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A prospective examination of sex differences in posttraumatic autonomic functioning

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A prospective examination of sex differences in posttraumatic autonomic functioning

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1. Introduction

Trauma exposure is very common, with lifetime prevalence rates up to 89% (Kilpatrick et al., 2013). Symptoms of posttraumatic stress disorder (PTSD) affect a substantial proportion of those exposed to trauma and include unwanted re-experiencing of the event, avoidance of trauma reminders, negative changes in thinking and mood, and hyperarousal (APA, 2013). Individuals with PTSD symptoms experience significant functional impairment across multiple domains, and they are at greater risk of having cardiometabolic diseases compared to the general population (Edmondson et al., 2013; Norman et al., 2006; Pacella et al., 2013). One of the proposed mechanisms underlying increased cardiometabolic disease incidence in PTSD is altered functioning of the autonomic nervous system, such as increased heart rate (HR) and blood pressure (BP), and decreased high frequency heart rate variability (HF-HRV; for reviews see Buckley and Kaloupek, 2001 and Michopoulos et al., 2015). While numerous cross-sectional studies have demonstrated that those with PTSD exhibit altered autonomic functioning (reviewed below), there is a paucity of longitudinal studies following individuals in the immediate aftermath of trauma. Further, it is well-established that PTSD rates are twice as high in women compared to men (Kessler et al., 1995), yet sex differences in autonomic functioning are relatively unknown among trauma-exposed populations.

There is a long-standing literature supporting an association between PTSD and autonomic deficits. Compared to healthy and trauma-exposed controls, individuals with PTSD demonstrate increased sympathetic
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their skin conductance response to a trauma interview was gathered at

to determine if skin conductance in the immediate post-trauma period was

sex differences in HR, HRV, and BP at this 2-week session, as well as to

this time. They completed a fear conditioning paradigm two weeks later

compared to men with PTSD (Inslicht et al., 2013), and high levels of

suggested low parasympathetic control prior to deployment was pre

necessary to better understand sex differences in autonomic functioning

may confer differential risk following trauma. Another study by Minassian et al. (2015)

demonstrated that high skin conductance response in the immediate

trauma aftermath is a strong prospective indicator of PTSD development

by Shaley et al. (1998, 2000) indicated that HR in the immediate trauma

aftermath was predictive of subsequent PTSD and that autonomic defi-

cits develop along with PTSD. Another study by Minassian et al. (2015)
suggested low parasympathetic control prior to deployment was pre-

dictive of subsequent PTSD among U.S. Marines. Recently, our group
demonstrated that high skin conductance response in the immediate

trauma aftermath is a strong prospective indicator of PTSD development

(Hinrichs et al., 2019). These findings are promising in terms of their

potential to identify PTSD risk, but much more is needed to develop

reliable biomarkers of autonomic functioning for use in the immediate

trauma aftermath.

Sex differences in PTSD prevalence are well-established, however,
little is known about sex differences in autonomic functioning among

trauma-exposed samples (for a review, see Seligowski et al., 2020). In a
study of assault survivors, women who experienced increased HR during

script-driven imagery were more likely to be given a PTSD diagnosis six
months later compared to men and compared to women who didn’t have
such a response (Klein et al., 2010). Among motor vehicle accident

survivors seen in the emergency department, no sex differences were

observed in HR, but women were more likely to have PTSD at follow-up
(Irish et al., 2011). In contrast, another study found that men with PTSD

exhibited higher HRV than trauma-exposed controls, and no effect in

women, but it is possible that these differences were due in part to

collecting HRV data during a dark-enhanced startle paradigm (Kamm-
walala et al., 2012). In terms of skin conductance, women with PTSD
appear to demonstrate higher responses during fear conditioning
compared to men with PTSD (Inslicht et al., 2013), and high levels of
progesterone have been implicated in extinction retention deficits
among women with PTSD (Pineles et al., 2016, 2018). Given the
apparent link between autonomic deficits and cardiometabolic disease
risk in PTSD, as well as the elevated prevalence of PTSD in women, it is
necessary to better understand sex differences in autonomic functioning
that may confer differential risk following trauma.

The current study used a prospective design to examine several as-
pects of posttraumatic autonomic functioning as part of the AURORA
study (McLean et al., 2020). Men and women were recruited from
emergency departments immediately following trauma exposure and
their skin conductance response to a trauma interview was gathered at
this time. They completed a fear conditioning paradigm two weeks later
where HR, HRV, and BP were assessed. We sought to identify potential
sex differences in HR, HRV, and BP at this 2-week session, as well as to
determine if skin conductance in the immediate post-trauma period was
associated with autonomic functioning two weeks later. Given limited
sex differences research in PTSD populations, we examined the effects of
both sex and 8-week PTSD status on these autonomic indices.

2. Methods

2.1. Participants and procedure

As part of the multi-site AURORA study, participants were recruited
from 22 emergency departments (EDs) across the U.S. immediately
following a traumatic event (see McLean et al., 2020 for additional
detail). Traumatic events included motor vehicle collisions, physical
assault, sexual assault, and serious accidents. Exclusion criteria were
intracranial injury, long bone fracture or significant extracranial hem-
orrhage, pregnancy, and admission due to intentional self-injury or
suicide attempt. Given the study’s aim of examining acute trauma re-
 sponses, participants were also excluded if they reported ongoing do-
mestic violence. Fear conditioning was completed in a sub-sample of
AURORA participants at one of four locations two weeks following the
emergency department visit. All participants provided informed consent
and the study was approved by each site’s Institutional Review Board.
See Fig. 1 for a CONSORT diagram outlining participant flow through
the study.

2.2. Measures

PTSD Checklist for DSM-5 (PCL-5; Weathers et al., 2013). The
PCL-5 is a 20-item self-report measure of PTSD symptom severity and
was administered eight weeks following the ED visit. Responses are on a
scale from 0 (not at all) to 4 (extremely). A total score was used to assess
overall PTSD severity. A score of 33 or higher was used to indicate
probable PTSD diagnosis (Bovin et al., 2016).

2.3. ED skin conductance response measurement

eSense (Mindfield Biosystems, Inc., Berlin, Germany). Using previ-
ously validated procedures (Hinrichs et al., 2017), skin conductance
levels were recorded with the eSense system during the initial ED visit.
eSense software was downloaded to an iPad and two Velcro electrodes,
which were connected to the iPad, were attached to the middle and
index finger. Participants were asked to describe the traumatic event
that brought them to the ED and eSense was used to continuously
measure skin conductance levels during the interview. This method has
been used previously in the ED among trauma-exposed adults (Hinrichs
et al., 2019).

2.4. 2-Week psychophysiological assessment

Approximately two weeks following the ED visit, participants
completed a follow-up session that included a seated BP assessment and
a fear-potentiated startle (FPS) paradigm, during which HR and HRV

Fig. 1. CONSORT diagram.
were obtained. The FPS paradigm is a Pavlovian fear conditioning paradigm that has been validated in clinical and nonclinical samples (e.g., Glover et al., 2011; Jovanovic et al., 2012; Norholm et al., 2006; Seligowski et al., 2018). It includes a habituation phase followed by fear acquisition and extinction phases. During habituation, 12 108 dB startle probes were delivered through headphones to assess baseline startle, and eight of these were delivered upon the termination of conditioned stimuli (CS), which were colored shapes presented on a computer screen. During acquisition, one of the shapes (CS+) was paired with an aversive unconditioned stimulus (US), while the other was not (CS-). The US was a 250 ms/140 p.s.i. air blast directed at the larynx. The CS+ and CS- were each presented for 6 s and the startle probe was presented at their termination. During acquisition, the CS+ and startle probe were followed by the US 0.5 s later. Habituation included four trials of each CS (not reinforced with air blasts) and four startle probes alone (noise alone [NA]). Acquisition included three conditioning blocks with four trials of each type (NA, CS+, CS-) in each block. Extinction included four blocks of four trials of each type (NA, CS+, [unreinforced], and CS-) in each block. All trials were on a fixed schedule and the inter-trial interval was 9–22 s.

Psychophysiological data were acquired using Biopac MP150 for Windows (Biopac Systems, Inc., Aero Camino, CA). HR and HF-HRV were continuously measured with Ag/AgCl electrodes in the Lead II placement of electrodes using the electrocardiogram (ECG) module, while eyeblink startle was indexed using the electromyogram (EMG) module, which was measured with two Ag/AgCl electrodes placed below the participant’s pupil.

3. Data processing and analysis

3.1. Psychophysiology data processing

Skin conductance data were acquired using eSense on the iPad at a sampling rate of 10 Hz and the data were exported using .csv files. Baseline skin conductance was measured for 2 min and followed by the participant’s narrative of their traumatic event. Skin conductance measurements were obtained throughout the narrative.

HR and HF-HRV data were processed using MindWare software, which identifies R-waves and R-R intervals (i.e., the time between heart beats), and detects artifacts, which were visually inspected and corrected (MindWare, Inc.). HR and HF-HRV were derived by spectral analysis of 1-min epochs with a Hamming windowing function and log transformed. Settings for the high frequency band were based on standard recommendations for HF-HRV data (0.12–0.40 Hz; Task Force, 1996). HR and HF-HRV values were averaged over the three acquisition blocks and four extinction blocks. The EMG sampling rate was 1 kHz, which was amplified by a gain of 2000, and filtered using 28 Hz and 500 Hz low and high pass filters; visual inspection was performed using MindWare. Following processing in MindWare, psychophysiological data were exported to SPSS. Consistent with American Heart Association guidelines, hypertension was defined as systolic BP ≥ 130 or diastolic BP ≥ 80 (hearts.org).

3.2. Data analysis

Eight-week PCL-5 data were available for 192 participants. Using a cutoff score of 33 (Bovin et al., 2016), a dichotomous PTSD variable was created to indicate probable versus no PTSD. Of those with 8-week PCL-5 data, 158 participants had useable BP from the 2-week session, 151 participants had useable HR and HF-HRV data from 2-week fear conditioning, and 141 had useable eSense data from the ED. Reasons for missingness included data quality (e.g., noisy ECG signals) and incomplete data acquisition (e.g., participant did not complete fear conditioning). A chi-square analysis was used to compare the prevalence of hypertension among men versus women. Six univariate analyses of variance (ANOVAs) were used to test the effects of sex and PTSD on systolic and diastolic BP, average HR during acquisition and extinction, and average HF-HRV during acquisition and extinction. These models controlled for age, race, and body mass index (BMI). Bivariate correlations were used to test associations among eSense and BP, HR, and HF-HRV. All analyses were conducted with SPSS v.24 and significance set to p < .05.

4. Results

Table 1 displays descriptive data among the total sample and by sex, and Table 2 displays bivariate correlations among the total sample. Of the 192 participants with 8-week PCL-5 data (Mage = 35.88, SD = 13.56), 78 (40.6%) met criteria for probable PTSD per the cutoff score of 33. There were no sex differences in putative PTSD diagnosis (χ² = 0.77, p = .380) or PCL-5 score (F[1190] = 0.74, p = .389). In terms of race, 44.3% (n = 85) identified as Non-Hispanic Black, 33.3% (n = 64) identified as Non-Hispanic White, 17.7% (n = 34) identified as Hispanic, and 4.7% (n = 9) identified as Non-Hispanic “other” racial category.

Men were significantly more likely to meet criteria for hypertension than women, χ² = 10.74, p = .001. Results of the first univariate ANOVA indicated a significant main effect of sex on systolic BP, F(1,140) = 20.51, p < .001, such that it was higher in men compared to women (Fig. 2). The PTSD by sex interaction was not significant, F(1,140) = 0.34, p = .564, and there were no significant findings for diastolic BP. There was not a significant sex difference in baseline HF-HRV, F(1,173) = 0.41, p = .523. Further, there were no significant sex differences in eyeblink startle to the CS + or CS- during acquisition or extinction (see Fig. 3), nor any significant PTSD by sex interactions when controlling for age, race, and BMI (p > .05).

There was a significant main effect of sex on HR during fear acquisition, F(1,138) = 14.80, p < .001. The PTSD by sex interaction was not significant. During fear extinction, there was a significant main effect of sex on HR, F(1,130) = 9.90, p = .002, as well as a significant PTSD by sex interaction, F(1,130) = 4.10, p = .045. Post-hoc tests for this interaction indicated that HR was significantly different for men versus women only in the PTSD group, F(1,123) = 10.94, p = .001, whereas there was no significant effect of PTSD within either sex. As depicted in Fig. 4, women demonstrated higher HR compared to men during both fear acquisition and extinction, and this effect was stronger in the PTSD group during extinction, where HR was significantly higher in women (M = 78.53) compared to men (M = 67.70).

There was a significant main effect of sex on HF-HRV during fear acquisition, F(1,138) = 4.60, p = .034. The PTSD by sex interaction was not significant, F(1,138) = 3.41, p = .067. During fear extinction, there was a significant PTSD by sex interaction, F(1,129) = 5.18, p = .025. Post-hoc tests for this interaction indicated that HF-HRV was significantly different for men versus women only in the PTSD group, F(1,122) = 7.20, p = .008, whereas there was no significant effect of PTSD within either sex. As depicted in Fig. 5, women demonstrated lower HF-HRV compared to men during fear acquisition, and this effect was stronger in the PTSD group during extinction, where HF-HRV was significantly lower in women (M = 5.41) compared to men (M = 6.49).

Next, we examined associations among eSense skin conductance in the ED and 2-week BP, HR, and HF-HRV. Skin conductance values were significantly and positively associated with HR and negatively associated with HF-HRV only among women who developed PTSD (p < .05; see Table 3), indicative of increased sympathetic arousal and decreased parasympathetic control during fear conditioning. When controlling for age, race, and BMI using linear regression models, only the association between baseline eSense and extinction HF-HRV in women with PTSD remained significant (β = −0.36, p = .006). This suggests that increased sympathetic arousal in the immediate aftermath of trauma was associated with worse parasympathetic responses to fear extinction in women but not men with PTSD. No significant findings were observed in men or in women without PTSD.
most women were pre-menopausal. While prior studies have found that individuals with PTSD demonstrate higher BP than those without PTSD is important to note that the average age in our sample was 35 and thus age groups and it is thought that decreasing estradiol levels as a result of menopause in women play a role (i.e., estradiol is cardioprotective and likely to be higher in healthy women compared to men (for a review, see Koenig et al., 2012) and further suggests that women may be more likely than men to experience these deficits. Given that HF-HRV has been shown to be highly traumatized as comparisons in prior work (e.g., most participants were in motor vehicle collisions). Similarly, BP was assessed with only one measurement and this occurred two weeks following trauma exposure. It is therefore possible that the higher levels of BP observed in prior PTSD studies were a result of more chronic PTSD symptoms and sympathetic hyperarousal, which our study did not capture.

In terms of HR and HF-HRV, our findings indicate that women demonstrated significantly higher BP and rates of hypertension, and thus most women were pre-menopausal. While prior studies have found that individuals with PTSD demonstrate higher BP than those without PTSD (for a review, see Buckley and Kaloupek, 2001), we did not observe an effect of PTSD status. One potential explanation is that our sample is not as highly traumatized as comparisons in prior work (e.g., most participants were in motor vehicle collisions). Similarly, BP was assessed with only one measurement and this occurred two weeks following trauma exposure. It is therefore possible that the higher levels of BP observed in prior PTSD studies were a result of more chronic PTSD symptoms and sympathetic hyperarousal, which our study did not capture.

5. Discussion

This study used a prospective design to examine sex differences in autonomic functioning among a sample of recently traumatized men and women. Sex differences were observed and varied by biomarker. While men demonstrated significantly higher BP and rates of hypertension, women demonstrated significantly higher HR and lower HF-HRV, and these effects were strongest among women who subsequently developed PTSD. Further, acute sympathetic arousal (indexed via skin conductance response) associated with HR and HF-HRV during fear conditioning but only among women who developed PTSD.

Our findings regarding BP and hypertension are consistent with what is commonly observed in the general population, such that men are more likely to experience hypertension (American Heart Association, heart.org). This sex difference is known to decrease among older age groups and it is thought that decreasing estradiol levels as a result of menopause in women play a role (i.e., estradiol is cardioprotective and may explain lower rates of hypertension in pre-menopausal women; for reviews, see Colafella and Denton, 2018; Regitz-Zagrosek et al., 2016). It is important to note that the average age in our sample was 35 and thus most women were pre-menopausal. While prior studies have found that individuals with PTSD demonstrate higher BP than those without PTSD (for a review, see Buckley and Kaloupek, 2001), we did not observe an effect of PTSD status. One potential explanation is that our sample is not as highly traumatized as comparisons in prior work (e.g., most participants were in motor vehicle collisions). Similarly, BP was assessed with only one measurement and this occurred two weeks following trauma exposure. It is therefore possible that the higher levels of BP observed in prior PTSD studies were a result of more chronic PTSD symptoms and sympathetic hyperarousal, which our study did not capture.
Our findings regarding sex differences in autonomic functioning may have clinical implications. Specifically, men and women differed in their sympathetic arousal, with men demonstrating higher BP and women demonstrating higher HR. Further, women demonstrated lower parasympathetic function than men. As mentioned above, autonomic deficits have been implicated in the increased risk of cardiovascular disease in PTSD. Our findings suggest that the specific autonomic mechanisms through which cardiovascular disease develops could differ for men versus women with PTSD. For example, there is preliminary evidence that blockade of the renin-angiotensin system (responsible for BP regulation) via ace-inhibitors and angiotensin receptor blockers is associated with decreased likelihood of PTSD diagnosis (Khoury et al., 2012; Seligowski et al., 2021). We recently observed a sex effect such that the protective effects of these medications may be greater among men versus women (Seligowski et al., 2021). Thus, medications targeting BP may be more effective in men versus women with PTSD because men are more likely to experience hypertension and therefore see an effect of such medications. Prospective trials of antihypertensive medications for PTSD are needed to further explore sex differences in their effects. Another possible avenue for future trials is to determine if the autonomic deficits we observed during extinction in women with PTSD translate to clinical outcomes (e.g., do women with PTSD experience less symptom reduction from exposure treatments than men?). Thus far, sex differences in exposure-based treatments have not been reported, but we are not aware of any trials that examined sex differences in autonomic functioning during these treatments.

While capturing acute trauma reactions with a prospective design is a strength of this study, an important limitation is that our sample is not as highly symptomatic as comparisons from the literature. For example, we did not see main effects of PTSD status on BP, HR, of HF-HRV and this may be due to the recency of trauma exposure and the absence of severe PTSD symptoms in this cohort. Another limitation relates to trauma type, such that the index trauma for most participants was a motor vehicle collision and the incidence of PTSD in that population is lower than that of other trauma types, such as interpersonal violence and combat exposure (Kessler et al., 2017). Additionally, while we used a recommended cutoff for provisional PTSD diagnosis (Bovin et al., 2016) at 8-weeks, the current study relied on self-reported symptoms and did not include a structured clinical interview of PTSD. Future studies with more robust PTSD assessment among individuals with a broader range of trauma exposure will be needed to replicate and extend our findings. Despite these limitations, this study adds to a very scant literature regarding both 1) prospective assessments of posttraumatic autonomic functioning and 2) sex differences in posttraumatic autonomic functioning.

The current study identified sex differences in multiple domains of autonomic functioning among a recently traumatized sample. Our findings suggest that men and women demonstrate different patterns of sympathetic arousal, with men exhibiting higher BP and women exhibiting higher HR. Women also exhibited worse parasympathetic function as indicated by lower HF-HRV during fear conditioning, as was particularly seen in women who developed PTSD. Acute sympathetic arousal indexed by skin conductance in the emergency department was associated with higher HR and HF-HRV among women who developed PTSD, suggesting it may be a useful biomarker of subsequent autonomic functioning in this population. Additional studies examining subsequent sex differences in cardiovascular risk as a result of differential autonomic mechanisms are warranted.

CRediT authorship contribution statement

Antonia V. Seligowski: Data processing, Formal analysis, Data interpretation, Writing – original draft. Elizabeth R. Steuber: Data processing, Formal analysis, Data interpretation, Writing – original draft. Rebecca Hinrichs: Data processing, Formal analysis. Mariam H. Reda: Data processing, Formal analysis. Charis N. Wiltshire: Data processing, Formal analysis, Writing – original draft.
processing, Formal analysis.

Cassandra P. Wanna: Data processing, Formal analysis.
Sterling J. Winters: Data processing, Formal analysis.
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Stacey L. House: Funding acquisition, recruitment, logistics.
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Anna M. Chang: Funding acquisition, recruitment, logistics.

Fig. 3a. FPS by sex during fear acquisition at 2-weeks
Figure 3b. FPS by sex during fear extinction at 2-weeks.

Declaration of competing interest

In the last three years GDS has received research funding from the NSF, NIH and LifeBell AI, and unrestricted donations from AliveCor, Amazon Research, the Center for Discovery, the Gordon and Betty Moore Foundation, MathWorks, Microsoft Research, the Gates...
Foundation, Google, One Mind Foundation, and Samsung Research. GDS has financial interest in AliveCor and receives unrestricted funding from the company. He is also the CTO of MindChild Medical and CSO of LifeBell AI and has ownership in both companies. These relationships are unconnected to the current work. In the past three years, LTG has served on the Scientific Advisory Board of Sage Bionetworks, for which she received a small honorarium. SLR reports grants from NIH during the conduct of the study; personal fees from SOBP (Society of Biological Psychiatry) paid role as secretary, other from Oxford University Press royalties, other from APP (American Psychiatric Publishing Inc.) royalties, other from VA (Veterans Administration) per diem for oversight committee, and other from Community Psychiatry paid board service, including equity outside the submitted work; and Leadership roles on Board or Council for SOBP, ADAA (Anxiety and Depression Association of America), and NNDC (National Network of Depression Centers). SS has received funding from the Florida Medical Malpractice Joint Underwriter’s Association Dr. Alvin E. Smith Safety of Healthcare Services Grant; Allergan Foundation; the NIH/NIA-funded Jacksonville Aging Studies Center (JAX-ASCENT; R33AG05654); and the Substance Abuse and Mental Health Services Administration (1H79TI083101-01); and the Florida Blue Foundation. CWJ reports no direct conflicts related to this paper. He has been an investigator on studies funded by Hologic Inc, Janssen, AstraZeneca, and Vapotherm, for which his department has received research funding. JJ receives consulting payments from

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**Fig. 5a.** Sex effect on HF-HRV during fear acquisition at 2-weeks

**Figure 5b.** Interaction effect on HF-HRV during fear extinction at 2-weeks.
Table 3  
Descriptives and bivariate correlations of study variables among women with PTSD.

<table>
<thead>
<tr>
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<th>9</th>
<th>10</th>
<th>11</th>
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</thead>
<tbody>
<tr>
<td>1. eSense Baseline (ED)</td>
<td>–</td>
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<tr>
<td>2. eSense Start (ED)</td>
<td>.918**</td>
<td>–</td>
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<tr>
<td>3. eSense Max (ED)</td>
<td>.988**</td>
<td>.825**</td>
<td>–</td>
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<tr>
<td>4. eSense End (ED)</td>
<td>.672**</td>
<td>.776**</td>
<td>.863**</td>
<td>–</td>
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<tr>
<td>5. BP – Systolic (2-weeks)</td>
<td>.067</td>
<td>.025</td>
<td>.056</td>
<td>.139</td>
<td>–</td>
<td></td>
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<tr>
<td>6. BP – Diastolic (2-weeks)</td>
<td>–.182</td>
<td>–.169</td>
<td>–.021</td>
<td>–.029</td>
<td>–.851**</td>
<td>–</td>
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<tr>
<td>7. HR – Acquisition (2-weeks)</td>
<td>.287</td>
<td>.390**</td>
<td>.339</td>
<td>.386</td>
<td>.237</td>
<td>–.256</td>
<td>–</td>
<td></td>
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<tr>
<td>8. HRV – Acquisition (2-weeks)</td>
<td>.259</td>
<td>.232</td>
<td>–.157</td>
<td>–.368</td>
<td>.268</td>
<td>.327</td>
<td>–.566**</td>
<td>–</td>
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<tr>
<td>9. HR – Extinction (2-weeks)</td>
<td>.369</td>
<td>.411*</td>
<td>.417*</td>
<td>.536*</td>
<td>.123</td>
<td>.207</td>
<td>.967**</td>
<td>.460*</td>
<td>–</td>
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<tr>
<td>10. HRV – Extinction (2-weeks)</td>
<td>–.491*</td>
<td>.341</td>
<td>–.301</td>
<td>–.530*</td>
<td>.029</td>
<td>.093</td>
<td>.401*</td>
<td>.877**</td>
<td>–.339</td>
<td>–</td>
<td></td>
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<tr>
<td>11. PTSD symptoms (8-weeks)</td>
<td>–.118</td>
<td>–.178</td>
<td>–.309</td>
<td>.235</td>
<td>.262</td>
<td>–.232</td>
<td>.331</td>
<td>.174</td>
<td>.387</td>
<td>.321</td>
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<tr>
<td>Mean</td>
<td>2.69</td>
<td>3.03</td>
<td>5.36</td>
<td>3.27</td>
<td>122.58</td>
<td>83.08</td>
<td>73.04</td>
<td>6.16</td>
<td>74.45</td>
<td>6.23</td>
<td>46.98</td>
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<tr>
<td>SD</td>
<td>1.87</td>
<td>2.10</td>
<td>3.34</td>
<td>1.75</td>
<td>18.95</td>
<td>13.95</td>
<td>11.60</td>
<td>1.05</td>
<td>11.20</td>
<td>1.13</td>
<td>10.50</td>
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<tr>
<td>Minimum</td>
<td>.43</td>
<td>.50</td>
<td>.62</td>
<td>.43</td>
<td>96.00</td>
<td>57.00</td>
<td>53.41</td>
<td>3.32</td>
<td>54.02</td>
<td>3.30</td>
<td>33.30</td>
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<tr>
<td>Maximum</td>
<td>8.80</td>
<td>6.95</td>
<td>14.90</td>
<td>6.58</td>
<td>171.00</td>
<td>118.00</td>
<td>94.96</td>
<td>7.49</td>
<td>92.81</td>
<td>7.80</td>
<td>7.80</td>
</tr>
</tbody>
</table>

Note. *p < .05; **p < .01; BP = blood pressure; HR = heart rate; HRV = heart rate variability.

Acknowledgements

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.nestr.2021.100384.

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References

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