The Relationship Between Gender and PTSD Symptoms: Anxiety Sensitivity as a Mechanism

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Abstract

Despite well-established gender differences in the rates of Posttraumatic Stress Disorder (PTSD), there is a lack of research examining malleable, gender-linked risk factors that could be targeted in interventions to reduce risk among women. One such risk factor is anxiety sensitivity (AS), or a fear of anxiety related sensations. AS is elevated in women compared to men and is related to PTSD symptom severity. The current study provided a preliminary examination of whether AS could partially explain the association between gender and PTSD symptoms. Baseline data from a randomized controlled trial for elevated AS was used to explore this question in a sample of trauma-exposed individuals ($N=37$; 22 with a current PTSD diagnosis). Bias-corrected bootstrap mediation revealed that AS helped statistically explain the relationship between gender and PTSD symptoms. These results promote the potential importance of AS as a mechanism in the PTSD gender discrepancy. Future work should investigate these relationships longitudinally and establish whether targeting AS in a prevention paradigm could reduce the risk of PTSD development in women.

Keywords: gender, risk, anxiety sensitivity, posttraumatic stress, PTSD

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1. Introduction

Following a traumatic event, many individuals experience a stress reaction, which can lead to the development of Posttraumatic Stress Disorder (PTSD). PTSD, a psychiatric disorder marked by symptoms of reexperiencing, avoidance, negative cognitions or mood, and hyperarousal, represents a considerable personal and economic burden (American Psychiatric Association, 2013). Although a lifetime history of trauma is relatively common, only 7.8% of those who experience a traumatic event develop PTSD (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Moreover, research has consistently demonstrated that women are twice as likely as men to develop PTSD (e.g., Kessler et al., 1995). Although gender differences exist in the types of traumatic events experienced (e.g., women are more likely to be victims of sexual assault) and the number of traumatic events experienced, neither of these provide an adequate explanation for the elevated risk for PTSD seen among women (Tolin & Foa, 2002). Our current lack of understanding of this discrepancy highlights the need for further investigation of factors that may contribute to gender differences in PTSD.

Identifying gender-linked risk factors that are malleable, referred to as variable risk factors (Kraemer et al., 1997), is particularly critical to potentially mitigating the higher risk of PTSD in women. Anxiety Sensitivity (AS) is one such variable risk factor. AS is a cognitive style that reflects an individual's tendency to interpret benign, anxiety-related sensations as harmful or dangerous. This maladaptive interpretation is believed to arise from the belief that these arousal sensations will have negative cognitive, physical, or social consequences (Reiss, Peterson, Gursky, & McNally, 1986). For example, an individual with high AS may interpret a racing heartbeat as indicative of an impending heart attack, whereas an individual with low AS

may interpret this merely as an uncomfortable sensation. Empirical work has established AS comprises three subfactors: cognitive, physical, and social concerns (Taylor et al., 2007). AS has been linked to a number of anxiety, mood, and stress disorders (see Olatunji & Wolitzky-Taylor, 2009 for a review), and has demonstrated malleability through intervention. For example, one study found a brief computerized treatment yielded significant reductions in AS, and in turn reductions in anxiety and depressive symptoms (Schmidt, Capron, Raines, & Allan, 2014). Importantly, studies assessing gender differences in AS have repeatedly shown women to report higher levels of AS in both clinical (Schmidt & Koselka, 2000) and nonclinical (Deacon, Abramowitz, Woods, & Tolin, 2003; Stewart, Taylor, & Baker, 1997) samples. Thus, although unclear why women display elevated levels of AS compared to men, AS is a gender-linked risk factor that can be ameliorated.

Empirical work has also linked AS with the development and maintenance of PTSD (Elwood, Hahn, Olatunji, & Williams, 2009; Taylor, 2003). Theoretically, elevated AS prior to a traumatic event could exacerbate distress during and after the trauma through a positive feedback cycle of increasing anxiety symptoms, as the individual fears both the traumatic event and the anxiety symptoms caused by the event. This may amplify their emotional response and contribute to the development of avoidance and other PTSD symptoms. It has also been demonstrated that after the experience of a traumatic event, AS and PTSD symptoms maintain each other bidirectionally in which individuals learn to fear anxiety-related sensations due to their association with a trauma (Marshall, Miles, & Stewart, 2010). Despite a plethora of research demonstrating AS to be related to both gender and PTSD symptoms, no studies have tested whether gender differences in AS may help explain the gender discrepancies in PTSD symptoms.

The current study presents a novel approach to understanding heretofore unexplained gender differences in PTSD symptoms. Women report greater AS compared to men (Deacon et al., 2003; Schmidt & Kosleka, 2000), and elevated AS has been established as a risk factor for PTSD (Elwood et al., 2009; Taylor, 2003). Additionally, previous research has demonstrated that AS serves to partially explain the greater report of general anxiety and mood symptoms, as well as agoraphobic avoidance, among women (Norr, Albanese, Allan, & Schmidt, 2015; Schmidt & Koselka, 2000). Therefore, we hypothesized a significant indirect path from gender to PTSD symptoms through AS in a trauma-exposed sample.

2. Method

2.1 Participants and Procedure

Thirty-seven individuals who endorsed a trauma history during a semi-structured clinical interview were selected from a larger sample of community participants ($N=108$) recruited to participate in AS intervention trial because they scored above the community mean on the Anxiety Sensitivity Index-3 (for procedural details, see Schmidt et al., 2014). Data for the current study were collected prior to treatment. Traumatic events reported included: serious accident/fire/explosion (29.7%), physical (13.5%) and sexual (32.4%) assault, combat (24.3%), and other (64.9%). Ages ranged from 18 to 68 ($M=43.41$, $SD=14.55$) and the majority self-identified as women (56.8%). Participants self-identified as White/Caucasian (62.2%), Black/African American (29.7%), other (5.4%; e.g., biracial), and Hispanic/Latino (2.7%). Education levels were self-reported as: some high school (2.7%), high school diploma or equivalent (18.9%), technical degree/some college (56.7%), or bachelor's/graduate degree (21.7%). Yearly family income was self-reported as: <\$10,000 (24.3%), \$10,000-\$40,000 (48.6%), \$40,000-\$100,000 (24.3%), or >\$100,000 (2.8%). Over half (59.5%) of participants

met criteria for a current PTSD diagnosis, as assessed through the SCID. All procedures were approved by the University's institutional review board and written informed consent was obtained prior to study enrollment.

2.2 Measures

Structured Clinical Interview for DSM-IV (SCID). The SCID is a psychometrically sound, semi-structured interview that was used to assess for DSM-IV psychiatric diagnoses and trauma history (First, Spitzer, Gibbon, & Williams, 1996). Trained doctoral student therapists administered the SCID and diagnoses were reviewed by a licensed clinical psychologist. Percentage agreement between clinical interviewers for a random sample of approximately 15% of these interviews resulted in high interrater agreement (over 80% with a kappa of .77).

Anxiety Sensitivity Index-3 (ASI-3). The ASI-3 is an 18-item self-report questionnaire that measures feared consequences of anxiety-related sensations and is a psychometrically sound and valid measure of anxiety sensitivity (Reiss, Peterson, Gursky, & McNally, 1986; Taylor et al., 2007)). Internal consistency in the current study was excellent for the total score ($\alpha=.94$), physical ($\alpha=.90$), cognitive ($\alpha=.95$), and social ($\alpha=.90$) subscales.

PTSD Checklist–Civilian Version (PCL-C). The PCL-C is a 17-item self-report measure assessing the DSM-IV symptoms for PTSD that has demonstrated good psychometric properties (Weathers, Litz, Herman, Huska, & Keane, 1993). In the present investigation, the PCL-C demonstrated excellent internal consistency ($\alpha=.92$).

3. Results

3.1 Preliminary Analyses

Participants were on average elevated on the ASI-3 and the PCL-C (Table 1). No threats to normality, homoscedasticity, or multicollinearity were found. Individuals with a PTSD

diagnosis had significantly higher PCL-C scores ($M=56.32$, $SD=15.25$) than those without PTSD ($M=44.00$, $SD=10.76$; $t_{(35)}=2.70$, $p=.01$). However, there was no difference in ASI-3 scores between those with a PTSD diagnosis ($M=33.73$, $SD=19.03$) and those without ($M=34.47$, $SD=16.34$; $t_{(35)}=0.12$, $p=.90$), likely due to the presence of comorbid anxiety pathology in both groups, as other anxiety disorders are also associated with elevated levels of AS (Olatunji & Wolitzky-Taylor, 2009). Chi-square tests revealed no differences ($ps>.05$) between groups on the presence of other anxiety diagnoses (i.e., panic, social anxiety, generalized anxiety, and obsessive-compulsive disorders), or race. A t-test revealed no difference between groups on age ($p=.65$). ASI-3 scores were significantly associated with PCL-C scores (Table 1). Women displayed marginally higher scores on the ASI-3 ($M=38.76$, $SD=17.66$) compared to men ($M=27.81$, $SD=16.37$; $t_{(35)}=1.93$, $p=.06$) and non-significantly higher scores on the PCL-C ($M=53.29$, $SD=14.68$) compared to men ($M=48.75$, $SD=14.97$; $t_{(35)}=0.92$, $p=.36$), although these relationships did not reach statistical significance likely due to the limited sample size. However, as it is well-established that a significant direct effect (gender to PCL-C scores) is not required for statistical mediation (Shrout & Bolger, 2002), and the literature has demonstrated a robust relationship between gender and AS (Deacon et al., 2003; Schmidt & Kosleka, 2000; Stewart et al., 1997), as well as PTSD symptoms (Elwood et al., 2009; Taylor, 2003), in larger samples, we proceeded with the mediation analysis.

3.2 Mediation Analyses

To test the hypothesis that there will be a significant indirect path from gender to PTSD symptoms through AS, PROCESS for SPSS (Hayes, 2012) was used to create a bias-corrected bootstrap mediation model with 5,000 random bootstrap samples, yielding 95% confidence intervals of the estimates. The values for the paths from the IV (gender) to the mediator (ASI-3)

and the paths from the mediator to the dependent variable (PCL-C) were computed, as well as the indirect effect. The overall model was statistically significant ($F(2, 34)=7.28, p=.002, R^2=.30$; Figure 1). As predicted, there was a significant indirect effect from gender to PCL-C scores through ASI-3 scores (indirect=5.03, 95% CI [0.44,13.26], $\kappa^2=.18$).

To explore potential unique indirect effects through AS subfactors, a parallel mediation model was tested with all three AS subfactors (cognitive, physical, and social). This model revealed no unique indirect effects through ASI-3 cognitive (indirect=0.35, 95% CI [-3.01,6.44]), ASI-3 physical (indirect=2.39, 95% CI [-0.37,10.08]), or ASI-3 social scores (indirect=2.18, 95% CI [-0.23,10.58]).

4. Discussion

The results of the current study provide preliminary evidence that AS is a variable risk factor that could partially explain elevated levels of PTSD symptoms among women documented in the literature. In support of our initial hypothesis, there was a significant indirect effect between gender and PTSD symptoms, indicating that AS statistically accounts for part of the relationship between gender and PTSD symptoms, which is consistent with a mediational process. These findings are consistent with previous work that has found elevated rates of AS among women (Deacon et al., 2003; Schmidt & Koselka, 2000), that AS is a risk factor for PTSD symptoms (Elwood, Hahn, Olatunji, & Williams, 2009; Taylor, 2003), and that AS partially explains the relationship between gender and symptoms of anxiety and mood psychopathology (Norr et al., 2015). Analyses revealed no unique indirect effects through any of the AS subfactors, likely due to the indirect effect being driven by the overlapping variance among the subfactors (representing AS overall) rather than their unique variances.

The findings from the current study help clarify our understanding of elevated PTSD symptoms among women. Prior work suggests that elevated levels of AS among women could result in elevated PTSD symptoms via a bidirectional escalation between AS and PTSD symptoms (Marshall et al., 2010). It is also possible that elevated AS at the time of the traumatic event could lead to greater PTSD symptoms through amplified emotional responding during the trauma (Taylor, 2003). Thus, although speculative, elevated AS among women could confer additional risk for elevated symptoms of PTSD through multiple avenues, suggesting that amelioration of elevated AS may yield substantial benefits for the prevention of PTSD symptoms among women.

Fortunately, prior research has demonstrated that AS can be reduced (e.g., Schmidt et al., 2014; Watt, Stewart, Birch, & Bernier, 2006), and that the reduction of AS can lead to subsequent reductions in PTSD symptoms (e.g., Olthuis, Watt, Mackinnon, & Stewart, 2014). Although AS interventions have been shown to prevent the development of anxiety psychopathology broadly (Schmidt et al., 2007), future research should determine if an AS intervention prior to the experience of a traumatic event can reduce the risk of future PTSD development in women.

It is important to consider the limitations of the current study. First, due to a relatively small sample size ($N=37$) the results of the current study should be considered with caution. However, given the novelty of the current investigation, these results are an important step forward in the literature. Second, as these data are not longitudinal in nature, we cannot claim true causal mediation, only the presence of a significant indirect effect, which is consistent with a mediational process. Future research should investigate temporal mediation to establish true causality. Third, given that the current sample consisted of high risk individuals (individuals with

elevated AS) these findings might not generalize beyond a high risk population. Fourth, given evidence of reciprocal relations between AS and PTSD (Marshall et al., 2010) it is possible that gender differences in PTSD in response to trauma give rise to gender differences in AS, which could be tested via a longitudinal design. Finally, given the small sample we were unable to investigate whether AS statistically mediated the relationship between gender and PTSD diagnostic status due to the limited variability caused by the dichotomous nature of gender and PTSD diagnostic status.

Despite these limitations, this study significantly advances the current literature by proposing AS as a potential risk factor that could help account for the gender discrepancy in the experience of PTSD symptoms. Given the malleable nature of AS, it appears to be a potentially ideal target for reducing the elevated risk of PTSD symptom development in women.

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Figure 1. Mediation model examining the indirect effect of gender on PCL-C through ASI-3. ASI-3=Anxiety Sensitivity Index-3; PCL-C=PTSD Checklist-Civilian Version; 95% confidence intervals presented in brackets.

Table 1

Descriptives and Intercorrelations

	1	2	3	4	<i>M</i>	<i>SD</i>
1. ASI-3 Total	--				34.03	17.76
2. ASI-3 Physical	.86*	--			10.24	6.98
3. ASI-3 Cognitive	.86*	.63*	--		11.89	7.44
4. ASI-3 Social	.80*	.54*	.52*	--	11.89	6.67
5. PCL-C	.55*	.51*	.40*	.48*	52.32	14.77

Note. ASI-3=Anxiety Sensitivity Index–3; PCL-C=PTSD Checklist–Civilian Version. * $p < .01$