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Resting Heart Rate Variability: Exploring Associations with Symptom Severity in Adults with Substance Use Disorders and Posttraumatic Stress

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Abstract

Objective: Substance use disorders (SUDs) and post-traumatic stress symptoms are commonly comorbid. Previous studies have established that those with SUD or post-traumatic stress disorder (PTSD) have lower high frequency-heart rate variability (HF-HRV) compared to controls, suggesting low HF-HRV may be a biomarker of a common physiological mechanism underlying both disorders. We evaluated HF-HRV as a potential biomarker of a common underlying process by testing whether lower HF-HRV related to greater severity of substance use and PTSD symptoms in individuals with both substance use disorders and at least four symptoms of PTSD.

Methods: HF-HRV was measured in 49 adults with SUD and at least four symptoms of PTSD. We performed a series of regressions controlling for age to test whether low HF-HRV was associated with greater SUD and PTSD symptom severity. SUD symptoms were measured by the Addiction Severity Index and PTSD symptoms were measured by the Clinician-Administered PTSD Scale and the PTSD Checklist.

Results: After controlling for age, low resting HF-HRV was significantly associated with drug and alcohol symptom severity but not PTSD symptom severity.

Conclusions: HF-HRV may be more sensitive to the severity of drug and alcohol use rather than PTSD. Findings may suggest that in PTSD populations, HF-HRV may primarily index comorbid SUD symptoms. HF-HRV could serve as an objective measure of substance use severity and should be further investigated as a predictor of outcomes in treatment for SUD.

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Keywords

heart rate variability; substance use; trauma; post-traumatic stress; PTSD; addiction

Introduction

Substance use disorder (SUD) and post-traumatic stress disorder (PTSD) commonly co-occur (Back, Waldrop, & Brady, 2009; Najavits et al., 2007). Individuals with SUD who also present with either full PTSD or subthreshold PTSD symptoms have a more chronic and costly clinical course (Back et al., 2009; Pietrzak, Goldstein, Southwick, & Grant, 2011; Vujanovic, Bonn-Miller, & Petry, 2016). Therefore, it is important to identify common mechanisms of pathology, potential points of intervention, and corresponding biomarkers for SUD and PTSD symptoms, to inform and track the effects of interventions on underlying biological processes.

Heart rate variability (HRV), or the time interval between heart beats, may be a useful biomarker of the difficulties with physiological flexibility and emotional regulation that are present in both SUD and PTSD (Appelhans & Luecken, 2006b; Balzarotti, Biassoni, Colombo, & Ciceri, 2017). High frequency-heart rate variability (HF-HRV) reflects increases and decreases in heart rate that occur with respiration, and is thought to indicate the strength of parasympathetic influence on the heart through the vagus nerve (Thayer, Hansen, Saus-Rose, & Johnsen, 2009). The vagus nerve acts as a tonic “brake” on the heart, and through it the parasympathetic system can quickly regulate cardiac output to meet situational demands (Porges, 2001). High levels of HF-HRV are thus thought to indicate greater ability to adjust physiological arousal and regulate emotion in response to situational demands, whereas low levels indicate autonomic inflexibility (Appelhans & Luecken, 2006a; Beauchaine & Thayer, 2015; Thayer & Lane, 2007).

Consistent with this, low HF-HRV has been shown to be associated with both SUDs and post-traumatic stress. For example, a recent meta-analysis found that alcohol dependence was associated with lower HRV (Quintana, McGregor, Guastella, Malhi, & Kemp, 2013), and a meta-analysis of seven studies found lower resting HF-HRV in individuals with PTSD as compared to healthy controls (Chalmers, Quintana, Abbott, & Kemp, 2014; Sammito, Thielmann, Zimmermann, & Böckelmann, 2015). There are several plausible pathways linking HF-HRV to SUD and PTSD. First, low HF-HRV may predispose individuals to SUD and PTSD as less able to regulate emotion may lead to more adverse impacts of trauma and more likelihood of using substances to cope with emotions. Consistent with this possibility, low HF-HRV predicts development of PTSD following combat-related trauma in veterans, although a corresponding study has not been conducted in SUDs (Minassian et al., 2015). It is also possible that trauma or substance use impairs parasympathetic functioning, either through a psychological mechanism such as chronic stress (Milivojevic & Sinha, 2018), or in the case of substances, physiological effects on cardiac health and regulation (Bau et al., 2010).

Although prior findings indicate HF-HRV differs between individuals with and without SUD and PTSD, it is not known whether HF-HRV changes in conjunction with changes in SUD

and PTSD symptom severity and vice versa. A relationship with symptom severity is an important characteristic of a putative biomarker, particularly if the ultimate goal is to measure the effect of interventions intended to improve physiological functioning. In addition, most studies to date have examined HF-HRV in individuals with either SUD or PTSD symptoms rather than both. One previous study that examined HF-HRV in individuals with comorbid alcohol use disorder (AUD) and PTSD versus PTSD alone did not find lower HF-HRV in the comorbid group as compared to the PTSD-alone group (Ray, Pyne, & Gevirtz, 2017). However, the substances examined were limited to alcohol, and the focus of the study was on categorical diagnosis rather than symptom severity. Therefore, our aim was to test the hypothesis that resting HF-HRV would be negatively correlated with both SUD and post-traumatic stress symptom severity in adults with comorbid SUDs and PTSD symptoms.

Methods

Recruitment

Participants were adults ages of 18 to 65, with substance dependence on any substance (other than exclusive nicotine dependence) per the *DSM-IV* (American Psychiatric Association [APA], 2000), history of trauma exposure per the *DSM-5* (APA, 2013) Criterion A, and at least four current symptoms of PTSD per the *DSM-5* (Criteria B-E). The Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1997) was conducted to determine current (past month) psychiatric diagnoses, including SUD diagnoses. Participants were seeking treatment for substance dependence and trauma-related symptoms at an outpatient research clinic. There was a complete discussion of the study with potential participants, after which informed consent was obtained as per the IRB-approved protocol. The study included individuals with subthreshold PTSD symptoms based on: (1) comparable rates of distress and impairment in full versus subthreshold PTSD (McLaughlin et al., 2015; Pietrzak et al., 2011); and (2) high rates of subthreshold PTSD in SUD populations (Najavits et al., 2003; Yarvis, Bordnick, Spivey, & Pedlar, 2005; Yarvis & Schiess, 2008). Exclusionary criteria included exclusive (only) nicotine dependence and alcohol or opioid dependence requiring detoxification. Data for the current analysis came from the baseline session of a clinical trial of a novel treatment for comorbid SUD and traumatic stress (NCT02461732; Vujanovic, Smith, Green, Lane, and Schmitz 2018).

Heart Rate Variability

Electrocardiograms were collected during a 5-minute rest period using disposable Ag/AgCL electrodes in a modified Lead II placement on the chest. Signals were amplified by a Biopac ECG100C amplifier (Biopac Systems, Inc., Goleta, CA) with a 35 Hz low pass notch filter and 0.05 Hz high pass filter, and sampled at 1,000 Hz by a Biopac MP150. We used Biopac AcqKnowledge software to detect R-waves using auto threshold detection with a noise rejection interval of 5% of the peak-to-peak range, and windowing of 40 to 120 beats-per-minute. Inter-beat intervals were edited using CardioEdit software and then HF-HRV was quantified using CardioBatch software (Brain-Body Center, University of Illinois at Chicago, Chicago IL) according to the moving polynomial method (Porges, 1985; Porges &

Bohrer, 1990) with standard adult HF-HRV settings: 2 Hz sample rate, frequency window of 0.12 – 0.40 Hz, and 30 s epoch length. We selected this method as frequency domain measures of HF-HRV are more appropriate for shorter recording times (i.e., 5 minutes) than time domain measures such as the standard deviation of NN intervals (SDNN; Xhyheri, Manfrini, Mazzolini, Pizzi, & Bugiardini, 2012).

Measures of SUD Severity

Addiction Severity Index-Lite (ASI; Cacciola, Alterman, McLellan, Lin, & Lynch, 2007; McLellan et al., 1992) is a semi-structured interview determined past-month SUD symptom severity for alcohol (ASI Alcohol) and drugs (ASI Drugs). The interview also assessed past-month prescription medication for physical and psychiatric health.

Measures of PTSD Severity

Clinician Administered PTSD Scale for *DSM-5* (CAPS-5; Weathers, Blake, Schnurr, Kaloupek, Marx, & Keane, 2015) is a 30-item structured interview that assesses frequency and intensity of each PTSD symptom to create an overall symptom rating (0 – 80), determined on a 5-point Likert-style scale (0 = *absent* to 4 = *extreme/incapacitating*). In the current study, the past-month time frame was used to assess current PTSD diagnosis and symptom severity.

PTSD Checklist for DSM-5 (PCL-5; Blevins, Weathers, Davis, Witte, & Domino, 2015) is a 20-item self-report measure of PTSD symptom severity with each item reflecting a symptom of PTSD. Respondents rate each item on a 5-point scale (0 = *not at all* to 4 = *extremely*) regarding how often the symptom bothered them in the past month.

Data Analytic Plan

We first evaluated associations between HF-HRV and the following potential confounds: age, years of education, monthly income, race, gender, and past-month use of prescription medication for physical and psychiatric health. Only age demonstrated a significant association ($p < .05$) with HF-HRV. Therefore, we controlled for age in all regression models. Our primary analysis consisted of four linear regressions with HF-HRV as the outcome variable, and ASI Drugs, ASI Alcohol, CAPS-5 Total, and PCL-5 Total as predictor variables in separate models. Each model also controlled for age by entering age in the first block and the predictor variable in the second block. Using Mahalanobis Distance, we found no multivariate outliers between outcome and predictor variables. Age was not multicollinear with any severity variables. Residuals of these regressions were normally distributed.

Results

Participant Characteristics

Of 49 participants, 25 were female ($M_{\text{age}} = 45.5$, $SD = 10.0$). Most participants identified as African American ($n = 37$) followed by White/Caucasian ($n = 10$) and Other/Unknown ($n = 2$). Average level of education was high school ($M = 12.99$ years, $SD = 2.36$). The most common SUD diagnoses were cocaine ($n = 34$), alcohol ($n = 20$), and cannabis ($n = 19$),

with 26 participants meeting criteria for multiple SUD diagnoses. Approximately 77% of participants had a full PTSD diagnosis ($n = 38$). Participants had a mean ASI Drugs score of 0.23 ($SD = 0.09$), mean ASI Alcohol score of 0.26 ($SD = 0.28$), mean CAPS-5 Total score of 37.39 ($SD = 11.57$), and mean PCL-5 Total score of 49.27 ($SD = 12.80$).

Primary Analysis

Table 1 presents results for the predictors of interest from the four separate regressions. ASI Drugs was significantly related to HF-HRV even after controlling for age, $\beta = -.34$, $t(45) = -2.79$, $p = .008$. ASI Alcohol was also significantly related to HF-HRV after controlling for age, $\beta = -.30$, $t(45) = -2.35$, $p = .02$. CAPS-5 Total and PCL-5 Total were not significantly related to HF-HRV, with or without controlling for age. Notably, this pattern of results remained the same when analyses were conducted with only the subsample meeting full diagnostic criteria for PTSD ($n = 38$).

Discussion

Lower HF-HRV related to greater SUD symptom severity in adults with SUD and at least four PTSD symptoms. Contrary to our hypothesis, HF-HRV was not significantly related to PTSD symptom severity, suggesting that HF-HRV may be more sensitive to SUD than PTSD symptomatology. If so, previous studies demonstrating differences between individuals with and without PTSD may have been primarily capturing unmeasured differences in substance use. Indeed, one study found that current PTSD was not associated with lower HF-HRV after controlling for demographic and health factors including history of substance dependence (Shah et al., 2013). This study, however, did not specify whether the primary contributor to HF-HRV was history of substance use or another variable. In contrast, Udo et al. (2013) showed that alcohol use was significantly negatively associated with HF-HRV at baseline even after adjusting for symptoms of depression and anxiety, suggesting substance use may be more strongly related to HF-HRV than emotional symptoms.

This study provides preliminary evidence that HF-HRV is a stronger marker of SUD severity than post-traumatic stress symptom severity in a comorbid SUD and post-traumatic stress population. Other measures of SUD severity, such as positive baseline urinalysis results and self-reported days of use, have been shown to predict treatment outcomes (Hanlon, O'grady, & Bateman, 2000; Poling, Kosten, & Sofuoglu, 2007). HF-HRV may have advantages over these markers of SUD severity, as it is a physiological measure rather than a subjective self-report. Our findings also provide indirect support for the idea of targeting HF-HRV in treatment, consistent with pilot studies suggesting HRV biofeedback may reduce substance related cravings during biofeedback (Eddie, Conway, Alayan, Buckman, & Bates, 2018), substance related cravings in those with low HRV (Eddie et al., 2014), depression symptoms in people with PTSD as compared to a progressive muscle relaxation group (Zucker, Samuelson, Muench, Greenberg, & Gevirtz, 2009) and PTSD symptoms when compared with treatment as usual (Tan, Dao, Farmer, Sutherland, & Gevirtz, 2011).

Limitations of the current study are a relatively small sample size ($n = 49$), which may have prevented detection of the effects of PTSD symptom severity or alcohol use severity on HF-

HRV. Second, the SUD measures may not have captured the full range of substance use severity, because drugs and alcohol severity were separately measured, ignoring potential additive effects of these substances in a sample where many participants demonstrated comorbid SUDs. Future work might employ more standardized, well-established general substance use severity measures and replicate these findings across specific substance classes. Third, we did not examine other measures of HRV, such as SDNN, proportion of NN intervals differing by 50 ms (pNN50) or root mean square of successive differences (RMSSD). Future work in this area should incorporate longer collection times to examine varied HRV indices. Fourth, some confounds that could have affected HRV were not assessed in the current study, including cardiovascular disease/risk factors (e.g., diabetes, hypertension), caffeine intake, and time of HRV measurement, among others (Billman, 2011). A comprehensive assessment of health-related factors was not part of the evaluation for this clinical trial of a novel behavioral treatment, but should be considered in extensions of this work. Fifth, the results presented here were a secondary analysis, not pre-specified at the outset of the parent clinical trial, and as such must be treated as preliminary. Finally, the cross-sectional design prevented conclusions about causality and did not allow testing whether changes in SUD or PTSD severity track with HF-HRV over time.

In conclusion, our results suggest HF-HRV should be investigated further as a potential biomarker of processes underlying symptom severity in SUD. Findings may suggest that in PTSD populations, HF-HRV may primarily index comorbid SUD symptoms. Ultimately, after further development, this potential biomarker may contribute to our ability to predict treatment outcomes for SUD, particularly in treatments involving physiological interventions, and to understand and target biological mechanisms involved in substance use. Therefore, this investigation presents an important, preliminary step in informing our understanding of HF-HRV in SUD populations with post-traumatic stress.

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Table 1.

Results of regressions

	<i>R</i> ² (overall model)	<i>F</i> (overall model)	<i>B</i>	<i>SE</i>	<i>β</i>	<i>t</i>	<i>p</i>
ASI Drugs	.38	<i>F</i> (2, 43) = 13.10	−7.18	2.57	−.34	−2.79	< .01 *
ASI Alcohol	.33	<i>F</i> (2, 44) = 10.96	−2.05	0.87	−.30	−2.35	.02 *
CAPS Total	.26	<i>F</i> (2, 45) = 7.87	−0.02	0.02	−.09	−0.70	.49
PCL Total	.26	<i>F</i> (2, 44) = 7.67	−0.02	0.02	−.10	−0.76	.45

Note. ASI = Addiction Severity Index; CAPS = Clinician Administered PTSD Scale; PCL = PTSD Checklist.

* *p* < .05.