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Posttraumatic Stress as a Moderator of the Association Between HPA-Axis Functioning and Alcohol Use Disorder Among a Community Sample of Women Currently Experiencing Intimate Partner Violence

Silvi C. Goldstein[1], **Melissa R. Schick**[1,2], **Lisa L. Weyandt**[1], **Tami P. Sullivan**[3], **Hans Saint-Eloi Cadely**[1], **Nicole H. Weiss**[1]

[1]Department of Psychology, University of Rhode Island

[2]Department of Psychiatry and Behavioral Sciences and the Department of Veterans Affairs Medical Center Mental Health Service, Medical University of South Carolina

[3]Department of Psychiatry, Yale University School of Medicine

Abstract

Women experiencing intimate partner violence (IPV) experience a heightened prevalence of alcohol use disorder (AUD). Hypothalamic–pituitary–adrenal (HPA)-axis functioning has been associated with increased risk for AUD in other populations, including individuals with posttraumatic stress disorder (PTSD) symptoms. The goal of the present study was to determine whether PTSD symptom severity exacerbates the relationship between HPA-axis functioning and AUD. Participants were 151 community women who had experienced physical or sexual IPV in the past 30 days by their current male partners and used any amount of alcohol or drugs. A two-phase emotion induction protocol was utilized: Neutral mood induction followed by randomly assigned negative, positive, or neutral emotion induction. Saliva cortisol samples were obtained immediately following the neutral mood induction (baseline HPA-axis functioning), 20 min following the individualized emotion induction script (HPA-axis reactivity), and 40 min post the emotionally evocative cue (HPA-axis recovery). Findings revealed that PTSD symptom severity moderated the relations between baseline HPA-axis functioning and HPA-axis recovery and log odds of meeting criteria for AUD. Specifically, baseline HPA-axis functioning was positively associated with log odds of meeting criteria for AUD at high (but not low) PTSD symptom severity, whereas HPA-axis recovery was negatively associated with log odds of meeting criteria for AUD at high (but not low) PTSD symptom severity. Results contribute to our understanding of the biological processes involved in the etiology and maintenance of AUD among women experiencing IPV—specifically the prominent role of PTSD symptom severity.

Correspondence concerning this article should be addressed to Nicole H. Weiss, Department of Psychology, University of Rhode Island, 142 Flagg Road, Kingston, RI 02881, United States. nhweiss7@gmail.com.

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This study was not preregistered. Code and materials are available upon request to the corresponding author.

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Keywords

intimate partner violence; alcohol use disorder; posttraumatic stress disorder; HPA axis; cortisol

Intimate partner violence (IPV) is an international public health concern associated with substantial personal and societal costs (Cadilhac et al., 2015). Alcohol use disorder (AUD) is one common correlate of IPV that is of particular clinical significance (Coker et al., 2002). An epidemiological study found a significantly higher prevalence rate of AUD among individuals who had experienced IPV (7.3%) versus those who had not experienced IPV (2.3%; Okuda et al., 2011). Moreover, studies find a clear positive association between alcohol use and IPV among women (Devries et al., 2014). For instance, women who had experienced IPV were twice as likely to report daily alcohol use compared to those with no history of IPV (Carbone-López et al., 2006). Further, women in the general population who experienced past-year IPV had a greater odds of moderate and severe (vs. mild) AUD (La Flair et al., 2012). Speaking to the directionality of this association, Martino et al. (2005) found that women who experienced IPV were at an increased risk for later heavy drinking, whereas earlier heavy drinking did not predict later IPV. Notably, alcohol-related harm among women experiencing IPV is linked to numerous negative outcomes including psychological distress (Sullivan & Holt, 2008), legal problems (Oberleitner et al., 2013), and economic strain (Peterson et al., 2018). The heightened prevalence and significant impacts of alcohol-related harm in women experiencing IPV underscore the importance of research in this area.

Among women experiencing IPV, hypothalamic–pituitary–adrenal (HPA)-axis functioning may be particularly important to assess in relation to AUD. Production of cortisol, which is triggered by stress-induced activation of the HPA-axis, has been studied extensively in relation to AUD (Stephens & Wand, 2012; Wand, 2008). Notably, studies report a correlation between cortisol responsivity and activity within the mesolimbic dopaminergic pathway, which underlies AUD development, maintenance, and exacerbation (Oswald et al., 2005; Stephens & Wand, 2012; Wand et al., 2007). Altered HPA-axis responsivity may be present before alcohol exerts toxic effects on the central nervous system and subsequently contribute to initial vulnerability to AUD (Stephens & Wand, 2012). Studies suggest that cortisol may facilitate neurotransmission of dopaminergic neurons and, consequently, the reward circuity involved in AUD (Saal et al., 2003). Allostatic alterations—or the process by which HPA-axis function attempts to restore to homeostasis thus elevating stress peptide levels—have been posited to, among other things, contribute to the maintenance of AUD by altering brain reward pathways, ultimately resulting in increasing alcohol cravings (Stephens & Wand, 2012). Given the association between HPA-axis functioning and AUD, it is important to investigate this relation among women experiencing IPV.

Past research has identified cortisol levels as a valid biomarker of HPA-axis functioning (Nicolson, 2007), including among women experiencing IPV (Feinberg et al., 2011; Kim et al., 2015; Pinna et al., 2014; Pinto et al., 2016). When faced with acute stressors, the HPA-axis activates to preserve the “fight-or-flight” response, leading to glucocorticoid release, such as cortisol release in the process of allostasis (McEwen & Wingfield, 2003). In turn,

this process can cause alterations in HPA-axis functioning, leading to changes in cortisol levels (Guilliams & Edwards, 2010). For instance, some individuals experience a lack of adaptation during chronic stress exposure (e.g., baseline cortisol levels are typically elevated and cortisol response to acute stress is blunted), resulting in excessive cortisol exposure following each stressful event. After repetitive activation, the system exhibits allostatic injury, marked by elevated basal cortisol levels and an inability to mount an acute response (McEwen & Gianaros, 2010; Stephens & Wand, 2012). Other studies suggest that chronic exposure to stressors may lead to periods of elevated cortisol levels that are not reduced appropriately by negative feedback inhibition (i.e., HPA-axis recovery), creating further HPA-axis abnormalities (Guilliams & Edwards, 2010). These shifts in cortisol levels can be observed in the laboratory across three phases: (a) a baseline HPA-axis functioning phase, which reflects unstimulated, nonstressed HPA activity; (b) an HPAaxis reactivity phase in which cortisol increases from baseline (i.e., preemotional stimuli) levels following the onset of emotional stimuli; and (c) an HPA-axis recovery phase in which cortisol levels return to baseline levels following the offset of the emotional stimuli (McEwen, 1998).

Importantly, existing literature underscores the potential role of posttraumatic stress disorder (PTSD) in attenuating HPA-axis functioning (Inslicht et al., 2006; Johnson et al., 2008; Pinna et al., 2014). PTSD is a severe psychiatric condition characterized by the development and persistence of intrusions, avoidance, alterations in mood and cognitions, and arousal and reactivity following exposure to a traumatic event (American Psychiatric Association [APA], 2013). PTSD is widespread among women experiencing IPV (Spencer et al., 2019), and the combination of IPV and PTSD among women is linked to deleterious outcomes, including increased alcohol use (Sullivan et al., 2016). Yehuda and LeDoux (2007) hypothesized that individuals with PTSD fail to achieve recovery and restitution of physiological homeostasis after exposure to trauma, yet, the literature is mixed regarding the specific nature of HPA-axis dysfunction in PTSD. For example, a systematic review found that several studies reported a positive association between PTSD and cortisol levels, while others either found no association or a negative association (Speer et al., 2019). In contrast, research consistently supports PTSD and AUD occur concomitantly (Debell et al., 2014). Indeed, Szabo et al. (2020) concluded that high levels of PTSD symptom severity, coupled with stress, dysregulate cortisol and increase risk of AUD, supporting a bidirectional impact, such that alcohol is used as a coping mechanism in response to distress and related HPA-axis dysfunction, but can also dysregulate HPA-axis functioning and cortisol levels itself. Given these findings, and that cortisol can serve as an objective index of stress reactivity—therefore functioning as a more rigorous tool for assessing the role of HPA-axis functioning—it is clinically relevant to further investigate the unique role of PTSD symptom severity in the relation between HPA-axis functioning and AUD.

Addressing a critical gap in the literature, the goal of the present study was to explicate whether PTSD symptom severity influences the strength of the relationship between HPA-axis functioning (i.e., baseline, reactivity, and recovery cortisol responding) and log odds of meeting criteria for AUD among a community sample of women experiencing IPV. We hypothesized that higher levels of PTSD symptom severity would strengthen the association between HPAaxis functioningandlogodds ofmeetingcriteriaforAUD,such thata positive association between HPA-axis functioning and AUD will be stronger for those

with higher PTSD symptoms. We chose to include both women with and without a PTSD diagnosis in the present sample because most trauma-exposed people experience some PTSD symptoms, though they may not rise to the threshold of PTSD diagnosis (Zlotnick et al., 2002). Among women experiencing IPV, the experience of PTSD symptoms is related to significant impairmentin functioning, even when they don't meet full diagnostic criteria for PTSD (Hellmuth et al., 2014). As such, it is important to include individuals who are experiencing subthreshold PTSD symptoms to fully capture the population of trauma-exposed women experiencing IPV.

Method

Study Overview

Data were collected as part of a larger study examining the proximal role and temporal ordering of emotion dysregulation in the relations between PTSD symptoms and substance use and Human Immunodeficiency Virus (HIV)/sexual risk. All procedures were reviewed and approved by the [redacted] Institutional Review Board. The larger study entailed (a) a baseline session, (b) an experimental session, (c) 30 days of experience sampling using interactive voice technology, and (d) a follow-up session. The present study extracted data from the baseline and experimental sessions. To limit participant burden, these sessions were conducted on separate days. Individual interviews were conducted in-person by bachelor's- or masters-level clinical psychology doctoral students in private offices to protect participants' safety and confidentiality. During the baseline session, participants completed a structured diagnostic assessment, answered self-report questionnaires, and received a standardized protocol for developing individualized emotion inductions. In the experimental session, participants underwent a two-phase emotion induction protocol. The first phase involved a neutral mood induction. The second phase was the presentation of a randomly assigned emotion induction (negative, positive, or neutral) delivered via headphones. Participants provided saliva samples at three points: Following the neutral mood induction, at 20 min postindividualized emotion induction, and at 40 min postindividualized emotion induction.

Participants

Recruitment materials were posted in community establishments throughout Providence County, Rhode Island such as grocery stores, laundromats, and shops; selected state offices such as the Office of Housing and Community Development; and waiting rooms, bathrooms, and exam rooms of urban-area primary care clinics; as well as in website postings (e.g., Craigslist). Eligibility was determined through a phone screen. Participants were women who had experienced physical or sexual victimization in the past 30 days by their current male partners and used any amount of drugs or alcohol during that time. Additional inclusion criteria were: (a) age 18 or older, (b) fluent in the English language, and (c) current involvement in a relationship of at least 6 months' duration with current contact at least twice a week. Exclusion criteria were (a) current mania/psychosis [assessed in the baseline session with the Structured Clinical Interview (SCID-5) for Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5); First & Williams, 2016], (b) current impairment in cognitive functioning (assessed in the baseline session using the

Mini-Mental Status Exam and requiring a score >24; Folstein et al., 1975), (c) self-reported current pregnancy, (d) colorblindness, (e) cardiovascular disease, and (f) residenceinashelter or grouphome.The final sampleincluded151women who participated in the baseline and experimental sessions (see Procedures); demographic characteristics are summarized in Table 1.

Measures

Diagnostic Measure—A computerized version of the SCID-5 was administered to establish AUD and PTSD diagnoses (First & Williams, 2016). The SCID-5, a gold standard semi-structured assessment instrument for psychiatric disorders, has been found to yield valid and reliable current and lifetime diagnoses across a variety of common psychiatric disorders, including AUD and PTSD. A recent assessment of interrater reliability of the SCID-5 found evidence of moderate to excellent reliability across major diagnostic categories, including kappas of .84 and .80 for AUD and PTSD, respectively (Osório et al., 2019). SCID-5 interviews were conducted by clinical psychology doctoral students trained to reliability with the principal investigator, a licensed clinical psychologist in the state of Rhode Island. All data were reviewed by the principal investigator. In the case of ambiguous responses, data were discussed by the principal investigator and interviewer until a consensus was reached.

Self-Report Measures

Traumatic Exposure.: The Life Events Checklist for DSM-5 (LEC-5; Weathers, Blake, et al., 2013) is a 17-item self-report measure designed to assess lifetime exposure to traumatic events. The LEC-5 assesses exposure to 16 traumatic events, with a final item assessing for any other stressful event not captured in the first 16 items. For each event, the respondent is asked to indicate if: (a) it happened to them, (b) they witnessed it, (c) they learned about it, (d) they experienced it as part of their job, (e) they are not sure if they experienced it, or (f) they did not experience it. Any of the first four response options indicated a positive Criterion A traumatic event endorsement (APA, 2013). The LEC has shown convergent validity with measures assessing traumatic exposure and psychopathology known to relate to traumatic exposure (Weathers, Blake, et al., 2013). In the present study, the LEC-5 was used to identify an index trauma for subsequent assessment of PTSD symptoms on the PTSD Checklist for DSM-5 (PCL-5) as well as a covariate (i.e., number of lifetime traumatic events), given evidence for the influence of trauma exposure in HPA-axis functioning (de Kloet et al., 2007; Klaassens et al., 2009).

Posttraumatic Stress Disorder Symptom Severity.: The PCL-5 (Weathers, Litz, et al., 2013) is a 20-item self-report measure that assesses past-month PTSD symptoms consistent with DSM-5 criteria (APA, 2013). Participants completed the PCL-5 in response to the most distressing traumatic event endorsed on the Life Events Checklist for the DSM-5. Each item was rated using a 5-point Likert-type scale (0 = *not at all*, 4 = *extremely*). Possible scores range from 0 to 80, with higher values indicating increased severity of PTSD symptoms, and with a recommended cut-off score of 31 or higher to identify probable PTSD diagnosis (Blevins et al., 2015; Bovin et al., 2016). The PCL-5 has excellent psychometric properties

(Blevins et al., 2015; Bovin et al., 2016; Wortmann et al., 2016). Cronbach's α was .99 in the current sample.

Menstrual Cycle.: Female participants were asked whether they were currently menstruating (yes/no). This variable was included as a covariate in the present study given evidence for an impact of menstrual cycle phase on cortisol (Kirschbaum et al., 1999).

Biological Measure—Salivary cortisol samples were obtained at three timepoints during the experimental session: (a) immediately following neutral mood induction (i.e., baseline HPA-axis functioning), (b) 20 min following the individualized emotion induction script (i.e., HPA-axis reactivity; given evidence that cortisol levels peak approximately 20 min after presentation of an emotionally evocative cue (see Dickerson & Kemeny, 2004; Nicolson, 2007), and (c) 40 min post emotionally evocative cue (i.e., HPA-axis recovery). Saliva samples were collected by instructing participants to pool saliva in their mouth, then transfer the saliva into a centrifuge tube with a Salivette. Approximately 0.5 mL of saliva was collected, then sealed, and stored in a freezer. All samples were assayed in duplicate for salivary cortisol offsite using a highly sensitive enzyme immunoassay. The test used 25 μL of saliva per determination, has a lower limit of sensitivity of 0.003 μg/dL, standard curve range from 0.012 μg/dL to 3.0 μg/dL, an average intra-assay coefficient of variation (CV) of 3.8%, and an average inter-assay CV of 5.1%. Following recommended guidelines (see Nicolson, 2007 for guidelines in assessing cortisol response to acute stimuli in the laboratory) data on the time of day of cortisol collection and time since last use of nicotine were collected and included as covariates in the study analyses.

Procedures

Baseline Session—Participants provided informed consent, followed by an interview using a computerized version of the SCID-5 and completed a battery of self-report measures that included the LEC-5 and the PCL-5. Prior to the baseline session, participants were randomly assigned to one of three emotion induction conditions (negative, positive, or neutral). For participants in the negative and positive emotion induction conditions, a standardized protocol for developing individualized emotion induction scripts was followed. Participants were asked to recall a recent or vivid event during which they became "very angry" (negative condition) or "very excited" (positive condition) that did not involve substances or trauma. This portion of the session was audio recorded so that the interviewer could subsequently create a script using the participant's own language. Participants were asked to picture the situation in their mind and try to remember as vividly as possible what the event entailed and their feelings at the time. Participants were then asked to describe the incident in as much detail as possible. The interviewer probed for key aspects of the event (e.g., time and place of the event, as well as emotions, thoughts, and bodily sensations experienced during the event).

Prior to the experimental session, a personalized script consisting of a series of autobiographical statements, appraisals, and emotional responses generated from the interview was recorded onto an audiotape. This script was approximately 1 min in length and the narrator wasconsistentacrossallscripts(theprincipal investigator). All scripts were

presented in a female voice with a neutral tone (to reduce reactivity giventhatthe sample is characterized by experiences of IPV with a male partner). The method for generating these individualized emotion induction scripts was based on procedures originally developed by Lang and colleagues (see Lang & Cuthbert, 1984; Levin et al., 1982). The script is designed to maximize emotional responses by depicting the events in a salient, emotion-focused form in second person, present tense. This procedure reliably induces emotional responses in trauma-exposed samples (Lang et al., 1983; Orr et al., 1993; Pitman et al., 1987; Tull et al., 2011, 2019).

Neutral scripts were also developed for this study. Consistent with Keane et al. (1998), the neutral script was standardized and consistent across participants. It provided a description of activities involved in getting up in the morning (e.g., brushing teeth, getting dressed). The neutral script was also approximately 1 min in length and similarly consisted of descriptions of morning events, as well as thoughts and feelings that a person may experience in response.

At the end of the baseline session, participants were instructed to abstain from alcohol and illicit drugs for a period of at least 4 days prior to the experimental session. This criterion reduces the risk for intoxication and acute withdrawal (see Coffey et al., 2006, 2011). Participants were compensated with $40 for completing the baseline session.

Experimental Session—At the start of the experimental session (approximately 4–7 days after the baseline session), participants' compliance with substance use restrictions was assessed. Specifically, a urine drug screen (iCup by Alere Toxicology Services) was administered at the start of the experimental session to test for metabolites of Tetrahydrocannabinol (THC), cocaine, opiates, amphetamines, benzodiazepines, methamphetamine, oxycodone, propoxyphene, barbiturates, and 3,4-methylenedioxy-methamphetamine (MDMA). To assess recent alcohol intoxication, expired air samples were analyzed (Alco-sensor IV, Intoximeters Inc., St. Louis, MO). Participants who tested positive for illicit drugs or who had a blood alcohol level >.01 were rescheduled. Due to the long half-life of THC metabolites, participants who tested positive for THC and reported marijuana use in the past 30 days, but not past 4 days, were allowed to participate in the experimental session. Participants were also asked to report whether they were currently menstruating. Following this, we induced a neutral mood by displaying colors, one after another, on a screen in front of the participants for 5 min. This procedure, called the "vanilla baseline procedure," has been found to produce a more neutral mood (e.g., less anxiety) compared with an absence of activities (i.e., having the participant sit still and do nothing for 5 min; Jennings et al., 1992). Participants provided the first salivary cortisol sample after neutral mood induction and then listened to the 1-min individualized emotion induction script developed during the baseline session. Once the tape was finished, participants were instructed to close their eyes and imagine vividly the event taking place in real-time for 1 min. Participants provided additional saliva samples 20- and 40-min postindividualized emotion induction. Participants were compensated $25 for completing the experimental session and were provided with a list of community resources. This study was not preregistered. Code and materials are available upon request to the corresponding author.

Analytic Strategy

To address the question of whether HPA-axis functioning [i.e., baseline, reactivity (reflected by a change score calculated by subtracting baseline cortisol values from cortisol values assessed 20 min postemotion induction), and recovery (reflected by a change score calculated by subtracting cortisol values assessed 20 min postemotion induction from cortisol values assessed 40 min postemotion induction) cortisol responding], PTSD symptom severity, and their interactions are associated with log odds of membership in the AUD group (defined as meeting criteria for mild, moderate, or severe AUD), three moderation analyses were conducted using the PROCESS SPSS macro (Model 1) as recommended by Hayes (2018). The PROCESS procedures use ordinary least squares regression and bootstrapping methodology, which confers more statistical power than do standard approaches to statistical inference and does not rely on distributional assumptions. Bootstrapping was done with 5,000 random samples generated from the observed covariance matrix to estimate bias-corrected 95% confidence intervals (CIs) and significance values. For interactions found to be significant, following the methods described by Aiken et al. (1991), we plotted regression slopes of differences in log odds of membership in the AUD group and conducted follow-up analyses to examine whether the slopes of the regression lines differed significantly from zero. All models included total number of lifetime traumatic events, time of day at which saliva data was collected, time since last cigarette, and current menstruation as covariates.

While the full sample was used for analyses examining the effect of baseline HPA-axis functioning, the sample was restricted only to those participants randomized to positive and negative emotion induction conditions for models examining HPA-axis reactivity and recovery. No significant differences were detected between the positive and negative emotion induction condition with respect to any of the three HPA-axis functioning indices; thus, to improve statistical power, these groups were combined. Sensitivity analyses revealed that we were adequately powered to detect medium effects $d = .56$, which corresponds to $r = .27$ and $R^2 = .07$; our effects were above this threshold.

Results

Moderation analyses testing the hypothesis that higher levels of PTSD symptom severity would strengthen the association between HPA-axis functioning and AUD, controlling for time of day of saliva sample collection, time since last cigarette use, number of lifetime traumatic experiences, and current menstruation, are summarized in Table 2. In the model examining baseline HPA-axis functioning, no significant main effects were detected for either baseline HPA-axis functioning, $b = 3.71$, $SE = 2.30$, $z = 1.62$, $p = .11$, 95% CI [–.79, 8.21], or PTSD symptom severity, $b = .02$, $SE = .01$, z = 1.68, $p = .09$, 95% CI [–.004, .05], predicting log odds of meeting criteria for AUD. However, the interaction of baseline HPA-axis functioning and PTSD symptom severity was significantly associated with the log odds of meeting criteria for AUD, $b = .29$, $SE = .14$, $z = 2.04$, p = .04, 95% CI [.01, .56]. As is summarized in Figure 1, analysis of simple slopes revealed that baseline HPAaxis functioning was significantly positively associated with log odds of meeting criteria for AUD at high, $b = 10.10$, $SE = 4.48$, $z = 2.26$, $p = .02$, 95% CI [1.32, 18.87], but not low,

$b = -2.67$, $SE = 3.18$, $z = -0.84$, $p = .40$, 95% CI [–8.90, 3.55], levels of PTSD symptom severity.

In the model examining HPA-axis reactivity, no significant main effects were detected for either HPA-axis reactivity, $b = -.43$, $SE = 5.71$, $z = -0.08$, $p = .94$, 95% CI [–11.62, 10.76], or PTSD symptom severity, $b = .01$, $SE = .01$, $z = 0.45$, $p = .66$, 95% CI [–.02, .03], predicting log odds of meeting criteria for AUD. Additionally, the interaction of HPA-axis reactivity and PTSD symptom severity was not significantly associated with log odds of meeting criteria for AUD, $b = -.01$, $SE = .33$, $z = -0.04$, $p = .97$, 95% CI [–.65, .62].

Finally, in the model examining HPA-axis recovery, no significant main effects were detected for either HPA-axis recovery, $b = -13.20$, $SE = 10.52$, $z = -1.26$, $p = .21$, 95% CI [–33.82, 7.42], or PTSD symptom severity, $b = .01$, $SE = .02$, $z = 0.59$, $p = .55$, 95% CI [–.02, .04]. However, the interaction of HPA-axis reactivity and PTSD symptom severity was significantly associated with the log odds of meeting criteria for AUD, $b = -1.64$, $SE = .73$, $z = -2.25$, $p = .02$, 95% CI [–3.07, −.21]. As is summarized in Figure 2, analysis of simple slopes revealed that HPA-axis recovery was significantly negatively associated with log odds of meeting criteria for AUD at high, $b = -51.18$, $SE = 20.88$, $z = -2.45$, $p = .01$, 95% CI [–92.11, −10.26], but not low, $b = 24.78$, $SE = 18.81$, $z = 1.32$, $p = .19$, 95% CI [–12.09, 61.65], levels of PTSD symptom severity.

Exploratory Analyses

Given recent calls to explore biological stress responsivity within racial and ethnic subgroups, we conducted exploratory analyses to examine potential differences in HPA-axis functioning, PTSD (symptom severity and diagnosis), and AUD. We found that there were no significant between-group differences with respect to basal cortisol, $F(5, 139) = 0.36$, $p = .88$, cortisol reactivity, $F(5, 139) = 1.72$, $p = .14$, cortisol recovery, $F(5, 137) = 0.20$, $p = .96$, or PTSD symptom severity, $F(5, 136) = 1.18$, $p = .32$, nor were there differences in the proportion of participants who met criteria for PTSD, $\chi^2(5) = 7.08$, $p = .22$, or AUD, $\chi^2(5) = 3.50$, $p = .62$, across racial/ethnic groups. We also examined correlations between our variables of interest by racial/ethnic group and largely did not find evidence for significant relations (rs from −.25 to .23, ps from .15 to .995), except between basal cortisol and PTSD symptom severity for Black participants ($r = .35$, $p = .03$).

Discussion

The purpose of the present study was to elucidate the combined role of PTSD symptomology and HPA axis function on risk for AUD among women experiencing IPV. Consistent with study hypotheses, baseline HPA-axis functioning was positively associated, and HPA-axis recovery negatively associated, with log odds of meeting criteria for AUD at high—but not low—levels of PTSD symptom severity. These findings advance research on the relation between HPA-axis functioning and AUD by highlighting the important influence of PTSD symptom severity in these associations.

Overall, our results extend previous findings concerning the association of HPA-axis functioning with AUD (Lee et al., 2018; Oswald et al., 2005; Stephens & Wand, 2012;

Wand, 2008; Wand et al., 2007) and PTSD (Speer et al., 2019; Szabo et al., 2020) separately, underscoring the moderating role of PTSD symptom severity in the relationships between both baseline HPA-axis functioning and HPA-axis recovery and AUD. Specifically, our findings suggest that severity of PTSD symptoms exacerbates the link between HPA-axis functioning and AUD among women experiencing IPV. This may suggest an important role of the mesolimbic dopamine reward pathway, which is involved in stress response and addiction (Cleck & Blendy, 2008; Sinha et al., 2005).

Of note, we found no significant main effect for HPA-axis reactivity predicting log odds of meeting criteria for AUD. Given evidence that allostatic injury can cause the HPA-axis to become less sensitive, marked by elevated basal levels and an inability to mount an acute response, following subsequent stressful episodes, it is possible that tests of HPA-axis reactivity among individuals experiencing chronic trauma, such as women experiencing IPV, do not capture HPA-axis functionality in the relation to AUD due to blunted cortisol activity (Guilliams & Edwards, 2010; McEwen & Gianaros, 2010; Sinha et al., 2009; Stephens & Wand, 2012). In fact, our finding that mean reactivity reflected a negligible decrease from baseline is consistent with previous work regarding blunted cortisol reactivity in AUD (see Table 1). This aligns with previous literature that finds significant blunted-to-absent cortisol reactivity among people with AUD (Adinoff et al., 2005) including after exposure to a psychological stressor (Lovallo et al., 2000). Indeed, blunted cortisol reactivity response has been found when using personalized stressful imagery among individuals with AUD (Sinha et al., 2009) along with suppressed cortisol in trauma-exposed veterans diagnosed with PTSD relative to controls (de Kloet et al., 2007). Decreased HPA-axis reactivity has been shown to be associated with increased maladaptive behaviors including alcohol use among those who have experienced significant life stressors (Kim, 2017; Walton et al., 2018). Furthermore, our result, which found that for participants with high PTSD symptom severity, HPA-axis recovery was negatively associated with greater odds of AUD, also suggests that blunted cortisol reactivity is a manifestation of allostatic load among IPV-exposed women who are exposed to chronic stressors.

Our significant findings among HPA-axis at baseline and recovery, but not reactivity, may be understood through the drinking to cope self-medication model (Khantzian, 1997). Individuals with PTSD may misuse alcohol to self-medicate a negative affective state, including anxiogenic and hyperarousal symptoms associated with the disorder (Gilpin & Weiner, 2017; Hawn et al., 2020). Indeed, alcohol has been identified as the most commonly used substance among individuals with PTSD (Shorter et al., 2015). One study found a longitudinal association between IPV, alcohol-related harm, and drinking to cope, such that drinking to cope mediates the relationship between IPV and both alcohol-related harm and consumption (Øverup et al., 2015). Research suggests that IPV-exposed women use alcohol to alleviate their PTSD symptoms, such that elevated PTSD symptom severity is related to greater likelihood of drinking and amount of alcohol consumed (Sullivan et al., 2020). While individuals without severe PTSD may experience a normal decrease in negative affect after experiencing an emotionally evocative cue (e.g., HPA-axis recovery), it is possible that those with severe PTSD symptoms do not. Thus, perhaps severe PTSD symptoms are influencing the relation between HPA-axis and AUD at baseline and recovery, rather than at reactivity, when individuals are attempting to manage and diminish the effects of a prolonged affective

state. Alternatively, perhaps this unexpected finding is due to additional barriers related to understanding HPA-axis reactivity (i.e., blunted reactivity); such that HPA-axis reactivity may be altered in ways that can persist into adulthood for individuals who have experienced early-life trauma (Dunlop & Wong, 2019).

Results of the present study help explain the influence of PTSD symptom severity on the strength of the relationships between HPA-axis functioning (i.e., baseline, reactivity, and recovery cortisol responding) and AUD among a community sample of women experiencing IPV. However, our study should be interpreted in the context of its limitations. First, our study only measured cortisol at three timepoints: Baseline, reactivity (i.e., 20-min postemotion induction), and recovery (i.e., 20-min postreactivity sample). This measure of cortisol reactivity should be noted as a limitation given that it is possible it may not fully capture timing and peak cortisol reactivity of the cortisol response curve. Future research may consider a repeated 10-min saliva sampling to better capture cortisol responsivity as has been done in other research (Gozansky et al., 2005). Second, we included current menstruation as a covariate in analyses given evidence suggesting that hormonal fluctuations regarding the menstrual cycle phase can influence HPA-axis functioning (Montero-López et al., 2018). However, we were limited in our assessment of menstrual cycle and menstrual phase; future work should include more thorough assessment to further elucidate the role of menstrual cycle-related cortisol fluctuations in relation to AUD. Lastly, our study assessed PTSD among a sample of community women experiencing IPV, thus our results may not generalize to other trauma-exposed samples, such as those identified by other index traumas or in different settings (e.g., treatment-seeking clinical settings); other clinical populations including people who have depression given than HPA-Axis may be blunted in depressed patients (Saxbe, 2008), which commonly co-occurs with both PTSD (Smith et al., 2016) and AUD (Grant et al., 2015); and among men for whom HPA-axis reactivity has been found to be stronger than among women (Saxbe, 2008). Relatedly, given that women suffer disproportionately from IPV (Caldwell et al., 2012), and the unique presentation of IPV across sexual minority populations, the current sample focused on women-identified individuals in heterosexual relationships. Future research should investigate the influence of PTSD on the relationship between HPA axis and AUD in different settings, among men, within specific racial and ethnic subgroups (Price et al., 2021), and across gender and sexual minorities.

Despite these limitations, the findings of the present study advance our understanding of the associations among HPA-axis functioning, PTSD symptom severity, and AUD among women experiencing IPV. Specifically, we found that baseline HPA-axis functioning was significantly positively associated with log odds of meeting criteria for AUD at high (but not low) PTSD symptom severity, whereas HPA-axis recovery was significantly negatively associated with log odds of meeting criteria for AUD at high (but not low) PTSD symptom severity. Our findings contribute a novel understanding of the biological processes involved in the development of AUD among women experiencing IPV—specifically the prominent role of PTSD symptom severity.

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Public Health Significance

This study highlights how PTSD symptom severity moderates the relationship between HPA-axis functioning and odds of meeting criteria for alcohol use disorder (AUD). Results contribute to our understanding of the biological processes involved in the etiology and maintenance of AUD among women experiencing IPV—specifically the prominent role of PTSD symptom severity.

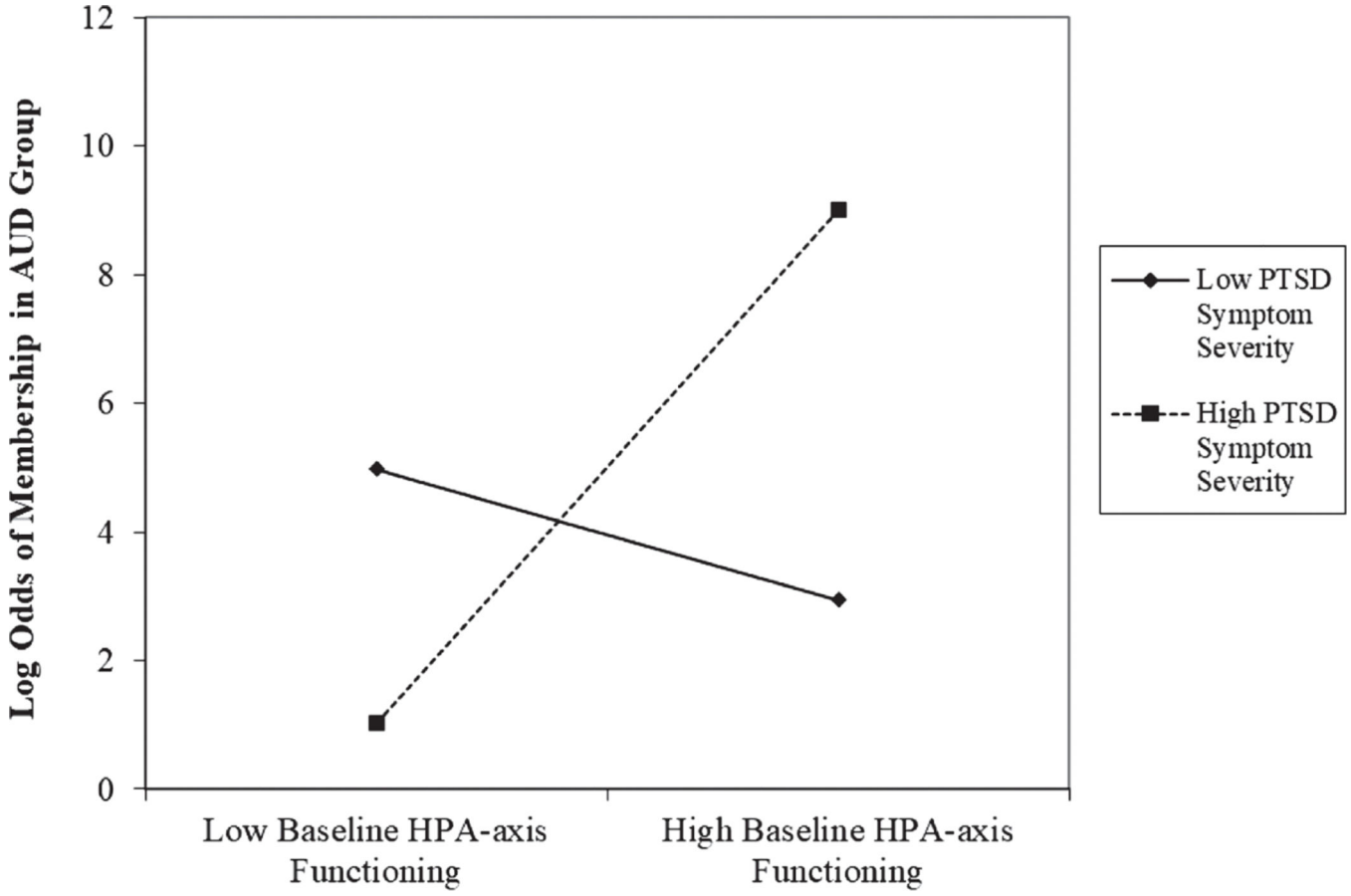

Figure 1.

Baseline HPA-Axis Functioning by PTSD Symptom Severity Interaction for Log Odds of Membership in the AUD Group

Note. HPA = hypothalamic–pituitary–adrenal; PTSD = posttraumatic stress disorder; AUD = alcohol use disorder.

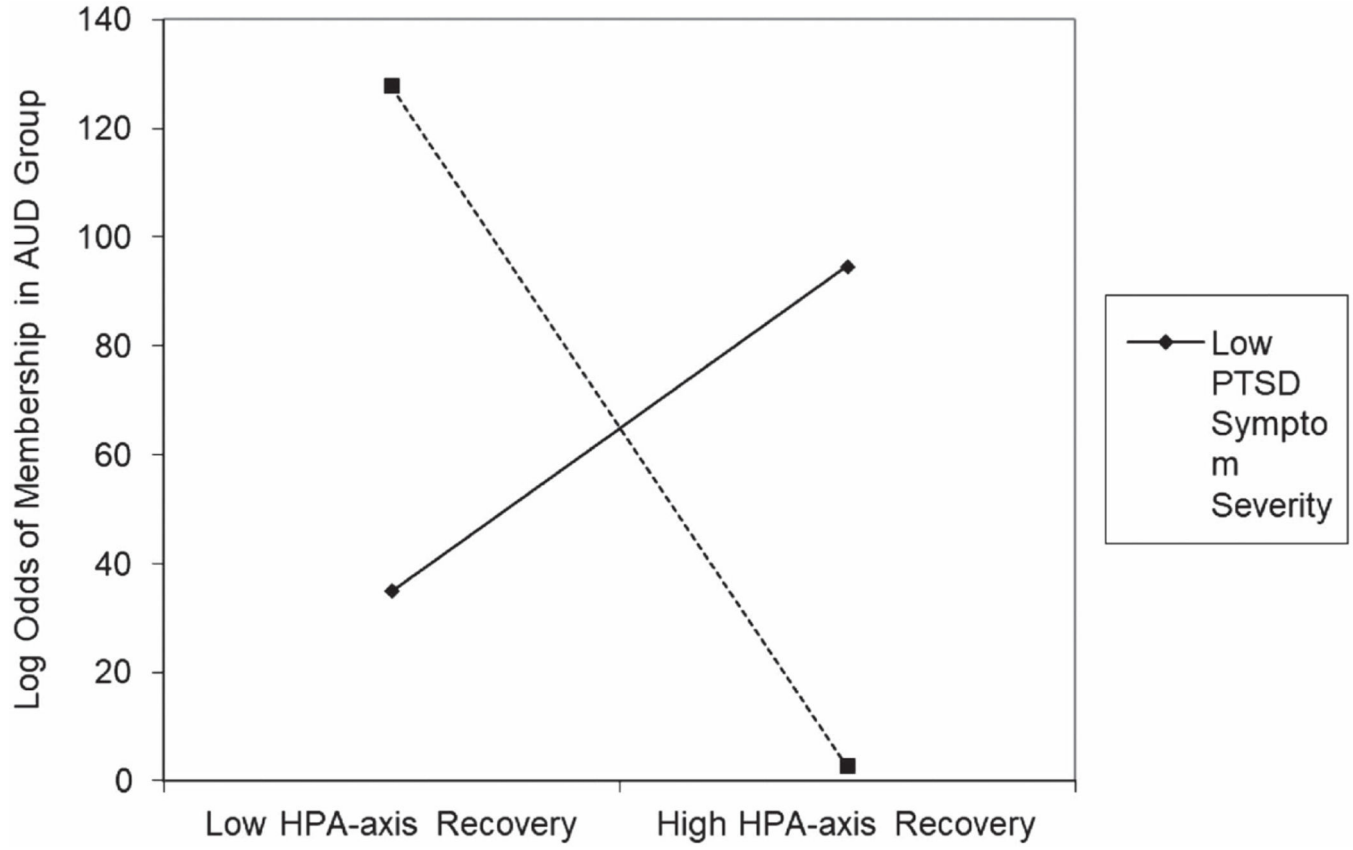

Figure 2.

HPA-Axis Recovery by PTSD Symptom Severity Interaction for Log Odds of Membership in the AUD Group

Note. HPA = hypothalamic–pituitary–adrenal; PTSD = posttraumatic stress disorder; AUD = alcohol use disorder.

Table 1

Sample Demographic and Descriptive Characteristics

Demographic and descriptive characteristics	*M (SD)*	Range	*n* (%)
Age	40.81 (11.64)	19–65	
Racial/ethnic background			
Black or African American			46 (30.5%)
White			65 (43.0%)
American Indian/Alaska Native			12 (7.9%)
Hispanic or Latina			17 (11.3%)
Not listed			9 (6.0%)
Prefer not to respond			2 (1.3%)
Years of education completed	12.39 (2.04)	6–18	
Employment			
Full time (35+ hr per week)			8 (5.3%)
Part time (less than 35 hr per week or sporadic employment)			17 (11.3%)
Unemployed			115 (76.2%)
Prefer not to respond			11 (7.3%)
Monthly household income	$1278.03 ($1594.52)	$0–$10416.67	
Relationship status			
Married			12 (7.9%)
Unmarried			113 (74.8%)
Separated or divorced			13 (8.6%)
Prefer not to respond			13 (8.6%)
Relationship length (in years)	6.08 (5.88)	0.5–30	
Days with partner per week	5.80 (1.89)	0–7	
Current AUD diagnosis			81 (53.6%)
Current PTSD diagnosis			67 (39.0%)
Lifetime PTSD diagnosis			103 (59.9%)
PTSD symptom severity	34.15 (21.79)	0–80	
Indices of HPA-axis functioning			
Baseline HPA-axis functioning (ug/dL)	0.21 (0.40)	0–4.32	

Demographic and descriptive characteristics	*M (SD)*	Range	*n* (%)
HPA-axis reactivity (ug/dL)	–0.02 (0.16)	−1.06–1.28	
HPA-axis recovery (ug/dL)	0.06 (0.99)	−1.19–11.76	
Covariates			
Number of lifetime traumatic events	6.04 (5.34)	0–17	
Time since last cigarette (in minutes)	234.89 (532.92)	10–4,320	
Current menstruation			18 (11.9%)

Note. AUD = alcohol use disorder; PTSD = posttraumatic stress disorder; HPA = hypothalamic–pituitary–adrenal.

Table 2

Summary of Moderation Analyses Predicting Membership in the AUD (vs. no AUD) Group

Variable of interest	*b*	*SE*	*z*	*p*	95% CI
Model 1: Baseline HPA-axis functioning (n = 93)					
Intercept	**1.49**	**.74**	**2.02**	**.04**	**[.04, 2.94]**
Baseline HPA-axis functioning	3.71	2.30	1.62	.11	[–.79, 8.21]
PTSD symptom severity	.02	.01	1.68	.09	[–.004, .05]
Baseline HPA-axis functioning × PTSD symptom severity	**.29**	**.14**	**2.04**	**.04**	**[.01, .56]**
Number of lifetime traumatic events	−.02	.05	−0.34	.74	[−.11, .08]
Time of day	−.62	.37	−1.68	.09	[−1.35, .11]
Time since last cigarette	.001	.001	1.16	.24	[.00, .002]
Current menstruation	.06	.79	0.07	.94	[–1.49, 1.60]
Model 2: HPA-axis Reactivity (n = 56)					
Intercept	1.42	1.02	1.39	.17	[−.58, 3.43]
HPA-axis reactivity	−.43	5.71	−0.08	.94	[−11.62, 10.76]
PTSD symptom severity	.01	.01	0.45	.66	[−.02, .03]
HPA-axis reactivity × PTSD symptom severity	−.01	.33	−0.04	.97	[−.65, .62]
Number of lifetime traumatic events	−.01	.07	−0.17	.86	[−.14, .12]
Time of day	−.59	.43	−1.38	.17	[−1.42, .25]
Time since last cigarette	.00	.001	−0.17	.87	[−.002, .002]
Current menstruation	.59	.94	0.62	.53	[–1.25, 2.43]
Model 3: HPA-axis recovery (n = 55)					
Intercept	**3.00**	**1.41**	**2.12**	**.03**	**[.23, 5.76]**
HPA-axis recovery	−13.20	10.52	−1.26	.21	[−33.82, 7.42]
PTSD symptom severity	.01	.02	0.59	.55	[−.02, .04]
HPA-axis recovery × PTSD symptom severity	**−1.64**	**.73**	**−2.25**	**.02**	**[−3.07, −.21]**
Number of lifetime traumatic events	−.06	.07	−0.82	.41	[−.21, .08]
Time of day	**−1.22**	**.56**	**−2.17**	**.03**	**[−2.32, −.12]**
Time since last cigarette	.00	.001	0.05	.96	[−.002, .002]
Current menstruation	−.89	1.17	−0.76	.45	[−3.99, 1.40]

Note. Bolded typeface indicates significance at the level $p < .05$. AUD = alcohol use disorder; HPA = hypothalamic–pituitary–adrenal; PTSD = posttraumatic stress disorder.