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Predictive Value of Heart Rate Measures on Posttraumatic Stress Disorder: A Critical Review of Select Recent Studies

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ABSTRACT

Posttraumatic stress disorder (PTSD) is characterized by maladaptive psychophysiological changes, such as a reduced vagal tone and hyperarousal, indicating autonomic nervous system dysfunction. In particular, physiological measures of heart rate, and heart rate variability (HRV) have been linked with PTSD expression, indicating that these measures may have diagnostic value. It remains unclear, however, whether altered heart rate and HRV contribute to the risk of PTSD development. This paper provides an overview of the present understanding of psychophysiological factors that may causally contribute to the manifestation of PTSD. The predictive value of heart rate and HRV measures are evaluated. The following sources of evidence are critically reviewed: relationships between momentary HRV components and PTSD symptom severity, predictions of PTSD development from post-trauma heart rate, and predictions of PTSD development from pre-trauma HRV. Available data challenge preliminary findings that abnormalities in heart rate and HRV currently offer reliable insight into PTSD development, but suggest that with additional research, there is a promising role for physiological biomarkers of autonomic dysregulation in risk prediction of future psychopathology.

Keywords: Posttraumatic stress disorder; Trauma; Heart rate; Heart rate variability; Psychophysiology; Autonomic nervous system; Risk

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Introduction

Posttraumatic stress disorder (PTSD) is a prevalent and pernicious mental health condition with an estimated lifetime risk of 8.7% among U.S. civilians (American Psychiatric Association [APA], 2013) and a 7.7% to 23% lifetime prevalence among U.S. combat veterans (Fulton et al., 2015; Lehavot et al., 2018). Heightened physiological arousal is a well-established and central feature of the disorder, with hypervigilance, exaggerated startle response, rapid heart rate, increased blood pressure, and shortness of breath symptoms associated with exposure to trauma cues (APA, 2013; Marshall et al., 2019; Shaffer & Ginsberg, 2017; Shaffer et al., 2014). PTSD is also associated with several psychological and cognitive symptoms, including dissociation, depersonalization, derealization, deficits in attention, concentration, and memory (APA, 2013; Clausen et al., 2017; McKinnon et al., 2016).

Posttraumatic Stress Disorder Development and Trajectory

By definition, PTSD develops as a consequence of "exposure to actual or threatened death, serious injury, or sexual violence" (APA, 2013, p. 271). Trauma exposure may occur in several ways, with the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*; APA, 2013) describing the direct experience of traumatic events, witnessing traumatic events, learning of traumatic events occurring to someone close, and repeated or extreme exposure to details of traumatic events as precipitating circumstances. Despite the presence of trauma as a diagnostic criterion, not everyone who experiences Criterion A trauma will develop PTSD (Santiago et al., 2014; Shah & Vaccarino, 2015). According to the National Institute of Health, 37.1% of individuals exposed to intentional trauma and 23.1% exposed to non-intentional trauma will meet DSM-5 criteria for PTSD diagnosis 12 months after trauma exposure (Santiago et al., 2014). Also concerning is the chronic nature of the disorder. Research on posttraumatic stress trajectories of World Trade Center tower survivors found that 17.1% of survivors diagnosed with PTSD had chronically elevated or worsening symptom severity persisting nearly two decades after 9/11 (Adams et al., 2019).

Even with the benefit of timely and appropriate treatment, a diagnosis of PTSD is associated with severe and persistent adverse psychological and physiological outcomes (Adams et al., 2019; APA, 2013; Murdoch et al., 2017). The disorder has been linked to increased rates of depression, suicide attempts and completion, homelessness, and substance use disorders, as well as impaired occupational and social functioning (Murdoch et al., 2017). Studies have demonstrated a positive relationship between PTSD and adverse physical health outcomes, including chronic pain, obesity, heart disease, heart failure, and stroke (Levine et al., 2014; Remch et al., 2018). Given the severity of long-term dysfunction and impairment associated with PTSD, accurate and early detection of individuals at risk of developing the disorder is critical for the prevention and amelioration of its clinical course.

Early Identification of Elevated PTSD Risk

As PTSD is known to have lasting negative psychological and physiological consequences, it is essential to identify individuals at risk of the disorder as early as possible so that appropriate intervention can be provided. Yet, to date, the mental health profession has had little ability to detect trauma survivors with heightened susceptibility to PTSD. The prediction of stress disorder development is complicated by the transdiagnostic nature of many PTSD symptoms outlined in the DSM-5, which address non-specific general distress rather than PTSD-specific symptomatology (Marshall et al., 2019). PTSD symptoms of general distress subsume a range of negative alterations in cognition and affect, including depressed and anxious mood, fear, guilt, shame, irritability, emotional volatility, and sleep disturbances (APA, 2013; Marshall et al., 2019). Non-specific components of PTSD overlap substantially with symptoms

characteristic of other trauma- and stressor-related disorders, anxiety disorders, and depressive disorders (APA, 2013). As a result, general symptoms of acute posttraumatic stress are not sensitive enough to determine which trauma survivors will develop PTSD (Beauchaine & Thayer, 2015; Goodman & Griffin, 2018).

In response to this limitation, physiological indicators of autonomic dysregulation characteristic of PTSD have been posited as a means of identifying trauma-exposed individuals at increased risk of developing the disorder (Beauchaine & Thayer, 2015). Heart rate, blood pressure, cortisol levels, and galvanic skin response have all been proposed as diagnostic biomarkers of PTSD pathophysiology (Beachaine & Thayer, 2015; Chou et al., 2014). Despite preliminary evidence from early research suggesting that heart rate measures may correspond with elevated PTSD risk, the predictive value of heart rate as a discriminative psychophysiological marker of PTSD susceptibility has not been established (Ehlers et al., 2010; Tan et al., 2011; Yehuda et al., 1998).

The present critical review was performed to elucidate this issue. This paper provides an overview of the psychophysiology of PTSD, physiological biomarkers of PTSD, and the utility of heart rate measures as indicators of autonomic nervous system functioning. Current evidence in the form of cross-sectional (Green et al., 2016), longitudinal prospective post-post (Price et al., 2014), and longitudinal prospective pre-post studies (Minassian et al., 2015) relating to the predictive relationship proposed between measures of heart rate and PTSD are critically examined.

Psychophysiology of PTSD

The autonomic nervous system, comprised of the sympathetic, parasympathetic, and enteric systems, regulates involuntary physiologic processes that include heart rate, blood pressure, respiratory rate, digestion, pupillary response, and sexual arousal (Ziegler, 2012). The sympathetic and parasympathetic nervous systems offer dual control over involuntary physiological processes, with each system asserting dominance under specific conditions (McCorry, 2007; Won & Kim, 2016). In the event that an individual encounters an acute stressor, the sympathetic nervous system responds most quickly, via sympathoneural and adrenomedullary responses (Carter & Goldstein, 2015). Activation of the sympathetic nervous system results in a heightened state of physiological arousal (i.e., the “fight or flight” response) that prepares the body for action (McCorry, 2007). Under normal conditions, once the acute stressor has passed, the sympathetic response quickly subsides and the parasympathetic nervous system is activated, returning the body to its pre-crisis state (i.e., “rest and digest”). However, when an individual experiences recurrent acute stress, the sympathetic nervous system becomes chronically activated, lacking the compensatory parasympathetic nervous system activity necessary to obtain and maintain physiological homeostasis (Won & Kim, 2016). Under persistently stressful situations, catecholamine levels increase and acetylcholine levels decrease, as is seen in individuals with PTSD (Sherin & Nemeroff, 2011; Won & Kim, 2016).

A core clinical feature of PTSD is sustained hyperactivity of the sympathetic branch of the autonomic nervous system, as demonstrated by elevations in heart rate, blood pressure, skin conductance, reduced heart rate variability, and cortisol levels, along with abnormalities in other physiological measures (Nemeroff, 2016; Shaffer et al., 2014; Sherin & Nemeroff, 2011). These pathophysiological changes manifest as symptoms of sustained hyperarousal, impaired responsiveness to stressors, and behavioral and emotional inflexibility characteristic of PTSD (APA, 2013; Beauchaine, 2001; Shaffer et al., 2014; Sherin & Nemeroff, 2011). That psychophysiological symptoms of PTSD persist long after the precipitating traumatic experience has ended indicates that individuals with PTSD

suffer from enduring pathological changes in autonomic functioning in response to recurrent stressors (APA, 2013; Shaffer & Ginsberg, 2017; Shaffer et al., 2014). The consistency with which these pathological changes are observed in individuals with PTSD suggests that signs of autonomic dysfunction may serve as a useful biomarker of PTSD development (Haag et al., 2019). In this review, heart rate variability will be examined as a potential biomarker of the pathophysiological underpinnings of PTSD.

Heart Rate Variability and Autonomic Nervous System Function

HRV, defined as the beat-to-beat variation in heart rate, is a reflection of autonomic flexibility (Billman, 2011; Thayer & Lane, 2000). HRV offers a dynamic measure of the highly coordinated sympathetic and parasympathetic modulation of cardiac function (Shaffer & Ginsberg, 2017; Thayer & Lane, 2000). Although the exact influences of sympathetic and parasympathetic control on variability remain controversial and are under continued investigation, HRV is nevertheless a clinically accepted measure of autonomic functioning widely used in the biomedical sciences (Billman, 2011; Ge et al., 2020; Malik et al., 1996). HRV is considered an indicator of general health, and reduced HRV has been linked to chronic pathological psychological and psychological conditions (Beauchaine & Thayer, 2015).

HRV indices most often used in PTSD research include the high-frequency components of HRV (HF-HRV), the low-frequency components of HRV (LF-HRV), and the ratio of LF-HRV to HF-HRV (LF/HF ratio; Ge et al., 2020; Haag et al., 2019; Shaffer et al., 2014). Research findings suggest the HF-HRV band is closely related to respiratory sinus arrhythmia and support its interpretability as a measure of parasympathetic activity (Berntson et al., 2007; Minassian et al., 2015; Shaffer et al., 2014). In contrast, autonomic influences underlying the LF-HRV band are actively debated and its interpretation remains controversial (Haag et al., 2019; Shaffer et al., 2014). Most recently, experts in the field have posited that LF-HRV is a measure of baroreflex activity and is controlled by dual influences of sympathetic and parasympathetic divisions of the autonomic nervous system (Rahman et al., 2011; Shaffer & Ginsberg, 2017; Shaffer et al., 2014). High LF-HRV is believed to reflect increased sympathetic nervous system activity (Quintana et al., 2016).

Interpretation of the LF/HF ratio remains the subject of active debate due to issues regarding the interpretation of the LF-HRV band described above. Still, LF/HF has been tentatively proposed to reflect sympatho/vagal balance (Schiweck et al., 2019; Shaffer & Ginsberg, 2017; Shaffer et al., 2014). Low LF/HF may indicate greater parasympathetic activation relative to sympathetic activation, and high LF/HF may indicate greater sympathetic activation relative to parasympathetic activation (Shaffer et al., 2014). Researchers have advised that the LF/HF ratio should be interpreted cautiously (Shaffer & Ginsberg, 2017; Shaffer et al., 2014).

Heart Rate Variability and PTSD

With relative consistency, research involving military veterans (Dennis et al., 2014; Park et al., 2017; Shah et al., 2013), disaster survivors (Adams et al., 2019; Lee et al., 2018; Tucker et al., 2012), and young adults (Rissling et al., 2016) has revealed that individuals with PTSD exhibit pathologically reduced HRV. However, observed relationships between other indices of HRV and PTSD have been less consistent. Most studies suggest that HF-HRV is reduced in individuals with PTSD (Chalmers et al., 2014; Cohen et al., 2000; Tucker et al., 2012; Wahbeh & Oken, 2013), while others have found no alterations in HF-HRV (Lee et al., 2018). Additionally, some research points toward elevated LF-HRV and LF/HF ratios among individuals with PTSD (Lakusic et al., 2007; Lee et al., 2018; Tucker et al., 2012), while others have failed to find an association (Chalmers et al., 2014). Evaluation of conflicting study results underscores the need for reliable methodology

and accurate interpretations in psychophysiological research.

Cross-Sectional Research on HRV and PTSD

Green and colleagues (2016) assessed the relationship between stress disorder symptoms and moment-to-moment measures of HRV in participants (*n* = 83; age range: 18-39 years) diagnosed with PTSD. Participants were recruited from a more extensive study of trauma and health (Dennis et al., 2014). The researchers hypothesized that moments of increased PTSD symptom severity would be associated with simultaneous reductions in HRV indices. To test this hypothesis, HF-HRV and LF-HRV measures were collected using 24-hour Holter monitor electrocardiogram recordings and PTSD symptoms were self-reported by patients using ecological momentary assessments prompted by random alarms. PTSD status was assessed by a licensed clinical psychologist using the Clinician-Administered PTSD Scale, which adheres to DSM-IV criteria for PTSD diagnosis. After controlling for smoking habits, alcohol use, and sleep disturbances, results showed that momentary PTSD symptom severity was associated with statistically significant concurrent reductions in LF-HRV but non-significant reductions in the HF-HRV. The authors concluded that the detected reduction in LF-HRV at the time of PTSD symptom experience indicated decreased parasympathetic control, and consequently, was supportive of the position that autonomic dysregulation, rather than behavioral health factors, directly underlie PTSD symptoms.

Green and colleagues' (2016) observation that greater momentary PTSD symptom severity was associated with reduced LF-HRV contradicts earlier findings of elevated LF-HRV among individuals with PTSD (Lakusic et al., 2007; Lee et al., 2018) and evidence that LF-HRV is predominantly regulated by sympathetic activity relative to parasympathetic under stressful conditions (Schaffer et al., 2014). Acute stress results in the withdrawal of parasympathetic activity in favor of sympathetic fight-or-flight responses, which is expected to manifest in a reduction if HF-HRV and elevation in LF-HRV (Berntson et al., 2007; McCorry, 2007; Minassian et al., 2015; Rahman et al., 2011; Shaffer et al., 2014). The authors suggested that their findings may be attributable to methodological differences between their study and earlier research associating PTSD with elevated LF-HRV but did not provide further explanation (Green et al., 2016). In a subsequent paper, the study authors proposed that inversely associated PTSD symptom severity and LF-HRV may be the result of delayed or attenuated autonomic recovery in response to acute stress (Dennis et al., 2016).

Additional limitations to the Green et al. (2016) study further diminish the ability to infer correlations between PTSD symptom severity and HRV indices. Only 83 individuals out of an original participant sample of 107 were included in the final analyses and reasons for exclusion were not disclosed. Given this, the validity of results may be compromised by selection bias. The biasing effects of self-selection should also be considered as individuals who do/do not choose to participate in mental health-related research may differ in motivation, intention, and other factors that could influence study outcomes. In addition, moment-to-moment PTSD symptoms were collected using a self-report survey. Self-report data are inherently vulnerable to feigning, malingering, inaccurate self-perception, and other response biases. Overall, problematic methodology and interpretation of results undermine the strength of the conclusions proposed by the Green et al. (2016) study.

Longitudinal Prospective, Post-Post Research on HRV and PTSD

While cross-sectional research provides crucial insight into the psychophysiology of PTSD, only limited inferences can be drawn from the results of correlational studies regarding the development of the disorder. Without establishing temporal precedence, a causal relationship between reduced HRV and PTSD

cannot be determined. Additional data is required to elucidate whether PTSD symptoms are driven by autonomic dysregulation occurring as a direct consequence of trauma exposure or a correlate of other PTSD vulnerability factors, such as behavioral health risks (e.g., smoking, alcohol dependence; Dennis et al., 2014). Determining the relative timing of PTSD and autonomic dysfunction is necessary if biomarkers of nervous system functioning, such as heart rate and HRV, are to be used in the early identification of trauma survivors at increased risk of developing PTSD.

To address this issue, research has begun to investigate whether physiological responses occurring immediately after trauma exposure predict subsequent PTSD symptoms. Longitudinal prospective studies following the developmental course of PTSD in the immediate aftermath of trauma, also described as 'post-post' studies, can be broadly categorized as those that examine the predictive value of heart rate in relation to PTSD with heart rate measures evaluated during the acute posttraumatic phase (typically within 72 hours of trauma exposure) and those that look at heart rate measures during exposure to idiographic trauma cues in the early posttraumatic phase (e.g., 1-month post trauma-exposure; Goodman & Griffin, 2018; Morris et al., 2016; Schmidt et al., 2015). Research examining heart rate alterations in the acute posttraumatic phase is the more common of the two designs (Morris et al., 2016).

One example of post-post longitudinal research is the Price et al. (2014) study, which explored the predictive value of two physiological responses to trauma (heart rate and cortisol levels) known to occur in response to acute stress and implicated in fear acquisition processes central to the development and maintenance of PTSD symptoms (Careaga et al., 2016). The authors hypothesized that increased heart rate in the acute posttraumatic phase would be associated with elevated PTSD symptoms at follow-up visits. The participant sample was comprised of 55 individuals who presented to a Level One trauma center emergency department after experiencing violent physical or sexual assault meeting PTSD Criterion A requirements. Individuals were assessed using the Standardized Trauma Interview (Foa & Rothbaum, 2001) and heart rate measures were obtained by nurses in the emergency department as part of an initial physical exam. PTSD symptoms were evaluated at four- and twelve-weeks post-trauma using the PTSD Symptom Scale, Interview Version (Foa et al., 1993).

Contrary to their hypothesis, the authors found no evidence that heart rate in the acute posttraumatic phase was associated with later development of PTSD symptoms (Price et al., 2014). This finding is inconsistent with prior research and results from a recent meta-analysis that assessed the relationship between heart rate and subsequent PTSD symptoms (Morris et al., 2016). Based on the collective analysis of 20 studies (combined n = 4656), including the Price et al. (2014) study, Morris and colleagues (2016) concluded there was relatively consistent evidence that elevated basal heart rate in the acute posttraumatic period was a positive predictor of PTSD symptoms. Although an aggregate positive correlation between higher acute posttraumatic heart rate and PTSD development was observed, it is notable that effect sizes from the individual studies comprising the meta-analysis ranged from -0.22 to 0.70, indicating that some of the research included in the full analysis did not independently find elevated heart rate to be a positive predictor of PTSD symptoms. Instead, some of the studies included in the analysis found elevated basal heart rate immediately post-trauma to be a negative predictor of PTSD development (Morris et al., 2016).

One potential explanation for the discrepancy between results from the Price et al. (2014) study and research demonstrating a positive correlation between heart rate and PTSD is the presence of dissociation in the acute posttraumatic period. In addition to autonomic

and neuroendocrine functioning, altered states of consciousness influence the degree of psychophysiological arousal experienced in response to trauma (Chou et al., 2018). Dissociation, one form of altered state of consciousness, is a survival response to serious threat, with female victims of rape and sexual assault showing particular vulnerability to experiencing dissociation in response to trauma (Nöthling et al., 2015). From an adaptive perspective, dissociation at the time of trauma and immediately after can be psychologically protective.

The co-occurrence of dissociation and trauma exposure is common enough that the current iteration of the DSM acknowledges a unique dissociative subtype of PTSD (PTSD+DS) characterized by dissociative symptoms of depersonalization, derealization, and emotional detachment (APA, 2013). Research has consistently demonstrated a link between dissociative states and reduced physiological arousal (Chou et al., 2018). Dissociation in response to trauma may result in physiological inhibition and lower heart rate in the acute posttraumatic period (Chou et al., 2014). If an individual experiences dissociation at the time of physiological assessment, heart rate is likely to be reduced. This could account for the negative relationship between heart rate and PTSD symptoms observed in some post-post studies, including the Price et al. (2014) study.

Longitudinal Prospective, Pre-Post Research on HRV and PTSD

Findings of elevated heart rate and reduced HRV among individuals with PTSD support the hypothesis that autonomic dysfunction driven by sympathetic overactivation and/or parasympathetic withdrawal is responsible for stress disorder symptom experience and expression (Chou et al., 2018; Shaffer & Ginsberg, 2017; Shaffer et al., 2014). However, the causal relationship between HRV and PTSD has received comparatively little consideration, primarily due to methodological challenges. Cross-sectional and post-post prospective study designs lack the structure necessary to determine whether PTSD symptoms are attributable to autonomic dysregulation occurring as a consequence of trauma exposure or if reduced HRV before trauma increases the risk of developing PTSD post-trauma. Possible explanations for pre-trauma HRV-driven risk of stress disorders include heightened glucocorticoid negative feedback and lower blood circulating cortisol levels resulting in reduced modulation of autonomic response to fear and stress (Gillie & Thayer, 2014; Morris et al., 2016) and low trait resilience to stress (Carnevali et al., 2018). To address this gap in knowledge, prospective studies, referred to as ‘pre-post’ studies, have looked at the predictive value of heart rate measures collected before trauma exposure on PTSD risk following trauma exposure (Schmidt et al., 2015).

Using a pre-post longitudinal design, Minassian and colleagues (2015) investigated the utility of HRV indices as autonomic biomarkers of PTSD susceptibility following trauma exposure. The researchers hypothesize that reduced HRV pre-trauma would be a significant risk factor for PTSD post-trauma and predicted that Marines who developed stress disorder symptoms following combat would present with reduced HRV prior to deployment. To test this hypothesis, LF-HRV, HF-HRV, and LF/HF measures were collected for a cohort of 2,160 U.S. Marines participating in the 2-phase Marine Resiliency Study. HRV components were measured using a finger photoplethysmograph for 5 minutes in a resting state. PTSD symptoms were evaluated at pre-deployment and again at three-month and six-month post-deployment visits using the Clinician-Administered PTSD Scale. As hypothesized, Minassian and colleagues (2015) found that lower pre-deployment HRV, as measured by an elevated LF/HF ratio, was a statistically significant predictor of post-deployment PTSD following adjustment for prior deployment-related combat exposure. Post-deployment PTSD prevalence was significantly greater among Marines with

high pre-deployment LF/HF ratios than Marines with lower pre-deployment LF/HF ratios.

The observation that greater LF/HF ratios significantly predict PTSD development is consistent with previous findings that LF/HF increases in response to autonomic dysregulation favoring sympathetic activation (Shaffer et al., 2014). As such, the results of the Minassian et al. (2015) study provide preliminary evidence that reduced HRV prior to trauma increases the risk of developing PTSD post-trauma. However, methodological limitations temper the conclusions that can be drawn with respect to the potential causal relationship between high pre-deployment LF/HF ratios, reduced HRV, and risk of post-deployment PTSD. First, post-deployment HRV measures were not collected for almost half of the participants who underwent pre-deployment physiological testing and reasons for attrition were not disclosed. The validity of the results may be compromised by attrition bias. Further, the demographic homogeneity of the participant group, young males serving as U.S. Marines, limits the generalizability of results to other populations.

The need for expeditious HRV data collection led researchers to use the less sensitive finger photoplethysmograph for HRV measures rather than the more sensitive electrocardiographic Holter monitors with respiratory bands used in other HRV/PTSD studies (Shaffer & Ginsberg, 2017). Additionally, health factors known to influence both PTSD risk and autonomic functioning, such as depression symptoms, previous non-combat related trauma exposure, physical health, and medical history, were not controlled for. Without controlling for potential confounding factors, it cannot be determined if LF/HF ratio is a unique biomarker of PTSD vulnerability or a correlate of a different, unaccounted for, causal mechanism. Despite these limitations, Minassian and colleagues' (2015) pre-post study provides valuable insight into the potential physiological etiology of PTSD. More rigorous and less heterogeneous research is required to verify these findings and clarify the relationship between pre-trauma HRV and PTSD vulnerability.

Conclusion

The present paper critically reviewed evidence of the suitability of HRV indices as predictors of increased PTSD vulnerability following exposure to Criterion A trauma. Early research on cardiac physiological biomarkers have shown, broadly, that elevated LF-HRV and LF/HF and reduced HRV and HF-HRV are associated with PTSD development and symptom severity, with sustained hyperactivity of the sympathetic nervous system and/or the withdrawal of parasympathetic modulation believed to underlie stress disorder symptoms. However, a recent study (Green et al., 2016) found LF-HRV to be reduced and HF-HRV to be unaltered in individuals diagnosed with PTSD, results which contradict widely-accepted models of autonomic dysregulation. These unexpected findings may be attributable to study procedures tapping into a measure of sympathetic hyperactivation independent of parasympathetic withdrawal or suggest attenuated autonomic recovery in response to acute stress. Future research will need to clarify the physiological pathways driving abnormalities observed in measures of LF-HRV, HF-HRV, and LF/HF before their association with stress disorder psychopathology can be fully understood.

A review of research (Price et al., 2014) exploring the predictive value of heart rate on PTSD development using a prospective post-post study design also revealed contradictory findings. Contrary to hypotheses, heart rate measured in the acute posttraumatic phase was not found to be associated with subsequent PTSD symptoms among a small cohort of trauma survivors exposed to severe physical or sexual violence (Price et al., 2014). These results were inconsistent with meta-analytic data demonstrating a small but reliable positive correlation between heart rate and PTSD symptoms (Morris et al., 2016). Dissociation, a possible cognitive modulator of heart rate

responsivity, has been proposed as a potential explanation for these findings. Taken together, post-post data suggest that heart rate collected shortly after trauma may offer predictive insight into future PTSD development if cognitive functioning peri-trauma is taken into account.

Lastly, prospective pre-post research (Minassian et al., 2015) investigating the utility of pre-trauma HRV measures as autonomic biomarkers of PTSD susceptibility following trauma exposure was reviewed. Consistent with prior research, greater LF/HF ratios were found to significantly predict PTSD development, an observation supporting sympathetic overactivation, rather than parasympathetic underactivation, as the foundation of PTSD-related autonomic dysfunction. Such results provide preliminary evidence that reduced HRV prior to trauma may increase PTSD vulnerability among U.S. Marines (Minassian et al., 2015).

Collectively, evidence from recent cross-sectional and longitudinal research challenge earlier findings that physiological measures of heart rate and HRV currently offer reliable insight into PTSD development. However, these same studies also suggest that with additional rigorous, data-driven research, pathophysiological biomarkers of autonomic dysregulation may contribute meaningfully to PTSD risk prediction in the future.

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