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The association of interpersonal trauma with somatic symptom severity in a primary care population with chronic pain: exploring the role of gender and the mental health sequelae of trauma

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Abstract

Objective—Female trauma survivors develop somatic symptoms more frequently than male survivors. We propose a model for somatic symptom development among trauma survivors, focusing on gender differences.

Methods—Among 597 urban primary care patients with chronic pain, we examined the association between somatic symptom severity and three interpersonal trauma types: 1) sexual trauma (ST), 2) intimate partner violence (IPV), and 3) childhood trauma history (≥ 3 adverse childhood experiences (3+ACE)). We developed a structural equation model in which PTSD, depression, and substance abuse were evaluated as potential mediators of the path between trauma exposure and somatic symptom severity, and explored the role of gender in this model.

Results: 350 (59%) respondents were female; the mean age was 47. Women reported significantly more somatic symptoms than men, although somatic symptoms were increased among all interpersonal trauma survivors. In models in which the potential intervening variables are

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considered in aggregate, we did not find a significant interaction between gender and trauma on somatic symptom severity, with the exception of 3+ACEs. A structural equation model showed that depression and substance abuse, for men, and depression, for women, were associated with somatic symptom severity. PTSD was not associated with somatic symptom severity. Paths from trauma exposures to mental health sequelae were stronger for men.

Conclusions: Women have more severe somatic symptoms. With the exception of 3+ACEs, the association between trauma and somatic symptoms is similarly amplified in both genders. Structural equation models showed the pathways differed by gender in function and strength of association.

Keywords

adult; pain; depression; psychosomatic medicine; stress disorders; post-traumatic; substance-related disorders

Introduction

Traumatic life events are prevalent; nationally representative surveys indicate approximately 75% of the U.S. population [1] experience at least one during their lifetime [2]. Trauma survivors disproportionately utilize the healthcare system [2], commonly presenting with medically unexplained physical symptoms [3]. Although a history of trauma is strongly associated with the development of somatic symptoms, [4, 5] not all survivors of trauma develop adverse mental health conditions or somatization. Reasons for these differences include genetic variation [6], personality type, and socio-demographic factors [7], but the mechanism linking trauma exposure to adverse mental health is incompletely understood. Because interpersonal trauma is strongly linked to somatic symptoms, [8, 9] examining how somatic symptoms develop among survivors of interpersonal trauma is a critical step towards the development of therapeutic interventions to decrease these symptoms.

Associations between trauma exposures and psychiatric diagnoses have been extensively explored in the literature. Post-traumatic stress disorder (PTSD), a well-described consequence of trauma exposure characterized by re-experiencing the trauma, avoidance and hyper-arousal [10], affects approximately 8% of all U.S. citizens during their lifetime [1]. Among trauma survivors, a lifetime history of PTSD has been reported among 8% of men and 20% of women [11]. PTSD is more likely to develop with increasing number of trauma exposures [1]. Depression is highly comorbid with PTSD among trauma survivors, found in approximately half of all trauma survivors with PTSD, possibly because both PTSD and depression are associated with dysphoria and disturbances in mood and cognition [12]. Likewise, substance abuse is elevated among trauma survivors, because trauma-exposed individuals may self-medicate to treat trauma-related stress symptoms, or conversely, because substance abusers are more likely to engage in risky behaviors that can precipitate trauma exposure [13]. PTSD and depression are highly co-morbid with substance abuse among trauma survivors, and may contribute to the development of substance dependence among trauma survivors [14, 15].

Somatic symptoms are also prevalent among trauma survivors with PTSD. For example, one-third of returning veterans with PTSD have severe somatic symptoms [16]. Somatic symptoms are also linked to depression, and are often the presenting complaint among primary care patients with depression [17]. Although highly associated in the literature, a direct relationship between somatic disorders and substance abuse is less well established [18]. Although numerous studies examine associations between trauma exposure, PTSD, depression, substance abuse, and somatic symptom severity, most do not explore pathways that may link these conditions.

Prior research has shown that somatic complaints are increased among women trauma survivors compared to men who have had traumatic experiences [5, 19, 20]. Gender differences in biologic factors such as sensitization of stress hormones and neuroendocrine differences between men and women have been proposed to mediate differences in somatic symptoms in men and women trauma survivors [21]. Alternatively, increased somatic symptoms among women may be explained by gender differences in trauma type or frequency. For example, there is a higher prevalence of sexual trauma among women compared to men [22]. However, men who have experienced sexual coercion are likely to have greater levels of somatization compared to men who do not experience sexual coercion [23]. Likewise, intimate partner violence (IPV) is more likely to be reported by women compared to men [24-26], and literature suggests that IPV is associated with increased somatic symptoms in both men [27, 28] and women [29]. Adverse childhood experiences (ACEs) may differentially affect men and women, although the data on this trauma exposure is less straightforward. Some studies have shown that women experience more ACEs compared to men [30], whereas others studies have shown no gender difference in the frequency of ACEs [31]. Research in women suggests that ACEs are associated with increased somatic symptoms [32-34], although less is known in men.

The gender differential among somatic symptoms in trauma survivors may be mediated by differences in the impact of other trauma sequelae on somatic symptoms. These sequelae include post-traumatic stress disorder (PTSD), depression and substance abuse, all of which are associated with increased somatic symptoms [18, 35, 36]. Women who survive trauma are more susceptible to the development of PTSD than men who survive trauma [37]. Similarly, depression and anxiety disorders, which are more common among women, are among the strongest predictors of somatoform disorders [38]. Conversely, men with extreme alcohol use are more likely to have somatization symptoms compared to women [39]. Thus, gender differentials in PTSD, depression and substance abuse, all prevalent conditions among trauma survivors, may in part explain the gender disparity in somatic symptoms in this population.

Limited information examines mechanisms that explain the complex interconnections between trauma, PTSD, depression and substance abuse, and somatic symptoms. We are not aware of a single model that incorporates all of these elements. Prior models have suggested that PTSD may partially, but incompletely, mediate the association between trauma exposures and substance abuse and depression [40-42] and that depression functions as a mediator between trauma exposure and drug use [43]. However, the role of these mental health sequelae of trauma as potential mediators in the association between trauma and

somatic symptom severity has been largely unexplored, other than one study examining PTSD in adolescents [42]. This study found that PTSD was a weaker mediator between trauma and mental health problem for males compared to females [42]. Most prior literature has not examined the role of gender in mediational models. Moreover, existing literature examining the association between trauma and somatic symptoms is limited in ability to describe gender differences due to study designs largely excluding women [16, 44] or men [45, 46].

To address this knowledge deficit, we developed a model to investigate the role of PTSD, depression, and substance use in the association between trauma (IPV, sexual trauma, and ACEs) and somatic symptoms in a primary care population with chronic pain. Of note, PTSD, depression and substance abuse are highly comorbid following trauma, with studies revealing that the likelihood of at least one other psychiatric diagnosis among persons with PTSD is 80% or higher [11, 47]. This suggests that these mental health sequelae of trauma together constitute a response to traumatizing events. Given the associations between PTSD, depression and substance abuse and somatic symptoms, the mental health sequelae of trauma could function as an intermediary in the association between traumatic exposures and somatic symptom severity, and may in part explain the gender difference in somatic symptoms observed in trauma survivors.

In the current study, we utilize several methodologies to explore the role of these potential intermediaries in explaining how gender impacts the association between potentially traumatizing events and somatic symptom severity. Specifically, we hypothesize that women will have a more exaggerated response to trauma compared to men, due to higher levels of somatic symptoms and higher rates of PTSD among women trauma survivors. We further examine a potential mediation model that explores whether the mental health sequelae of trauma, conceptualized as PTSD, depression and substance abuse, mediate somatic symptom severity among survivors of trauma. Finally, we examine whether this mediation model functions similarly between men and women.

Materials and Methods

Study Sample

Cross-sectional data were collected from the *PTSD, Addictions, Chronic Pain and Trauma* (PACT) study. The details of this study's design have been previously described [48]. Briefly, the PACT study was designed to establish the prevalence of cooccurring chronic pain, substance use disorder, and PTSD among patients presenting for primary care to an urban, academic medical center between 2005 and 2007. Trained research assistants invited consecutive, English-speaking patients, between 18 and 60 years of age, presenting to primary care clinics, to enroll in the study. After obtaining verbal consent, 2,194 patients were administered a short screening survey [49] to determine the presence of chronic pain. Of these, 822 (37%) were eligible, of whom 620 (75%) agreed to participate in the study, and 597 (96%) completed the interview. The Boston University Medical Center Institutional Review Board approved the study and a Certificate of Confidentiality was obtained from the National Institutes of Health. We selected a population of individuals with chronic pain in order to ensure our sample was enriched in trauma exposures, the mental health sequelae of

trauma, and somatic symptoms. This sampling strategy increased our power to understand the interrelationships between these variables.

Variables

To construct our main independent variable (interpersonal trauma exposure), we examined three independent traumatic exposures: 1) Intimate partner violence (IPV), 2) sexual trauma (ST), and 3) adverse childhood experiences (ACEs). Although a number of traumas were assessed during the PACT study interview, we selected these three traumas for evaluation given literature that strongly links interpersonal trauma to somatic symptoms. IPV was defined as one or more affirmative responses to questions adapted from the Add Health Home Questionnaire [50]; 2) ST was defined as sexual assault and molestation, based on exposures defined in the Composite International Diagnostic Instrument's (CIDI) PTSD module [51]; 3) ACEs were described from a 10-item scale adapted from Felitti [31], which includes measures of childhood abuse childhood household dysfunction, and has been shown to correlate to numerous adult mental and physical health problems. Participants were first asked to consider their experiences “while you were growing up, during the first 18 years of life.” Questions about childhood abuse included such items as “Did a parent or other adult in the home...” “...often push, grab, shove or slap you?” or “...ever touch you in a sexual place or make you touch them when you did not want them to?” Questions about childhood household dysfunction included items such as “Did you live with anyone who was a problem drinker or alcoholic?” or “Did a household member go to prison?” Our measure of 3 or more ACEs (3+ACEs) was dichotomized at the sample median. We also created a composite variable that described any of the above (IPV, ST, 3+ACEs).

Population descriptors included self-identified gender, age (in years), race/ethnicity (black, Hispanic, white, other), educational level, marital status, (partnered, divorced, single), employment status (employed, unemployed), and health insurance status (dichotomized in this analysis as insured/uninsured).

Intervening variables, i.e., those that were proposed to describe the mental health sequelae of trauma, included PTSD, depression and substance abuse. These variables were chosen because of their previously reported associations with trauma, somatic symptoms, and gender. PTSD was defined based on the CIDI version 2.1 PTSD Module [51], a validated instrument designed to measure psychiatric diagnoses based on DSM-IV criteria. PTSD could be in response to any trauma, not just the traumas included in the independent variable as described above. Depression was measured by the Patient Health Questionnaire (PHQ)-9, a screening questionnaire validated to measure depression symptoms and severity in racially and ethnically diverse primary care populations [52, 53]. Substance abuse was defined by meeting DSM-IV criteria for any lifetime drug (including prescription) abuse or dependence measured using the CIDI version 2.1 module on drug disorders and/or past year alcohol dependence as measured with the CIDI-short form [54]. Data on alcohol abuse and prior dependence was not measured to reduce respondent burden.

The dependent variable was somatic symptom severity, based on the PHQ-15 question survey, which is validated to measure the severity of somatic symptoms. Scores are categorized into minimal, low, medium, and high, and correlate with DSM-IV diagnosis of

somatization disorder [55]. The PHQ-15 was organized into categories to ensure that the outcomes measured in this research were clinically relevant. Due to the high somatic symptom severity in our chronic pain population, we combined the minimal and low categories into one category ('low'). Of note, one question on this scale refers to menstrual cramps and is thus appropriate for women only, giving women potentially higher scores on this instrument. However, given the validity and reliability of this instrument to detect somatoform disorders among both men and women [56], it is an appropriate tool for measuring this outcome.

Statistical Methods

We utilized several statistical techniques to investigate the association between trauma and somatic symptom severity in men and women. We first used chi-square tests for categorical data and t-tests for measurement data to examine associations between gender and demographics, trauma exposures, somatic symptom severity, and our proposed intervening variables (PTSD, depression, and substance abuse). We then used multivariable proportional odds regressions for ordinal categorical dependent variables [57] creating sequential models investigating the role of gender in the association between trauma and somatic symptom severity. The odds ratios from these models reflect the increase (or decrease) in the odds of being in a higher somatic symptom severity category, for a one unit increase in the independent variable. We model the outcome of a one category increase of more severe somatic symptoms, based on clinical class (low, medium, or high), which ensures that our models describe clinically relevant differences between groups. The proportional odds models allow for the examination of the effect of each of the proposed trauma exposures independently as well as in aggregate.

We developed three separate models for our three identified interpersonal trauma exposures (sexual trauma, IPV, and 3+ACE), as well as a model examining a history of any of these exposures. For these models we examined the interaction between gender and the trauma exposure on the outcome of somatic symptom severity, controlling for age, race, education and employment status. We then added as a group each of the three identified variables measuring the mental health sequelae of interpersonal trauma (substance abuse, depression and PTSD). In these models, we examined the following: First, if the association between interpersonal trauma and somatic symptom severity differed by gender, we would expect a significant interaction term. Second, if the proposed variables describing the mental health sequelae of trauma were correctly positioned in the proposed causal pathway, i.e., the variables intervene between interpersonal trauma exposure and somatic symptom severity, placing these variables in the model would attenuate, and perhaps eliminate, the association between trauma exposure and somatic symptom severity for both genders. These models could thus support positioning the mental health sequelae of interpersonal trauma as intervening variables between trauma exposure and somatic symptom severity, which would establish the logic of the structural equation model described below.

Finally, we developed and tested a structural equation model to explore the role of the proposed intervening variables in the association between interpersonal trauma and somatic symptom severity. The structural equation model allows for examination of the

directionality of the data, and the interrelationships between constructs tested in the model. In the structural equation model, we examined a latent construct comprised of all three types of interpersonal trauma exposure (IPV, sexual trauma, and 3+ACEs). We then examined paths from trauma exposure to each of the three intervening variables (PTSD, depression, and substance abuse), and paths from each of the three intervening variables to somatic symptom severity. We also included the direct path from interpersonal trauma to somatic symptom severity in the model. The three intervening variables in this model are dichotomous, and were modeled through a probit link. Model fit was evaluated through the comparative fit index (CFI >.95 is acceptable) and root mean square error of approximation (RMSEA<.05 is acceptable) [58]. Associations are described through standardized regression coefficients. We first fit a full model that included all possible pathways between model variables, and then used a backward elimination process to develop a parsimonious model that maintained good model fit.

To test whether the associations between interpersonal trauma, the three intervening variables, and somatic symptom severity differ by gender, we compared the fit of a two-group SEM model that allowed path coefficients to differ for males and females to a two-group SEM model that forced path coefficients to be the same for males and females. To further examine differences between males and females, we tested whether each individual pathway in the models differed for males and females by comparing a constrained versus unconstrained model. Finally, we developed a parsimonious two-group model using backward elimination. The structural equation modeling was performed using MPlus Software, Version 6.1; all other statistical analyses were performed using SAS Software, Version 9.3. P-values of <.05 were considered statistically significant in all analyses.

Results

Among 597 respondents, 59% were women and 41% were men. As shown in Table I, women were slightly more likely to be of Black or African-American race. Other key demographic features did not differ significantly by gender. As expected, interpersonal trauma frequencies and types differed significantly between women and men. Women were more likely than men to report a history of sexual trauma and intimate partner violence, but were equally likely to report three or more adverse childhood experiences. With respect to the main dependent variable, somatic symptom severity, women reported significantly more severe somatic symptoms than men. Only 23% of the women reported low somatic symptoms, compared to 42% of women reporting high somatic symptoms. Among the men, the reverse pattern was seen, with a higher proportion of men (47%) reporting low somatic symptoms compared to high somatic symptoms (24%).

Table II shows the results of the independent multivariable proportional odds regression equations. In model 1 of each panel, the association between interpersonal trauma exposure and somatic symptom severity is shown, with a term examining the interaction of gender and trauma. A single model for each interpersonal trauma is presented, with the results for men and women stratified to underscore our findings with respect to gender. Each of these models showed that trauma exposure was associated with increased odds of more severe somatic symptoms. We found no significant interaction between gender and trauma on

somatic symptom severity, with the exception of the model for 3+ACEs. Exposure to 3+ACEs was associated with 2.8 times the odds of having more severe somatic symptoms for women but this result was non-significant for men. Thus, with the exception of 3+ACEs, gender did not act as an effect modifier in the association between interpersonal trauma and somatic symptom severity.

In model 2 of each panel, we add the intervening variables proposed to comprise the mental health sequelae of interpersonal trauma. These proposed intervening variables were added in aggregate. Concordant with our hypothesis, we found that including the aggregated mental health sequelae of interpersonal trauma variables attenuated the association between trauma and somatic symptoms severity for both genders. This result suggests, but does not prove, that these variables may be correctly positioned as intervening variables in an exploratory pathway between interpersonal trauma exposure and somatic symptom severity, which was then tested in the structural equation models below.

Figure I shows the results of our final structural equation model with standardized path coefficients from both the full and reduced models, p-values and model fit statistics. The model provides good fit to the data, with CFI=0.985 and RMSEA=0.029. The model shows significant, positive paths from the latent interpersonal trauma variable to all three proposed intervening variables (PTSD, depression, substance use). However, of the three proposed intervening variables, only depression was significantly associated with somatic symptom severity. The direct path from interpersonal trauma to somatic symptom severity was also significant, indicating that the association between trauma and symptom severity that cannot be fully explained by the proposed intervening variables.

Fitting a two-group SEM by gender showed that the path coefficients significantly differed for males and females ($p=0.003$). A series of analyses examining each pathway in the model individually showed that associations between trauma and depression ($p<0.001$), trauma and PTSD ($p<0.001$), and trauma and substance abuse ($p=0.002$) are significantly stronger in males compared to females, while associations from depression to somatic symptom severity ($p=0.536$), PTSD to somatic symptom severity ($p=0.460$), substance use ($p=0.394$) to somatic symptom severity, and the direct path between trauma exposure and somatic symptom severity ($p=0.823$), did not significantly differ between males and females.

Figures II and III show the results of both the full and reduced two-group models, with path coefficients, p-values and model fit statistics provided, by gender. Our final parsimonious two-group model provided good fit to the data (CFI=0.988, RMSEA=0.028), and showed significant paths from the latent trauma variable to all three intervening variables for both males and females. However, males (Figure II) showed significant pathways from interpersonal trauma to somatic symptom severity through both depression and substance abuse, and after accounting for these indirect paths there was no direct path between trauma and somatic symptoms. In contrast, females (Figure III) showed a significant pathway from interpersonal trauma to somatic symptoms only through depression, and a significant direct effect of trauma on somatic symptom severity remained after controlling for the three proposed intervening variables.

Our structural equation models describe the association between interpersonal trauma and somatic symptoms. The models show that the association between interpersonal trauma and depression, PTSD, and substance abuse are stronger in men than in women. For men, depression and substance abuse form an intervening response to interpersonal trauma, and are correctly positioned in the pathway between trauma exposure and somatic symptom severity. For women, depression is correctly positioned in this pathway, and a direct pathway is also shown. For both genders, although PTSD comprises a response to interpersonal trauma, it is not correctly positioned as an intervening variable and thus does not mediate the association between interpersonal trauma exposure and somatic symptom severity.

Discussion

Our results concur with prior reports in that women have greater and more severe somatic symptoms than men independent of trauma exposure [17]. However, our findings extend prior work by showing that, although the association between interpersonal trauma and somatic symptom severity is increased in both men and women, the strength and the function of the pathways that mediate this amplification differ by gender. With the exception of the exposure for 3+ACEs, we did not find a statistical interaction between gender and interpersonal trauma. The finding that ACEs were significantly associated with greater somatic symptoms in women warrants exploration. This may be due to the fact that childhood trauma such as physical abuse is associated with greater adverse effects, such as depression, in women versus men [59], or adult males might be more likely to receive treatment for the sequelae of childhood abuse [60].

For the remaining traumas, we found that gender did not modify the overall association between interpersonal trauma and somatic symptom severity. Gender is a confounder in the association between trauma and somatic symptom severity, reflected by the greater somatic symptom severity noted among women. However, both men and women who survive trauma experience more severe somatic symptoms compared to those who are not trauma survivors. Moreover, both the function and strength of the pathways between the variables in the model differ between men and women. Our finding that men had stronger associations between interpersonal trauma and the proposed intervening variables compared to women is particularly intriguing, unique in the literature, and warrants further exploration. It may be that the proposed intervening variables are more closely associated with trauma exposure for men compared to women, because women more readily develop these sequelae in response to other stimuli not measured in these models. Moreover, the fact that a direct pathway between trauma exposure and somatic symptoms was noted for women but not for men, suggests that there may be an unmeasured intermediary between trauma exposure and somatic symptoms, that may more accurately describe trauma sequelae for women but not for men. It should be noted although the associations between trauma and the proposed intervening variables were stronger in men, the frequencies of these mental health responses to trauma were as expected, with men showing higher levels of substance abuse and women showing a (nonsignificant) trend towards greater frequency of PTSD and depression.

Gender does appear to play a role in the amount and severity of somatic symptoms, perhaps as a function of the instruments that measure these constructs or in the biology of experiencing pain. Compared to men, women have reported greater pain to experimental pain stimuli, differ in brain activation in response to pain, and respond differently to analgesics [61, 62]. Biological mechanisms thought to underlie these differences include hormonal influences, sex differences in the endogenous opioid system, and differences in neurotransmitters including dopamine and serotonin [62]. Nonbiologic mechanisms such as sex role beliefs, strategies in coping with pain, and expectancies related to pain may also explain gender differences [63].

Given our finding that gender does not usually moderate the effect of interpersonal trauma on somatic symptom severity, our models offer a potential explanation for the observed gender disparity. Men and women both experience mental health sequelae in response to trauma. Prominent features of these sequelae are depression, substance abuse and PTSD. Depression appears to be a prominent mediator for both men and women; whereas substance abuse is a prominent mediator only for men. Thus, one might presume differences in somatic symptoms may be due partially to differences in mediator variables. In fact, data from other studies suggest that substance abuse is a more prominent feature of the mental health sequelae of trauma among men [39], and depression is more prominent among women [37, 38]. However, the associations between trauma exposures and mental health sequelae of interpersonal trauma were stronger for men compared to women. Thus, our data suggest that it is the increased prevalence of overall trauma among women in our sample compared to men – rather than stronger associations between variables in the proposed models -- that is likely to account for greater somatic symptom severity among women trauma survivors compared to men.

Although we cannot determine directionality of association in our cross-sectional sample, a large burden of epidemiologic data, including data from children, strongly suggest that the proposed pathway functions with the traumatizing event as a precipitant, and the subsequent adverse mental health outcomes (depression, PTSD and substance abuse) as following events [64]. However, it should be noted that traumatic events do not always precede the development of psychiatric diagnoses. For example, substance abuse, in particular, may occur as both a consequence of trauma as well as a precipitant of traumatizing experiences [64]. Nevertheless, our structural equation model suggests a pathway in which trauma is conceptualized as preceding the development of both PTSD and intervening variables (depression and substance abuse), which further precedes the development of more severe somatic symptoms. Future study using longitudinal data may determine whether this pathway is confirmed in other samples.

Our study offers a novel finding, that for both men and women, PTSD may not be correctly positioned in the pathway between trauma exposure and somatic symptom severity. This finding is relevant in light of prior reports showing an association between PTSD and somatic symptom severity [16, 65]. Although our data do not directly address this, we suggest that high rates of co-occurrence between PTSD and depression [66] and substance abuse [67, 68] may account for this finding. Further, our data show that there is a direct path between interpersonal trauma exposure and somatic symptom severity for women. Thus, for

women, the antecedent trauma, or other unmeasured intervening variables, rather than PTSD, may account for the previously reported effect of PTSD on somatic symptoms. Whereas PTSD is an interpersonal trauma sequela, it does not appear to function as part of an intervening trauma response leading to increased somatic symptom severity.

Our study also suggests a possible target to improve somatic symptoms in trauma survivors. Decreasing depressive symptoms for both men and women, and additionally focusing on substance abuse among men after trauma may mitigate somatization and perhaps other consequences of trauma. Studies of treatment of somatization have shown that behavioral treatment effectiveness is lower compared to other psychological conditions such as depression and that short-term behavioral treatments generally lack sustained effectiveness [69]. Limited medication trials suggest that selective serotonin reuptake inhibitors (SSRIs) may be effective in treating medically unexplained symptoms [70], although studies of fibromyalgia, which shares characteristics with somatoform disorders, have demonstrated anti-depressants, particularly tri-cyclic antidepressants, to be only moderately effective [71]. Thus, our work suggests another target for treatment of somatization should be the treatment of intervening mental health sequelae of traumatizing events, (i.e., depression and substance use disorder) among trauma survivors with high levels of somatization symptoms. Because our data also demonstrated the expected association between trauma exposure and PTSD, we suggest these treatments should also incorporate guideline-concordant treatment of PTSD. Additionally, our models showed an additional direct pathway between trauma exposure and somatic symptoms for women, suggesting that there are mediators of somatic symptom severity other than depression that are important for women, but were not fully described in this study.

Limitations

There are several important limitations to this work. These data are cross-sectional. Thus causality cannot be established and directionality of association may only be inferred. Subjects were recruited from a single institution, serving a predominantly urban and minority population seeking primary care, which may reduce the generalizability of our results. Further, our results may not translate to trauma survivors who do not suffer from chronic pain. In addition, PTSD measurement in this study used any index trauma and not the specific traumas used in this analysis. It is possible that if we were able to accurately model PTSD in response to a specifically assessed trauma, our results would potentially reveal stronger linkages to the other variables in the model. However, it should be noted that our model did demonstrate an association between PTSD and the trauma exposures as defined. Moreover, the traumas studied (ACEs, IPV and sexual trauma) are likely to contribute to vulnerability to PTSD with subsequent trauma.

Additionally, we only examined interpersonal traumas in this research. Although these traumas are most linked to somatic symptoms, it is possible that our effects may have been stronger had we included other forms of violence, as trauma exposure tends to be cumulative and associated with greater somatic symptoms [9]. Finally, although our measure captured clinically relevant somatic symptom severity, we were not able to measure other key elements of somatoform disorder, such as dissociation. Future research should examine

these pathways including additional measures such as the somatoform dissociation questionnaire [72, 73] to ensure that clinically relevant aspects of somatoform disorder are examined.

Strengths and Conclusions

There is a paucity of literature that directly examines the role of gender in the somatic presentations seen in survivors of trauma. This analysis suggests that an intervening mental health response to interpersonal trauma mediates the association between trauma and somatic symptoms, and adds to the body of literature exploring complex relationships in psychiatric manifestations found in trauma survivors [74]. This work further suggests that PTSD may not be a mediator between interpersonal trauma exposure and somatic symptom severity, but rather that depression and substance abuse from an intervening response to mediate this association. Thus, our analyses show that in both men and women somatic symptoms are elevated after interpersonal trauma. However, the frequencies of interpersonal trauma, and both the strength and content of the pathways that mediate these associations, appear to function differently by gender.

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Highlights

- We explore gender differences in somatic symptom severity among trauma survivors.
- Women have more severe somatic symptoms than men overall.
- Structural equation models showed differences in trauma-somatic symptom pathways.
- In women, depression (neither PTSD nor substance abuse) mediated the pathway.
- In men, substance abuse and depression (not PTSD) mediated this pathway.

Figure I.

Structural equation model examining the association between interpersonal trauma exposure and somatic symptom severity – overall sample.

All paths from observed to latent variables had $p < .001$. Paths represented by dotted lines are not significant. Depression is correctly positioned as an intervening variable. A direct pathway from interpersonal trauma to somatic symptom severity is shown. PTSD and substance abuse are associated with interpersonal trauma, but do not intervene between interpersonal trauma exposure and somatic symptom severity.

Model Fit Statistics: Full Model: CFI 0.987 RMSEA 0.029; Reduced Model: CFI: 0.985, RMSEA 0.029

Figure II.

Structural equation model examining the association between interpersonal trauma exposure and somatic symptom severity - Males.

All paths from observed to latent variables had $p < .001$. Paths represented by dotted lines are not significant. For males, depression and substance abuse are correctly positioned as intervening variables. PTSD is not an intervening variable.

Model Fit Statistics: Full Model: CFI 0.989, RMSEA 0.028; Reduced Model: CFI 0.988, RMSEA 0.027

Figure III.

Structural equation model examining the association between interpersonal trauma exposure and somatic symptom severity - Females.

All paths from observed to latent variables had $p < .001$. Paths represented by dotted lines are not significant.

For females, depression is correctly positioned as an intervening variable. There is also a direct pathway from interpersonal trauma exposure to somatic symptoms. PTSD is not an intervening variable.

Model Fit Statistics: Full Model: CFI 0.989, RMSEA 0.028; Reduced Model: CFI 0.988, RMSEA 0.027

Table I**Participant Demographics And Key Findings by Gender**

	Male N=247		Female N=350		p-value
	<u>Mean</u>	<u>SD</u>	<u>Mean</u>	<u>SD</u>	
Age	45.12	9.33	46.27	9.77	.15
	<u>N</u>	<u>%</u>	<u>N</u>	<u>%</u>	
Race/Ethnicity					
Black/African American	146	59%	217	62%	.02
Hispanic/Latino	28	11%	31	9%	
White	52	21%	51	15%	
Other	20	8%	50	14%	
Education Level					
Less than High School	72	29%	94	27%	.54
High School and more	175	71%	256	73%	
Marital Status					
Partnered	74	30%	92	26%	.57
Separated/Divorced	78	32%	121	35%	
Single	95	38%	137	39%	
Employment					
Employed	100	40%	136	39%	.69
Unemployed	147	60%	214	61%	
Insurance					
Yes	177	72%	254	73%	.76
No	70	28%	95	27%	
INTERPERSONAL TRAUMA EXPOSURES					
Sexual Trauma					
Yes	42	17%	170	49%	<.001
No	204	83%	177	51%	
IPV Victimization					
Yes	103	42%	198	57%	<.001
No	144	58%	152	43%	
ACE 3+					
Yes	107	43%	142	41%	.50
No	140	57%	208	59%	
Any Interpersonal Trauma Exposure					
Yes	152	62%	253	73%	<.010
No	94	38%	95	27%	
SOMATIC SYMPTOM SEVERITY					
Low	116	47%	81	23%	<.001
Medium	72	29%	122	35%	
High	59	24%	147	42%	
MENTAL HEALTH SEQUELAE RESPONSE TO OF INTERPERSONAL TRAUMA					

	Male N=247		Female N=350		p-value
	<u>Mean</u>	<u>SD</u>	<u>Mean</u>	<u>SD</u>	
Substance Abuse					
Yes	58	23%	52	15%	<.010
No	189	77%	298	85%	
Current PTSD					
Yes	43	17%	80	23%	.11
No	204	83%	270	77%	
Depression					
Yes	93	38%	156	45%	.09
No	154	62%	194	55%	

Table II
Results of multivariate proportional odds regression models predicting an increase in of one clinical class of somatic symptom severity, by interpersonal trauma type

Model 1 is a single model for each trauma type examining the interaction between gender and trauma type, showing results stratified by gender. Model 2 is a single model for each trauma type that additionally assesses the role of the proposed mediators that describe the mental health sequelae of interpersonal trauma. All models control for age, race, education and employment status.

PANEL A – Sexual Trauma, Yes vs. No	Model 1 (aOR, 95%CI)	Model 2 (aOR, 95% CI)
For Females	2.05 (1.36, 3.08)	1.64 (1.07, 2.51)
For Males	2.61 (1.38, 4.91)	1.84 (0.96, 3.55)
Sexual trauma*sex interaction p-value	.53	.76
<i>Mental Health Response to Interpersonal Trauma</i>		
Substance Abuse		1.97 (1.28, 3.02)
PTSD		1.64 (1.09, 2.48)
Depression		2.80 (2.00, 3.91)
PANEL B – IPV, Yes vs. No	Model 1 (aOR, 95%CI)	Model 2 (aOR, 95%CI)
For Females	1.90 (1.27, 2.84)	1.48 (0.97, 2.24)
For Males	1.89 (1.17, 3.07)	1.38 (0.83, 2.29)
IPV*sex interaction p-value	.99	.84
<i>Mental Health Response to Interpersonal Trauma</i>		
Substance Abuse		1.99 (1.30, 3.05)
PTSD		1.69 (1.13, 2.54)
Depression		2.68 (1.92, 3.74)
PANEL C – 3+ACEs, Yes vs. No	Model 1 (aOR, 95%CI)	Model 2 (aOR, 95%CI)
For Females	2.76 (1.81, 4.21)	2.18 (1.41, 3.38)
For Males	1.45 (0.89, 2.35)	1.10 (0.66, 1.82)
3+ACEs 3+*sex interaction p-value	.05	.04
<i>Mental Health Response to Interpersonal Trauma</i>		
Substance Abuse		2.01 (1.31, 3.08)
PTSD		1.61 (1.07, 2.42)
Depression		2.68 (1.92, 3.75)
PANEL D- Any Interpersonal Trauma Exposure, Yes vs. No.	Model 1 (aOR, 95%CI)	Model 2 (aOR, 95%CI)
For Females	2.84 (1.83, 4.43)	2.34 (1.46, 3.73)
For Males	2.04 (1.24, 3.33)	1.31 (0.77, 2.22)
Any interpersonal trauma*sex interaction p-value	.32	.10
<i>Mental Health Response to Interpersonal Trauma</i>		
Substance Abuse		1.94 (1.26, 2.98)
PTSD		1.68 (1.12, 2.53)
Depression		2.76 (1.97, 3.87)