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Anxiety Sensitivity as a Moderator of the Association Between Premenstrual Symptoms and Posttraumatic Stress Disorder Symptom Severity

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Abstract

Experience of premenstrual symptoms may be an important factor involved in understanding posttraumatic stress disorder (PTSD) symptom vulnerability. Correlations between PTSD and premenstrual dysphoric disorder (PMDD) have been identified in epidemiological studies, however, the nature of this relation is not clear. The current study examined the incremental validity of premenstrual symptoms, as well as their interaction with anxiety sensitivity, in the prediction of PTSD symptom severity above and beyond other theoretically relevant covariates. A community sample of trauma-exposed women ($N = 63$) completed questionnaires assessing premenstrual symptoms, anxiety sensitivity, and neuroticism and were administered the Clinician-Administered PTSD Scale to assess PTSD symptom severity. A series of hierarchical linear regressions revealed that premenstrual symptoms uniquely predicted PTSD total, reexperiencing, avoidance, and hyperarousal symptoms above and beyond other theoretically relevant covariates (i.e., number of potentially traumatic events, neuroticism, panic attack history, and anxiety sensitivity). Additionally, anxiety sensitivity emerged as a moderator of the association between premenstrual symptoms and PTSD symptom severity such that greater premenstrual symptoms were associated with greater PTSD total, reexperiencing, and numbing symptom severity for individuals high, but not low, in anxiety sensitivity. Experience of premenstrual symptoms may be

an important sex-specific mechanism involved in increasing vulnerability for PTSD symptoms, particularly among women high in anxiety sensitivity.

Keywords

anxiety sensitivity; premenstrual symptoms; posttraumatic stress disorder

Premenstrual syndrome (PMS) requires the presence of one or more moderate to severe psychological (e.g., anxiety) and physical (e.g., headache) symptoms that occur regularly during the premenstrual phase (Milewicz & Jedrzejuk, 2006). Premenstrual dysphoric disorder (PMDD) is an extreme variant along the continuum of premenstrual symptoms that requires the presence of five or more psychological and physical symptoms, which occur regularly during the week prior to menses, remit with the onset of menses, and are absent the week following menses. Significant impairment related to these symptoms is also required for a PMDD diagnosis (American Psychological Association, 2000; Halbreich, Borenstein, Pearlstein, & Kahn, 2003). Premenstrual distress, or severity of premenstrual symptoms, may be a sex-specific vulnerability that uniquely or, in combination with other risk factors, increases PTSD symptom severity. Indeed, research has indicated significant relations between PTSD and PMDD (Pilver, Levy, Libby, & Desai, 2011; Wittchen, Perkonig, & Pfister, 2003). For example, a large cross-sectional study found that individuals with a history of trauma with (OR = 8.14) or without (OR = 2.84) PTSD were more likely to have PMDD (Pilver et al., 2011). Two prospective reports found that PTSD or a history of potentially traumatic events at baseline predicted increased incidence of subsequent PMDD or PMS at the follow-up assessment (Perkonig, Yonkers, Pfister, Lieb, & Wittchen, 2004; Wittchen et al., 2003). Although history of traumatic events and PTSD demonstrated unique risks, subthreshold PMDD at baseline emerged as the strongest predictor of subsequent PMDD (Perkonig et al., 2004).

Collectively, the existing empirical literature does not elucidate the nature of the relation between PTSD and premenstrual symptoms. It is possible that (a) trauma and/or PTSD symptoms increase the risk for experience of premenstrual symptoms, (b) that the experience of premenstrual symptoms increases risk for increased PTSD symptom severity, or (c) some third factor influences both problems (Monteleone et al., 2010; Rasmusson et al., 2006).

There are a number of interrelated streams of evidence that link PTSD and premenstrual symptoms. First, women who experience greater premenstrual symptoms may be more likely to respond to a traumatic event with increased emotional reactivity, which could increase their likelihood for developing PTSD symptoms. For example, some literature has shown that women with PMDD evidence increased dysregulation of the stress response system, regardless of cycle phase, as compared with control women (Girdler et al., 1998). Second, it is possible that premenstrual symptoms may represent a proxy/marker for an underlying neuroendocrine and hormonal vulnerability that influences PTSD symptomatology. For example, low estrogen levels have been associated with extinction deficits in fear learning (Glover et al., 2012) and low levels of allopregnanolone, a

neuroactive metabolite of progesterone, have been associated with increased PTSD reexperiencing symptoms (Rasmusson et al., 2006), as well as PMS (Monteleone et al., 2000). Additionally, menstrual cycle phase, particularly the luteal phase, influences the consolidation of emotional memories and fear learning (Bryant et al., 2011). Finally, it is possible that these two symptom profiles reflect an underlying shared affective vulnerability. PMS and PTSD symptoms also may have reciprocal effects on one another. For example, one prospective study found that global perceived stress during the premenstrual phase predicted premenstrual symptoms, but also that premenstrual symptoms predicted perceived stress (Woods et al., 1998). It is important for research in this area to further explore the nature of these relations, as well as extend such research to examine how sex-specific mechanisms interact with other factors in relation to PTSD symptom severity.

Anxiety sensitivity (AS), or the fear of anxiety and its physical, psychological, and social consequences (McNally, 2002), is one potential factor that may be useful for understanding the nature of the relation between premenstrual symptoms and PTSD. Elevated levels of AS are consistently related both concurrently (Collimore, McCabe, Carleton, & Asmundson, 2008; Stephenson, Valentiner, Kumpula, & Orcutt, 2009) and prospectively (Elwood, Mott, Williams, Lohr, & Schroeder, 2009; Feldner, Zvolensky, Schmidt, & Smith, 2008) with greater PTSD symptom severity. Moreover, some evidence indicates that AS and PTSD symptoms likely influence one another bidirectionally. For example, Marshall, Miles, and Stewart (2010) conducted a longitudinal examination of AS and PTSD symptoms within a sample of physical injury survivors at three time points (shortly after injury, 6 months, and 12 months) and found that AS predicted future PTSD symptom severity and vice versa.

There also is a small, but growing, literature linking AS and premenstrual symptoms. Specifically, individuals high on AS retrospectively and prospectively report more severe premenstrual symptoms on the Menstrual Distress Questionnaire (MDQ; Moos, 1968), a self-report measure of premenstrual symptoms, as compared with individuals low on AS (Sigmon, Rohan, Boulard, Dorhofer, & Whitcomb, 2000). Similarly, higher levels of premenstrual distress are related to increased reactivity to laboratory-induced physiological arousal (i.e., carbon dioxide-enriched air challenge; Nillni, Rohan, Bernstein, & Zvolensky, 2010).

Thus far, there are no published studies explicitly linking PMDD/PMS, AS, and PTSD symptom severity. However, we might expect that premenstrual symptoms would predict PTSD symptom severity among individuals high in AS. Individuals high in AS are more sensitive to bodily changes occurring during the premenstrual phase, as suggested by increased premenstrual symptom reports in this group of women (Sigmon et al., 2000). This sensitivity also may influence their perception of PTSD symptom severity. Alternatively, biological vulnerability (i.e., hormone changes) underlying the experience of increased premenstrual symptoms (Halbreich, 2003) and PTSD symptoms (Rasmusson et al., 2006) may serve as an additional PTSD-relevant vulnerability among at-risk women (i.e., high in AS) by way of alterations of sex-specific hormones. In other words, it is important to understand if AS, a well-understood cognitive risk factor for PTSD, and premenstrual distress exert unique, as well as synergistic, effects on PTSD symptom severity.

The first aim of the present study was to examine the incremental validity of premenstrual symptoms in regard to PTSD symptom severity in a community sample of trauma-exposed women. Specifically, we examined the unique effect of premenstrual symptoms on PTSD symptom severity above and beyond theoretically relevant covariates, including trait-level neuroticism (Fauerbach, Lawrence, Schmidt, Munster, & Costa, 2000), history of potentially traumatic events (PTEs; Breslau et al., 1999), and a history of nonclinical panic attacks (Marshall-Berenz, Vujanovic, & Zvolensky, 2011). Second, the current study examined the interactive effects of premenstrual symptoms and AS on PTSD symptom severity. It was hypothesized that: (a) premenstrual symptoms would evidence an incremental unique effect on PTSD symptom severity above and beyond neuroticism, number of lifetime PTEs, and history of nonclinical panic attacks; and (b) AS would moderate the relation between premenstrual symptoms and PTSD symptom severity, such that greater levels of premenstrual symptoms would be associated with greater PTSD symptom severity among individuals high, but not low, in AS. A similar pattern of results was expected across PTSD symptom clusters (i.e., reexperiencing, effortful avoidance, emotional numbing, and hyperarousal).

Method

Participants

Participants were 63 women ($M_{age} = 21.0$ years, $SD = 6.1$, $range = 18-46$) recruited from the greater Burlington, Vermont community who met criteria for lifetime trauma exposure according to *DSM-IV-TR* PTSD Criterion A on the Clinician-Administered PTSD scale (CAPS; Blake et al., 1995). In terms of current psychopathology, 68.3% ($n = 43$) of the sample met criteria for one or more current (past month) Axis I diagnosis as measured by the Structured Clinical Interview for *DSM-IV* Axis I Disorders–Non-Patient Version (SCID-I/NP; First, Spitzer, Gibbon, & Williams, 1994). Current PTSD diagnosis and symptoms were assessed via the CAPS (Blake et al., 1995). Individuals scored an average of 11.78 ($SD = 14.51$) on the CAPS severity ratings (frequency + intensity), indicating relatively low levels of PTSD symptoms (e.g., minimum score of 45 reliably corresponds to a PTSD diagnosis; Weathers, Ruscio, & Keane, 1999), with only 1.6% ($n = 1$) meeting full criteria for PTSD. See Table 1 for complete participant characteristics.

Data were collected as part of a larger laboratory investigation on nonclinical panic attacks and biological challenge responding that oversampled individuals with nonclinical panic attacks, and as such, 49.2% of the total sample met criteria for past 2-year nonclinical, unexpected panic attacks (i.e., “out of the blue” panic attacks without a panic disorder diagnosis). Exclusionary criteria for the original study included: (a) current or past cardiopulmonary (chronic) illness (e.g., asthma); (b) current acute respiratory illness (e.g., bronchitis); (c) pregnancy; (d) seizure disorders; (e) past participation in a CO₂ study; (f) current psychotropic medication use; (g) lifetime panic disorder diagnosis; (h) psychosis; and (i) current serious suicidal ideation. Inclusionary criteria for the current study included: (a) being female, (b) lifetime PTSD Criterion A trauma exposure, and (c) having completed the Menstrual Distress Questionnaire in the larger study.

Measures

The *Structured Clinical Interview for DSM–IV Axis I Disorders–Non-Patient Version* (SCID-I/NP; First et al., 1994) was administered to assess inclusion/exclusion criteria (i.e., panic attack history, suicidal ideation, and exclusionary Axis I diagnoses). Of the SCID-I/NP administrations, 20% were reviewed by the principal investigator (PI) of the larger study (E.B.) to ensure interrater agreement on symptom codings and diagnoses, with no diagnostic disagreements noted.

The *Clinician-Administered PTSD Scale* (CAPS; Blake et al., 1995) is considered the “gold standard” for PTSD assessment (Weathers, Keane, & Davidson, 2001) and was administered in the current study to determine current (past month) PTSD symptom severity (i.e., frequency and intensity) and diagnosis. The CAPS Life Events Checklist was used to determine the number of lifetime PTEs. Past work has found that the Life Events Checklist has good test–retest reliability, good convergent validity with the Traumatic Life Events Checklist (Kubany et al., 2000), and significant associations with PTSD symptom severity (Gray, Litz, Hsu, & Lombardo, 2004). All reported events were summed to index the number of PTEs experienced. Criteria A1 and A2 were assessed for individuals’ “worst” traumatic event only. Of the CAPS administrations, 20.2% were reviewed by the PI of the larger study (E.B.) to ensure interrater reliability, with no cases of diagnostic disagreement. The current study utilized the sum of the frequency and intensity ratings for the total CAPS severity score, along with severity scores for the four-symptom clusters that have been supported in several confirmatory factor analyses (i.e., reexperiencing, effortful avoidance, emotional numbing, and hyperarousal; King, Leskin, King, & Weathers, 1998).

The *Anxiety Sensitivity Index–III* (ASI-III; Taylor et al., 2007) is an 18-item measure on which respondents indicate, on a 5-point Likert-type scale (0 = *very little* to 4 = *very much*), the degree to which they are concerned about possible negative consequences of anxiety symptoms (e.g., “*It scares me when my heartbeats rapidly*”). The current study utilized the total ASI-III score, as it represents the global-order AS factor, and internal consistency of ASI-III total score in the current sample was excellent ($\alpha = .91$).

The *Big Five Inventory* (BFI; John & Srivastava, 1999) is a 44-item self-report measure assessing the Big Five personality traits (i.e., Extraversion, Agreeableness, Conscientiousness, Neuroticism, and Openness; John, 1989). Participants indicate, on a 5-point Likert-type scale (1 = *disagree strongly* to 5 = *agree strongly*), the degree to which phrases corresponding to the adjectives considered to be markers of the five personality domains applies to them. The current study utilized the Neuroticism sub-scale of the BFI (e.g., “*is depressed, blue*”) to index participants’ general tendency to experience negative mood states, and internal consistency of BFI-N total score in the current sample was good ($\alpha = .88$).

The *Menstrual Distress Questionnaire* (MDQ; Moos, 1968) is a 47-item self-report measure that assesses retrospective premenstrual symptom severity on a 7-point Likert scale (0 = *did not experience* to 7 = *severe or intense experience*). For each symptom, respondents are instructed to select which rating best describes “[their] experience during the week immediately before [their] last menstrual cycle.” The current study used the MDQ total

score as a measure of global premenstrual distress, and internal consistency of MDQ total score in the current sample was excellent ($\alpha = .95$).

Procedure

Participants were recruited via flyers and advertisements placed in local newspapers in the greater Burlington, Vermont community. Interested participants, who provided informed consent, completed a screening session to determine eligibility. In this session they were administered the SCID-I/NP and the CAPS by a trained graduate student, and eligible participants were invited to complete the self-report measures. They were compensated \$10 for their participation. This study was approved by the IRB at the University of Vermont.

Data-Analytic Plan

Analyses were conducted in IBM SPSS Statistics 19.0, and all tests were two-tailed with an alpha level set at .05. First, zero-order correlations were conducted among variables of interest. Second, a series of hierarchical multiple regressions were conducted to examine the unique effect of premenstrual symptoms (MDQ total score), and the interactive effects of AS (ASI-III total score) and premenstrual symptoms on PTSD total symptom severity (i.e., CAPS total score) and revised symptom cluster severity scores (i.e., CAPS reexperiencing, avoidance, numbing, and hyperarousal scores; King et al., 1998). The number of PTEs, neuroticism (BFI-N) score, and past 2-year nonclinical panic attacks (yes/no; panic group) were entered in Step 1, ASI and MDQ total scores were mean-centered and entered in Step 2, and their interaction term was entered into Step 3. Significant interactions were examined graphically (Aiken & West, 1991) and statistically (Holmbeck, 2002) post hoc.

Results

Descriptive Statistics and Zero-Order Correlations

See Table 2 for descriptive statistics and zero-order correlations among study variables. MDQ total scores evidenced low to moderate (r s range from .27 –.55) significant correlations with number of traumas endorsed, panic attack status group, BFI-Neuroticism, and ASI-III (p s < .05). MDQ was significantly positively related to all PTSD symptom severity outcomes (p s < .05), whereas ASI-III and number of PTEs were only significantly positively related to total PTSD severity and PTSD reexperiencing severity (p s < .05), and BFI-N was significantly positively related to all PTSD symptom severity outcomes (p s < .05) with the exception of PTSD avoidance severity. Finally, panic attack group was significantly positively related only to PTSD hyperarousal severity (p < .05).

Unique and Interactive Effects of Premenstrual Symptoms and Anxiety Sensitivity on PTSD Symptom Severity

Complete regression results are reported in Table 3. For CAPS total symptom severity, Step 1 of the model (BFI-Neuroticism, number of PTEs, and panic group) accounted for 25% of unique variance with only BFI-Neuroticism evidencing a significant unique effect (p < .01). Step 2 of the model (MDQ and ASI) accounted for an additional 13% of unique variance with only MDQ evidencing significant unique variance (p < .01). Step 3 of the model (MDQ

× ASI) accounted for an additional 5% of unique variance ($t = 2.04$, $\beta = .24$, $sr^2 = .08$, $p < .05$).

See Figure 1 for a graphical representation of the MDQ × ASI interaction on PTSD symptom severity. As depicted, individuals with increased levels of both AS and premenstrual symptoms reported the greatest PTSD symptom severity. Post hoc probing analyses of the simple slopes and interactions supported the significant moderating role of ASI in the relation between MDQ and PTSD symptom severity. Specifically, the association between MDQ total score and PTSD symptom severity was statistically significant when ASI total score was high ($t = 3.97$, $\beta = .66$, $sr^2 = .24$, $p < .001$), while controlling for covariates, such that individuals who reported more premenstrual symptoms reported the greatest PTSD symptoms severity. However, the relation between MDQ total score and PTSD symptom severity was not statistically significant when ASI total score was low ($t = 1.37$, $\beta = .25$, $sr^2 = .04$, $p = .18$), while controlling for covariates.

For CAPS reexperiencing symptom severity, Step 1 of the model (BFI-Neuroticism, number of PTEs, and panic group) accounted for 25% of unique variance with BFI-Neuroticism ($p < .01$) and number of PTEs ($p < .05$) evidencing significant unique effects. Step 2 of the model (MDQ and ASI) accounted for an additional 9% of unique variance with only MDQ evidencing significant unique effect ($p < .05$). Step 3 of the model (MDQ × ASI) accounted for an additional 7% of unique variance ($t = 2.50$, $\beta = .30$, $sr^2 = .11$, $p < .05$).

See Figure 2 for a graphical representation of the MDQ × ASI interaction on PTSD reexperiencing symptom severity. As depicted, individuals with increased levels of both AS and premenstrual symptoms reported the greatest PTSD reexperiencing symptom severity. Post hoc probing analyses of the simple slopes and interactions supported the significant moderating role of ASI in the relation between MDQ and PTSD reexperiencing symptom severity. Specifically, the association between MDQ total score and PTSD reexperiencing symptom severity was statistically significant when ASI total score was high ($t = 3.61$, $\beta = .60$, $sr^2 = .20$, $p < .01$), while controlling for covariates, such that individuals who reported more premenstrual symptoms reported the greatest PTSD reexperiencing symptom severity. However, the relation between MDQ total score and PTSD reexperiencing symptom severity was not statistically significant when ASI total score was low ($t = .53$, $\beta = .10$, $sr^2 = .01$, $p = .60$), while controlling for covariates.

For CAPS avoidance symptom severity, Step 1 of the model (BFI-Neuroticism, number of PTEs, and panic group) did not account for any significant variance. Step 2 of the model (MDQ and ASI) accounted for an additional 8% of unique variance with only MDQ evidencing a significant unique effect ($p < .05$). Step 3 of the model (MDQ × ASI) was not significant.

For CAPS hyperarousal symptom severity, Step 1 of the model (BFI-Neuroticism, number of PTEs, and panic group) accounted for 18% of unique variance with only BFI-Neuroticism evidencing a significant effect ($p < .01$). Step 2 of the model (MDQ and ASI) accounted for an additional 20% of unique variance with only MDQ evidencing a significant effect ($p < .01$). Step 3 of the model (MDQ × ASI) was not significant.

For CAPS numbing symptom severity, Step 1 (BFI-Neuroticism, number of PTEs, and panic group) accounted for 15% of unique variance with only BFI-Neuroticism evidencing a significant effect ($p < .05$). Step 2 of the model (MDQ and ASI) did not account for any significant unique variance. Step 3 of the model (MDQ $\times$ ASI) accounted for an additional 7% of unique variance ($p < .05$).

See Figure 3 for a graphical representation of the MDQ $\times$ ASI interaction on PTSD numbing symptom severity. As depicted, individuals with increased levels of both AS and premenstrual symptoms reported the greatest PTSD numbing symptom severity. Post hoc probing analyses of the simple slopes and interactions supported the significant moderating role of ASI in the relation between MDQ and PTSD numbing symptom severity. Specifically, the association between MDQ total score and PTSD numbing symptom severity was statistically significant when ASI total score was high ($t = 2.57$, $\beta = .48$, $sr^2 = .11$, $p < .05$), while controlling for covariates, such that individuals who reported more premenstrual symptoms reported the greatest PTSD numbing symptom severity. However, the relation between MDQ total score and PTSD numbing symptom severity was not statistically significant when ASI total score was low ($t = -.07$, $\beta = -.02$, $sr^2 = .00$, $p = .94$), while controlling for covariates.

Given the high proportion of individuals experiencing nonclinical panic attacks ($n = 49.2\%$), we examined the interaction between panic group and MDQ total score, while controlling for BFI-Neuroticism, number of PTEs, and anxiety sensitivity in order to examine whether this effect was specific to anxiety sensitivity. Results revealed no significant Panic Group $\times$ MDQ interactions on any of the PTSD symptom severity outcomes ($ps > .05$). Additionally, the pattern of main effects for MDQ remained the same and panic group exhibited no significant main effects ($ps > .05$).

Discussion

Previous research suggests a relation between premenstrual symptoms and PTSD symptoms (Pilver et al., 2011). However, there are limited data on whether premenstrual symptoms uniquely predict PTSD symptom severity above and beyond other theoretically relevant factors as well as how they may interact with other risk factors. The current study examined the incremental validity as well as the interactive effects of premenstrual symptoms and AS on trauma symptom severity among a sample of trauma-exposed women.

Consistent with past studies (Pilver et al., 2011; Wittchen et al., 2003), premenstrual symptoms uniquely predicted PTSD total, reexperiencing, avoidance, and hyperarousal symptom severity above and beyond number of PTEs, neuroticism, panic attack history, and AS. The present findings extend the literature by demonstrating that premenstrual symptoms and AS evidence a synergistic effect on PTSD total, reexperiencing, and numbing symptom severity. Specifically, among individuals high in AS, greater premenstrual symptoms were related to greater PTSD symptom severity, particularly reexperiencing and numbing symptom. This relation was not evident for individuals low on AS, suggesting that the experience of premenstrual symptoms may be a PTSD-relevant vulnerability factor only among specific at-risk individuals (i.e., high AS). One potential explanation for this finding

is that premenstrual symptoms may play a maintaining role in PTSD symptoms among individuals high in AS. Given that individuals high on AS are more likely to misinterpret physiological sensations as dangerous, they may be more likely to notice and misinterpret physiological sensations that occur naturally during the premenstrual phase or misattribute them to external trauma cues, thereby maintaining PTSD symptoms.

Several important study limitations should be noted. First, this study used only retrospective reports of typical premenstrual symptoms, which may be less accurate than prospective reporting (Logue & Moos, 1986; Marvan & Cortes-Iniestra, 2001). For example, women report greater premenstrual symptoms when measured retrospectively as compared with when they were measured prospectively on the same premenstrual phase using the MDQ (Marvan & Cortes-Iniestra, 2001). Therefore, it is possible that the premenstrual distress reported in this study may be inflated, and future work would benefit from replicating this study using a prospective assessment (e.g., daily tracking) of premenstrual symptoms. Second, this study did not assess current or past clinical forms of premenstrual distress (i.e., PMDD). Future research would benefit from comparing nonclinical forms of premenstrual distress to individuals with current or past PMDD. Third, the nature of the relation between premenstrual and PTSD symptoms is not well understood and cannot be answered within the context of the current study. Specifically, this study does not clarify the causal nature of the relationship between premenstrual symptoms and PTSD symptoms. Future studies using experimental methodologies and longitudinal designs will be useful for understanding the direction and potential causal and bidirectional nature of the observed relations. Fourth, this was a small sample, which may have reduced power to detect main effects, and future studies should replicate these findings with larger samples, as well as examine other factors that may be related to menstrual distress and PTSD symptom severity (e.g., age). Finally, several factors may have influenced the generalizability of these findings: (a) the present sample was a relatively homogenous (e.g., primarily Caucasian) group of young adults; (b) PTSD severity was low in this sample; and (c) prevalence of nonclinical panic attacks was over-sampled in this study due to the nature of the larger investigation. Previous studies have found an association between panic attacks and PTSD symptomatology. For example, 69% of trauma-exposed individuals seeking treatment for their PTSD symptoms experienced panic attacks, particularly the cognitive symptoms of panic (e.g., fear of going crazy; Falsetti & Resnick, 1997). However, the high percentage of individuals experiencing nonclinical panic attacks in this nontreatment-seeking sample may have inflated the amount of individuals high in interoceptive sensitivity, which may have impacted study findings. Additionally, given the well-documented finding of anxiety sensitivity as a risk factor for panic psychopathology (Schmidt, Zvolensky, & Maner, 2006), and to a lesser extent PTSD (Elwood et al., 2009), it is possible that anxiety sensitivity serves as a mechanism underlying both panic and PTSD pathology. Indeed, post hoc analyses that examined the interaction of panic group and premenstrual symptoms lends support to the unique role of anxiety sensitivity, as opposed to general experience of nonclinical panic attacks, in PTSD symptom severity. It will be important for future work to include more diverse samples and individuals with more severe clinical presentations.

The present findings suggest that premenstrual symptoms may be an important sex-specific mechanism involved in increasing vulnerability for PTSD symptoms among women.

Clinically, monitoring premenstrual symptoms among women with PTSD may have relevant treatment implications, particularly if they experience premenstrual exacerbation of PTSD symptoms. Future work is needed to better understand how premenstrual symptoms as well as the menstrual cycle are related to the etiology and/or maintenance of PTSD symptomatology.

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Figure 1.
Premenstrual Symptoms $\times$ Anxiety Sensitivity on PTSD Total Symptom Severity.

Figure 2.
Premenstrual Symptoms $\times$ Anxiety Sensitivity on PTSD Reexperiencing Symptom Severity.

Figure 3.
Premenstrual Symptoms $\times$ Anxiety Sensitivity on PTSD Numbing Symptom Severity.

Table 1Participant Characteristics ($N = 63$)

Age, Mean (<i>SD</i>)	21.02 (6.07)
Race, Number (%)	
Caucasian	59 (93.7)
Asian	1 (1.6)
Hispanic/Latino	1 (1.6)
Biracial	1 (1.6)
Marital status, number (%)	
Single	60 (95.2)
Married	1 (1.6)
Divorced	1 (1.6)
Highest education, number (%)	
High School/GED	10 (15.9)
Some college	45 (71.4)
Graduated college	5 (7.9)
Graduate degree	2 (3.2)
Axis I diagnoses, number (%)	
Major depressive disorder	4 (6.3%)
Bipolar II	1 (1.6%)
Dysthymic disorder	4 (6.3%)
Social phobia	8 (12.7%)
Specific phobia	6 (9.5%)
Obsessive compulsive disorder	3 (4.8%)
Generalized anxiety disorder	9 (14.3%)
Alcohol abuse	5 (7.9%)
Alcohol dependence	3 (4.8%)
Substance abuse	5 (7.9%)
Substance dependence	6 (9.5%)

Table 2
Descriptive Statistics and Zero-Order Correlations Among Predictor and Criterion Variables

	1	2	3	4	5	6	7	8	9	10	<i>M (SD) or %</i>
1. Number of PTEs	1	-.004	.11	-.05	.31*	.31*	.35**	.19	.21	.22	7.02 (4.08)
2. BFI-Neuroticism		1	.35**	.62**	.55**	.41**	.38**	.22	.32*	.38**	25.53 (7.02)
3. Panic group			1	.37**	.27*	.24	.19	.04	.20	.27*	56%
4. ASI-III				1	.33*	.28*	.31*	.21	.17	.21	17.50 (11.21)
5. MDQ					1	.57**	.53**	.33*	.33*	.58**	95.05 (35.98)
6. CAPS symptom severity						1	.94**	.71**	.74**	.83**	11.48 (14.46)
7. CAPS reexperiencing severity							1	.66**	.65**	.68**	4.60 (5.91)
8. CAPS avoidance severity								1	.35**	.49**	1.50 (2.54)
9. CAPS numbing severity									1	.42**	1.86 (3.83)
10. CAPS arousal severity										1	3.52 (5.29)

Note. Total number of PTEs = lifetime potentially traumatic events endorsed on the Clinician Administered PTSD Scale (CAPS)–Life Events Checklist; BFI-Neuroticism = Big Five Inventory–Neuroticism subscale; Panic Group = panic attack group status (i.e., meets criteria for a past 2-year history of nonclinical, unexpected panic attacks); ASI-III = Anxiety Sensitivity Index–III total score; MDQ = Menstrual Distress Questionnaire Total Score; CAPS symptom, reexperiencing, avoidance, numbing, and hyperarousal severity = CAPS–Total PTSD symptom, reexperiencing, avoidance, numbing, and hyperarousal severity (frequency + intensity).

* $p < .05$.

** $p < .01$.

Table 3
Main and Interactive Effects of Premenstrual Symptoms and Anxiety Sensitivity Predicting PTSD Symptoms

	<i>R</i> ²	95% CI	<i>R</i> ²	<i>t</i>	β	<i>sr</i> ²	<i>p</i>
DV: CAPS–Total score							
Step 1	.25	.07, .43	.245				.001
BFI–Neuroticism				3.35	.42	.11	.002
PTEs				1.85	.22	.06	.07
Panic group				.45	.06	.00	.66
Step 2	.38	.20, .56	.131				.04
MDQ				3.31	.49	.17	.002
ASI				.06	.01	.00	.96
Step 3	.42	.25, .59	.047				.02
MDQ by ASI interaction term				2.04	.24	.08	.04
DV: CAPS–Reexperiencing							
Step 1	.25	.08, .41	.246				.001
BFI–Neuroticism				3.03	.38	.14	.004
PTEs				2.60	.31	.11	.01
Panic group				.05	.01	.00	.96
Step 2	.34	.17, .51	.089				.036
MDQ				2.58	.38	.11	.01
ASI				.66	.09	.00	.51
Step 3	.41	.25, .57	.071				.02
MDQ by ASI interaction term				2.50	.30	.11	.02
DV: CAPS–Avoidance							
Step 1	.08	–.04, .20	.082				.19
BFI–Neuroticism				.09	.02	.00	.93
PTEs				–.13	–.02	.00	.90
Panic group				–.89	–.12	.01	.38
Step 2	.16	.01, .31	.082				.09
MDQ				2.09	.36	.08	.04
ASI				.80	.13	.01	.43

	<i>R</i> ²	95% CI	<i>R</i> ²	<i>t</i>	β	<i>sr</i> ²	<i>p</i>
Step 3	.17	.02, .32	.010				.42
MDQ by ASI interaction term				.81	.11	.01	.42
DV: CAPS-Hyperarousal							
Step 1	.18	.01, .34	.182				.01
BFI-Neuroticism				2.69	.35	.12	.009
PTEs				1.02	.13	.02	.31
Panic group				.92	.12	.02	.36
Step 2	.38	.20, .56	.20				.001
MDQ				4.00	.59	.24	<.001
ASI				-.72	-.10	.01	.47
Step 3	.38	.21, .55	.005				.52
MDQ by ASI interaction term				.64	.08	.01	.52
DV: CAPS-Numbing							
Step 1	.15	-.01, .31	.151				.03
BFI-Neuroticism				2.67	.35	.11	.01
PTEs				.45	.06	.00	.66
Panic group				.57	.08	.01	.57
Step 2	.20	.04, .36	.047				.22
MDQ				1.68	.28	.05	.10
ASI				-.56	-.09	.01	.58
Step 3	.26	.09, .43	.066				.04
MDQ by ASI interaction term				2.15	.28	.08	.04