The Predictive Value of Complex PTSD Symptoms on Resting High-Frequency Heart Rate Variability

Janey Elizabeth Dike

Thesis submitted to the faculty of the Virginia Polytechnic Institute and State University in partial fulfillment of the requirements for the degree of

Master of Science
In
Psychology

Russell T. Jones
Bruce H. Scarpa-Friedman
Julie C. Dunsmore

December 9, 2020
Blacksburg, Virginia

Keywords: Interpersonal violence, trauma, heart rate variability

The Predictive Value of Complex PTSD Symptoms on Resting High-Frequency Heart Rate Variability

Janey Elizabeth Dike

ABSTRACT

Although the negative consequences of traumatic exposure across various domains of functioning have been well-documented, gaps and discrepancies continue to exist in the understanding of the impact of complex trauma, such as interpersonal violence (IPV), and how outcomes may vary across diverse populations and identities. In this cross-sectional study investigating the impact of traumatic exposure on physiological domains of functioning, a sample of female-identifying college students completed a number of self-reported measures (assessing past and present trauma exposure, complex posttraumatic stress disorder [CPTSD] symptoms, racial-ethnic minority status, and age of onset of first traumatic exposure) and provided resting high-frequency heart rate variability (hfHRV) data, which served as a biomarker for the potential impact of trauma exposure on physiological domains. Correlational and multiple regression analyses were conducted to determine the strength of relationships between variables and the predictive value of the models. Results indicated endorsement of IPV trauma was significantly associated with earlier age of onset, more severe levels of CPTSD symptoms, and higher hfHRV, but not racial-ethnic minority status. Racial-ethnic minority status was significantly related to more severe CPTSD symptoms. Type of trauma exposure was the only variable that emerged as having predictive value for changes in hfHRV. These findings suggest that experiencing IPV may have unique implications for trauma symptomatology and functioning above and beyond other forms of traumatic exposure, but that continued research must be conducted in order to draw more robust conclusions about the effects of exposure on physiological regulation across various racial-ethnic identities.

The Predictive Value of Complex PTSD Symptoms on Resting High-Frequency Heart Rate Variability

Janey Elizabeth Dike

GENERAL AUDIENCE ABSTRACT

Research has highlighted the consequences that extremely negative, stressful experiences, also called traumatic events, can have on the way humans think, emote, behave, and physically react. It can be more difficult to draw conclusions about the effects of interpersonal violence (IPV), or violence that occurs at the hands of another (i.e., family, partner, or community violence), due to the complex, severe, and long-term nature of symptoms that survivors experience. There is also limited research about what complex trauma looks like across diverse populations. This study aimed to investigate the impact of traumatic exposure on physiology, which falls under biology and broadly includes the functions of living things. A sample of female-identifying college students completed a number of self-reported measures (assessing trauma exposure, complex posttraumatic stress disorder [CPTSD] symptoms, racial-ethnic minority status, and age of onset of first traumatic exposure) and provided resting high-frequency heart rate variability (hfHRV) data, which measures variation in time between beats and served as a measure for the potential impact of trauma exposure on physiology. Results suggested that IPV exposure was associated with earlier age of first traumatic experience, more severe levels of CPTSD symptoms, and higher hfHRV. Racial-minority status was significantly related to more severe CPTSD symptoms. Type of trauma exposure significantly predicted changes in hfHRV. Findings suggest that experiencing IPV may uniquely influence trauma symptoms and functioning above and beyond other forms of traumatic exposure. Continued research will allow for stronger conclusions about the effects of traumatic exposure on physiology across various racial-ethnic identities.

Table of Contents

List of Figures

List of Tables

Introduction

For years, research has highlighted the high rates of interpersonal violence and sexual assault against women, and the #MeToo movement has brought widespread public and media attention to the negative effects that survivors endure (Basile et al., 2011; Mendes et al., 2018; Smith et al., 2018; Tjaden & Thoeness, 2000). Research has demonstrated that the costs of interpersonal violence are high, both for individuals and society; it is estimated that the economic burden of rape alone across the United States surpasses 3 trillion dollars, with the majority of these expenses withdrawing from medical, government, and work productivity domains (Peterson et al., 2017). Despite public awareness of the prevalence and maladaptive consequences following trauma exposure, sexual assault rates among American women remain high, with research estimating that almost one in five women have been raped in their lifetime, over two out of five have experienced some form of sexual violence, and over half have been physically assaulted, either as a child or as an adult (Smith et al., 2018; Tjaden & Thoeness, 2000). Research has demonstrated that female college-aged students may be particularly at risk for experiencing sexual assault; studies suggest that approximately 38 percent of heterosexual females were between the ages of 18 and 24 when they first experienced rape or attempted rape (Smith et al., 2018; Walters et al., 2011). The Department of Justice reports that female college students are three times more likely than the general population to experience sexual violence (Sinozich & Langton, 2014). An abundance of research has demonstrated that the consequences of traumatic experiences do not stop following exposure; survivors face complex and variable challenges across cognitive, emotional, social, and physiological domains and are often at risk for chronic, long-term consequences, such as revictimization and severe symptomatology. In an effort to build upon existing research, this empirical study serves to analyze the relationships between trauma symptomatology and physiology among trauma-exposed participants. It also investigates the influence of traumatic exposure type by studying the impact of how experiences of interpersonal violence may lead to lasting effects across domains of functioning.

Literature Review

Interpersonal Violence and Complex Posttraumatic Stress Disorder Symptoms

Interpersonal violence (IPV), which includes sexual, physical, and domestic violence, both in adulthood and childhood, varies from single-incident (i.e., car crash) and non-interpersonal traumas, both in the nature of its frequency and severity of outcomes (Elliott, 2003). Specifically, IPV is more strongly associated with severe and complex trauma symptomatology than single-incident and non-interpersonal traumas (Ford et al., 2006). A history of childhood IPV is related to both higher levels of cumulative trauma experiences in adulthood and a presentation of trauma symptoms that are severe and complex (Cloitre et al., 2009; Scott, 2007). The link between childhood abuse and impairment in adulthood is illustrated through the increased risk of revictimization, suicidal tendencies, substance use, and other mental health issues, such as depression (Classen et al., 2005; Felitti et al., 2019).

The literature points to a number of severe and maladaptive consequences that arise following exposure to IPV. On an individual level, survivors of interpersonal violence are susceptible to developing posttraumatic stress disorder (PTSD) symptoms following trauma (DePierro et al., 2013). Individuals can experience a range of symptoms and reactions to traumatic events, which are captured in the heterogeneous nature of PTSD symptoms (Bonanno & Mancini, 2012). The range of symptoms that can be experienced has led researchers to suggest that trauma symptoms, like traumatic experiences, exist on a spectrum, with complex presentations emerging that may not be fully represented by quintessential diagnostic trauma criteria (Hegadoren et al., 2006). Symptoms found to be more indicative of a complex PTSD (CPTSD) presentation, and that have been tied to experiences of IPV, include broad categories of affect dysregulation, problems related to negative self-concept, and interpersonal issues (Cloitre et al., 2013; Palic et al., 2016).

Additionally, the cognitive model of trauma suggests that those who develop PTSD symptoms tend to misappraise stimuli or situations as threatening and have difficulty distinguishing between threat and safety cues (Ehlers & Clark, 2000; Wicking et al., 2016). This threat misappraisal is associated with many of the quintessential PTSD symptoms observed in survivors of trauma, such as hyperarousal, reexperiencing, and avoidance of triggering stimuli (American Psychiatric Association, 2013). Research has shown that the effects of these

maladaptive cognitive and emotional stress symptoms can lead to serious health consequences; exposure to interpersonal violence has been associated with long-term physical health consequences, such as chronic pain, gastrointestinal problems, and cardiovascular and autoimmune disease (Breiding et al., 2008; Dichter et al., 2011; Dube et al., 2008; Stöckl & Penhale, 2015; Suglia et al., 2015). Similarly, populations who experienced maltreatment and sexual abuse in childhood have been found to have elevated rates of heart disease and cancer (Felitti et al., 2019). In order to gain more robust knowledge about the short and long-term sequelae following trauma exposure, it is crucial to examine how individuals respond to traumatic experiences and trauma-related stimuli across cognitive, emotional, behavioral, and physiological domains. In sum, trauma not only affects mental health, but physical health as well.

Trauma and Physiology

As evidenced by PTSD criteria in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013), alterations in physiology and arousal are classic features of posttraumatic stress.

The Generalized Unsafety Theory of Stress. The Generalized Unsafety Theory of Stress (GUTS), which focuses on the relationship between physiology and chronic stress, posits that the experience of chronic stress is due to a generalized perception of unsafety. Perceptions of unsafety can affect responses and cause dysregulation on psychological and physiological levels. These dysregulated reactions to prolonged threat can then lead to impairments in the ability to learn safety cues (Brosschot et al., 2018). In the context of PTSD, this perception of unsafety reflects the tendency to generalize perceived fears across broad categories of stimuli and to avoid internal or external stimuli that lead to reminders of the event (i.e., traumatic experience involving sexual abuse leading to avoidance of all interpersonal physical touch; traumatic experience involving choking leading to avoidance of all activities resulting in shortness of breath). The GUTS model helps to explain how exposure to stressors can have ongoing effects, cognitively, emotionally, behaviorally, and physiologically, that persist long after the actual stressor or traumatic experience has ended.

Hypothalamic Pituitary Adrenal Axis and Heart Rate Variability. In line with the GUTS model and the evidence supporting both psychological and physiological changes in

response to long-term exposure to threat or perceived threat, it is important to look at how stress affects the body's regulatory systems. The body's stress responses are mediated by the endocrine system via the hypothalamic pituitary adrenal (HPA) axis. The HPA axis functions to help the body respond to internal (i.e., digestion, immune system) and external (i.e., stressful stimuli) changes (Fries et al., 2009). HPA axis activity also contributes to the development of stress disorders, such as PTSD (Wessa et al., 2006). In line with the mind-body connection, the HPA axis is regulated by the hippocampus, amygdala, and prefrontal cortex (Fries et al., 2009). These regions of the brain are particularly relevant to trauma-exposed populations, as hyperreactivity and impairments have been observed in these regions in response to trauma exposure and trauma-related stimuli (Shin et al., 2006). The HPA axis is particularly relevant in that women's HPA axis activity has generally been found to be more sensitized to stressors than male counterparts (Meewisse et al., 2007).

As the HPA axis affects major organs and systems in the body when it is activated by stress, it may play a role in influencing cardiac activity (Stephens & Wand, 2012). Heart rate variability (HRV) serves as an index of vagal functioning and can be used as a non-invasive measure that assesses variations in beat-to-beat interval and can indicate stress-related regulation problems (Thayer et al., 2009; Tulppo et al., 1996; Schubert et al., 2009). The magnitude of variation can be analyzed within different bands of frequency (i.e., low frequency, high frequency, etc.). High-frequency HRV (hfHRV) generally falls between 0.15-0.40 Hz and can provide a measure of tonic cardiac vagal activity when measured during resting baseline (Berntson et al., 1993; Hill et al., 2017; Van der Kolk, 2003). This high-frequency band parameter provides an index of respiratory sinus arrhythmia (RSA; Akselrod et al., 1981; Shaffer & Ginsberg, 2017). Ideally, collecting five-minute resting period measurements can provide the most accurate assessment of cardiac vagal regulation, with a recommended minimum suggestion of one-minute recording time, for calculating high-frequency HRV frequency-domain values (Schaffer & Ginsberg, 2017).

High HRV has been found to be associated with elevated executive function performance, more effective regulation in emotional and physiological domains, enhanced social engagement, and higher overall measures of mental and physical health (Kemp & Quintana, 2013; McCraty & Shaffer, 2015; Thayer et al., 2009). In contrast, low hfHRV has been associated with an impaired ability to identify safety cues and physiological stress reactions to

non-stressful (neutral) stimuli, supporting the concept of generalized unsafety (Brosschot et al., 2018; Van der Kolk, 2003). These stress reactions occur as low hfHRV is associated with the body's reduced abilities to effectively manage internal and environmental stressors (Kim et al., 2018). Lower resting HRV has been found in trauma-exposed populations who exhibit symptoms of chronic stress and PTSD (Van der Kolk, 2003). In IPV-exposed populations specifically, researchers pointed to a negative correlation between baseline HRV and PTSD severity, supporting the theory that lower HRV may be related to chronic dysregulation in parasympathetic activity, thus explaining some posttraumatic responses to stressors (Hauschildt et al., 2011). Therefore, because of the association between cardiac patterns and PTSD symptomatology and the potential long-term negative health outcomes of this relationship, it is important to examine the connection between trauma outcomes and hfHRV following trauma exposure.

Race, Stress, and Trauma

Racial identity plays an important role in one's vulnerability to exposure to traumatic experiences and to the development of posttraumatic stress symptoms. There has been evidence that African American, Afro-Caribbean, Latinx, and Indigenous populations in the United States tend to experience elevated rates of PTSD as compared to White populations, whereas Asian populations tend to experience lower rates of PTSD (Alegría et al., 2013; Kisely et al., 2017). Disparities in prevalence rates can occur for a variety of reasons, such as experiences of discrimination, more barriers to healthcare and mental health services, and a higher chance of experiencing chronic stressors. In fact, research has demonstrated that perceived discrimination is linked to higher rates of PTSD (Asnaani & Hall-Clark, 2017). In a study of Black, Asian, Latinx, and Indigenous individuals, approximately half of individuals described an incident of racial discrimination or harassment as invoking stress for at least a 2-month period, and about half of participants reported that the interaction significantly affected them (Carter & Forsyth, 2010). Even in traumatic events not associated with discrimination, the profile of PTSD in racial minorities has been found to include CPTSD symptomatology, such as shame, self-blame, dissociation, and extreme hyperarousal (Hall-Clark et al., 2016). This could be partially explained by the types of traumatic events that minority groups are more likely to experience, with African American and Afro-Caribbean people being more likely to experience or witness

interpersonal violence (Alegría et al., 2013), Black participants being more likely to experience sexual violence, Latino people experiencing higher rates of physical violence, Asian populations being more likely to experience organized forms of violence, and White respondents being more likely to report accidents or physical injuries with respect to other racial-ethnic groups (McLaughlin et al., 2019). Although nuances arise across racial-ethnic groups as to which types of exposure and symptoms they will experience, these collective findings suggest overall that racial-ethnic minorities in the United States are likely to experience additional types of traumatic or stressful events in contrast to their White counterparts, and that the symptoms they may experience following exposure are likely to be more complex, severe, and harmful across emotional and physical domains.

A review of studies on the accumulation of stressful experiences among racial-ethnic minorities suggests that exposure to chronic stressors leads to dysregulation of the HPA axis, which results in the body engaging in maladaptive coping mechanisms that can have negative effects over time, though the direction of this dysregulation is not always consistent (Berger & Sarnyai, 2015). More specifically, in one study assessing the physical toll of such stressors in African American undergraduate college students, self-reported perceived discrimination was inversely related to resting hfHRV (Hill et al., 2017). This finding aligns with the idea that ongoing or frequent exposure to stressors may reduce the body's ability to regulate and cope with perceived or actual threats. Even in research that did not overtly inquire about discriminatory experiences based on racial-ethnic identity, lower HRV has been found in minority samples. In one study, researchers concluded that while lower baseline hfHRV was related to aging in White participants, young African American participants demonstrated similarly low levels of hfHRV, indicating that their cardiac vagal functioning was operating at a less effective level than what would be expected given their ages (Choi et al., 2006). In another, all HRV frequency measures in a 24-hour period were found to be significantly lower in African American and Hispanic participants than White participants, and some parameters of lower HRV were significantly related to lower socioeconomic status (Lampert et al., 2005). Overall, these findings indicate that individuals who are members of racial-ethnic minority groups may be at higher risk for dysregulated stress response symptoms due to a number of intersecting factors, including, but not limited to, exposure risk, the experience of severe and complex symptomatology, discrimination, biological characteristics, and intersecting group membership

such as socioeconomic status. These factors may also leave racial-ethnic minorities more vulnerable to maladaptive outcomes across domains following exposure to traumatic events.

Age of Onset of Traumatic Experiences

Age of onset represents the age of an individual at the time of their first reported exposure to a potentially traumatic event. As discussed in the context of interpersonal violence, age of first traumatic exposure has been determined to be an important variable when considering outcomes related to trauma, such as complexity and chronicity of PTSD symptoms and revictimization. Robust research suggests that a significant number of individuals report their first traumatic event as occurring during childhood or adolescence, particularly when the traumatic exposure was sexual or interpersonal in nature (DePierro et al., 2013; McCutcheon et al., 2010; Schoedl et al., 2010). In one outpatient sample of participants who had experienced some form of violence, researchers found that exposure before the age of 12 was a significant predictor for more severe posttraumatic and depressive symptoms as an adult (Schoedl et al., 2010). In a longitudinal study focusing on women who endorsed trauma, traumatic experiences that were sexual in nature and earlier ages of onset were both associated with risk for the subsequent development of PTSD (McCutcheon et al., 2010). Regarding revictimization risk, researchers have also pointed to evidence that early age of onset is associated with more severe levels of child sexual abuse and of PTSD symptoms (Filipas & Ullman, 2006). Thus, earlier experiencing or witnessing of traumatic events, particularly those of an interpersonal nature (i.e., sexual or physical abuse) can lead to chronic and negative health and safety outcomes, such as revictimization and more severe symptomatology.

Racial-ethnic differences emerge related to exposure to childhood maltreatment and early age of first traumatic exposure, with Black and Hispanic populations being more likely to report traumatic events in childhood than other racial-ethnic groups (Roberts et al., 2011) and Hispanic and non-Hispanic Black adolescents reporting higher rates of polyvictimization (multiple types of traumatic experiences) as compared to non-Hispanic Whites (López et al., 2017). In line with the literature, research conducted in an African American sample demonstrated that those who had experienced traumatic experiences, particularly those that were interpersonal in nature, were more likely to exhibit more severe levels of depression and PTSD as adults (Dunn et al., 2017).

Earlier age of onset has also been linked to lower HRV, with one study finding that reported emotional abuse in childhood and a history of major depressive disorder among female participants predicted lower resting hfHRV in adulthood (Stone et al., 2018). Although age of onset is associated with several variables that are linked to low hfHRV, such as interpersonal traumatic exposure and race, there is little research on how age of onset may contribute to changes in hfHRV above and beyond other contributing factors.

Gaps in the Literature

A number of studies have investigated trauma exposure, and specifically sexual assault trauma, among female undergraduate students. Despite these strides in knowledge, and despite the strong connection between traumatic experiences and changes in physiological arousal and dysregulation, there is currently a dearth in the literature regarding the influence of complex posttraumatic stress symptoms on physiological outcomes such as hfHRV, and how these relationships may be maintained. Additionally, there are mixed findings within the existing research surrounding how physiological variables are influenced by the severity of posttraumatic symptoms or type of traumatic exposure; some researchers suggest that conflicting and null results may be a product resulting from the nature of chronically-stressed IPV populations and the tendency for downward regulation or "blunted" responses following prolonged exposure to stressors (DePierro et al., 2013; Rohleder et al., 2008; Wessa et al., 2006). The current study aimed to begin filling these gaps in the literature by exploring the relationships between both physiological and emotional domains in the context of interpersonal trauma exposure and complex posttraumatic stress symptoms.

Most current methodology in the trauma literature also focuses on a diagnostic approach to symptomatology over a Research Domain Criteria (RDoC) perspective. The RDoC research framework incorporates both behavioral and biological components of functioning across several domains and takes into account the heterogeneity of individual differences within disorders (National Institute of Mental Health, 2013). Inclusion criteria for studies often involves meeting for a PTSD diagnosis and group comparisons are often made between PTSD vs. non-PTSD samples rather than between samples who have experienced different types of trauma (i.e., interpersonal) or who have a spectrum of symptomatology experiencing, particularly in studies where physiological measures are observed in trauma-exposed populations (Freed & D'Andrea,

2015; Meewisse et al., 2007; Van der Kolk, 2003). The present investigation utilized the multi-level analytic approach of the RDoC framework and aimed to measure the severity of CPTSD symptoms across the sample, rather than assessing whether participants met DSM-5 diagnostic criteria. Rather than coding PTSD as present or absent and using this as a predictor variable, the study examined type of exposure as a variable (i.e., IPV, non-IPV, or control/no trauma exposure) to assess for the potential predictive value of traumatic exposure experiences. Additionally, although CPTSD symptoms have become more prevalent over the last decade in the trauma literature, there is still much to be learned in terms of the pervasiveness of complex trauma symptoms and how severe experiences of negative internal emotions (i.e., shame, blame, low self-worth), dissociation, and severe dysregulation in emotional and physical domains may play a role in stress physiology.

Last, there is a general scarcity of racial-ethnic representation and inclusive conclusions in psychological studies, including those focused on traumatic experiences. The current study aimed to take an intersectional approach by incorporating gender and racial-ethnic identities into the analysis and considering essential differences in what trauma presentations look like across groups, and why these differences may occur (i.e., individual, social, societal influences).

This study served to explore associations between complex posttraumatic stress symptoms, different types of traumatic exposure, self-identified race, and age of first traumatic experience, and examined how they are related to baseline hfHRV. This connection between CPTSD severity and physiology, in addition to considerations regarding individuals' experiences (i.e., gender; racial identity; age of onset) may lead to insights concerning long-term health outcomes and the maintenance and perpetuation of trauma-related symptoms in IPV-exposed populations and in those most vulnerable to exposure or the development of trauma symptomatology.

In summation, the specific aims of the current investigation were to:

1. Examine the relationships between the variables of interest, including CPTSD symptoms, racial minority status, type of traumatic exposure, age of onset of first traumatic exposure, and baseline hfHRV.
2. Investigate the predictive value of CPTSD symptoms, racial minority status, type of traumatic exposure, and age of onset of first traumatic exposure on baseline hfHRV.

Method

The study investigated the relationship between complex posttraumatic stress symptom severity and physiological outcomes, as measured through baseline hfHRV, across participants who have experienced IPV trauma, non-IPV trauma, or who reported no history of exposure to a potentially traumatic event. In order to determine identity-related and experience-related factors that may further contribute to physiological and cardiac outcomes, racial-ethnic minority status and age of onset of first traumatic experience (if applicable) was also investigated in relation to hfHRV. It should be noted that although data collection was completed prior to the development of the current study, all hypotheses were developed prior to data cleaning and analyses, and prior to knowledge of total available sample size.

Hypotheses

1. The relationships between IPV trauma exposure and CPTSD symptoms, racial minority status, age of onset, and baseline hfHRV will be stronger than among the no trauma and non-IPV trauma exposure groups.
2. CPTSD symptoms, racial minority status, and exposure type (no reported trauma; non-interpersonal violence trauma; IPV trauma) will all emerge as significantly and negatively predictive of baseline hfHRV based on existing literature surrounding the impact of chronic stress and trauma.
3. In participants who report at least one traumatic experience, it is predicted that age of onset of first traumatic experience will emerge as significantly predictive of hfHRV outcomes, with earlier age of onset predictive of lower hfHRV.

Of note, the current investigation was developed within a larger fear-learning study, entitled “POST 3,” which served as a collaborative project between the Department of Psychology’s Mind-Body and Stress and Coping Labs at Virginia Tech (Huskey, 2020). The project received approval from the Virginia Tech IRB. All participating research assistants completed online training related to human subjects and in-person, supervised training on all laboratory procedures. This study consisted of an online survey and in-laboratory data collection session wherein participants were observed during a safety learning paradigm. Several physiological measures were collected during the in-laboratory session, including

electrocardiogram, electromyogram, electrodermal, and respiration activity data. These data were collected during a baseline period as well as during three different phases: habituation, acquisition, and extinction. A number of neurocognitive measures were also collected following the fear-learning paradigm. For the purposes of this investigation, only select measures on the self-reported online survey and resting hfHRV data collected during the baseline period were analyzed.

Participants

A sample of 36 self-identifying female adults were recruited from Virginia Tech's undergraduate and graduate student population and completed both the online survey and in-laboratory portions of the POST 3 study. This sample size was determined sufficient for the included analyses through a priori calculations via G*Power, which assumes a sample size of at least 25 to achieve a medium effect size ($f^2 = 0.35$) and moderate power (1 - β error probability = .80) when implementing a linear multiple regression with four predictor variables (complex PTSD symptoms; exposure group; race; age of onset of first traumatic exposure; Faul et al., 2007).

Recruitment and Screening Procedure

Participants were recruited to participate in the online survey portion of the study via Virginia Tech's online SONA system and flyer distribution throughout the Virginia Tech campus. Following completion of the survey, participants who endorsed major medical and hearing impairments were excluded. This exclusion criterion was monitored using the Mind-Body Lab Health History Questionnaire (MBL-HHQ), which was administered via Qualtrics. Additional exclusion criteria included participant endorsement of serious medical issues, such as cardiorespiratory and cardiovascular conditions, a history of seizures, epilepsy, neurological conditions, or brain disorders. Cases were evaluated individually based on the responses to the MBL-HHQ prior to being recruited to the in-laboratory portion of data collection. Participants were recruited for the in-laboratory study from the sample of participants who completed the online survey in its entirety. For this investigation, and consistent with research aims, only female participants who completed the in-laboratory portion of the study were included in the analyses. Completion of the online survey was incentivized via a raffle for a gift card and

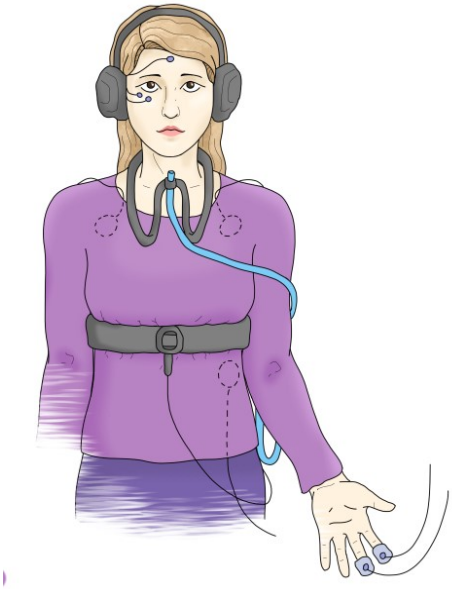
participants who completed the in-laboratory portion of the study were given the choice of SONA course extra-credit or monetary compensation for their participation.

In-Laboratory Procedure

Survey participants who met eligibility criteria were invited to complete the in-laboratory portion of data collection. The in-laboratory portion of the study took place in Williams Hall on the Virginia Tech campus and took approximately 2.5 hours to complete per participant. Survey participants who were invited to complete the in-laboratory portion of the study were met by a research assistant. After reviewing the informed consent form with the participant, the research assistant left the room to allow the participant to review and sign the form. Then, the participant read and signed the informed consent form. In order to control for various extraneous variables, each participant then completed a short survey assessing for recent health behaviors (Appendix E), such as intake of food, caffeine, alcohol, vigorous exercise, sleep data, and menstrual cycle information, and a research assistant recorded the participant height and weight. Participants then completed a hearing test (tones ranged from 12-22 kHz) and a color-blindness test at the computer monitor. Next, researchers attached electrodes to the participants for electromyogram, electrocardiogram, and electrodermal activity measures and fit participants with a respiration belt. The participants were given headphones to wear and the air blast harness was also fitted over their head. For the purposes of this investigation, only the electrocardiogram and respiration belt (Figure 1.) were used to capture the physiological variables required for analyses, but these various devices for data collection are described in order to best convey the participants' environment for the baseline period, which occurred after the attachment procedures. To capture baseline measurements, including hfHRV, participants were asked to sit in the computer chair in the study room. Research assistants left the room during the baseline period and turned off the light switch. Participants were asked to sit still for five minutes in the darkened study room in order to collect resting hfHRV and respiratory data. Participants were monitored by research assistants through a two-way mirror and adjustments were made to the electrode attachments and respiration belt as necessary.

Figure 1

Physiological Data Collection Methods



Measures

Complex PTSD Symptoms

Complex PTSD symptoms were assessed through the Self-Report Inventory for Disorders of Extreme Stress, which was included in the online survey portion of the study (SIDES-SR; Van der Kolk, 1996). This measure is a 45-item instrument that assesses symptoms across six dimensions, including disorders of affect regulation, amnesia and dissociation, somatization, disruptions in self-perception, disorders in relationships with others, and disrupted systems of meaning. The SIDES-SR is designed to capture symptoms and functional impairment that have been found to be related to complex forms of trauma exposure (i.e., chronic, early onset, interpersonal) and life adversity. The SIDES-SR was utilized by capturing overall, composite scores for each participant. Total scores across all six subscales can range from 0-135 points, as each item is scored on a scale of 0-3. This composite score is recommended for the self-reported version of the SIDES in order to capture a measure of overall symptom severity (Spinazzola, 2019). In a sample of participants recruited from outpatient clinics and community settings, the measure demonstrated good inter-rater reliability (kappa coefficient = 0.81), good internal consistency ($\alpha = 0.53$ to 0.96; Pelcovitz et al., 1997), and good construct validity (Zlotnick & Pearlstein, 1997).

DSM-5 PTSD Symptoms

A measure assessing quintessential, standardized PTSD symptomatology was included in analyses for standardization purposes and to test for multicollinearity with the SIDES-SR. The Posttraumatic Stress Disorder Checklist for DSM-5 (PCL-5; Weathers et al., 2013) contains 20 self-report items that assess for symptoms that are reflective of DSM-5 criteria for a PTSD diagnosis. Researchers examined the measure's psychometric properties in two studies with undergraduate student samples: in the first study, the PCL-5 demonstrated good internal consistency ($\alpha = 0.94$) and test-retest reliability ($r = 0.82$); the second study also demonstrated the PCL-5 had good internal consistency ($\alpha = 0.95$) and validity (Blevins et al., 2015).

Trauma Exposure

The Trauma History Screen (THS), which was included in the online survey, was used to assess for a history of potentially traumatic events. The Trauma History Screen is a 14-item self-report measure that screens for a variety of potentially traumatic events (e.g., car accident, natural disaster, physical attack; Carlson et al., 2005). The THS was shown to have high test-retest reliability ($r = .87$) in a sample of female university students and good convergent validity ($r = .73$) in young adults (Carlson et al., 2011). Kappa values were high for this measure in terms of categorization of traumatic experiences, including the category for interpersonal violence (Carlson et al., 2011). Previous research has used this measure to parse out experiences of interpersonal violence from other types of traumatic events (Carlson et al., 2013).

Age of First Exposure

Age of onset of first traumatic experience was also assessed using the THS. In addition to the 14-item screener for various potentially traumatic experiences, the THS asks for additional information for each positive endorsement, including the age the respondent was when the traumatic event occurred (i.e., "Your age when this happened:____").

Race

Participant race and ethnicity was captured on the online survey and respondents were encouraged to check off which race(s) or ethnic group(s) they identified with (i.e., Native American or Alaska Native; Asian; Black or African American; Native Hawaiian or Pacific Islander; White; Hispanic/Latino(a); I choose not to answer; Other [please specify]). Participants were able to check off multiple racial-ethnic categories with which they identified.

Resting High-Frequency Heart Rate Variability (hfHRV)

To measure baseline, or resting, hfHRV, an electrocardiogram (ECG) was used through the BIOPAC and Acqknowledge systems (gain set to 2000 with a maximum bandwidth of .05 to 150 Hz). Electrodes were attached to participants below their left and right collarbones and below their left rib cage. A respiration belt was also fitted around the participants' torsos, near the bottom of their sternums (Figure 1). This respiration data are typically utilized to assess for any influence on hfHRV values. Resting measures of HRV were collected during a five-minute baseline period that occurred towards the beginning of the in-laboratory session. HfHRV falls on a frequency spectrum (0.15-0.40 Hz) and measures cardiac vagal control during rest. To calculate high-frequency HRV specifically, interbeat intervals (IBIs) were measured. IBIs represent the distance in milliseconds between successive peak-wave (R-wave) ranges. Spectral analysis, a statistical method used for time series data analysis, was enacted using a Fast-Fourier Transform (FFT) function in the Kubios HRV Standard software, Version 3.2.0, 2019 (Standard, 2018). FFT calculated the power ($ms^{2)}$, which is the signal energy, for the high-frequency band. The natural logarithm value was then derived for each participant from the FFT calculation; when hfHRV is analyzed using the natural logarithm (ln), it has been found to represent cardiac vagal activity (Berntson et al., 1993).

Of note, a disruption in the methodological procedures occurred in the collection of resting hfHRV data. Although the study was designed to collect resting hfHRV data over the course of a five-minute baseline, technical issues reportedly occurred, resulting in only one minute of baseline measures being recorded for approximately half of the included participants. As such, baseline hfHRV measures reflect resting data over the course of one minute for all participants included in the analysis. Although this served as a limitation regarding validity, the one-minute recording falls within the minimum recommendation for hfHRV data collection (Shaffer & Ginsberg, 2017).

Data Analysis

All analyses were conducted in SPSS version 22 software (IBM Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0). Descriptive statistics for all variables of interest were examined to determine normality of distributions and outliers. Skewness and kurtosis were examined, with all variables falling between acceptable values of -2.0 and 2.0. No significant multivariate outliers were identified using Mahalanobis Distance. Regarding missing

data, one RSA value was missing and one age of onset value was missing among those who reported experiencing a traumatic event. Little's Missing Completely at Random (MCAR) test for missing values was conducted with the two aforementioned variables in order to determine whether there were any patterns or relationships between the missing data (Little, 1988). This test was not significant, indicating that the missing data were random (chi-square = 3.179; p = 0.204). As such, these missing data were not included in analyses.

Two variables were dummy-coded for analysis. Racial minority status was coded into dichotomous variables, with 0 representing participants who identified as White and 1 representing participants who identified as a racial-ethnic minority (i.e., endorsed at least one non-White identity). Due to a small, largely homogenous sample, all racial-ethnic minority participants (n=11) were included together in one group for analyses. Out of the 11 participants who reported racial-ethnic minority statuses, six identified as "Asian," one identified as "Hispanic/Latino(a)," two identified as "Asian, White," one identified as "Asian, White, Hispanic/Latino(a)," and one identified as "Other (not specified)".

The trauma group variable was also dummy-coded, with 0 representing no endorsement of traumatic experiences, 1 representing endorsement of only a non-IPV experience(s), and 2 representing endorsement of at least one IPV experience. Based on literature surrounding IPV and complex PTSD, the trauma groups were ordered such that higher values are likely indicative of more complex trauma experiences (Cloitre et al., 2009; Elliott, 2003; Ford et al., 2006). The three types of trauma experiences were synthesized into one variable rather than three separate dummy-coded variables due to limited sample size. As such, the trauma group variable will be interpreted as a synthesized, overall predictor rather than comparing how three distinct trauma groups may compare in their predictive value. Although a larger sample size is necessary for a robust comparison test, a univariate analysis of variance was conducted using Tukey's test (Tukey, 1977) in order to gauge differences between the trauma groups prior to the dummy-coded variable being included in the regression model. Although results indicated that there were no statistically significant differences between the three groups, the mean differences between the no trauma and IPV trauma groups approached significance (p = 0.069). It is anticipated that with a larger sample size in this multiple group comparison, significant differences would emerge between groups, particularly between the no trauma and IPV trauma groups, and thus,

the dummy-coded trauma group variable was still included in the correlation and regression analysis.

Correlational statistics were examined to determine significant relationships among variables. The variables of interest for the regression analyses (hfHRV, racial identity, complex PTSD symptoms, trauma group, age of onset) were included in correlation analyses. Of note, the age of onset variable correlations were only computed with the participants who endorsed a traumatic experience. In addition to these constructs, additional variables were included in the correlation computations for exploratory purposes. In order to examine how distinct trauma groups may be related to other variables in the analyses, three separate dummy coded variables representing each trauma group were also analyzed for correlational purposes only (i.e., No Trauma [0 = not in this group; 1 = part of this group]; Non-IPV Trauma [0 = not in this group; 1 = part of this group], IPV Trauma [0 = not in this group; 1 = part of this group]). A composite score variable for the PCL-5 was also included in the correlation table, as it reflects a measure of standardized posttraumatic stress disorder symptoms that align with the DSM-5.

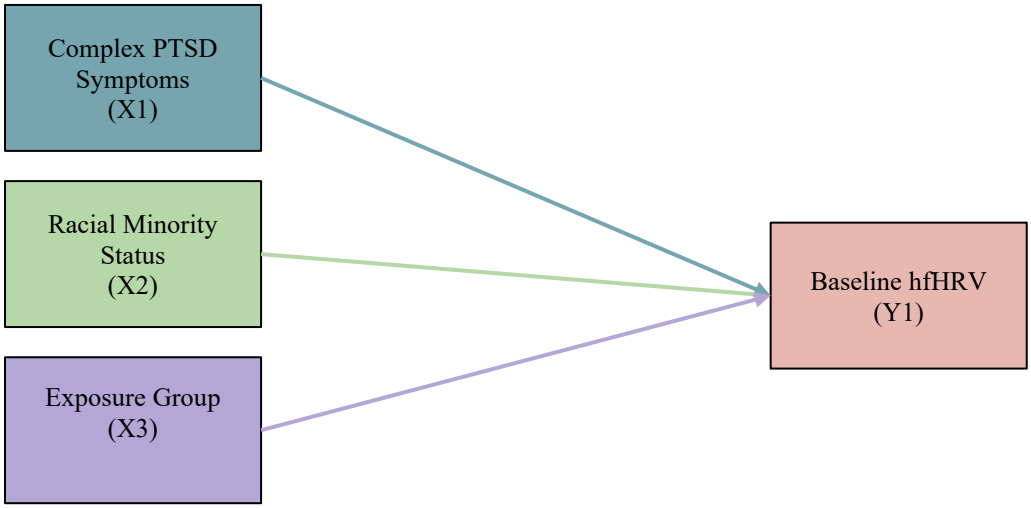
Presence of multicollinearity between the SIDES-SR and PCL-5 was examined in order to determine if one measure of PTSD symptoms should be included in the regression analyses over the other (i.e., if multicollinearity existed, the more standardized and widely used PCL-5 would be used in analyses over the SIDES-SR). Generally, a correlation coefficient greater than 0.8 between two independent variables suggests the presence of significant multicollinearity (Berry et al., 1985; Vatcheva, 2016). No significant multicollinearity was determined between these two variables, or any other variables included in the analyses ($r[35] = 0.717$, $p < .01$). As such, the SIDES-SR was included in the regression model in order to capture more complex trauma symptomatology.

A series of multiple linear regression analyses were conducted. For the first set of regression analyses, complex PTSD symptoms (composite SIDES-SR score), racial minority status, and trauma group were added as independent variables, with baseline hfHRV (represented by RSA) tested as the dependent variable. Data from all participants were utilized. Baseline hfHRV was regressed onto the independent variables in three different blocks using the Enter method. This method computes the predictive values of each variable and the additional value that adding each variable individually to the model contributed. This analysis allowed for the determination of the unique contributions of each variable, above and beyond effects of those

entered prior. The regression analysis was then analyzed again with the inclusion of the same independent and dependent variables, but using the Stepwise method. This final method allowed the statistical software to make automatic decisions at every step about the inclusion of independent variables based on a minimum significance threshold, and to remove variables that became insignificant after adding additional independent variables that better accounted for the variance in the outcome variable. This highlights the unique contributions of each independent variable, above and beyond the effects of those entered prior. A model diagram for this set of regression analyses is illustrated below in Figure 2.

Figure 2

Regression Analyses Conducted with all Participants

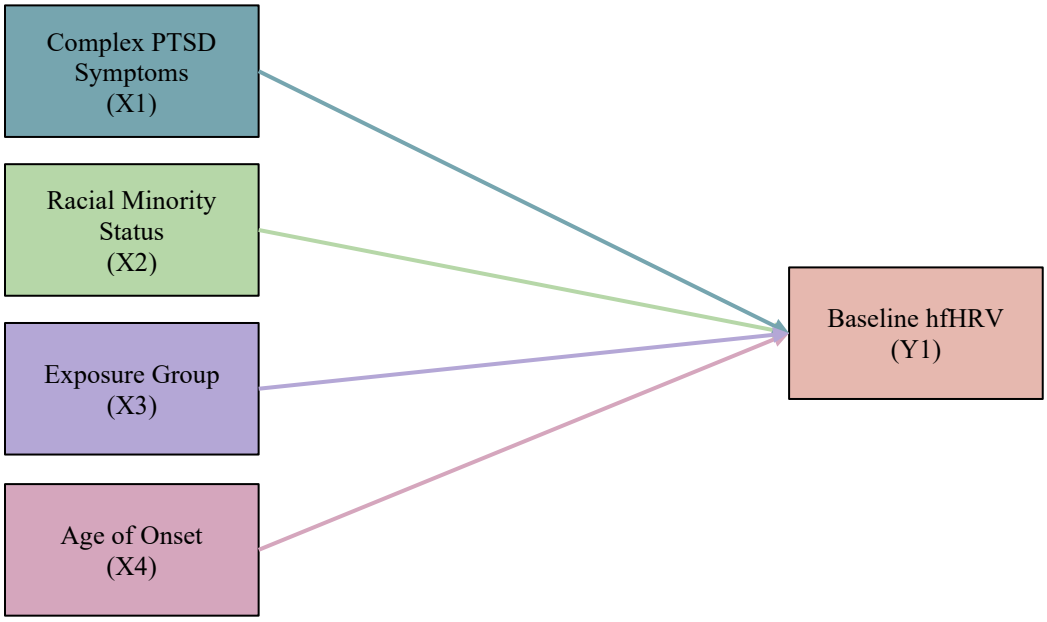


Note. Model equation: $\hat{Y}_1 = B_0 + B_1X_1 + B_2X_2 + B_3X_3$

The second set of regression analyses was conducted with only the participants who endorsed a traumatic experience. With this reduced dataset, a fourth variable, age of onset, was added to the analysis. This variable was added in order to analyze the predictive value that age of first traumatic experience may contribute to baseline hfHRV. This second set of analyses utilized the Enter method. A model diagram for this set of regression analyses is illustrated below in Figure 3.

Figure 3

Regression Analyses Conducted with all Trauma-Exposed Participants.



Note. Model equation: $\hat{Y}_1 = B_0 + B_1X_1 + B_2X_2 + B_3X_3 + B_4X_4$

Results

Descriptive Statistics

The total sample size included in the descriptive and correlational analyses was 36, with the exception of one piece of missing data each from the RSA and age of onset variables. Within this group of female-identifying participants, 25 (69.44%) identified as White and 11 (30.56%) identified as a racial-ethnic minority. The majority of participants reported that they were between 18-21 years of age (n = 33; 91.67%), with the three remaining participants reporting ages of 22 or 25 years (M age = 19.69; SD = 1.74). Nine (25.00%) participants did not endorse experiencing any lifetime trauma, 15 (41.67%) participants reported experiencing non-IPV trauma, and 12 (33.33%) participants reported having experienced at least one IPV trauma. Of participants who endorsed trauma exposure, 10 (38.46%) individuals reported age of onset as occurring between ages 3-12 years. The most condensed ages of onset occurred between ages 13-17 years, with 11 (42.31%) participants reporting their first traumatic experience during these teenage years. Only 5 (19.23%) participants reported age of onset as occurring in adulthood (18 years or older). The mean reported age of onset was 13.04 years (SD = 4.75). On the SIDES-SR measure of CPTSD symptoms, the minimum reported score was 0 and the maximum reported score was 55 (M = 15.81, SD = 12.97). HfHRV values ranged from 4.90-8.43 $\ln(ms^2)$ with a mean of 6.78 (SD = 0.94).

Descriptive statistics demonstrated similar presentations in the subset of participants who reported being exposed to potentially traumatic events: 19 (70.4%) identified as White and 8 (29.6%) identified as a racial-ethnic minority; ages ranged from 18-25 years old (M = 19.56; SD = 1.577); and the minimum reported SIDES-SR score was 1 and the maximum was 55 (M = 16.85; SD = 12.55). The average age of onset among the non-IPV exposed participants was 14.67 years (range: 8-20 years) and the average age of onset among the IPV exposed participants was 10.81 years (range: 3-19 years).

Pearson Correlation Analyses

Bivariate correlation coefficients were calculated across the variables of interest in order to assess for significant relationships. CPTSD symptoms, as represented by the composite score on the SIDES-SR, were significantly related to racial-ethnic minority status ($r[35] = 0.345$, $p < .05$). They were also significantly and negatively correlated with age of onset ($r[25] = -0.509$, $p < .01$).

Reported trauma-exposure was significantly associated with several constructs. When using the three-level dummy-coded trauma group variable in analysis, trauma group was significantly and negatively correlated with age of onset ($r[25] = -0.409$, $p < .05$) and positively correlated with hfHRV ($r[34] = 0.385$, $p < .05$). Correlation coefficients were also examined between three individually dummy-coded variables for each type of trauma exposure (no reported trauma, non-IPV exposure, and IPV trauma). Endorsing no trauma exposure was not significantly associated with any variables of interest. Endorsing non-IPV trauma was significantly and positively related to age of onset ($r[25] = 0.409$, $p < .05$). Last, endorsing IPV trauma was positively associated with hfHRV ($r[34] = 0.385$, $p < .05$) and CPTSD symptoms ($r[35] = 0.389$, $p < .05$), and negatively associated with age of onset ($r[25] = -0.409$, $p < .05$). Age of onset was significantly lower among the IPV trauma group ($M = 10.81$, $SD = 5.36$) than among the non-IPV trauma group ($M = 14.67$, $SD = 3.60$); $t(24) = -2.193$, $p = .038$.

First Set of Regression Analyses

The first regression analysis was conducted using the Enter method and hfHRV was regressed onto CPTSD symptoms, racial-ethic minority status, and trauma-exposure group level. Due to one piece of missing physiological data, 35 participants were included in the analysis. Because each proposed predictor variable was entered in three different blocks, thus adding each independent variable one at a time, three separate models were generated. In the first model, CPTSD symptoms explained 0.0% of the variance in an individual's hfHRV ($R^2 = 0.00$, adjusted $R^2 = -0.30$) and did not indicate a significant contribution to the variance ($\Delta R^2 = 0.00$, $p = .981$). In the second model, CPTSD symptoms and racial-ethnic minority status together explained approximately 8.5% of the variance in hfHRV ($R^2 = 0.085$, adjusted $R^2 = 0.028$). The addition of the racial-ethnic minority status variable in the model did not contribute to a significant change in the explained variance ($\Delta R^2 = 0.085$, $p = .095$). In the third and final model, CPTSD symptoms, racial-ethnic minority status, and trauma group explained an estimated 20.0% of the variance in hfHRV ($R^2 = 0.20$, adjusted $R^2 = 0.123$). The addition of the trauma group variable to the model explained an additional and significant 11.5% of the variance in hfHRV ($\Delta R^2 = 0.115$, $p < .05$).

Due to the low predictive value of multiple variables included in the regression model, but demonstration of significant predictive value in the trauma group variable, the same variables were analyzed via the Stepwise method in order for the statistical software to automatically

include or exclude independent variables based on a minimum significance threshold. At the first and only step of the model analysis, the trauma group variable was included and CPTSD symptoms and racial-ethnic minority status variables were excluded. Alone, the trauma group value accounted for approximately 14.8% of the variance in participant hfHRV ($R^2 = 0.148$, adjusted $R^2 = 0.123$, $\Delta R^2 = .148$, $p < .05$). Thus, the final Stepwise model included hfHRV regressed onto the reported trauma group variable.

Second Set of Regression Analyses

The second set of regression analyses were completed only with participants who endorsed a history of exposure to potentially traumatic events. Due to two pieces of missing data (one hfHRV value and one age of onset value), two participants who endorsed traumatic experiences were excluded from analysis and the total sample size from which results were derived was 25.

The regression utilized the Enter method in order to determine the unique predictive contribution of each of the variables of interest. In the first output model, CPTSD was tested as a predictor of variation in hfHRV. Results indicated that CPTSD symptoms explained approximately 0.4% of the variance in hfHRV ($R^2 = 0.004$, adjusted $R^2 = -0.040$) and did not indicate a significant contribution to the variance ($\Delta R^2 = 0.004$, $p = .9773$). The second model regressed hfHRV onto CPTSD symptoms and racial-ethnic minority status, and together, these variables explained approximately 5.8% of the variance in hfHRV ($R^2 = 0.058$, adjusted $R^2 = -0.028$). Adding racial-ethnic minority status into the model did not contribute to a significant change in the explained variance ($\Delta R^2 = 0.054$, $p = .272$). In the third model, CPTSD symptoms, racial-ethnic minority status, and trauma group together explained an estimated 11.4% of the variance in hfHRV ($R^2 = 0.114$, adjusted $R^2 = -0.012$). Adding the trauma group variable to the model did not result in a significant change in the predictive value of the model ($\Delta R^2 = 0.056$, $p = .262$). The fourth and final model tested the prior variables with the addition of age of onset. The collection of all four independent variables contributed to explaining approximately 12.7% of the variance in hfHRV ($R^2 = 0.127$, adjusted $R^2 = -0.048$). The addition of age of onset also did not significantly contribute to the estimated predicted value of the model ($\Delta R^2 = 0.013$, $p = .598$). Thus, no model included in this regression analysis emerged as significant and no variable served as a significant predictor for hfHRV in the trauma-exposed subset.

Discussion

This investigation examined the relationship between varying outcomes of potentially traumatic exposure and how individual identity and experiences may contribute to unique trauma sequelae in a sample of female college students. The study aimed to specifically investigate the way that exposure to chronic or interpersonal potentially traumatic events and the experience of complex symptomatology may have lasting impact on physiological domains and the body's ability to respond adaptively to both internal and external stressors.

Although participants were not recruited specifically based on endorsement of traumatic experiences, trauma exposure was evident in this general sample, with 3 out of 4 participants endorsing potentially traumatic events and 1 in 3 participants endorsing IPV experiences. In line with hypotheses, endorsement of IPV trauma was significantly associated with higher levels of CPTSD symptoms. Interestingly, of the highest 10 SIDES-SR composite scores, seven were endorsed by participants who reported IPV exposure. Neither inclusion in the no trauma group nor the non-IPV trauma group was significantly related to CPTSD symptoms. Endorsing IPV trauma was also significantly associated with an earlier age of onset and endorsing non-IPV trauma was associated with a later age of onset. These relationships may have emerged due to the nature of IPV, in that violence is enacted at the hands of a (typically) known or trusted other, and is often not a single-incident experience (i.e., the violence may occur repeatedly and could occur over a period of time; Elliott, 2003). As was the case with this sample, experiences of IPV are likely to occur during vulnerable development periods of childhood and adolescence, as well as in adulthood (Cloitre et al., 2009). Further, the average age of onset for participants who endorsed IPV was around age 10 years, which was approximately 4 years earlier than the average age of onset for those in the non-IPV group. Taken together, those who endorse experiences of IPV are likely to be at risk at an earlier age for developing severe and complex symptomatology, such as negative and complicated feelings of shame and self-blame, negative self and world perceptions, dissociation, and difficulties in social relationships. These symptoms can be particularly harmful, as previous research points to the pervasive nature of internalizing emotions, such as shame, that are related to feelings of worthlessness and powerlessness, social isolation, and maladaptive, self-blaming beliefs about the self and one's role in the context of the traumatic event (Dorahy et al., 2013; Freed & D'Andrea, 2015; La Bash & Papa, 2014).

In contrast with expectations, endorsement of IPV was not significantly related to racial-ethnic minority status. Based on existing literature, it was expected that racial-ethnic minorities would be more at risk for experiences of IPV than White participants, but no differences emerged between racial-ethnic groups and types of potentially traumatic experiences (Alegría et al., 2013; Kisely et al., 2017). The only variable that racial-ethnic minority status was significantly related to was CPTSD symptoms, with an endorsement of a racial-ethnic minority identity being correlated with higher levels of CPTSD symptoms. These findings may be indicative of several factors. One drawback in this sample of college students was a lack of representation across racial-ethnic groups. This homogeneity may be due to a number of variables, including, but not limited to, the predominantly White geographic community surrounding the university and the lack of equitable access to higher education in the United States across varying racial-ethnic backgrounds. As such, only 11 participants reported racial-ethnic minority identities. Of these participants, the majority (6) identified as Asian, one identified as Hispanic/Latino(a), three identified with multiple racial-ethnic identities, and one did not specify. No participants reported African American, Afro-Caribbean, or Indigenous identities. In the trauma literature, Asian individuals are less likely to develop PTSD following exposure to potentially traumatic events as compared to other racial-ethnic groups and may be more likely to experience organized or political forms of violence, as opposed to interpersonal, which may reflect the lack of an association with the IPV group (McLaughlin et al., 2019). Despite this, endorsement of racial-ethnic minority status in this study was significantly associated with more severe CPTSD symptoms. Although this relationship is in opposition to prevalence rates of PTSD in Asian populations, one possible explanation is that the SIDES-SR may be capturing symptoms that more closely align with the negative outcomes of experiencing discrimination and racism than quintessential diagnostic PTSD symptoms (Hall-Clark et al., 2016). Within this sample, caution should be exercised when drawing conclusions or providing explanations for results in order to avoid generalizing the experiences of various racial-ethnic minorities, especially as their experiences of trauma and discrimination in the United States may not be analogous. Although these participants were combined in analyses for statistical purposes, it should be noted that there are inherent nuances across individual, group, and cultural experiences of trauma and mental health. It is likely that this sample size is too small to make

valid conclusions about racial-ethnic minority experiences, especially for participants who identified as Hispanic/Latino(a) or who have multiple racial-ethnic identities.

Endorsement of IPV trauma was also related to higher levels of resting hfRHV via correlation analyses. Though significant, the direction of the relationship was inconsistent with expectations. In line with the GUTS model, it was expected that participants who had experienced IPV traumatic events would be more likely to experience stress that was chronic and complex, and that this prolonged exposure to stress would lead to cardiac vagal dysregulation (Kim et al., 2008; Van der Kolk, 2003). This dysregulation would be reflected in lower hfHRV, as the body would not be as adept to respond to stressors in the environment (Brosschot et al., 2018; Van der Kolk, 2003). Instead, the relationship between IPV trauma endorsement and hfHRV was positive, potentially indicating greater flexibility and adaptive functioning in response to stressors (Kemp & Quintana, 2013; McCraty & Shaffer, 2015; Thayer et al., 2009). Rationale for this relationship may be reflected in the experiences of the participants. Although IPV is related to chronic, ongoing stressors and survivors are often at risk for revictimization and more severe psychopathology, it may be that the participants in this study have not been exposed to repeated or long-term IPV, or that they possess protective and resiliency factors that have buffered against the effects of traumatic exposure. These protective factors could range from environmental factors, like social support and access to healthcare services, to background and identity, such as socioeconomic status, race, sexual identity, etc. Since all participants in the study were currently enrolled in a 4-year university or graduate program, it is likely that they have access to resources or sources of support that may not be present in a community sample or a sample recruited specifically from a low socioeconomic background. Also of note, due to recruitment via SONA, many of the participants were also psychology majors, which may be related to enhanced knowledge and reduced stigma about mental health services and understanding symptomatology. Any of these factors could also contribute to continued abilities to decipher between safety cues and dangerous stimuli, leading to continued adaptive stress responses. Higher hfHRV may also be more present among participants who reported IPV exposure as an adaptive response. Participants who reported IPV exposure experiences in the past may have catalyzed learning of more physiological and cognitive coping strategies, which are both associated with higher hfHRV (Thayer et al., 2009). Greater coping capacity resulting

from prior IPV may explain the results in this sample particularly if they are not experiencing ongoing internal or external stressors or threats.

This pattern was reflected in the regression analyses as well. In the regression model with all study participants included, the three-level dummy-coded trauma group variable emerged as the only independent variable with significant predictive value in explaining variations in hfHRV. Again, however, the standardized coefficient ($\beta = 0.385$) indicated that higher dummy-coded values, which theoretically reflected exposure to more complex traumatic events, predicted higher hfHRV. This may indicate that the participants who endorsed IPV experiences have not experienced chronic stress at a level that would result in downward regulation of hfHRV. In this case, a sample of participants who were recruited based on a history of IPV exposure (i.e., clinical sample, sample utilizing domestic violence resources or services, etc.) may better represent communities suffering from chronic stressors and threat. Or, it may be that experiencing IPV predicted higher hfHRV because potentially traumatic exposure led participants' regulatory systems to adapt in a way that would allow for more efficient responses to stressors.

Interestingly, CPTSD symptoms and racial-minority status did not emerge as significant predictors in the regression analyses with all participants. Although the SIDES-SR does include a subscale for somatization, these symptoms reflect more externally tangible or obvious indicators of health such as chronic pain, fainting, nausea, shortness of breath, etc., and may not be as useful for capturing the underlying mechanisms or consequences of stress on the body (i.e., inflammation, blood pressure, etc.; Van der Kolk, 1996). Relating to racial-ethnic minority status, much of the research with significant findings on low hfHRV and traumatic experiences has been conducted with Black/African American participants (Choi et al., 2006; Hill et al., 2017; Lampert et al., 2005). This investigation did not have any participants who reported this racial-ethnic identity. This suggests that Black/African American participants may be more susceptible to lower hfHRV than other racial-ethnic minorities or may be experiencing other factors (i.e., discrimination, historical trauma, barriers to healthcare) in a way that is specific to the Black community and that will not be equally captured in other minority groups.

CPTSD, racial-minority status, age of onset, and trauma exposure group did not serve as significant predictors of change in hfHRV in the subset of trauma-exposed participants. Small sample size, a relatively homogenous group of participants, and lack of a trauma-specific sample

likely contributed to a lack of significance. Additionally, it is most likely that there are other unknown variables that better account for changes in hfHRV in this sample.

Clinical Implications

Although the majority of variables in the analysis did not contribute predictive value on hfHRV, type of traumatic exposure, particularly endorsement of IPV, did present as an important variable both in terms of being a predictor of change in hfHRV and in its association with more severe CPTSD symptoms and earlier age of onset. These relationships demonstrate how interpersonal violence may have severe and lasting effects on people across various domains of functioning, including emotional, cognitive, social, and physiological, and that these outcomes may begin to develop at younger ages than what would be expected from other forms of traumatic exposure, though more research is necessary in order to draw more conclusive decisions about directionality of change. These findings align with previous research and suggest that mental health care providers should consider the widespread impact that traumatic experiences may have on clients and should focus on various domains of functioning, both in terms of treatment targets and in identifying maintenance of symptomatology. In line with addressing functional impairment across domains, more research focused on the impact of traumatic experiences on physiological domains and how it may affect dysregulation in the stress response system can help answer questions about how other posttraumatic stress symptoms are exacerbated and maintained. More robust and replicated findings in terms of how trauma variables may relate to physiological outcomes can influence the way that researchers and clinicians understand and treat maladaptive symptomatology following trauma exposure. Findings from this investigation may also underscore the importance of viewing trauma from a holistic functioning, severity, and spectrum-based approach (i.e., RDoC; National Institute of Mental Health, 2013), rather than from a diagnostic approach, as the nature of the trauma and severity of symptoms emerged as the most significant and interrelated variables in analyses.

Limitations and Future Directions

Several limitations arose throughout the current study. First and foremost, the investigation included a limited sample size. Although appropriate power with a moderate effect size was obtained in order to conduct regression analyses, several alterations to the variables were carried out in order to include the variables of interest. For example, the trauma exposure variable for the purposes of regression analyses was created using three-level dummy coding

since there was not enough power to test for differences between groups. Although higher numbers were intended to indicate more complex, interpersonal experiences of trauma, this is a loose interpretation since conceptually, experiences cannot actually be scaled against one another (i.e., one unit between no trauma and non-IPV does not equal the same unit of measurement between non-IPV trauma and IPV trauma). Since endorsement of traumatic experiences was not included in the recruitment strategy, the types of traumatic exposure were not equal across the three different levels.

Additionally, due to a largely homogenous sample and limited racial-ethnic diversity, all of the participants who endorsed racial-ethnic minority status were grouped together, so correlational results and predictive value coefficients should be interpreted with caution. There were too few endorsements of any one racial-ethnic group besides "White" with which valid analyses could be conducted. Importantly, there is compelling and evidence-based research that demonstrates racial-ethnic disparities in trauma exposure and outcomes, particularly health outcomes, but these disparities were not reflected in the outcomes of this investigation. Relatedly, although estimations can be made about the effect that experiences of discrimination and racism may have had on trauma-adjacent results in the study, no racial stress or perceptions of discrimination measures were specifically included in the study's survey measures.

Regarding methodology, the investigation was cross-sectional in nature. In regression analyses, longitudinal data are ideal in order to more accurately determine directional effects. As such, our knowledge about the predictive relationship between trauma exposure type and hfHRV is limited in the conclusions that can be drawn. Another methodological limitation arose during data collection. As mentioned above, a shorter duration of ECG measures was recorded during resting baseline than the planned 5-minutes. Although some evidence suggests that 1-minute of recordings is sufficient, more valid hfHRV measures may have been captured with a longer recording. With regard to analytic methods, this study did not control for respiration rate in the duration in which hfHRV data were sampled, although some evidence suggests that respiration rate is not related to hfHRV and is not always appropriate to use as a covariate during baseline (Denver et al., 2007).

Lastly, limitations were present in terms of data that was not collected or utilized in this investigation, but would be useful in addressing future questions related to trauma and PTSD. Although age of onset and type of exposure was examined, further analyses could incorporate

frequency and longevity of traumatic experiences and assess for polyvictimization (i.e., experiencing more than one type of traumatic event). These variables may contribute to experiences of chronic stress and generalized unsafety, particularly if they occur over long periods of time or across vulnerable developmental periods. Additionally, the study yielded limited results about the physiological impact that traumatic experiences may have had on participants. More in-depth studies may benefit from investigating additional physiological markers, such as startle response or cortisol awakening response, in a trauma-specific sample in order to gain a more comprehensive picture of regulatory stress response systems. In tandem, incorporation of general health markers (i.e., reports of heart and autoimmune disease onset and symptoms; measures of inflammation; blood pressure, etc.), particularly over time, can provide more robust data about how outcomes of traumatic exposure may translate to and impact physical domains of functioning. In line with the aims of the study, more intentional data collection surrounding racial and minority stress, both in the context of experiencing traumatic events and also in experiencing everyday discrimination and harassment, will serve to advance research on underrepresented groups and bridge the gap in mental and physical healthcare disparities.

References

Akselrod, S., Gordon, D., Ubel, F. A., Shannon, D. C., Berger, A. C., & Cohen, R. J. (1981). Power spectrum analysis of heart rate fluctuation: a quantitative probe of beat-to-beat cardiovascular control. *science*, *213*(4504), 220-222.

Alegría, M., Fortuna, L. R., Lin, J. Y., Norris, F. H., Gao, S., Takeuchi, D. T., Jackson, J. S., Shrout, P. E., & Valentine, A. (2013). Prevalence, risk, and correlates of posttraumatic stress disorder across ethnic and racial minority groups in the United States. *Medical care*, *51*(12), 1114–1123.

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.

Asnaani, A., & Hall-Clark, B. (2017). Recent developments in understanding ethnocultural and race differences in trauma exposure and PTSD. *Current Opinion in Psychology*, 14, 96-101.

Basile, K. C., Black, M. C., Breiding, M. J., Chen, J., Merrick, M. T., Smith, S. G., ... & Walters, M. L. (2011). National Intimate Partner and Sexual Violence Survey; 2010 Summary Report. Centers for Disease Control.

Berger, M., & Sarnyai, Z. (2015). "More than skin deep": stress neurobiology and mental health consequences of racial discrimination. *Stress*, 18(1), 1-10.

Berntson, G. G., Cacioppo, J. T., & Quigley, K. S. (1993). Respiratory sinus arrhythmia: autonomic origins, physiological mechanisms, and psychophysiological implications. *Psychophysiology*, 30(2), 183-196.

Berry, W. D., Feldman, S., & Stanley Feldman, D. (1985). *Multiple regression in practice* (No. 50). Sage.

Blevins, C. A., Weathers, F. W., Davis, M. T., Witte, T. K., & Domino, J. L. (2015). The posttraumatic stress disorder checklist for DSM-5 (PCL-5): Development and initial psychometric evaluation. *Journal of traumatic stress*, 28(6), 489-498.

Bonanno, G. A., & Mancini, A. D. (2012). Beyond resilience and PTSD: Mapping the heterogeneity of responses to potential trauma. *Psychological Trauma: Theory, Research, Practice, and Policy*, 4(1), 74.

Breiding, M. J., Black, M. C., & Ryan, G. W. (2008). Chronic disease and health risk behaviors associated with intimate partner violence—18 US states/territories, 2005. *Annals of Epidemiology*, 18(7), 538-544.

Brosschot, J. F., Verkuil, B., & Thayer, J. F. (2017). Exposed to events that never happen: Generalized unsafety, the default stress response, and prolonged autonomic activity. *Neuroscience & Biobehavioral Reviews*, 74, 287-296.Carlson, E. B., Garvert, D. W., Macia, K. S., Ruzek, J. I., & Burling, T. A. (2013). Traumatic stressor exposure and post-traumatic symptoms in homeless veterans. *Military Medicine*, 178(9), 970-973.

Carlson, E., Palmieri, P., Smith, S., Kimerling, R., Ruzek, J., & Burling, T. (2005). *The Trauma History Screen (THS)*.

Carlson, E. B., Smith, S. R., Palmieri, P. A., Dalenberg, C., Ruzek, J. I., Kimerling, R., ... & Spain, D. A. (2011). Development and validation of a brief self-report measure of trauma exposure: The Trauma History Screen. *Psychological Assessment*, 23(2), 463.

Carlson, E. B., Garvert, D. W., Macia, K. S., Ruzek, J. I., & Burling, T. A. (2013). Traumatic stressor exposure and post-traumatic symptoms in homeless veterans. *Military medicine*, 178(9), 970-973.

Carter, R. T., & Forsyth, J. (2010). Reactions to racial discrimination: Emotional stress and help-seeking behaviors. *Psychological Trauma: Theory, Research, Practice, and Policy*, 2(3), 183–191.

Choi, J. B., Hong, S., Nelesen, R., Bardwell, W. A., Natarajan, L., Schubert, C., & Dimsdale, J. E. (2006). Age and ethnicity differences in short-term heart-rate variability. *Psychosomatic medicine*, 68(3), 421-426.

Classen, C. C., Palesh, O. G., & Aggarwal, R. (2005). Sexual revictimization: A review of the empirical literature. *Trauma, Violence, & Abuse*, *6*(2), 103-129.

Cloitre, M., Stolbach, B. C., Herman, J. L., Kolk, B. V. D., Pynoos, R., Wang, J., & Petkova, E. (2009). A developmental approach to complex PTSD: Childhood and adult cumulative trauma as predictors of symptom complexity. *Journal of Traumatic Stress*, 22(5), 399-408.

Cloitre, M., Garvert, D. W., Brewin, C. R., Bryant, R. A., & Maercker, A. (2013). Evidence for proposed ICD-11 PTSD and complex PTSD: A latent profile analysis. *European Journal of Psychotraumatology*, *4*(1), 20706.

Denver, J. W., Reed, S. F., & Porges, S. W. (2007). Methodological issues in the quantification of respiratory sinus arrhythmia. *Biological psychology*, 74(2), 286-294.

DePierro, J., D'andrea, W., & Pole, N. (2013). Attention biases in female survivors of chronic interpersonal violence: relationship to trauma-related symptoms and physiology. *European journal of psychotraumatology*, *4*(1), 19135.

Dichter, M. E., Cerulli, C., & Bossarte, R. M. (2011). Intimate partner violence victimization among women veterans and associated heart health risks. *Women's Health Issues*, 21(4), S190-S194.

Dorahy, M. J., Corry, M., Shannon, M., Webb, K., McDermott, B., Ryan, M., & Dyer, K. F. (2013). Complex trauma and intimate relationships: The impact of shame, guilt and dissociation. *Journal of Affective Disorders*, 147(1-3), 72-79.

Dube, S. R., Fairweather, D., Pearson, W. S., Felitti, V. J., Anda, R. F., & Croft, J. B. (2009). Cumulative childhood stress and autoimmune diseases in adults. *Psychosomatic Medicine*, 71(2), 243.

Dunn, E. C., Nishimi, K., Powers, A., & Bradley, B. (2017). Is developmental timing of trauma exposure associated with depressive and post-traumatic stress disorder symptoms in adulthood?. *Journal of psychiatric research*, 84, 119–127.

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy*, 38(4), 319-345.

Elliott, L. (2003). Interpersonal violence. *Journal of General Internal Medicine*, 18(10), 871-872.

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, *39*, 175-191.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., ... & Marks, J. S. (2019). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, 56(6), 774-786.

Filipas, H. H., & Ullman, S. E. (2006). Child sexual abuse, coping responses, self-blame, posttraumatic stress disorder, and adult sexual revictimization. *Journal of Interpersonal Violence*, 21(5), 652-672.

Ford, J. D., Stockton, P., Kaltman, S., & Green, B. L. (2006). Disorders of extreme stress (DESNOS) symptoms are associated with type and severity of interpersonal trauma exposure in a sample of healthy young women. *Journal of Interpersonal Violence*, *21*(11), 1399-1416.

Freed, S., & D'Andrea, W. (2015). Autonomic arousal and emotion in victims of interpersonal violence: Shame proneness but not anxiety predicts vagal tone. *Journal of Trauma & Dissociation*, 16(4), 367-383.

Fries, E., Dettenborn, L., & Kirschbaum, C. (2009). The cortisol awakening response (CAR): facts and future directions. *International Journal of Psychophysiology*, 72(1), 67-73.

Hall-Clark, B., Sawyer, B., Golik, A., & Asnaani, A. (2016). Racial/ethnic differences in symptoms of posttraumatic stress disorder. *Current Psychiatry Reviews*, 12(2), 124-138.

Hauschildt, M., Peters, M. J., Moritz, S., & Jelinek, L. (2011). Heart rate variability in response to affective scenes in posttraumatic stress disorder. *Biological Psychology*, 88(2-3), 215-222.

Hegadoren, K. M., Lasiuk, G. C., & Coupland, N. J. (2006). Posttraumatic stress disorder part III: health effects of interpersonal violence among women. *Perspectives in Psychiatric Care*, 42(3), 163-173.

Hill, L. K., Hoggard, L. S., Richmond, A. S., Gray, D. L., Williams, D. P., & Thayer, J. F. (2017). Examining the association between perceived discrimination and heart rate variability in African Americans. *Cultural diversity & ethnic minority psychology*, 23(1), 5–14.

Huskey, A. M. (2020). *Computational and Human Learning Models of Generalized Unsafety* (Doctoral dissertation, Virginia Tech).

IBM Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp.

Kemp, A. H., & Quintana, D. S. (2013). The relationship between mental and physical health: insights from the study of heart rate variability. *International Journal of Psychophysiology*, 89(3), 288-296.

Kim, H. G., Cheon, E. J., Bai, D. S., Lee, Y. H., & Koo, B. H. (2018). Stress and Heart Rate Variability: A Meta-Analysis and Review of the Literature. *Psychiatry investigation*, *15*(3), 235–245.

Kisely, S., Alichniewicz, K. K., Black, E. B., Siskind, D., Spurling, G., & Toombs, M. (2017). The prevalence of depression and anxiety disorders in indigenous people of the Americas: A systematic review and meta-analysis. *Journal of Psychiatric Research*, *84*, 137-152.

La Bash, H., & Papa, A. (2014). Shame and PTSD symptoms. *Psychological Trauma: Theory, Research, Practice, and Policy*, 6(2), 159.

Lampert, R., Ickovics, J., Horwitz, R., & Lee, F. (2005). Depressed autonomic nervous system function in african americans and individuals of lower social class: a potential mechanism of race- and class-related disparities in health outcomes. *American Heart Journal*, 150(1), 153–160.

Liddell, B. J., Kemp, A. H., Steel, Z., Nickerson, A., Bryant, R. A., Tam, N., ... & Silove, D. (2016). Heart rate variability and the relationship between trauma exposure age, and psychopathology in a post-conflict setting. *BMC psychiatry*, 16(1), 133.

Little, R. J. (1988). A test of missing completely at random for multivariate data with missing values. *Journal of the American Statistical Association*, 83(404), 1198-1202.

López, C. M., Andrews III, A. R., Chisolm, A. M., de Arellano, M. A., Saunders, B., & Kilpatrick, D. (2017). Racial/ethnic differences in trauma exposure and mental health disorders in adolescents. *Cultural diversity and ethnic minority psychology*, 23(3), 382.

McCraty, R., & Shaffer, F. (2015). Heart rate variability: new perspectives on physiological mechanisms, assessment of self-regulatory capacity, and health risk. *Global advances in health and medicine*, 4(1), 46-61.

McCutcheon, V. V., Sartor, C. E., Pommer, N. E., Bucholz, K. K., Nelson, E. C., Madden, P. A., & Heath, A. C. (2010). Age at trauma exposure and PTSD risk in young adult women. *Journal of traumatic stress*, 23(6), 811–814.

McLaughlin, K. A., Alvarez, K., Fillbrunn, M., Green, J. G., Jackson, J. S., Kessler, R. C., ... & Alegría, M. (2019). Racial/ethnic variation in trauma-related psychopathology in the United States: a population-based study. *Psychological medicine*, 49(13), 2215-2226.

Meewisse, M. L., Reitsma, J. B., De Vries, G. J., Gersons, B. P., & Olff, M. (2007). Cortisol and post-traumatic stress disorder in adults: systematic review and meta-analysis. *The British Journal of Psychiatry*, 191(5), 387-392.

Mendes, K., Ringrose, J., & Keller, J. (2018). # MeToo and the promise and pitfalls of challenging rape culture through digital feminist activism. *European Journal of Women's Studies*, 25(2), 236-246.

National Institute of Mental Health (2013). *Research Domain Criteria (RDoC)*. Retrieved October 13, 2019, from https://www.nimh.nih.gov/research/research-funded-by-nimh/rdoc/index.shtml

Palic, S., Zerach, G., Shevlin, M., Zeligman, Z., Elklit, A., & Solomon, Z. (2016). Evidence of complex posttraumatic stress disorder (CPTSD) across populations with prolonged

trauma of varying interpersonal intensity and ages of exposure. *Psychiatry Research*, *246*, 692–699.

Pelcovitz, D., Van der Kolk, B., Roth, S., Mandel, F., Kaplan, S., & Resick, P. (1997). Development of a criteria set and a structured interview for disorders of extreme stress (SIDES). *Journal of traumatic stress*, *10*(1), 3-16.

Peterson, C., DeGue, S., Florence, C., & Lokey, C. N. (2017). Lifetime economic burden of rape among US adults. *American Journal of Preventive Medicine*, 52(6), 691-701.

Roberts, A. L., Gilman, S. E., Breslau, J., Breslau, N., & Koenen, K. C. (2011). Race/ethnic differences in exposure to traumatic events, development of post-traumatic stress disorder, and treatment-seeking for post-traumatic stress disorder in the United States. *Psychological medicine*, 41(1), 71–83.

Rohleder, N., Chen, E., Wolf, J. M., & Miller, G. E. (2008). The psychobiology of trait shame in young women: Extending the social self preservation theory. *Health Psychology*, 27(5), 523.

Schoedl, A. F., Costa, M. C. P., Mari, J. J., Mello, M. F., Tyrka, A. R., Carpenter, L. L., & Price, L. H. (2010). The clinical correlates of reported childhood sexual abuse: an association between age at trauma onset and severity of depression and PTSD in adults. *Journal of child sexual abuse*, 19(2), 156-170.

Schubert, C., Lambertz, M., Nelesen, R. A., Bardwell, W., Choi, J. B., & Dimsdale, J. E. (2009). Effects of stress on heart rate complexity—a comparison between short-term and chronic stress. *Biological Psychology*, 80(3), 325-332.

Scott, S. T. (2007). Multiple traumatic experiences and the development of posttraumatic stress disorder. *Journal of Interpersonal Violence*, 22(7), 932-938.

Shaffer, F., & Ginsberg, J. P. (2017). An overview of heart rate variability metrics and norms. *Frontiers in public health*, 5, 258.

Shin, L. M., Rauch, S. L., & Pitman, R. K. (2006). Amygdala, medial prefrontal cortex, and hippocampal function in PTSD. *Annals of the New York Academy of Sciences*, 1071(1), 67-79.

Sinozich, S., & Langton, L. (2014). Rape and sexual assault victimization among college-age females, 1995-2013. *Washington, DC: US Department of Justice, Office of Justice Programs, Bureau of Justice Statistics.*

Smith, S. G., Zhang, X., Basile, K. C., Merrick, M. T., Wang, J., Kresnow, M. J., & Chen, J. (2018). The National Intimate Partner and Sexual Violence Survey: 2015 Data Brief–Updated Release.

Spinazzola, J. (2019). Structured Interview for Disorders of Extreme Stress (SIDES) & Self-Report Inventory for Disorders of Extreme Stress (SIDES-SR) Manual. Unpublished manuscript). *The Trauma Center*, Brookline, MA.

Standard, H. R. V., & Premium, H. R. V. (2018). Kubios HRV.

Stephens, M. A., & Wand, G. (2012). Stress and the HPA axis: role of glucocorticoids in alcohol dependence. *Alcohol research:current reviews*, 34(4), 468–483.

Stöckl, H., & Penhale, B. (2015). Intimate partner violence and its association with physical and mental health symptoms among older women in Germany. *Journal of Interpersonal Violence*, 30(17), 3089-3111.

Stone, L. B., Amole, M. C., Cyranowski, J. M., & Swartz, H. A. (2018). History of childhood emotional abuse predicts lower resting-state high-frequency heart rate variability in depressed women. *Psychiatry research*, 269, 681-687.

Suglia, S. F., Sapra, K. J., & Koenen, K. C. (2015). Violence and cardiovascular health: a systematic review. *American Journal of Preventive Medicine*, *48*(2), 205-212.

Thayer, J. F., Hansen, A. L., Saus-Rose, E., & Johnsen, B. H. (2009). Heart rate variability, prefrontal neural function, and cognitive performance: the neurovisceral integration perspective on self-regulation, adaptation, and health. *Annals of Behavioral Medicine*, 37(2), 141-153.

Tjaden, P. G., & Thoennes, N. (2000). Full report of the prevalence, incidence, and consequences of violence against women. Centers for Disease Control and Prevention.

Tukey, J. W. (1977). *Exploratory data analysis* (Vol. 2, pp. 131-160).

Tulppo, M. P., Makikallio, T. H., Takala, T. E., Seppanen, T. H. H. V., & Huikuri, H. V. (1996). Quantitative beat-to-beat analysis of heart rate dynamics during exercise. *American Journal of Physiology-Heart and Circulatory Physiology*, 271(1), H244-H252.

Van der Kolk, B. A. (1996). Structured interview for disorders of extreme distress–Self-report [SIDES-SR]. *Unpublished manuscript*.

Van der Kolk, B. A. (2003). The neurobiology of childhood trauma and abuse. *Child and Adolescent Psychiatric Clinics of North America*, 12(2), 293-318.

Vatcheva, K. P., Lee, M., McCormick, J. B., & Rahbar, M. H. (2016). Multicollinearity in Regression Analyses Conducted in Epidemiologic Studies. *Epidemiology (Sunnyvale, Calif.)*, *6*(2), 227.

Walters, M., Chen, J., & Breiding, M. (2011). National Intimate Partner and Sexual Violence Survey 2010: Findings on Victimization by Sexual Orientation. Center for Victim Research.

Weathers, F. W., Litz, B. T., Keane, T. M., Palmieri, P. A., Marx, B. P., & Schnurr, P. P. (2013). The PTSD checklist for DSM-5 (PCL-5)–standard [measurement instrument]. *Retrieved from http://www.ptsd.va.gov.*

Wessa, M., Rohleder, N., Kirschbaum, C., & Flor, H. (2006). Altered cortisol awakening response in posttraumatic stress disorder. *Psychoneuroendocrinology*, 31(2), 209-215.

Wicking, M., Steiger, F., Nees, F., Diener, S. J., Grimm, O., Ruttorf, M., … Flor, H. (2016). Deficient fear extinction memory in posttraumatic stress disorder. *Neurobiology Of Learning And Memory*, 136, 116–126.

Zlotnick, C., & Pearlstein, T. (1997). Validation of the structured interview for disorders of extreme stress. *Comprehensive Psychiatry*, *38*(4), 243-247.

Table 1

Descriptive Statistics

Variable	N	Range	Minimum	Maximum	Mean		Std. Deviation	Variance	Skewness		Kurtosis	
	Statistic	Statistic	Statistic	Statistic	Statistic	Std. Error	Statistic	Statistic	Statistic	Std. Error	Statistic	Std. Error
Participant Age	36	7	18	25	19.69	.290	1.737	3.018	1.609	.393	3.093	.768
Racial Minority	36	1.00	0.00	1.00	.3056	.07786	.46718	.218	.881	.393	-1.299	.768
hfHRV	35	3.53	4.90	8.43	6.7837	.15858	.93818	.880	-.085	.398	-.675	.778
SIDES TOTAL	36	55.00	0.00	55.00	15.8056	2.16202	12.97210	168.275	1.313	.393	1.618	.768
Trauma Group	36	2.00	0.00	2.00	1.0833	.12833	.76997	.593	-.146	.393	-1.261	.768
No Trauma Group	36	1.00	0.00	1.00	.2500	.07319	.43916	.193	1.206	.393	-.582	.768
Non-IPV Trauma Group	36	1.00	0.00	1.00	.4167	.08333	.50000	.250	.353	.393	-1.989	.768
IPV Trauma Group	36	1.00	0.00	1.00	.3333	.07968	.47809	.229	.738	.393	-1.544	.768
Age of Onset	26	17.00	3.00	20.00	13.0385	.93064	4.74536	22.518	-.542	.456	-.626	.887
Valid N (listwise)	25											

Table 2

Full Sample Pearson Correlations

Variable	hfHRV	Participant Age	Racial Minority	SIDES TOTAL	PCL TOTAL	Trauma Group	No Trauma Group	Non-IPV Trauma Group	IPV Trauma Group	Age of Onset
hfHRV	—									
Participant Age	-.053	—								
Racial Minority	-.277	.189	—							
SIDES TOTAL	-.004	.018	.345*	—						
PCL TOTAL	-.010	-.068	.255	.717**	—					
Trauma Group	.385*	-.087	-.152	.322	.273	—				
No Trauma Group	-.260	.140	.035	-.142	-.121	-.824**	—			
Non-IPV Trauma Group	-.141	-.112	.173	-.247	-.208	-.093	-.488**	—		
IPV Trauma Group	.385*	-.011	-.213	.389*	.329*	.854**	-.408*	-.598**	—	
Age of Onset	-.173	-.179	-.059	-.509**	-.235	-.409*	.c	.409*	-.409*	—

*p < 0.05 (2-tailed). **p < .01 (2-tailed). c. cannot be computed

Table 3

No Trauma Group Pearson Correlations

Variable	hfHRV	Participant Age	Racial Minority	SIDES TOTAL	PCL TOTAL
hfHRV	—				
Participant Age	-.210	—			
Racial Minority	-.408	-.151	—		
SIDES TOTAL	-.293	.217	.484	—	
PCL TOTAL	-.561	.055	.878**	.750*	—

*p < 0.05 (2-tailed). **p < .01 (2-tailed).

Table 4

Non-IPV Trauma Group Pearson Correlations

Variable	hfHRV	Participant Age	Racial Minority	SIDES TOTAL	PCL TOTAL	Age of Onset
hfHRV	—					
Participant Age	-.153	—				
Racial Minority	-.240	.024	—			
SIDES TOTAL	.231	-.510	.491	—		
PCL TOTAL	.281	-.288	.292	.754**	—	
Age of Onset	-.095	.256	.117	-.462	-.274	—

**p < .01 (2-tailed).

Table 5

IPV Trauma Group Pearson Correlations

Variable	hfHRV	Participant Age	Racial Minority	SIDES TOTAL	PCL TOTAL	Age of Onset
hfHRV	—					
Participant Age	.197	—				
Racial Minority	-.060	.773**	—			
SIDES TOTAL	-.483	.109	.529	—		
PCL TOTAL	-.305	-.062	.050	.598*	—	
Age of Onset	-.029	-.423	-.536	-.358	.030	—

*p < 0.05 (2-tailed). **p < .01 (2-tailed).

Table 6

Fisher Z-Transformation Among Significant Correlations Between Trauma Exposure Groups

	Participant Age and Racial Minority Status					PCL Total and Racial Minority Status				
	n	r	z	p	Cohen's q	n	r	z	p	Cohen's q
No Trauma	9	-0.151	0.352	0.725	-0.176	9	0.878	2.132*	0.033	1.066
Non-IPV Trauma Groups	15	.024				15	.292			
No Trauma	9	-0.151	2.276*	0.023	-1.004	9	0.878	2.499*	0.012	1.317
IPV Trauma Groups	12	.773				12	.050			
Non-IPV Trauma	15	0.024	2.239*	0.025	-1.18	15	0.292	0.569	0.57	0.251
IPV Trauma Groups	12	.773				12	.050			

*p < 0.05 (2-tailed).

Table 7

*Trauma Group * Racial Minority Crosstabulation*

		Participant Race		
		White	Racial Minority	Total
Trauma Group	No Trauma	6	3	9
	Non-IPV Trauma	9	6	15
	IPV Trauma	10	2	12
Total		25	11	36

Note. $\chi^2 = (2, N = 36) = 1.754$, p = .416

Table 8

T-Test for Significant Age of Onset Differences between the IPV and Non-IPV Trauma Group

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Interval of the Difference	
									Lower	Upper
Age of Onset	Equal Variances Assumed	3.393	.078	-2.193	24	.038*	-3.84848	1.75473	-7.47008	-.22689
	Equal Variances not Assumed			-2.063	16.416	.055	-3.84848	1.86504	-7.79406	.09709

*$p < 0.05$ (2-tailed).

Table 9

First Set of Regression Analyses Predicting hfHRV, Enter Method

	B	SE *B*	t	p	R Square	ΔR
Model 1						
SIDES_TOTAL	0	0.013	-0.024	0.981	0	0
MODEL 2						
SIDES_TOTAL	0.007	0.013	0.534	0.597	0.085	0.085
Racial_Minority	-0.63	0.366	-1.722	0.095		
MODEL 3						
SIDES_TOTAL	-0.005	0.013	-0.337	0.738		
Racial_Minority	-0.408	0.363	-1.123	0.27	0.2	0.115*
Trauma_Group	0.451*	0.213	2.113	0.043		

*$p < 0.05$.

Table 10

First Set of Regression Analyses predicting hfHRV, Stepwise Method

	B	SE *B*	t	p	R Square	ΔR
Model 1						
SIDES_TOTAL	0.463*	0.193	2.398	0.022	0.148	.123*

*$p < 0.05$.

Table 11

Second Set of Regression Analyses predicting hfHRV (Trauma-Only Subset), Enter Method

	B	SE *B*	t	p	R Square	ΔR
Model 1						
SIDES_TOTAL	0.004	0.015	0.291	0.773	0.004	0.004
MODEL 2						
SIDES_TOTAL	0.009	0.015	0.586	0.564	0.058	0.054
Racial_Minority	-0.491	0.436	-1.126	0.272		
MODEL 3						
SIDES_TOTAL	-0.004	0.019	-0.197	0.845		
Racial_Minority	-0.276	0.471	-0.587	0.564	0.114	0.056
Trauma_Group	0.547	0.474	1.154	0.262		
MODEL 4						
SIDES_TOTAL	-0.008	0.021	-0.381	0.707		
Racial_Minority	-0.278	0.479	-0.581	0.568	0.127	0.013
Trauma_Group	0.501	0.49	1.023	0.318		
Age_of_Onset	-0.026	0.048	-0.535	0.598		

*$p < 0.05$.

Appendix A

Trauma History Screen (THS)

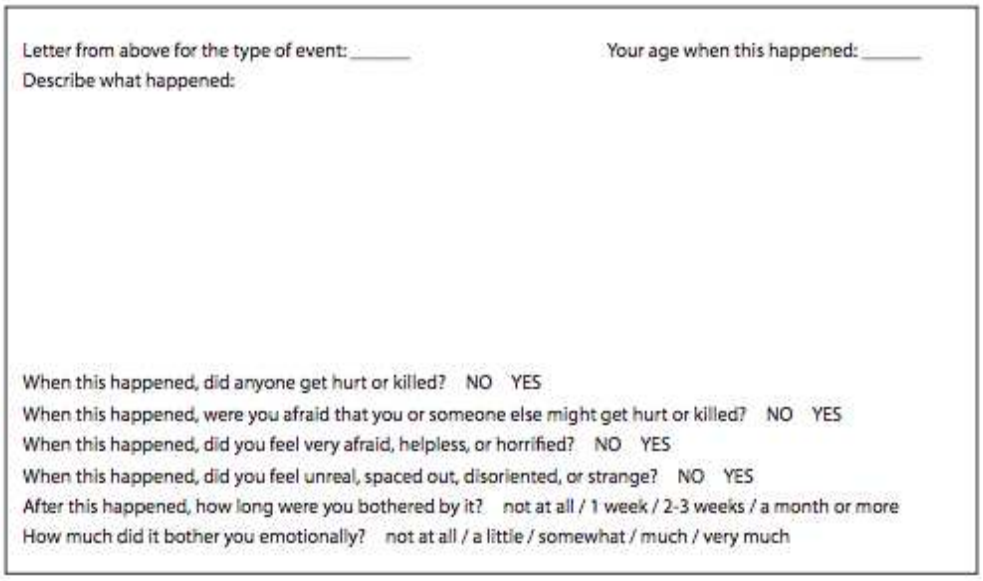
Trauma History Screen

The events below may or may not have happened to you. Circle "YES" if that kind of thing has happened to you or circle "NO" if that kind of thing has not happened to you. **If you circle "YES" for any events:** put a number in the blank next to it to show how many times something like that happened.

Event	Circle "YES" if that kind of thing has happened to you	Circle "NO" if that kind of thing has not happened to you	Number of times something like this has happened
A. A really bad car, boat, train, or airplane accident	YES	NO	_____ times
B. A really bad accident at work or home	YES	NO	_____ times
C. A hurricane, flood, earthquake, tornado, or fire	YES	NO	_____ times
D. Hit or kicked hard enough to injure - as a child	YES	NO	_____ times
E. Hit or kicked hard enough to injure - as an adult	YES	NO	_____ times
F. Forced or made to have sexual contact - as a child	YES	NO	_____ times
G. Forced or made to have sexual contact - as an adult	YES	NO	_____ times
H. Attack with a gun, knife, or weapon	YES	NO	_____ times
I. During military service - seeing something horrible or being badly scared	YES	NO	_____ times
J. Sudden death of close family or friend	YES	NO	_____ times
K. Seeing someone die suddenly or get badly hurt or killed	YES	NO	_____ times
L. Some other sudden event that made you feel very scared, helpless, or horrified	YES	NO	_____ times
M. Sudden move or loss of home and possessions	YES	NO	_____ times
N. Suddenly abandoned by spouse, partner, parent, or family	YES	NO	_____ times

Did any of these things really bother you emotionally? NO YES

If you answered "YES", fill out one or more of the boxes on the next pages to tell about EVERY event that really bothered you.



Note. Green arrows indicate traumatic experiences that qualify as interpersonal violence (IPV).

Appendix B

Racial Identity Measure

Note. Participants may check off multiple boxes.

Appendix C

Self-Report Inventory for Disorders of Extreme Stress (SIDES-SR)

SIDES-SR 4/2002

Subject ID : _____ Date: _____ Visit: _____ Collected by: _____

SIDES-SR

Instructions:

What follows are descriptions of difficulties that some people experience. After each statement please indicate: 1) whether it has ever been true for you; 2) if yes, how much you have been bothered by that problem in the past month; 3) if no, choose NA for "Not applicable" as the severity rating for the past month.

1. Small problems get me very upset. For example, I get angry at a minor frustration. I cry easily.

This has been true for me Yes No

How much have you been bothered in the last month?

None; not at all	0
Sometimes I overreact a little	1
Sometimes I get very upset, or everything upsets me more than it used to	2
Often I get extremely upset, have tantrums	3
Not applicable	NA

2. I find it hard to calm myself down after I become upset and have trouble getting back on track

This has been true for me Yes No

How much have you been bothered in the last month?

None; not at all	0
I get momentarily upset	1
It keeps coming back to me hour after hour	2
I get completely consumed by it	3
Not applicable	NA

3. When I feel upset, I have trouble findings ways to calm myself down.

This has been true for me Yes No

How much have you been bothered in the last month?

None; not at all	0
I need to make special efforts to calm myself (e.g. talking, sports, listening to music)	1
I need to stop everything and focus all my energy on calming down	2
I need to resort to extreme measures, like getting drunk, taking drugs, or doing other harmful things to my body	3
Not applicable	NA

8. Since the experience, or as long as I can remember, I have been in accidents or near accidents.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
Occasional accidents causing harm or pain but not requiring medical attention	1
One accident or episode requiring medical attention	2
More than one serious accident or episode requiring medical attention	3
Not applicable	NA

9. I find myself careless about making sure that I am safe.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I think about the risks involved in relationships or situations, but do it anyway	1
I take undue risks regarding the people I am with or places I visited	2
I keep company with people who I know could be dangerous; not taking measures to protect myself in dangerous situations	3
Not applicable	NA

10. I have deliberately tried to hurt myself (like burning or cutting myself).

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I hit or kick objects	1
I hurt myself deliberately (pinching, scratching, hitting, banging) without serious damage	2
I hurt myself deliberately in ways that cause serious physical damage	3
Not applicable	NA

11. I have thought about killing myself.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I was preoccupied, but had no plan	1
I made gestures or was chronically preoccupied with plans	2
I made one or more serious suicide attempts	3
Not applicable	NA

12. I make active efforts to keep myself from thinking about sex.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I try not to think about sex	1
I work very hard not to think about sex	2
I will not tolerate any thoughts about sex	3
Not applicable	NA

13. It bothers me to be touched in general.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
it sometimes bothers me	1
It often or regularly bothers me	2
I simply could not stand it	3
Not applicable	NA

14. It bothers me to be touched in a sexual way.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
Sometimes it bothers me	1
It often or regularly bothers me	2
I simply could not stand it	3
Not applicable	NA

15. I actively avoid sex.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I find myself making excuses	1
I try not to have sex	2
I don't have sex	3
Not applicable	NA

16. I find myself thinking about sex more than I want to.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I think about it too much	1
It distracts me from what I should be doing	2
I am obsessed with it	3
Not applicable	NA

17. I find myself driven to engage in sexual activities without really feeling that I had a choice.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I feel the urge, but I do not act on it	1
I feel compelled to, but I force myself to stop	2
I engage in compulsive sex	3
Not applicable	NA

18. I am active sexually in ways that I know put me in danger.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I am a bit careless	1
I talk myself into ignoring the danger or I only see the danger afterwards	2
I knowingly put myself in danger	3
Not applicable	NA

19. I expose myself to situations that might be dangerous, e.g. I get involved with people who might hurt me. I got to places that are not safe. I drive too fast.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I am a bit careless	1
I talk myself into ignoring the danger or I only see the danger afterwards	2
I knowingly put myself in danger	3
Not applicable	NA

20. There are parts of my life that I cannot remember, or I am confused about what happened, or I am unsure whether certain important things did or did not happen to me.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
There are a few memory lapses	1
There are important gaps in my memory; there are missing periods	2
I have no memory for days, months, or years of my life.	3
Not applicable	NA

21. I have difficulty keeping track of time in my daily life.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
At times, I have difficulty making or keeping track of schedules	1
I regularly show up in the wrong place at the wrong time	2
I am unable to keep track of my daily life	3
Not applicable	NA

22. I 'space' out when I feel frightened or under stress.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I am withdrawn at times	1
I go into my own world and do not let other people in	2
I feel like I stop existing	3
Not applicable	NA

23. I sometimes feel so unreal that it is as if I am living in a dream, or not really there, or behind a glass wall.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I feel unreal at times but I can easily be brought back	1
I feel unreal a lot and have difficulty getting back	2
I regularly feel totally disconnected from my surroundings	3
Not applicable	NA

24. I sometimes feel like there are two people living inside me who control how I behave at different times.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I am a very different person in different settings	1
It feels like different parts of me are in competition over how I should behave	2
There are separate parts of me that take control at different times	3
Not applicable	NA

25. I have the feeling that I basically have no influence on what happens to me in my life.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I do not take initiative in routine activities	1
At times, I do not bother to keep appointments, do not go out, do not return phone calls, do not take care of myself (e.g. my personal hygiene, shopping, eating.)	2
I simply do not bother to take care of myself	3
Not applicable	NA

26. I feel that I have something wrong with me after what happened to me, that can never be fixed.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I feel wounded, but that I can get better	1
I feel that parts of me are damaged but some parts of me still function	2
I feel like I am a permanently damaged person	3
Not applicable	NA

27. I feel chronically guilty about all sorts of things.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I feel more responsible than I need to for things that go wrong	1
I blame myself for things that go wrong even when I had nothing to do with it	2
I blame myself and punish myself for whatever goes wrong, even when I have nothing to do with it	3
Not applicable	NA

28. I am too ashamed of myself to let people get to know me. (How far did you go to hide from others? Did you avoid talking with people? Make up a cover story?)

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I make up stories to hide things I'm ashamed of	1
I avoid letting most people know who I really am for fear that they'll get to know me	2
I let no one get close to me to make sure they won't find out who I really am	3
Not applicable	NA

29. I feel set apart and very different from other people.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I feel quite different from people around me	1
I feel different from others and distant, estranged, or alienated from them	2
I feel like I am from another planet and don't belong anywhere	3
Not applicable	NA

30. People make too big a deal about the dangerousness of situations that I get involved in.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
Minimal	1
Moderate	2
Severe	3
Not applicable	NA

31. I have trouble trusting people.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I am guarded and am suspicious of people's motives	1
People need to prove themselves over and over again before I let my guard down	2
I don't trust anybody	3
Not applicable	NA

32. I avoid having relationships with other people.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I arrange to have lots of time by myself	1
I do not initiate contact with others. I do not make phone calls or write letters	2
I do not return phone calls, reply to letters. I stop conversations as soon as I can	3
Not applicable	NA

33. I have difficulty working through conflicts in relationships.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I am quiet or avoid situations that might cause conflict, or I am easily hurt and offended	1
I have trouble hearing other viewpoints, or have difficulty standing up for myself	2
I quit jobs and relationships without negotiating, I threaten to sue people if they offend me, I can't stand it if people disagree with me	3
Not applicable	NA

34. I find that other traumatic experiences keep happening to me.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
I find myself occasionally hurt in relationships	1
I repeatedly find myself hurt in relationships	2
I am seriously hurt by people I love or thought I could trust	3
Not applicable	NA

35. I have hurt other people in ways similar to how I was hurt.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
People have told me once or twice that I am hurtful	1
People have told me several times that I am hurtful, or I deliberately hurt people	2
I seriously hurt or injure other people in ways that are similar to ways I have been hurt myself	3
Not applicable	NA

36. I have trouble with (circle item that apply), yet doctors have not found a clear cause for it.

a) vomiting
b) abdominal pain
c) nausea
d) diarrhea
e) intolerance of food

This has been true for me Yes No

How much have you been bothered in the last month?

None; not at all	0
It is a minor problem that bothers me a little but does not affect my daily life	1
It is a serious enough problem to affect my daily life	2
It is a disabling problem that severely limits my daily life	3
Not applicable	NA

37. I suffer from chronic pain (circle items that apply), yet doctors could not find a clear cause for it.

a) in your arms and legs
b) in your back
c) in your joints
d) during urination
e) headaches
f) elsewhere

This has been true for me Yes No

How much have you been bothered in the last month?

None; not at all	0
It is a minor problem that bothers me a little but does not affect my daily life	1
It is a serious enough problem to affect my daily life	2
It is a disabling problem that severely limits my daily life	3
Not applicable	NA

38. I suffer from (circle items that apply), yet doctors have not found a clear cause for it.

a) shortness of breath
b) palpitations
c) chest pain
d) dizziness

This has been true for me Yes No

How much have you been bothered in the last month?

None; not at all	0
It is a minor problem that bothers me a little but does not affect my daily life	1
It is a serious enough problem to affect my daily life	2
It is a disabling problem that severely limits my daily life	3
Not applicable	NA

39. I suffer from trouble with (circle items that apply), yet doctors have not found a clear cause for it.

a) remembering things
b) swallowing
c) losing your voice
d) blurred vision
e) actual blindness
f) fainting and losing consciousness
g) seizures and convulsions
h) being able to walk
i) paralysis or muscle weakness
j) urination

This has been true for me Yes No

How much have you been bothered in the last month?

None; not at all	0
It is a minor problem that bothers me a little but does not affect my daily life	1
It is a serious enough problem to affect my daily life	2
It is a disabling problem that severely limits my daily life	3
Not applicable	NA

40. I suffer from (circle items that apply), yet doctors have not found a clear cause for it.

a) burning sensations in your sexual organs or rectum (not during intercourse)
b) impotence
c) irregular menstrual periods
d) excessive pre-menstrual tension
e) excessive menstrual bleeding

This has been true for me	Yes	No	
None; not at all			0
It is a minor problem that bothers me a little but does not affect my daily life			1
It is a serious enough problem to affect my daily life			2
It is a disabling problem that severely limits my daily life			3
Not applicable			NA

41. I feel hopeless and pessimistic about the future.

This has been true for me	Yes	No	

How much have you been bothered in the last month?

None; not at all	0
I get discouraged and lose interest in planning for myself	1
I don't see a future and go through the motions of living	2
I feel condemned and have no future left	3
Not applicable	NA

42. I do not expect to be able to find happiness in love relationships.

This has been true for me	Yes	No	

How much have you been bothered in the last month?

None; not at all	0
I sometimes feel distant and disconnected from my loved ones	1
I go through the motions of relationships, but feel numb	2
I don't feel part of the human race, and cannot imagine ever loving anybody	3
Not applicable	NA

43. I am unable to find satisfaction in work.

This has been true for me	Yes	No	

How much have you been bothered in the last month?

None; not at all	0
Sometimes it is a routine, but I can find reason to keep going	1
I have difficulty finding meaning in work or I cannot think of work that would be meaningful	2
Work is pointless	3
Not applicable	NA

44. I believe that life has lost its meaning.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
Sometimes it seems pointless	1
I cannot think of a good reason, but I keep on living	2
I live in a huge void	3
Not applicable	NA

45. There have been changes in my philosophy or religious beliefs—or in the religious beliefs or philosophical beliefs I grew up with.

This has been true for me	Yes	No

How much have you been bothered in the last month?

None; not at all	0
My beliefs have changed, but it was a normal progression of life	1
I am disillusioned with the religious beliefs I grew up with	2
I hate the religious beliefs I grew up with	3
Not applicable	NA

Appendix D

PTSD Checklist for DSM-5 (PCL-5)

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.

In the past month, how much were you bothered by:	Not at all	A little bit	Moderately	Quite a bit	Extremely
1. Repeated, disturbing, and unwanted memories of the stressful experience?	0	1	2	3	4
2. Repeated, disturbing dreams of the stressful experience?	0	1	2	3	4
3. Suddenly feeling or acting as if the stressful experience were actually happening again (as if you were actually back there reliving it)?	0	1	2	3	4
4. Feeling very upset when something reminded you of the stressful experience?	0	1	2	3	4
5. Having strong physical reactions when something reminded you of the stressful experience (for example, heart pounding, trouble breathing, sweating)?	0	1	2	3	4
6. Avoiding memories, thoughts, or feelings related to the stressful experience?	0	1	2	3	4
7. Avoiding external reminders of the stressful experience (for example, people, places, conversations, activities, objects, or situations)?	0	1	2	3	4
8. Trouble remembering important parts of the stressful experience?	0	1	2	3	4
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)?	0	1	2	3	4
10. Blaming yourself or someone else for the stressful experience or what happened after it?	0	1	2	3	4
11. Having strong negative feelings such as fear, horror, anger, guilt, or shame?	0	1	2	3	4
12. Loss of interest in activities that you used to enjoy?	0	1	2	3	4
13. Feeling distant or cut off from other people?	0	1	2	3	4
14. Trouble experiencing positive feelings (for example, being unable to feel happiness or have loving feelings for people close to you)?	0	1	2	3	4
15. Irritable behavior, angry outbursts, or acting aggressively?	0	1	2	3	4
16. Taking too many risks or doing things that could cause you harm?	0	1	2	3	4
17. Being "superalert" or watchful or on guard?	0	1	2	3	4
18. Feeling jumpy or easily startled?	0	1	2	3	4
19. Having difficulty concentrating?	0	1	2	3	4
20. Trouble falling or staying asleep?	0	1	2	3	4

Appendix E

Mind-Body Laboratory Health History Questionnaire (HHQ)

A very brief medical history must be obtained as part of the experimental protocol. It is very important that you be completely honest. This information will be kept strictly confidential.

1. What is your age, height, weight, and gender?

Age: _____ years

Height: _____ feet, _____ inches

Weight: _____ pounds

Sex: ___M ___F

2. Since birth, have you ever been hospitalized or had any major medical problems?

___ Yes ___ No

If Yes, briefly explain:

3. Have you ever experienced a concussion or lost consciousness due to a blow to the head?

___ Yes ___ No

If Yes, briefly explain:

4. Have you ever had problems that required you to see a counselor, psychologist, or psychiatrist?

___ Yes ___ No

If Yes, briefly explain:

5. Do you use tobacco products of any kind?

___ Yes ___ No

If Yes, describe what kind how often/much:

6. Have you ever been diagnosed with a psychological disorder?

___ Yes ___ No

If Yes, briefly explain:

7. Do you currently have or have you ever had any of the following?

___ Yes ___ No Strong reaction to cold weather

___ Yes ___ No Circulatory problems

___ Yes ___ No Tissue disease

___ Yes ___ No Skin disorders (other than facial acne)

___ Yes ___ No Arthritis

___ Yes ___ No Asthma

___ Yes ___ No Lung problems

___ Yes ___ No Cardiovascular disorder/disease

___ Yes ___ No Diabetes

___ Yes ___ No Hypoglycemia

___ Yes ___ No Hypertension (high blood pressure)

___ Yes ___ No Hypotension (low blood pressure)

___ Yes ___ No Hepatitis

___ Yes ___ No Neurological problems

___ Yes ___ No Epilepsy or seizures

___ Yes ___ No Brain disorder

___ Yes ___ No Stroke

If you responded Yes to any of the above conditions, briefly explain:

8. Have you ever been diagnosed as having:

___ Yes ___ No Learning deficiency or disorder

___ Yes ___ No Reading deficiency or disorder

___ Yes ___ No Attention deficit disorder

___ Yes ___ No Attention deficit hyperactivity disorder

___ Yes ___ No Autism Spectrum Disorder or Asperger's

9. Do you have:

___ Yes ___ No Claustrophobia (extreme fear of small closed spaces)

___ Yes ___ No Blood phobia (extreme fear of needles or blood)

___ Yes ___ No Fear of medical settings (e.g. hospital or doctor)

___ Yes ___ No Phobia of any type (if Yes, briefly explain:)

___ Yes ___ No Generalized anxiety disorder

___ Yes ___ No Anxiety disorder of any type (if Yes, briefly explain:)

If you responded Yes, briefly explain here:

10. List any over-the-counter or prescription medications you are currently taking:

11. List the symptoms that these drugs are treating

12. List any other medical conditions that you have or have had in the past:

13. What is your average daily caffeine consumption (approximate number of cups/glasses of coffee, tea, or caffeinated soda)?

14. What is your average weekly alcohol consumption (approximate number of alcoholic beverages)?

15. How many hours of sleep do you average per night?

16. On average, how often do you engage in physical activity for at least 30-minute sessions? (Circle one)

1- Never; 2- Rarely; 3-One to two days per week; 4-Three to four days per week;

5-Five to six days per week; 6-Seven days per week

17. Have you ever fainted? If so, explain. (When, what was likely to have caused it, how often does this occur?)