^2 < 0.001$.

The model predicting post-stressor alpha power at the right midfrontal electrode site was significant, $F(4, 75) = 42.843$, $p < 0.001$, $R^2 = 0.707$, and pre-stressor alpha power predicted post-stressor alpha power, $\beta = 0.843$, $t = 12.992$, $p < 0.001$, $sr^2 = 0.696$. However, post-stressor alpha power was not predicted by PTSD symptoms, $\beta = 0.087$, $t = 1.091$, $p = 0.279$, $sr^2 = 0.005$; anxiety symptoms $\beta = 0.040$, $t = 0.488$, $p = 0.627$, $sr^2 = 0.001$; nor depressive symptoms, $\beta = -0.119$, $t = -1.332$, $p = 0.187$, $sr^2 = 0.007$.

The model predicting post-stressor alpha power at the left lateral frontal electrode site was significant, $F(4, 75) = 35.023$, $p < 0.001$, $R^2 = 0.664$, and pre-stressor alpha power predicted post-stressor alpha power, $\beta = 0.813$, $t = 11.664$, $p < 0.001$, $sr^2 = 0.645$. PTSD symptoms predicted post-stressor alpha, $\beta = -0.174$, $t = -2.022$, $p = 0.047$, $sr^2 = 0.019$, such that greater alpha power (i.e., less neural activity) was associated with fewer PTSD symptoms. There were no relationships between post-stressor alpha power and anxiety symptoms $\beta = 0.092$, $t = 1.045$, $p = 0.300$, $sr^2 = 0.005$; nor depressive symptoms, $\beta = 0.115$, $t = 1.194$, $p = 0.237$, $sr^2 = 0.007$.

The model predicting post-stressor alpha power at the right lateral frontal right electrode site was significant, $F(4, 75) = 71.663$, $p < 0.001$, $R^2 = 0.801$, and pre-stressor alpha power predicted post-stressor alpha power, $\beta = 0.893$, $t = 16.853$, $p < 0.001$, $sr^2 = 0.794$. However, post-stressor alpha power was not predicted by PTSD symptoms, $\beta = -0.077$, $t = -1.163$, $p = 0.249$, $sr^2 = 0.004$; anxiety symptoms $\beta = 0.063$, $t = 0.924$, $p =
0.359, $sr^2 = 0.002$; nor depressive symptoms, $\beta = 0.039$, $t = -0.531$, $p = 0.597$, $sr^2 = 0.001$.

**Frontal Cortical Asymmetry as a Predictor of Distress Psychopathology**

We tested a structural equation model wherein we tested both the direct and indirect effects of pre-stressor mid frontal cortical asymmetry, as well as the direct effect of post-stressor frontal cortical asymmetry on distress psychopathology (Figure 4). Fit indices of this model suggested that it fit our data, $\chi^2 (df = 4) = 2.504$, $p = 0.644$, CFI = 1.00 (exact fit), TLI = 1.027 (exact fit), SRMR = 0.030 (acceptable fit), RMSEA < 0.001 (90% CI [< 0.001, 0.112]; exact fit). However, considering the low power these analyses achieved, these fit indices should be interpreted with caution. Greater pre-stressor mid frontal cortical asymmetry predicted greater distress psychopathology, $\beta = 0.313$, SE = 10.028, $p = 0.049$; however, post-stressor mid frontal cortical asymmetry did not, $\beta = -0.231$, SE = 10.449, $p = 0.147$. Pre-stressor mid frontal cortical asymmetry predicted post-stressor mid frontal cortical asymmetry, $\beta = 0.677$, SE = 0.081, $p < 0.001$. 
Figure 4. Structural equation model testing both the direct and indirect effects of pre-stressor mid frontal cortical asymmetry, as well as the direct effect of post-stressor mid frontal cortical asymmetry on distress psychopathology.

We also tested a structural equation model wherein we tested both the direct and indirect effects of pre-stressor lateral frontal cortical asymmetry, as well as the direct effect of post-stressor frontal cortical asymmetry on distress psychopathology (Figure 5). Fit indices of this model suggested that it fit our data, $\chi^2 (df = 4) = 1.045$, $p = 0.903$, CFI = 1.00 (exact fit), TLI = 1.047 (exact fit), SRMR = 0.021 (acceptable fit), RMSEA < 0.001 (90% CI [< 0.001, 0.058]; exact fit). However, considering the low power these analyses achieved, these fit indices should be interpreted with caution. Greater pre-stressor lateral frontal cortical asymmetry predicted greater distress psychopathology, $\beta = 0.472$, SE = 6.39, $p = 0.007$; however, post-stressor lateral frontal cortical asymmetry did not, $\beta = -0.242$, SE = 6.618, $p = 0.160$. Pre-stressor lateral frontal cortical asymmetry
predicted post-stressor lateral frontal cortical asymmetry, $\beta = 0.740$, SE = 0.073, $p < 0.001$.

**Figure 5.** Structural equation model testing both the direct and indirect effects of pre-stressor lateral frontal cortical asymmetry, as well as the direct effect of post-stressor lateral frontal cortical asymmetry on distress psychopathology.

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**Post-hoc Power Analysis of Structural Equation Models**

According to MacCallum et al. (1996), based on the sample sizes in our analyses and our model fits, we achieved the following levels of power for our structural equation model analyses: HF-HRV model (Figure 3), exact fit, $df = 2, n = 101$, power < 0.112; mid frontal cortical asymmetry model (Figure 4), exact fit, $df = 4, n = 78$, power < 0.112; lateral frontal cortical asymmetry model (Figure 5), exact fit, $df = 4, n = 78$, power < 0.112. According to Kline (2016), a sample size of $n = 118$ falls within the range that can
be defined as a medium-sized sample (i.e., 100 – 200 participants) and a sample size of \( n = 78 \) falls in a range that can be considered small (i.e., less than 100 participants).

**Baseline High-Frequency Heart Rate Variability as a Predictor of Change in Frontal Cortical Asymmetry**

We performed multiple regression analyses to test whether baseline HF-HRV predicted change in frontal cortical asymmetry. Pre-stressor frontal cortical asymmetry and baseline HF-HRV were entered as predictors and post-stressor frontal cortical asymmetry was entered as the outcome. The model predicting post-stressor mid frontal cortical asymmetry was significant, \( F(2, 68) = 27.301, p < 0.001, R^2 = 0.453 \), and pre-stressor frontal cortical asymmetry predicted post-stressor frontal cortical asymmetry, \( \beta = 0.672, t = 7.383, p < 0.001, sr^2 = 0.452 \). However, baseline HF-HRV was not associated with post-stressor mid frontal cortical asymmetry, \( \beta = 0.026, t = 0.171, p = 0.865, sr^2 < 0.001 \). The model predicting post-stressor lateral frontal cortical asymmetry was significant, \( F(2, 68) = 40.290, p < 0.001, R^2 = 0.550 \), and pre-stressor frontal cortical asymmetry predicted post-stressor frontal cortical asymmetry, \( \beta = 0.742, t = 8.977, p < 0.001, sr^2 = 0.452 \). However, baseline HF-HRV was not associated with post-stressor lateral frontal cortical asymmetry, \( \beta = -0.019, t = -0.225, p = 0.823, sr^2 < 0.001 \).

**Discussion**

The current study tested the relationships between distress psychopathology (including PTSD, anxiety, and depressive symptoms) and physiological markers of trait emotion regulation (baseline high frequency heart rate variability [HF-HRV]) and neural markers of approach and avoidance motivation in response to a stressor (frontal cortical
asymmetry and alpha power at midfrontal and lateral frontal brain areas). Our overall pattern of results is somewhat contradictory. Contrary to our hypotheses, greater HF-HRV was associated with higher negative affect after the stressor but was unrelated to symptom measures nor the distress psychopathology latent factor. Further, participants exhibited on average lower neural activity at right frontal regions following the stressor, suggesting a decrease in avoidance motivation. Only lower left frontal activity (i.e., less approach motivation) following the stressor was associated with fewer PTSD symptoms, but there was also a trend towards a significant association with greater anxiety symptoms. These results should be interpreted with caution given the low power achieved by our analyses.

**High Frequency Heart Rate Variability**

Contrary to our hypotheses, and much of the prior research on the relationships between HF-HRV and affect, the current study found a positive relationship between baseline HF-HRV and negative affect, such that greater HF-HRV was associated with greater negative affect following the stressor. Further, contrary to our hypotheses, we did not find any associations between HF-HRV and distress psychopathology, either assessed as a single latent factor or as independent symptom measures. Prior studies have largely demonstrated that HF-HRV is inversely related to negative affect and distress psychopathology (Beauchaine & Thayer, 2015), leading to the proposition that baseline HF-HRV is a marker of trait-like emotion regulation ability (Porges, 2007; Thayer & Lane, 2000). Lower baseline HF-HRV has been found to be associated with depressive symptoms (Kemp et al., 2010; Koch et al., 2019; Rottenberg, 2007), anxiety symptoms (Chalmers et al., 2014; Wang et al., 2023), and PTSD symptoms (A. A. Campbell et al.,
However, some have cautioned against conflating heart rate variability with emotion regulation, as there is evidence that it might more accurately be described as a marker of an individual’s trait-like capacity to respond flexibly to challenges (Appelhans & Luecken, 2006; Thayer & Lane, 2000). This nuanced distinction might help explain some of the contradictory results in the HF-HRV literature, including those of the current study.

Whereas the emotion regulation model of heart rate variability predicts that individuals with higher baseline levels of HF-HRV would be better able to down-regulate their emotional responses to stressors, and thus experience lower negative affect, the flexibility model predicts that individuals with higher baseline HF-HRV would be better able to access a wider repertoire of responses, which may or may not include down-regulation of negative emotions, depending on what would be adaptive for that individual in that specific context. Ode et al. (2010) tested the emotion regulation model of heart rate variability against the flexibility model and found evidence for the latter. Namely, they did not find that HF-HRV predicted neuroticism nor negative affective experiences (e.g., stress reactivity, negative affect), but instead, that individuals with lower levels of baseline HF-HRV experience stronger relationships between neuroticism and negative affective experiences. Ode et al. (2010) suggest that individuals with lower HF-HRV are less able to flexibly respond to stressors, thus trait neuroticism has a larger effect on those individual’s affect. Alternatively, they argue that baseline HF-HRV might have a mitigating effect such that individuals with higher HF-HRV might not be as likely to experience negative affect, even when trait neuroticism is high. This does not necessarily mean that individuals with higher HF-HRV experience less emotional reactivity than
those with lower HF-HRV, but rather that individuals with higher HF-HRV are better able to respond flexibly to reduce the negative affect that might result from stressful experiences.

There is further evidence against HF-HRV as a marker of emotion regulation, rather than flexibility, in several studies that, similar to the current study, have found associations between higher HF-HRV and greater negative affect. For example, Butler et al. (2006) found that higher baseline HF-HRV was associated with greater negative affect in response to a distressing social interaction, and that HF-HRV increased when participants were instructed to employ an emotion regulations strategy. They conclude that experiencing and expressing negative affect during the interaction was adaptive and reflective of flexible responding. Other studies have found that higher baseline HF-HRV is associated with greater daily negative affect among people with asthma and low perceived self-efficacy (T. S. Campbell et al., 2006), and that women with eating disorders have higher baseline HF-HRV and negative affect compared to healthy controls (Het et al., 2015). Thus, although our results are in the minority of the overall heart rate variability literature (Vaessen et al., 2021), they are not without precedent.

There are a number of situations in which greater experience of negative affect might be considered an adaptive response. Emotional Processing Theory (Foa & Kozak, 1986) posits that anxiety and fear-related disorders develop as a result of excessive avoidance of situations and stimuli that induce negative affect (e.g., fear, anxiety, panic, etc.); thus, the foundation of exposure therapy in treating these disorders involves gradual exposure to avoided stimuli to weaken the relationship between the stimulus and the negative affective response (Foa et al., 1989). Exposure therapy requires some experience
of negative affect for the necessary learning to occur (Craske et al., 2014). Further, major depressive disorder is characterized by reduced emotional reactivity to both positive and negative stimuli (Bylsma et al., 2008; Rottenberg et al., 2005), suggesting a role of maladaptive blunted emotional responding in the development of depression. Thus, it should not be assumed that heightened negative affect reported in the laboratory is always an indicator of maladaptive responding, and more work is needed to clarify the contexts in which experiencing negative affect reflects the adaptive flexibility that HF-HRV supposedly assesses.

Finally, other moderators might affect the relationship between HF-HRV, negative affect, and psychopathology symptoms. For example, Campbell et al. (2019) found that as age increases, the strength of the relationship between heart rate variability and PTSD symptoms also increases, thus caution should be taken when comparing samples of different ages. Campbell et al. (2019) also found that the type of symptom measure used (e.g., one assessing DSM symptoms or not) can also affect the strength of the relationship between heart rate variability and psychopathology. The PCL-5 is the only measure used in the current study that directly assesses DSM-5 psychopathology symptoms, whereas the BAI and CES-D both assess a broader range of anxiety and depressive symptoms that might not have a one-to-one correspondence to symptoms listed in the DSM. Lastly, use of antidepressant medication might also impact the relationship between heart rate variability and psychopathology, as some studies have demonstrated that selective serotonin reuptake inhibitors might decrease depressive and anxiety symptoms, without affecting heart rate variability (Kemp et al., 2010; van Zyl et
al., 2008). Although the current study excluded participants that reported current psychiatric medication use, we did not assess past medication use.

**Frontal Cortical Asymmetry in Response to a Stressor**

Contrary to our hypotheses, there was no change in frontal cortical asymmetry after the stressor, compared to before the stressor, despite self-reported increases in negative affect. Given that frontal cortical asymmetry is calculated a difference score, changes in frontal cortical asymmetry can reflect changes in either left-sided neural activity, right-sided activity, or both. Thus, we also examined changes at left and right frontal cortical sites independently. Contrary to our hypotheses, we found that there was a decrease in activity at right frontal sites following the stressor but, there was no change in neural activity in left frontal regions. Prevailing theories of frontal cortical asymmetry as an index of avoidance motivation (Coan & Allen, 2004; Reznik & Allen, 2018) would suggest that frontal cortical asymmetry decreases during stress, as neural activity in right frontal regions increases, reflecting increases in avoidance motivation and negative affect. Some prior studies have found evidence of these expected changes in laboratory stress or negative mood induction paradigms (Papousek et al., 2011, 2014; Zhang et al., 2018). Other studies have found that periods of greater stress in individual’s lives are associated with lower frontal cortical asymmetry (e.g., Lewis et al., 2007). However, other studies demonstrated contrary effects of stress on frontal cortical asymmetry. For example, Verona et al. (2009) found that laboratory stressors increased left frontal activity. Thus, the literature on the effects of stress on frontal cortical asymmetry remains mixed.
Notably, a number of studies have found that trait-like moderators can influence the effect that stress has on frontal cortical asymmetry. For example, Papousek et al. (2019) found that individuals with lower trait positive affect experience increases in right frontal activation following a laboratory psychosocial stressor, while individuals with higher trait positive affect experienced sustained levels of left frontal activation. Further, Pérez-Edgar et al. (2013) found that individuals with greater attentional bias towards threat experienced greater right frontal activation during stress. Thus, emotion-related individual differences can impact the effect that stressors have on frontal cortical asymmetry, which is consistent with the proposition that frontal cortical asymmetry is useful to assessing such individual differences (Reznik & Allen, 2018).

Beyond individual differences, methodological differences might also play a role in inconsistencies in literature. The laboratory paradigms used to invoke stress in studies of frontal cortical asymmetry vary widely and include psychosocial performance stressors (e.g., Papousek et al., 2019), physiological stressors meant to induce physical discomfort (e.g., Zhang et al., 2018), and a combination of the two (e.g., Verona et al., 2009). Further, other studies have used various emotional stimuli to evoke negative affect. These have included images (Harmon-Jones, 2007; Schöne et al., 2016), film clips (Lopez-Duran et al., 2012; Papousek et al., 2014), and audio recordings (Papousek et al., 2011). Given that frontal cortical asymmetry is sensitive to the cognitive processes occurring at the time it is recorded (Coan et al., 2006; Papousek & Schulter, 2004), it is reasonable to expect that different tasks would have different effects on frontal cortical asymmetry. Frontal cortical asymmetry is also sensitive to the specific emotions evoked by a task. In particular, anger has a special role in frontal cortical asymmetry research given its status
as a negative affective state with an approach-based motivational direction (Carver & Harmon-Jones, 2009). Numerous studies have demonstrated that anger is associated with greater left frontal activity and higher frontal cortical asymmetry scores (Harmon-Jones, 2003, 2007). Thus, the extent to which a particular task evokes anger, as opposed or in addition to, other negatively valanced emotions, can determine the direction of change in frontal cortical asymmetry that a task can invoke. Finally, the phase in the stress response in which EEG data are recorded (i.e., during anticipation of stress vs. during active experience of stress vs. recovering from stress) might also influence frontal cortical asymmetry. In a review of studies assessing EEG markers of stress reactivity, Vanhollebeke et al. (2022) note the considerable variability in timing of EEG recordings, which they attribute to the inconsistencies in results in the broader literature.

The current study sought to capture changes in frontal cortical asymmetry in response to a personally relevant stressor. Based on prior research indicating that recall of autobiographical memories (Labouvie-Vief et al., 2003), recall of associated sensory information (Lang, 1979), and verbal speech (Kirschbaum et al., 1993) can increase stress reactivity, we used a stressful event recall and speech task to elicit a stress response and associated negative affect. Given that neural activity in the EEG during speech preparation would conflate neural activity related to the affective response to the memory content with that of the cognitive task of preparing the speech and, given that recording EEG during the speech itself is precluded due to movement artifact, we only recorded EEG data immediately prior to and immediately following the stressor. Thus, the phase of the stress response to which our post-stressor EEG recording most closely corresponds is the beginning of recovery following the stressor. Although we anticipated a persistence
of negative affect in the minutes following the stressor (e.g., by way of rumination about
the recalled stressful event), it is possible that such an effect was attenuated, and
participants experienced reductions in avoidance motivation in the eight minutes
following the stressor, resulting in corresponding reductions in right frontal neural
activity.

**Frontal Cortical Asymmetry and Symptom Measures**

Contrary to our hypotheses, we found no associations between post-stressor
frontal cortical asymmetry and either PTSD or anxiety symptoms. However, consistent
with our hypotheses, there was a trend towards an association between greater post-
stressor frontal cortical asymmetry and fewer depressive symptoms. Much of the
literature on frontal cortical asymmetry has centered on its association with depressive
symptoms (Allen & Reznik, 2015; Coan & Allen, 2004) and most studies have found an
inverse relationship between the two (Thibodeau et al., 2006). Although we only found a
nonsignificant trend in support of this relationship, our results suggest that greater frontal
cortical asymmetry following a stressor is associated with fewer depressive symptoms.
We did not find evidence of such a relationship between frontal cortical asymmetry and
either PTSD or anxiety symptoms, which is a departure from some prior studies
(Mathersul et al., 2008; Meyer et al., 2015; Thibodeau et al., 2006), but consistent with
others that have similarly failed to find relationships between frontal cortical asymmetry
(Harrewijn et al., 2016; Lin et al., 2021; Metzger et al., 2004).

Further contrary to our hypotheses, analyses of alpha power at left and right
frontal regions separately revealed that lower neural activity in left frontal regions
following the stressor was associated with fewer PTSD symptoms. Conversely and
consistent with our hypotheses, there was a nonsignificant trend towards a relationship between lower left frontal activity post-stressor and greater anxiety symptoms. Although such nonsignificant trends should be interpreted with caution, it is notable that these associations were in opposing directions given that PTSD and anxiety disorders are highly co-morbid. The potential relationship between anxiety and less left frontal activity is consistent with most prior studies of anxiety (Thibodeau et al., 2006). The literature on PTSD and frontal cortical asymmetry is considerably more mixed (Meyer et al., 2015), and while some studies have found relationships between relatively greater right frontal activity and PTSD symptoms (e.g., Meyer et al., 2018; Rabe et al., 2006), others have not (Metzger et al., 2004).

As noted previously, methodological differences, such as stress provocation task and timing of EEG recording, should be considered when comparing the results of the current study and the broader literature. In addition, although depressive, PTSD, and anxiety symptoms are highly comorbid, and might be related to the same higher-order latent construct (Elhai et al., 2011; Krueger & Markon, 2006; Watson, 2005), the differences in the symptomatology of these disorders should also be considered. PTSD, in particular, is characterized by both avoidance-related symptoms (e.g., avoidance of trauma reminders, less interest in pleasurable activities) and approach-related symptoms (e.g., chronic feelings of anger, outward displays of aggression). Thus, the provocation of one symptom type over another would lead to competing expectations regarding the lateralization of frontal neural activity. It is possible that stressor used in the current study elicited anger in participants, and thus those who experienced less anger (i.e., less approach-motivation reflected by lower left frontal activity), also exhibited fewer PTSD
symptoms. Such conjectures should be made with caution however, and only to
demonstrate the need for further study to disentangle these contradictory results.

**Frontal Cortical Asymmetry and the Distress Psychopathology Latent Factor**

Contrary to our hypotheses, and the results of our analyses examining
independent symptom scores, we found a relationship between frontal cortical asymmetry
and distress psychopathology such that both greater mid frontal and lateral frontal cortical
asymmetries prior to the stressor were associated with greater distress psychopathology.
There were no associations between post-stressor frontal cortical asymmetry and the
distress psychopathology latent factor. These findings are a departure from much of the
previous literature, which, as noted previously, suggests that frontal cortical asymmetry is
associated with less psychopathology (Meyer et al., 2015; Thibodeau et al., 2006). Given
that anger is associated with greater frontal cortical asymmetry (Harmon-Jones, 2007;
Harmon-Jones & Gable, 2018), it is possible that these results reflect potentially high
levels of anger experienced by the participants in the current study prior to the stressor.
This explanation would be consistent with our findings that less left neural frontal
activity was associated with fewer PTSD symptoms. It is also possible that these results
are related to the symptom scales used to derive the distress psychopathology latent
factor. Notably, the Beck Anxiety Inventory (Beck et al., 1988) is primarily composed of
items assessing physiological symptoms of anxiety. Although such symptoms are
associated with the avoidant motivation directional typically characteristic of anxiety, the
way in which physiological anxious arousal is reflected in frontal cortical asymmetry
remains unclear. For example, Thoma et al. (2021) found that individuals with panic
disorder, which is also characterized by high levels of physiological symptoms, display
greater right than left frontal alpha power (i.e., greater frontal cortical asymmetry) compared to healthy controls. Thus, the extent to which frontal cortical asymmetry indexes the psychological vs. physiological concomitants of distress psychopathology remains to be clarified. It is imperative, however, given the small sample size and low achieved power, to interpret the current structural equation model results with caution. These results should be replicated in an independent, larger sample before conclusions regarding the relationship between frontal cortical asymmetry and distress psychopathology are reliably drawn.

**Implications**

The current study has several implications. First, although theories of frontal cortical asymmetry and high frequency heart rate variability posit that both reflect processes that play a role in distress psychopathology, the results of the current study suggest that they might not both be associated with the same aspects of distress psychopathology. Second, placed in the context of the broader literature, the results of the current study highlight the need to better understand the physiological effects of the stress paradigms used in psychophysiological research and the cognitive-affective processes that occur during and after those stressors. Third, our diverging results regarding frontal cortical asymmetry and independent alpha power at right and left frontal sites suggest that it might be useful to assess both types of markers of neural activity in future studies. Similarly, our diverging results regarding the distress psychopathology latent factor and the independent symptom scales suggest that it might be useful to assess both in future studies, particularly given that certain physiological markers might have different relationships with different symptom types.
Strengths

The current study had several strengths. First, it tested the relationships between distress psychopathology and two proposed physiological markers of emotion regulation/flexibility and approach-avoidance motivation, which have been widely studied independently but have remained underexamined together. Second, consistent with emerging models of psychopathology such as HiTOP and RDoC, which offer alternatives to the DSM-5, this study took a dimensional, transdiagnostic approach to assessing psychopathology and underlying psychophysiological processes. Finally, to increase ecological validity, we utilized a personally relevant stressor that might serve as a better proxy for the real-world stressors individuals with histories of trauma exposure might experience (e.g., reminders if stressful or traumatic experiences) compared to other laboratory paradigms.

Limitations

The current study also had several limitations. First, as noted above, although the stressor we used was designed to be personally relevant and reflective of stressful experiences individuals who experienced trauma are likely to encounter in their daily lives (i.e., reminders of past trauma or stressful experiences), it was not designed to elicit specific moods. Self-reported affect suggested that the task was effective in increasing negative affect and decreasing positive affect, but different individuals might have experienced different moods which might differentially impact frontal cortical asymmetry (e.g., anger vs. sadness). Second, we did not collect descriptive data on the stressor that participants recalled during the stressor. Factors such as the type of event they recalled or how long ago it was experienced might affect an individual’s stress
response. Third, also as noted above, we were only able to assess frontal cortical asymmetry immediately prior to and following the stressor, and it is possible that we captured recovery from the stressor rather than reactivity to it. Fourth, due to technical issues, we were unable to analyze heart rate variability data prior to and following the stressor, precluding us from comparing how the stressor affected heart rate variability and frontal cortical asymmetry. Fifth, although we instructed participants to refrain from consuming tobacco, drugs, alcohol, or caffeine for 2 hours prior to arriving to the lab for this study, we did not confirm compliance with these instructions upon participants’ arrival. Sixth, the current study did not employ a no-trauma control group to test whether the effects of the stressor on frontal cortical asymmetry were specific to individuals who experienced trauma. Seventh, although we took a dimensional approach to assessing psychopathology, only about 22% of our sample met likely criteria for an anxiety disorder and only about 24% met likely criteria for PTSD. Thus, it is likely that we did not capture the full range of symptom severity, nor did we have sufficient power to test whether the strength of the relationship between physiological markers and psychopathology increases as symptom severity increases. Eighth, although the loading of PTSD, anxiety, and depressive symptoms onto a distress latent factor has been reliably demonstrated (Elhai et al., 2011; Krueger & Markon, 2006; Watson, 2005), the current study employed three symptom scales that were developed independently and whose fit to a single latent factor has not been previously tested. Although the structural equation models in the current study fit the data well, this should be interpreted with caution given our small sample size. A different measure, which was designed to broadly assess distress psychopathology, such as the Inventory of Depression and Anxiety Symptoms – II
(Watson et al., 2012) might have been more appropriate to use for these purposes. Finally, although the current study sought to specifically focus on college students, given that young adulthood and time in college represent critical developmental windows, the generalizability of our results to the broader population might be limited.

**Future Directions**

The results of the current study suggest some future research directions. First, as noted above, more work is needed to clarify the possible diverging physiological effects of various laboratory stress paradigms. Second, the effects of specific, personally relevant mood inductions (e.g., recall and speech about an event that made an individual angry vs. sad, etc.) on frontal cortical asymmetry should be clarified and the relationships between psychopathology and frontal cortical asymmetry in response to various moods should be tested. Third, it might be valuable to conduct between-group analyses between individuals who experienced changes in frontal cortical asymmetry in response to a stressor and those who did not, or alternatively between those who experienced an increase in right frontal activity as opposed to an increase in left frontal activity. Finally, more work is needed to determine the contexts in which a greater negative response might be adaptive and the effects of such contexts on high frequency heart rate variability should be tested. These research directions can help determine which physiological processes, under which circumstances, can predict which forms of psychopathology. Particular attention should be paid to establishing whether certain psychophysiological processes are related to specific symptoms or higher order transdiagnostic latent factors. Clarifying these issues might contribute to our etiological understanding of distress
disorders and might help develop psychophysiological assessment tools that can aid with diagnosis and treatment.

**Conclusion**

The current study tested the relationships between distress psychopathology (assessed both as a transdiagnostic latent factor and as PTSD, anxiety, and depressive symptoms independently), baseline high frequency heart rate variability, and frontal cortical asymmetry in response to a personally relevant stressor among college students who experienced trauma. Our key findings included a positive relationship between HF-HRV and negative affect post-stressor, a decrease in right frontal neural activity following the stressor, and an inverse relationship between left frontal activity post-stressor and PTSD symptoms. Much of our results contradict those of prior studies and raise further questions regarding the use of these psychophysiological tools in understanding psychopathology.
Table 1. Participant demographics

<table>
<thead>
<tr>
<th>Participant Demographics</th>
<th>n (%)</th>
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</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>48 (40.7)</td>
</tr>
<tr>
<td>Female</td>
<td>70 (59.3)</td>
</tr>
<tr>
<td>Race/ethnicity</td>
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<tr>
<td>Asian/Pacific Islander</td>
<td>56 (47.5)</td>
</tr>
<tr>
<td>Black, non-Hispanic</td>
<td>15 (12.7)</td>
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<tr>
<td>Hispanic/ Latinx</td>
<td>23 (19.5)</td>
</tr>
<tr>
<td>White, non-Hispanic</td>
<td>14 (11.9)</td>
</tr>
<tr>
<td>Other</td>
<td>5 (4.2)</td>
</tr>
<tr>
<td>Multiple</td>
<td>5 (4.2)</td>
</tr>
<tr>
<td>Age, M (SD)</td>
<td>19.1 (2.84)</td>
</tr>
</tbody>
</table>
Table 2. Participants clinical characteristics

<table>
<thead>
<tr>
<th>Sample Clinical Characteristics</th>
<th>M (SD)</th>
<th>Range</th>
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<tbody>
<tr>
<td>Symptom Severity</td>
<td></td>
<td></td>
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<tr>
<td>PTSD (PCL-5)</td>
<td>20.51 (15.42)</td>
<td>0 – 59</td>
</tr>
<tr>
<td>Anxiety (BAI)</td>
<td>13.87 (9.67)</td>
<td>0 – 44</td>
</tr>
<tr>
<td>Depressive (CESD)</td>
<td>19.08 (10.26)</td>
<td>0 – 46</td>
</tr>
<tr>
<td>Number of Traumatic Events</td>
<td>2.84 (1.71)</td>
<td>1 – 10</td>
</tr>
<tr>
<td>Experiences (LEC-5)</td>
<td></td>
<td></td>
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<tr>
<td>Type of Trauma Experienced</td>
<td>n (%)</td>
<td></td>
</tr>
<tr>
<td>Natural Disaster</td>
<td>70 (59.3)</td>
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<tr>
<td>Fire or Explosion</td>
<td>12 (10.2)</td>
<td></td>
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<tr>
<td>Transportation Accident</td>
<td>52 (44.1)</td>
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<tr>
<td>Serious Accident</td>
<td>24 (20.3)</td>
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<tr>
<td>Exposure to Toxic Substance</td>
<td>5 (4.2)</td>
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<tr>
<td>Physical Assault</td>
<td>58 (49.2)</td>
<td></td>
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<tr>
<td>Assault with a Weapon</td>
<td>12 (10.2)</td>
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<tr>
<td>Sexual Assault</td>
<td>21 (17.8)</td>
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<tr>
<td>Other Unwanted Sexual Experience</td>
<td>41 (34.7)</td>
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<tr>
<td>Combat Exposure</td>
<td>1 (0.8)</td>
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<tr>
<td>Captivity</td>
<td>2 (1.7)</td>
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<tr>
<td>Life-threatening Illness or Injury</td>
<td>10 (8.5)</td>
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<tr>
<td>Severe Human Suffering</td>
<td>5 (4.2)</td>
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<tr>
<td>Sudden Violent Death</td>
<td>5 (4.2)</td>
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<tr>
<td>Sudden Accidental Death</td>
<td>8 (6.8)</td>
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</tr>
<tr>
<td>Causing a Serious Injury, Harm, or Death</td>
<td>6 (5.1)</td>
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Note: BAI = Beck Anxiety Inventory, CESD = Center for Epidemiological Studies Depression Scale, LECD-5 = Life Events Checklist – 5, PCL-5 = PTSD Symptom Checklist – 5
Table 3. Descriptive statistics of physiological variables

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<thead>
<tr>
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<th>M (SD)</th>
<th>Range</th>
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<tbody>
<tr>
<td>Baseline High Frequency Heart Rate Variability</td>
<td>6.476 (1.117)</td>
<td>3.81 – 9.07</td>
</tr>
<tr>
<td>Pre-stressor Mid Frontal Cortical Asymmetry</td>
<td>0.021 (0.110)</td>
<td>-0.25 – 0.23</td>
</tr>
<tr>
<td>Pre-stressor Lateral Frontal Cortical Asymmetry</td>
<td>0.045 (0.184)</td>
<td>-0.42 – 0.42</td>
</tr>
<tr>
<td>Post-stressor Mid Frontal Cortical Asymmetry</td>
<td>0.039 (0.105)</td>
<td>-0.25 – 0.32</td>
</tr>
<tr>
<td>Post-stressor Lateral Frontal Cortical Asymmetry</td>
<td>-0.008 (0.184)</td>
<td>-0.44 – 0.46</td>
</tr>
<tr>
<td>Pre-stressor Left Mid Frontal Alpha Power</td>
<td>1.032 (0.604)</td>
<td>-0.46 – 1.97</td>
</tr>
<tr>
<td>Pre-stressor Right Mid Frontal Alpha Power</td>
<td>1.042 (0.591)</td>
<td>-0.38 – 1.97</td>
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<tr>
<td>Pre-stressor Left Lateral Frontal Alpha Power</td>
<td>0.769 (0.658)</td>
<td>-0.76 – 2.42</td>
</tr>
<tr>
<td>Pre-stressor Right Lateral Frontal Alpha Power</td>
<td>0.737 (0.619)</td>
<td>-0.68 – 2.38</td>
</tr>
<tr>
<td>Post-stressor Left Mid Frontal Alpha Power</td>
<td>1.132 (0.553)</td>
<td>-0.28 – 2.02</td>
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<tr>
<td>Post-stressor Right Mid Frontal Alpha Power</td>
<td>1.194 (0.546)</td>
<td>-0.28 – 2.82</td>
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<tr>
<td>Post-stressor Left Lateral Frontal Alpha Power</td>
<td>0.841 (0.587)</td>
<td>-0.64 – 2.40</td>
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<tr>
<td>Post-stressor Right Lateral Frontal Alpha Power</td>
<td>0.822 (0.580)</td>
<td>-0.84 – 2.53</td>
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Table 4. Bivariate correlations between self-report variables

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<tr>
<td></td>
<td>.448***</td>
<td>.606***</td>
<td>.263**</td>
<td>.253**</td>
<td>-.063</td>
<td>-.034</td>
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| Note: *p ≤ 0.05, **p ≤ 0.01, ***p ≤ 0.001
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<tbody>
<tr>
<td>1. HF-HRV</td>
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<tr>
<td>2. Pre-Stressor Mid Frontal Cortical Asymmetry</td>
<td>.015</td>
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<tr>
<td>3. Pre-Stressor Lateral Frontal Cortical Asymmetry</td>
<td>.024</td>
<td>.768***</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Post-Stressor Mid Frontal Cortical Asymmetry</td>
<td>.030</td>
<td>.668***</td>
<td>.577***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Post-Stressor Lateral Frontal Cortical Asymmetry</td>
<td>&lt;.001</td>
<td>.512***</td>
<td>.748***</td>
<td>.587**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Pre-stressor Left Mid Frontal Alpha Power</td>
<td>.198</td>
<td>-.002</td>
<td>.011</td>
<td>.002</td>
<td>.066</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Pre-stressor Right Mid Frontal Alpha Power</td>
<td>.193</td>
<td>-.205</td>
<td>.147</td>
<td>.125</td>
<td>.135</td>
<td>.949**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Pre-stressor Left Lateral Frontal Alpha Power</td>
<td>.102</td>
<td>-.335**</td>
<td>-.300**</td>
<td>.023</td>
<td>-.101</td>
<td>.557***</td>
<td>.506***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Pre-stressor Right Lateral Frontal Alpha Power</td>
<td>.111</td>
<td>-.004</td>
<td>.079</td>
<td>.047</td>
<td>.215</td>
<td>.969***</td>
<td>.685***</td>
<td>.813***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Post-stressor Left Mid Frontal Alpha Power</td>
<td>.114</td>
<td>-.136</td>
<td>-.183</td>
<td>-.256*</td>
<td>-.186</td>
<td>.735**</td>
<td>.725***</td>
<td>.561***</td>
<td>.657***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Post-stressor Right Mid Frontal Alpha Power</td>
<td>.141</td>
<td>.094</td>
<td>.100</td>
<td>.129</td>
<td>.119</td>
<td>.814***</td>
<td>.836***</td>
<td>.710***</td>
<td>.827***</td>
<td>.774***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Post-stressor Left Lateral Frontal Alpha Power</td>
<td>.088</td>
<td>-.311**</td>
<td>-.353**</td>
<td>-.149</td>
<td>-.296**</td>
<td>.436***</td>
<td>.421***</td>
<td>.798***</td>
<td>.601***</td>
<td>.807***</td>
<td>.651***</td>
<td></td>
</tr>
<tr>
<td>13. Post-stressor Right Lateral Frontal Alpha Power</td>
<td>.086</td>
<td>-.025</td>
<td>.014</td>
<td>-.006</td>
<td>-.146</td>
<td>.569***</td>
<td>.583***</td>
<td>.773***</td>
<td>.892***</td>
<td>.795***</td>
<td>.834***</td>
<td>.819***</td>
</tr>
</tbody>
</table>

Note: *p ≤ 0.05, **p ≤ 0.01, ***p ≤ 0.001
Figure 1. The effect of the stressor on self-reported positive and negative affect.
Figure 2. The effect of the stressor on mid and lateral frontal cortical asymmetry
Figure 3. Structural equation model of high-frequency heart rate variability (HF-HRV) predicting distress psychopathology.
Figure 4. Structural equation model testing both the direct and indirect effects of pre-stressor mid frontal cortical asymmetry, as well as the direct effect of post-stressor mid frontal cortical asymmetry on distress psychopathology.
Figure 5. Structural equation model testing both the direct and indirect effects of pre-stressor lateral frontal cortical asymmetry, as well as the direct effect of post-stressor lateral frontal cortical asymmetry on distress psychopathology.
Appendix I. Study Self-Report Measures

PTSD Checklist (PCL-5)

Instructions: Below is a list of problems that people sometimes have in response to a very stressful experience. Please read each problem carefully and then circle one of the numbers to the right to indicate how much you have been bothered by that problem in the past month.

<table>
<thead>
<tr>
<th>In the past month, how much were you bothered by:</th>
<th>Not at all</th>
<th>A little bit</th>
<th>Moderately</th>
<th>Quite a bit</th>
<th>Extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Repeated, disturbing, and unwanted memories of the stressful experience?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>2. Repeated, disturbing dreams of the stressful experience?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3. Suddenly feeling or acting as if the stressful experience were actually happening again (as if you were actually back there reliving it)?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4. Feeling very upset when something reminded you of the stressful experience?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5. Having strong physical reactions when something reminded you of the stressful experience (for example, heart pounding, trouble breathing, sweating)?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6. Avoiding memories, thoughts, or feelings related to the stressful experience?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7. Avoiding external reminders of the stressful experience (for example, people, places, conversations, activities, objects, or situations)?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8. Trouble remembering important parts of the stressful experience?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9. Having strong negative beliefs about yourself, other people, or the world (for example, having thoughts such as: I am bad, there is something seriously wrong with me, no one can be trusted, the world is completely dangerous)?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10. Blaming yourself or someone else</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
for the stressful experience or what happened after it?

<table>
<thead>
<tr>
<th>Question</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>11. Having strong negative feelings such as fear, horror, anger, guilt, or shame?</td>
<td>0-4</td>
</tr>
<tr>
<td>12. Loss of interest in activities that you used to enjoy?</td>
<td>0-4</td>
</tr>
<tr>
<td>13. Feeling distant or cut off from other people?</td>
<td>0-4</td>
</tr>
<tr>
<td>14. Trouble experiencing positive feelings (for example, being unable to feel happiness or have loving feelings for people close to you)?</td>
<td>0-4</td>
</tr>
<tr>
<td>15. Irritable behavior, angry outbursts, or acting aggressively?</td>
<td>0-4</td>
</tr>
<tr>
<td>16. Taking too many risks or doing things that could cause you harm?</td>
<td>0-4</td>
</tr>
<tr>
<td>17. Being “superalert” or watchful or on guard?</td>
<td>0-4</td>
</tr>
<tr>
<td>18. Feeling jumpy or easily startled?</td>
<td>0-4</td>
</tr>
<tr>
<td>19. Having difficulty concentrating?</td>
<td>0-4</td>
</tr>
<tr>
<td>20. Trouble falling or staying asleep?</td>
<td>0-4</td>
</tr>
</tbody>
</table>
**Beck Anxiety Inventory (BAI)**

Instructions: Below is a list of common symptoms of anxiety. Please carefully read each item in the list. Indicate how much you have been bothered by that symptom during the past month, including today, by circling the number in the corresponding space in the column next to each symptom.

<table>
<thead>
<tr>
<th>1. Numbness or tingling</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Feeling hot</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3. Wobbliness in legs</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4. Unable to relax</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>5. Fear of worst happening</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6. Dizzy or lightheaded</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7. Heart pounding/ racing</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>8. Unsteady</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>9. Terrified or afraid</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>10. Nervous</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>11. Feeling of choking</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>12. Hands trembling</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>13. Shaky/ unsteady</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Fear of losing control</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>---</td>
<td>-----------------------</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>14.</td>
<td>Difficulty in breathing</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>15.</td>
<td>Fear of dying</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>16.</td>
<td>Scared</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>17.</td>
<td>Indigestion</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>18.</td>
<td>Faint/ lightheaded</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>19.</td>
<td>Face flushed</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>20.</td>
<td>Hot/ cold sweats</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>
Center for Epidemiologic Studies Depression Scale (CES-D)
Instructions: Below is a list of the ways you might have felt or behaved. Please tell me how often you have felt this way during the past week.

<table>
<thead>
<tr>
<th></th>
<th>Rarely or none of the time (less than 1 day)</th>
<th>Some or a little of the time (1-2 days)</th>
<th>Occasionally or a moderate amount of time (3-4 days)</th>
<th>Most or all of the time (5-7 days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I was bothered by things that usually don’t bother me.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2. I did not feel like eating; my appetite was poor.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3. I felt that I could not shake off the blues even with help from my family or friends.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4. I felt I was just as good as other people.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>5. I had trouble keeping my mind on what I was doing.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6. I felt depressed.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7. I felt that everything I did was an effort.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>8. I felt hopeful about the future.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>9. I thought my life had been a failure.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>10. I felt fearful.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>11. My sleep was restless.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>12. I was happy.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>13. I talked less than usual.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>14. I felt lonely.</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
15. People were unfriendly. | 0 | 1 | 2 | 3  
16. I enjoyed life.      | 0 | 1 | 2 | 3  
17. I had crying spells. | 0 | 1 | 2 | 3  
18. I felt sad.         | 0 | 1 | 2 | 3  
19. I felt that people dislike me. | 0 | 1 | 2 | 3  
20. I could not get “going.” | 0 | 1 | 2 | 3  


