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Effects of daily posttraumatic stress disorder symptoms on heart rate variability

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Abstract

Objective: Posttraumatic stress disorder (PTSD) is common, debilitating, and associated with increased risk for health problems, including cardiovascular disease (CVD). PTSD is related to poor autonomic function indicated by reduced heart rate variability (HRV). Yet very little work has tested the timescale or direction of these effects, given most evidence comes from cross-sectional studies. Documentation of when effects occur and in what direction can shed light on mechanisms of CVD risk and inform treatment. The present study of 169 World Trade Center (WTC) responders, oversampled for PTSD, tested how daily PTSD symptoms were associated with autonomic function as reflected through HRV.

Methods: Participants (N=169) completed surveys of PTSD symptoms 3x/day at 5-hour intervals for 4 days while also wearing ambulatory monitors to record electrocardiograms to derive HRV (i.e., mean absolute value of successive differences [MAVSD] between beat-to-beat intervals).

Results: HRV did not predict PTSD symptoms. However, PTSD symptoms during a 5-hour interval predicted reduced HRV at the next 5-hour interval ($\beta = -0.09$, 95% CI [−0.16, −0.02], $p = .008$). Results held adjusting for baseline age, current heart problems, and current PTSD diagnosis.

Conclusions: Findings underscore growing awareness that PTSD symptoms are not static. Even their short-term fluctuations may affect cardiovascular functioning, which could have more severe impacts if disruption accumulates over time. Research is needed to determine if momentary interventions can halt increases in PTSD symptoms or mitigate their impact on cardiovascular health.

Keywords

posttraumatic stress disorder; heart rate variability; ecological momentary assessment; multilevel modeling; first responders; World Trade Center

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Data are available upon request and all analysis code is available in Supplemental Digital Content 1. Analyses were not pre-registered.

Following the 9/11 attacks on the World Trade Center (WTC), thousands of people responded to assist with rescue, recovery, and cleanup [1], and in the process were exposed to traumatizing events and environmental toxins. In the years since, many developed posttraumatic stress disorder (PTSD), a condition characterized by recurring, intrusive thoughts of a trauma, avoidance of reminders of it, negative alterations in cognitions and mood, and alterations in arousal and reactivity [2]. A study of over 28,000 responders assessed 2-3 years after the attack found rates of PTSD between 6.2% for police and as high as 21.2% for non-traditional responders [3]. Even more than a decade after the attack, a substantial portion of responders continued to have symptoms consistent with ongoing PTSD [4].

PTSD can be a debilitating condition on its own [5, 6], but it also increases risk for a number of medical comorbidities [7, 8]. A robust literature has linked PTSD and cardiovascular disease (CVD). Across five prospective cohort studies (401,712 total participants), effect sizes of PTSD on incident CVD and/or cardiac mortality ranged from 1.46 to 3.28 [9-13]. These effects were evident even after controlling for a host of potentially confounding sociodemographic and comorbid mental health factors (e.g., gender, race/ethnicity, depression). An increased odds of CVD has also been observed in WTC responders. Specifically, exposure to 9/11 trauma and toxins (i.e., early exposure on the day of the attacks or prolonged work at the cleanup site) is linked with increased rates of CVD in firefighters who responded to the attacks [14]. Together, these studies provide strong evidence PTSD may be a distinct risk factor for the onset of CVD.

One potential mechanism linking PTSD and CVD may be dysfunction of the autonomic nervous system (ANS), which is composed of the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). The SNS is involved in the “fight-or-flight” response and is activated during times of stress or physical exertion. The PNS is involved in the “rest-and-digest” response and is responsible for conserving energy and returning cardiovascular activity to baseline via vagal innervation. One biomarker of SNS and PNS functioning is heart rate variability (HRV), i.e., variation in the time between heartbeats, as measured by the beat-to-beat (R-R) interval. Reduced HRV can occur with excessive SNS activity and decreased PNS activity [15, 16], and strongly predicts elevated risk for cardiac events and cardiovascular-related mortality [17].

Across multiple studies, individuals with PTSD show reduced HRV compared to controls. In a 2013 meta-analysis, individuals with PTSD exhibited reduced high-frequency HRV (HF-HRV) and low-frequency-HRV (LF-HRV) compared to those without PTSD (Hedge’s $g = -2.27$ and -1.72, respectively) [18]. However, this meta-analysis revealed high levels of heterogeneity and excluded unpublished research, which may have biased findings. A larger, more recent meta-analysis that included unpublished data confirmed those with PTSD have lower HF-HRV and LF-HRV at rest ($g = -0.23$ and -0.27, respectively), as well as lower HF-HRV during stress ($g = -0.24$) [19]. These results suggest PTSD is associated with dysfunction of the ANS — specifically lower parasympathetic activity and greater sympathetic activity — as evidenced by reductions in HRV compared to controls.

However, whether PTSD leads to reduced HRV and vice versa, including the timeframe over which these effects occur, is unclear. Although PTSD is associated with autonomic dysfunction, autonomic dysfunction also predicts PTSD. For example, low- to high-frequency (LF:HF) HRV ratio predicts post-deployment PTSD among combat soldiers [20], and higher heart rate immediately after trauma exposure predicts later development of PTSD [21]. To date, all but a few studies examining PTSD symptoms and HRV have been cross-sectional or have relied on assessments with long intervals between them, obscuring how and when effects occurred. Two exceptions are studies that have considered momentary associations between HRV and PTSD in naturalistic settings. These studies (both based on the same sample) found PTSD symptoms were associated with reduced HRV during wake [22] and sleep [23]. However, this sample consisted of young adults, who have different PTSD and/or HRV patterns compared to mid-life adults [24]. These studies also did not examine lagged associations to better infer temporal sequencing between PTSD and HRV. As such, it is unclear if momentary PTSD symptoms predict concurrent or later HRV, and/or vice versa.

Using novel methodologies such as ecological momentary assessment (EMA), reciprocal relationships between PTSD and HRV can be assessed non-invasively in real-time contexts. Examining these pathways in more naturalistic settings may confirm one part of the mechanism by which PTSD psychopathology is linked to CVD with greater ecological validity than laboratory-based studies. Such findings also would suggest that short-term exacerbations of PTSD symptoms are more important for HRV than previously recognized. EMA also may reveal a new way to conceptualize intervention efforts, via deployment of strategies in moments when individuals experience heightened symptoms [25].

The primary aim of the current study was to use EMA to examine bidirectional associations between PTSD symptoms and HRV in WTC responders. We hypothesized that greater PTSD symptoms within a 5-hour interval would be associated with reduced HRV concurrently. We also hypothesized PTSD symptoms would predict subsequent reductions in HRV, and likewise, lower HRV would predict subsequent increases in PTSD symptoms. As a secondary aim, to establish specificity, we also examined similar bidirectional associations between PTSD and heart rate (HR).

Methods

Participants

The target sample consisted of 202 WTC responders (M_{age} = 54.28, SD = 9.69) recruited from the Long Island site of World Trade Center Health Program between October 2014 and February 2016 for a study on PTSD and health. Participants in the larger study were oversampled for current PTSD, such that 39 (19%) had a current diagnosis of PTSD. Detailed information about the larger study and full sample characteristics have been reported elsewhere [26, 27]. Of the full 202 sample, 33 participants were removed due to missing heart rate variability data or daily PTSD data across all days (see data processing below), resulting in a final sample of 169 participants for analyses.

In the current study, the mean age of the final sample of 169 participants was 54.04 (*SD* = 9.62). The sample was primarily male (83%, *n* = 140), White (89%, *n* = 150), and non-Hispanic (82%, *n* = 139), and had an average of 14.71 (*SD* = 2.22) years of education. Most participants were non-smokers (90%, *n* = 152) and did not have a history of heart problems (85%, *n* = 144). Thirty participants (18%) had a current diagnosis of PTSD, and 63 (37%) had a lifetime diagnosis of PTSD. Mean PCL-5 scores were moderate to high (*M* = 39.27, *SD* = 17.02). The study was approved by the Stony Brook University Committees on Research Involving Human Subjects, and all participants provided written informed consent.

Procedure

Participants first completed diagnostic interviews during a baseline assessment. Following the baseline assessment, participants were trained on the EMA protocol, as well as on how to wear the heart monitor (see below for details). The heart monitor was worn for 4 days, and the PTSD assessments were completed 3x/day for 7 days; however, only the 4 days of PTSD data with corresponding HRV data were used for the current study. Participants were trained to complete surveys on an iPod provided by the research coordinator. Participants' schedules for the upcoming week were reviewed, and the participant and study coordinator agreed upon times to complete the assessments with approximately 5-hour intervals between them. Although a few variations of assessment time occurred, almost all assessment times tended to be at the same fixed time for all the participants (i.e., mid-morning, afternoon, evening). Alarms were set for these times so participants would be prompted to complete surveys. The average EMA compliance rate across participants was 93.8% (ranging from 55% to 100%), indicating high levels of compliance after training. Participants were also followed up with a call the day after study initiation to answer any questions related to the EMA protocol.

Measures

Baseline measures.

Demographics and medical status.: During baseline interviews, demographic information was collected. A checklist was also used to record any self-reported history of medical diagnoses, including hypertension or any cardiovascular conditions.

PTSD diagnosis.: The Structured Clinical Interview for DSM-IV (SCID) [28] was administered by experienced master's-level interviewers, who were closely supervised by two clinical psychologists (R.K. and C.R.). The interviews were administered to all participants during baseline assessment. PTSD diagnosis was operationalized as meeting the DSM-IV diagnostic criteria [2]. Previous assessments of reliability of the trained interviewers in this population demonstrated good inter-rater agreement ($\kappa = 0.82$) [4].

EMA measures.

Daily PTSD symptoms.: Eight items selected from the PCL-5 [29] were used to assess PTSD symptoms three times per day. Participants were instructed to rate each symptom in reference to "the past 5 hours" on a 5-point Likert scale from 1 (*not at all*) to 5 (*extremely*), and the 8 items were then averaged together. The eight items were selected

based on the four-factor (i.e., intrusion, avoidance, numbing, and hyperarousal) emotional numbing model of PTSD symptoms [30]. This model was selected for the present study because (1) it has received strong empirical support [31-33] and (2) it was very similar to the four-factor model proposed in the DSM-5. Based on factor analytic results in each PTSD symptom dimension and an intention of balancing reliability and validity to avoid the "attenuation paradox" [34], two items were chosen for each PTSD dimension. The eight items used in the EMA were: "I had repeated, disturbing and unwanted memories of the stressful experience" and "I felt very upset because something reminded me of the stressful experience" (intrusion); "I avoided memories, thoughts, or feelings related to the stressful experience" and "I avoided external reminders of the stressful experience" (avoidance); "I felt distant or cutoff from other people" and "I had strong negative feelings" (numbing); and "I felt jumpy or easily startled" and "I was 'super alert' or watchful or on guard" (hyperarousal). Given the repeated assessments of PTSD symptoms, reliability for this abbreviated version of the PCL-5 was calculated within a multilevel framework [35]. Specifically, in the present sample, the within-person reliability (i.e., reliability of change) was R_C = .78 and the between-person reliability was R_{KF} = .99.

Daily heart rate variability (HRV).: Ambulatory electrocardiography (ECG) signals were recorded in naturalistic settings for four days using the Actiheart device [36]. Participants were instructed to use the monitor during waking periods and were trained on placement of electrodes and the monitor during their baseline visit. Each morning, the recorder was fixed to participants' upper chest using electrodes placed just below the apex of the sternum and a second electrode running horizontally from the monitor as lateral as possible, given prior work suggesting such placement is associated with higher ECG amplitude and reduced noise from movement artifacts [37].

Actiheart signals were band-pass filtered (10-35 Hz), sampled at a frequency of 128 Hz, and processed with a QRS detection method algorithm [36]. Additional filtering contrasted each beat-to-beat (R-R) interval with the average of its two neighbors (i.e., average of the prior and subsequent R-R intervals). Intervals exceeding a 20% difference from the average of its neighbors (either above or below the 20% threshold) were flagged in SAS software, as were intervals less than 300 ms or greater than 1800 ms. Flagged intervals that were between 1.6 and 2.4 times the length of its neighbors were split into two intervals. All other flagged intervals were deleted.

After this filtering, heart rate variability (HRV) was calculated using the mean absolute value of successive differences (MAVSD) between consecutive R-R intervals averaged across the 5-hour period that preceded the EMA surveys. As a supplemental metric of HRV, we also calculated the root mean square of successive differences (RMSSD) between consecutive R-R intervals averaged across the 5-hour period that preceded the EMA surveys. Heart rate (HR) was calculated as the mean number of R-R intervals per minute averaged across the 5-hour period preceding the EMA surveys. Any 5-hour period with more than 50% missing heart rate data was treated as missing for the entire interval. On average, people had a mean of 9.18 (SD = 2.54) out 12 possible data points with both valid HRV and PTSD data across the study period (i.e., 3 five-hour waking segments per day x 4 days), indicating that most 5-hour segments provided usable data for analyses. Lagged variables were created within

each day. However, lags from evening to morning surveys were excluded because of the qualitative difference of heart rate and symptom experiences during sleep from daytime.

Statistical Analysis Plan

To address the study aims, longitudinal two-level multilevel modeling (MLM) was conducted, with Level 1 being the 5-hour repeated assessment level, and Level 2 being the person level. PTSD symptom severity was calculated at each assessment by averaging the eight PCL items. All MLM analyses were conducted using the *nlme* package [38] in R [39]. All models were estimated with random intercepts and random slopes to account for expected between-person differences. Given the repeated assessments of the current study, all the models were estimated with an autocorrelation structure of order 1 (AR[1]) [40]. R code is available in the Supplemental Digital Content.

The associations between PTSD symptom severity and HRV were examined in two ways. First, the effects of HRV on PTSD were estimated. Two separate models, with one assessing concurrent associations and the other assessing lagged associations, were specified. For the concurrent model, PTSD symptom severity was regressed on HRV recorded during the same period. For the lagged model, PTSD symptom severity was regressed on one-time lagged HRV (i.e., HRV during the previous period). Second, the effects of PTSD on HRV were estimated in a similar fashion (i.e., HRV was regressed on concurrent and one-time lagged PTSD symptom severity). To establish specificity of PTSD-HRV associations, we also examined similar models with HR (i.e., R-R Interval) as a concurrent or lagged predictor and outcome in separate models.

To further examine the robustness of the associations between PTSD and HRV, the aforementioned MLM analyses were then repeated covarying for age [41], any current heart problems [42], and current PTSD diagnosis [19]. Given multiple tests, an alpha level of < .01 was used for establishing significance. Standardized regression estimates were calculated using the *effectsize* R package [43]. Following the recommendations by Singer and Willett (2003) [44], for significant effects, we calculated the proportion reduction in variance (PRV) [45] as an index of effect size using the equation: PRV = [σ^2(null model with no predictors) - σ^2 (model of interest)] / σ^2(null model with no predictors), where σ^2 represents within-person (Level 1) variance estimated in the present study.

Results

Mean momentary PTSD scores were 1.57 (*SD* = 0.73, range = 1-5; skewness = 1.60, kurtosis = 2.34; Figures S1-S2, Supplemental Digital Content). Mean HRV values measured via MAVSD were 18.62 (*SD* = 10.86, range = 3.03 to 102.38; skewness = 1.96, kurtosis = 6.51; Figures S3-S4). Mean HRV values measured via RMSSD were 35.38 (*SD* = 18.90, range = 5.93 to 133.97; skewness = 1.14, kurtosis = 1.56; Figures S5-S6). Mean HR (i.e., R-R interval) values were 732.24 (*SD* = 101.17, range = 471.33 to 1076.22; skewness = 0.32, kurtosis = −0.33). There was a strong positive correlation between the two metrics of HRV (i.e., MAVSD and RMSSD): r = .90, 95% CI [0.89, 0.91], p < .001. Intraclass correlation coefficients revealed 80% of the variance in PTSD, 64% of the variance in HRV

(MAVSD), 59% of the variance in HRV (RMSSD), and 55% of the variance in HR was at the between-person level, respectively.

Table 1 presents concurrent and lagged associations between PTSD, HRV, and HR. Neither HR nor HRV in the prior or concurrent time period were significantly associated with PTSD. Similarly, concurrent PTSD was not associated with HR or HRV. However, PTSD during a given time interval had a significant effect on HRV during the next 5-hour interval (i.e., increased PTSD symptoms during a 5-hour interval predicted lower HRV [MAVSD] in the subsequent 5-hour interval: $\beta = -0.09$, 95% CI [−0.16, −0.02], $p = .008$; accounting for 11.4% of the within-person variance; Figure 1). (Results were nearly identical when using RMSSD as an alternative metric of HRV: $\beta = -0.11$, 95% CI [−0.19, −0.04], $p = .003$; accounting for 6.9% of the within-person variance; in Table S1, Supplemental Digital Content). After covarying for age, current heart problems, and current PTSD diagnosis determined via the SCID, the association between momentary PTSD and subsequent HRV remained statistically significant ($\beta = -0.09$, 95% CI [−0.16, −0.02], $p = .012$). All other associations remained statistically non-significant after adjusting for the same covariates. Given the positive skew of PTSD symptoms and HRV and non-normal residuals, a sensitivity analysis with the natural log of HRV was also performed; results were essentially unchanged.

Discussion

PTSD can exact a major toll on health, including increased risk for CVD. A number of cross-sectional studies show PTSD is associated with poor autonomic function, as indexed by lower HRV [18, 19], with effects similar to other CVD risk factors such as BMI and age. Yet the time scale and direction of associations between PTSD and reduced HRV has not been well studied or understood given most evidence is based on cross-sectional research.

The present study of 169 trauma-exposed WTC responders documented in near real-time the close link between changes in PTSD symptoms throughout the day and effects on HRV in the following hours. Specifically, across a 4-day period of monitoring, we documented how periods of increased PTSD during a 5-hour interval predicted decreased HRV in the subsequent 5-hour interval. Although effects were small, the fine-grained resolution of this study suggests that even small effects may accumulate to produce major clinical impact. Two major implications stand out.

First, this window into the timescale of effects is not only novel but has important clinical significance. Assessment and treatment of PTSD rarely considers day-to-day fluctuations of symptoms. Emerging evidence shows PTSD symptoms are not static but rather show high degrees of variability around a person’s average within and across days [22, 46-48]. Findings from the current study reveal the impact of this variability: on average, spikes in PTSD symptoms degraded autonomic function, at least temporarily. If one conceptualized PTSD symptoms as a stressor in and of itself, then findings from this study merge with a body of literature showing how stress affects HRV [49] and how certain interventions (e.g. slow-breathing, yoga, mindfulness-based stress reduction) may mitigate that impact [50, 51].

Whether this dynamic has more enduring, cumulative impacts on autonomic function and risk for CVD could not be tested in our study, as this requires longer follow-up. Yet, such a hypothesis is plausible, and underscores that short-term fluctuations in PTSD may not merely represent noise. Rather, they may represent a micro-process through which PTSD exacts its long-term negative effects on health. Fluctuations in PTSD impact other processes important for health, such as sleep [52] and inflammation [53, 54], but the present findings suggest one particular pathway by which they can have negative effects: via autonomic dysregulation. This finding has important potential implications, as autonomic imbalance (i.e., overactive SNS and/or underactive PNS) is robustly associated with increased chronic disease risk, premature aging, and all-cause mortality [55, 56].

Perhaps most importantly, our findings augment other literature showing ill effects of these short-term spikes in PTSD [57-59] and create more support for developing strategies to address short-term PTSD elevations. For example, deploying in-the-moment interventions may help mitigate the ill effects of PTSD on cardiovascular health. Previous studies show that use of a momentary “worry outcomes journal” and stress management software are effective at reducing anxiety, and may show similar promise for treating PTSD [60, 61]. HRV biofeedback has also been shown to help reduce PTSD symptoms in soldiers [62], which may be a promising option for deployment in real-time contexts.

Second, the direction of effects is noteworthy. A significant body of work posits that disruptions in autonomic function may provoke PTSD symptoms [20, 21]. However, our results suggest this is not the case, at least in the short-term. There was no evidence that reduced HRV led to subsequent changes in PTSD. Such clarification is important, because it helps to establish how PTSD symptoms may be driving poor autonomic health, rather than the reverse. Interestingly, these effects of PTSD on HRV may not be immediate, but rather take several hours to manifest. However, additional work is needed to understand the optimal measurement intervals. The direction of the effects we observed underscores and merges with a large literature showing how treatment of PTSD not only benefits mental health but also physical health markers [63].

Directions for Future Research

Despite the novelty of the findings and their clinical implications, study limitations qualify these conclusions. Most importantly, effects were small, precluding power to test for moderators of these effects. It may be that only certain individuals are susceptible to impacts of PTSD on autonomic function. For example, women are at higher risk for developing lower HRV after trauma exposure [64, 65]. In addition, our sample consisted of mostly White male responders, limiting generalizability. Future studies should investigate whether results hold in samples with a greater percentage of women and racial/ethnic minorities, and with different types of trauma exposure. It may also be that unmeasured variables – such as daily stress – were responsible for both PTSD symptoms as well as degraded autonomic function, making the observed relationship spurious. Future studies would benefit from testing whether PTSD predicts other types of multisystem physiological dysregulation, such as elevated blood pressure, flattened diurnal cortisol slopes, or altered inflammatory biomarker activity.

Regarding limitations of our HRV measurement approach, we used a relatively low sampling frequency of 128 Hz for HRV, which may have obscured effects. Further, participants only wore the Actiheart device during waking hours. Those with PTSD have reduced HRV during sleep [70]. We also did not collect data on physical activity patterns during the day, which may have affected both PTSD and HRV. Finally, to increase reliability, we aggregated HRV within 5-hour intervals, but HRV more proximal to PTSD assessments (e.g., within 30-60 minutes) may also be important to assess in future studies.

Conclusion

With these caveats in mind, our results nevertheless suggest, for the first time, that the impact of PTSD symptoms on cardiovascular functioning happens on an almost hourly basis. The long-term consequences of these effects remain to be evaluated, but addressing these symptom fluctuations may be an important target for future interventions and may have downstream benefits on cardiovascular health [55]. Ecological momentary interventions (EMIs), where coping strategies are deployed electronically in real time, may be a promising direction for future research. Cognitive-behavioral EMIs are feasible and effective at reducing mental health symptoms [71]. Taken together, our results demonstrate the long-standing aftermath that 9/11 has had on the health of responders who assisted with rescue and clean-up efforts. Given the large global burden of anxiety disorders such as PTSD [72], particularly among first responders, finding ways to mitigate their impacts is of critical public health importance.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Conflicts of Interest and Source of Funding:

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Abbreviations

ANS	autonomic nervous system
BMI	body mass index
CVD	cardiovascular disease
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders, fourth edition

ECG	electrocardiography
EMA	ecological momentary assessment
EMI	ecological momentary intervention
HF-HRV	high-frequency heart rate variability
HR	heart rate
HRV	heart rate variability
LF-HRV	low-frequency heart rate variability
MAVSD	mean absolute value of successive differences
PCL-5	PTSD Checklist for DSM-5
PNS	parasympathetic nervous system
PTSD	posttraumatic stress disorder
RMSSD	root mean square of successive differences
R-R interval	beat-to-beat interval
SCID	Structured Clinical Interview for DSM-IV
SNS	sympathetic nervous system
WTC	World Trade Center

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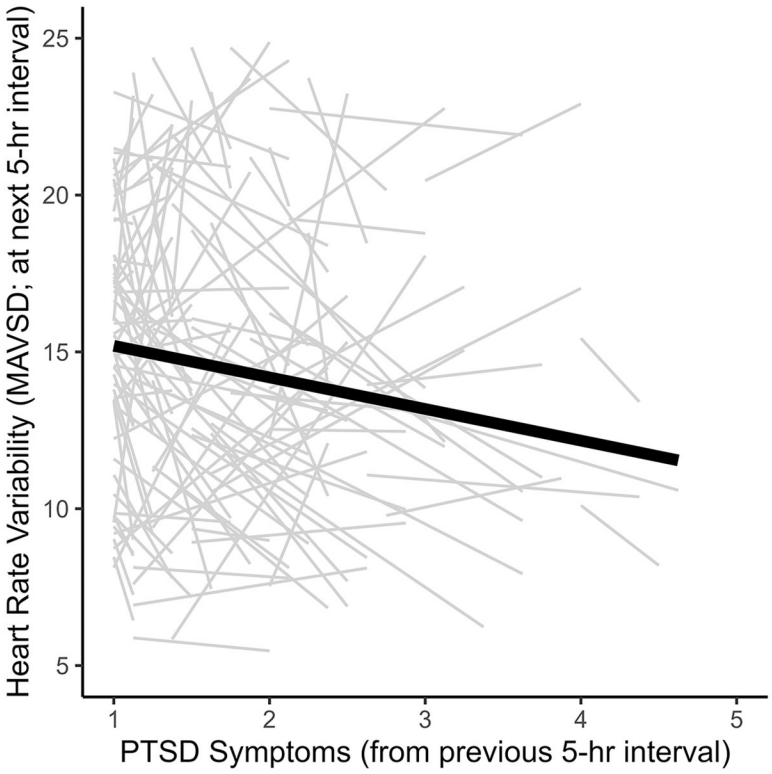

Figure 1. Association between PTSD and Subsequent HRV

Note. PTSD = posttraumatic stress disorder, MAVSD = mean absolute value of successive differences. Light gray lines represent the association between PTSD symptoms (from the previous 5-hour interval) and heart rate variability (HRV measured via MAVSD; at the next 5-hour interval) for each individual participant in the sample (i.e., the random slopes). The black line represents the average negative association between PTSD and HRV (MAVSD) pooled across the full sample ($\beta = -0.09$, 95% CI [−0.16, −0.02], $p = .008$).

Table 1.

Fixed-Effect Standardized Estimates for HR and HRV Predicting PTSD, and PTSD Predicting HR and HRV (Concurrent and Lagged Effects)

Outcome: PTSD							
	β	***95% CI***	***p***	***N_{ID}***	***Obs.***	***R^2 (cond.)***	***R^2 (marg.)***
Concurrent effects							
HR	<0.01	[−0.03, 0.04]	.72	169	1608	0.81	<0.01
HRV (MAVSD)	−0.01	[−0.05, 0.03]	.50	166	1524	0.82	<0.01
Lagged effects							
HR	0.02	[−0.02, 0.05]	.30	169	1548	0.82	<0.01
HRV (MAVSD)	−0.02	[−0.07, 0.03]	.47	166	1469	0.83	<0.01
Outcome: HR							
	β	***95% CI***	***p***	***N_{ID}***	***Obs.***	***R^2 (cond.)***	***R^2 (marg.)***
Concurrent effects							
PTSD	<0.01	[−0.07, 0.07]	.98	169	1608	0.53	<0.01
Lagged effects							
PTSD	0.01	[−0.06, 0.08]	.78	169	1513	0.54	<0.01
Outcome: HRV (MAVSD)							
	β	***95% CI***	***p***	***N_{ID}***	***Obs.***	***R^2 (cond.)***	***R^2 (marg.)***
Concurrent effects							
PTSD	−0.01	[−0.07, 0.06]	.89	166	1524	0.59	<0.01
Lagged effects							
PTSD	**−0.09**	**[−0.16, −0.02]**	**.008**	**166**	**1442**	**0.61**	**0.01**

Note. Bold values represent statistically significant effects ($p < .01$). Each model was estimated separately (i.e., concurrent vs. lagged effects, HR vs. HRV), but is presented together for brevity. For lagged effects, the independent variable was one-time lagged to reflect the previous recording period, which was used to predict the dependent variable at the next recording period. PTSD = posttraumatic stress disorder, HR = heart rate (R-R Interval), HRV = heart rate variability (i.e., mean absolute value of successive differences [MAVSD]), β = standardized regression estimate, 95% CI = 95% confidence interval, p = *p*-value, N_{ID} = number of persons included in the analysis; Obs. = total number of repeated measures observations at Level 1, R^2 (cond.) = conditional R-squared (i.e., percentage of within-person variance accounted for by both fixed and random effects), R^2 (marg.) = marginal R-squared (i.e., percentage of within-person variance accounted for by the fixed effects only). All results indicate unadjusted associations (i.e., non-transformed dependent variables and models without covariates).