

Abuse-Related Injury and Symptoms of Posttraumatic Stress Disorder as Mechanisms of Chronic Pain in Survivors of Intimate Partner Violence

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ABSTRACT

Objective. To examine the role of abuse-related injury and posttraumatic stress disorder (PTSD) symptom severity in mediating the effects of assaultive intimate partner violence (IPV) severity, psychological IPV severity, and child abuse severity on chronic pain severity in women survivors of IPV.

Methods. Using data collected from a community sample of 309 women survivors of IPV, structural equation modeling was used to test a theoretical model of the relationships among the key variables.

Results. The theoretical model accounted for almost 38% of the variance in chronic pain severity. PTSD symptom severity was a significant mediator of the relationships of both child abuse severity (beta = 0.13) and assaultive IPV severity (beta = 0.06) with chronic pain severity. Lifetime abuse-related injury was also a significant mediator of the relationships between both child abuse severity (beta = 0.05) and assaultive IPV severity (beta = 0.06) and chronic pain severity. Child abuse severity made the largest significant contribution to the model (beta = 0.35). Assaultive IPV severity had a significant indirect effect (beta = 0.12) on chronic pain severity while psychological IPV severity had a significant direct effect (beta = 0.20).

Conclusions. Management of chronic pain in IPV survivors requires attention to symptoms of PTSD, abuse-related injury, and lifetime experiences of violence. Ensuring that acute pain from injury is adequately treated and followed over time may reduce the extent of chronic pain in abused women. The results also support the importance of routine assessment for IPV and child abuse.

Key Words. Women; Chronic Pain; Intimate Partner Violence; Abuse-Related Injury; Posttraumatic Stress Disorder; Child Abuse

Introduction

For women, the lifetime prevalence of physical assault including forced sex and other forms of physical violence from an intimate partner is 25–30% [1]. Such assaultive intimate partner violence (IPV) is almost always accompanied by psychological coercion and degradation [1]. Globally,

experiences of assaultive violence are associated with increased likelihood of physical and mental health problems [2]. In a number of controlled studies, higher rates of both chronic pain [3,4] and disability related to chronic pain [5,6] have been documented in women who have experienced IPV than in women who have not. In clinical populations of women, a history of lifetime physical and/or sexual abuse has been associated with gastrointestinal or pelvic pain [7,8].

Pain is a subjective response to signals transmitted to the brain through the spinal cord. However,

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pain perception may be exacerbated or inhibited by affective and cognitive factors from the brain that modulate the transmission of nociceptive information at the spinal cord level [9]. Peripheral sensitization of pain pathways by persistent, inadequately treated acute pain can lead to hyperresponsiveness and dysregulation of pain inhibitory mechanisms [10]. Even minor injuries can progress to chronic pain [11]. Annually, 40–60% of abused women sustain injuries especially to the head, neck, and face [12]. Among women attending family practice clinics, a history of IPV-related injury increased the odds of generalized chronic pain [5]. Chronic pain in abused women may be partially explained by neuropathic changes related to physical injury.

Chronic pain also frequently occurs in the absence of injury, and may be understood as an outcome of a complex biopsychosocial stress response generated from physical and/or psychosocial trauma [13]. IPV involves chronic exposure to repeated traumatic events, such as battering or sexual assault, within an environment of humiliation, coercion, and control [14]. Traumatic events provoke an acute stress response [15]. Chemical mediators such as hormones, neurotransmitters, and cytokines are secreted by the sympathetic nervous system, hypothalamic-pituitary-adrenal (HPA) axis, or immune system to protect vital body functions in a process of allostasis [16]. When one mediator is increased or decreased, compensatory nonlinear changes occur among other mediators [16]. Paradoxically, these protective mediators can cause pathophysiological changes (i.e., allostatic overload) when they are chronically elevated in response to multiple, severe, or sustained stressors [16] such as in IPV. Genetics, early development, and lifestyle behaviors influence the onset and course of pathophysiological changes. One negative health consequence of allostatic overload is chronic pain [13], associated with an immune response in which proinflammatory cytokine levels are increased [17]. Shifts in cytokine levels are affected by cortisol levels which are dysregulated in chronic stress [18].

Posttraumatic stress disorder (PTSD), an anxiety disorder characterized by distressing memories or emotions about trauma, avoidance of trauma reminders, and elevated arousal [19], has been proposed to mediate the relationship between trauma exposure and health outcomes [20]. Chronic pain associated with PTSD is likely to be more intense and to result in more distress, higher levels of interference, and more disability

than chronic pain not associated with PTSD [21]. Two-thirds of battered women are estimated to experience PTSD [22]. PTSD is associated not only with specific assaults but also with living in a dangerous environment [14]. PTSD has been associated with increased HPA axis sensitivity to negative feedback and lower basal cortisol levels that lead to compromised immune response [17]. Specifically, PTSD symptoms were found to mediate the relationship between IPV and proinflammatory cytokines, offering support for the association between PTSD and an increased inflammatory response consistent with chronic pain in abused women [17]. PTSD has also been found to mediate the relationships between violence and physical health symptoms, with PTSD more strongly predicting pain-related symptoms vs nonpain-related symptoms [23].

Other research suggests that the development of PTSD symptoms in response to trauma may be linked to preexisting low cortisol levels that arise from exposure to trauma early in life [24]. Early abuse has been associated with persistent changes in the neuroendocrine stress response system and immune system dysregulation [25]. The degree and pattern of these changes may be influenced by factors such as age, type of maltreatment, and parental responsiveness [26]. However, in comparison to women with no abuse history, the risk of experiencing PTSD symptoms is 4.7 times greater for women who experienced childhood physical or sexual abuse, 4.9 times greater for those who experienced adult physical or sexual assault, and 12.4 times greater for those who were victimized both as children and adults [27]. Thus, child abuse history may be an important factor in understanding PTSD in abused women. Child maltreatment, particularly physical abuse [28] and sexual abuse [29], also has been associated with chronic pain in adult women.

In an earlier analysis, we confirmed the bivariate associations between chronic pain severity in women survivors of IPV and child abuse, IPV severity, abuse-related injury, and PTSD symptoms [30]. However, no research has been conducted to explore the multivariate relationships among these variables. Based on the foregoing review of the literature, we developed and tested a theoretical model (see Figure 1) to examine the role of abuse-related injury and PTSD symptom severity in mediating the effects of assaultive IPV severity, psychological IPV severity, and child abuse severity on chronic pain severity in women survivors of IPV.

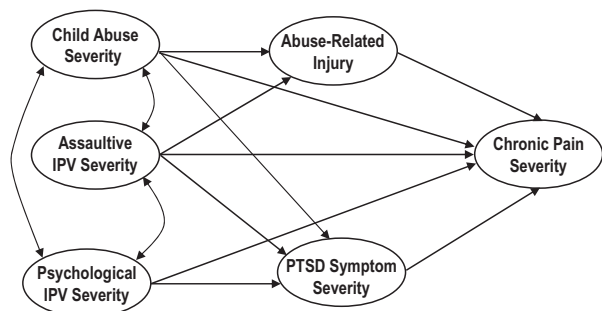


Figure 1 Theoretical model.

Methods

Baseline data from the Women's Health Effects Study, an ongoing prospective investigation of women's physical and mental health in the early years after leaving an abusive male partner [31], were used to test the theoretical model using structural equation modeling (SEM) techniques. A volunteer community sample of 309 adult women, English-speaking IPV survivors was recruited from three Canadian provinces using community advertising in local newspapers, community settings such as libraries and recreation centers, and in shelters and health and social service agencies. Inclusion criteria were having 1) separated from an abusive partner (index partner) for at least 3 months, but no more than 3 years prior to recruitment; and 2) screened positive on the modified abuse assessment screen (AAS) [32]. A "yes" response to one or more of four questions on the AAS asking about physical abuse, sexual abuse, fear, and control in the woman's relationship with the index partner was considered positive for exposure to IPV. After receiving informed consent, registered nurses conducted in-depth abuse histories and health assessments using computer-assisted data entry between June 2004 and January 2006 [31].

For the 309 women in the sample, the mean age was 39.4 (standard deviation [SD] = 9.80; range 19–63), the mean years of education was 13.4 (SD = 2.60, range 6–22), and the mean personal income in Canadian dollars was 20,391 (SD = 17,145; range 0–95,000). Approximately half the women were employed (45%, $N = 139$) and 56.3% ($N = 174$) were parenting a child under the age of 18 years. Although the majority of women were Caucasian, 16.8% ($N = 51$) self-identified as members of a visible minority group. On average, women had been living separately from their abusive partners for 20 months (SD = 10.2; range 3–40).

Measures of Latent Variables

Child abuse severity was measured by three scores from the Childhood Trauma Questionnaire (CTQ) [33], one each for emotional, physical, and sexual abuse. For the five items on each scale, participants were asked to rate the frequency of specific childhood events on a 5-point Likert scale, with higher scores indicating a greater severity of abuse. The CTQ has established reliability and validity among adult substance abusers, adolescent psychiatric inpatients, female health maintenance organization (HMO) members [33], and among women exposed to IPV [34]. In this study, Cronbach's alpha was 0.90 for emotional abuse, 0.89 for physical abuse, and 0.96 for sexual abuse.

Assaultive IPV severity was measured by the total score from the physical abuse scale of the Index of Spouse Abuse (ISA) [35] that contains 11 self-report items related to experiences of physical and sexual battering by the index partner. *Psychological IPV severity* was measured using the total score of the nonphysical abuse scale of the ISA that contains 19 items regarding experiences of psychological abuse from the index partner. The ISA has well-established reliability and validity [36], and in this study, internal consistency was 0.84 for the physical abuse scale and 0.83 for the nonphysical abuse scale. *Abuse-related injury* was measured by four questions regarding frequency of lifetime abuse-related: 1) hospitalizations; 2) visits to the emergency department; 3) broken bones; and 4) head injuries with loss of consciousness. Due to skewed distributions, extreme outliers, and a large number of zeros, scores were recoded on a scale of 1 to 4, where 1 reflects no injury and 2, 3, and 4 reflect increasing frequency of injury.

PTSD symptom severity was measured using total scores for each of three symptom clusters (intrusion, avoidance, hyperarousal) on the 17-item Davidson Trauma Scale (DTS) [37]. Women who reported a past traumatic event rated the symptoms of PTSD experienced in the past week by frequency and severity on 4-point scales and applicable item responses were summed for each cluster. Reliability and validity of the DTS has been demonstrated in a variety of trauma populations [38]. Internal consistency in this study was 0.90 for intrusion, 0.91 for avoidance, and 0.89 for hyperarousal.

Chronic pain severity was measured using three scores from the Chronic Pain Grade scale, namely, pain intensity, pain-related disability, and number of disability days in past 6 months [39].

Participants rated their current pain intensity, worst pain intensity in past 6 months, and average pain intensity in past 6 months on separate scales from 0 to 10. Using standard scoring, the pain intensity score was calculated by multiplying the mean of the three intensity items by 10 [39]. Pain disability was similarly derived from three items measuring past 6 months: pain-related interference with daily activities; change in ability to take part in recreational, social, and family activities; and change in ability to work, including housework. Disability days were measured by self-report of number of days lost in past 6 months from usual activities (work, school, or housework) because of pain. Reliability and validity of the Chronic Pain Grade scale has been well established [40]. In this study, internal consistency was 0.84 for the pain intensity scale and 0.93 for the pain disability scale.

Analysis

Analysis of the hypothesized model was carried out using SEM techniques. As the relationship between age and chronic pain is well established [41], we controlled for age by including age as a manifest variable (not in figure). Additionally, *child abuse severity*, *assaultive IPV severity*, and *psychological IPV severity* were allowed to intercorrelate freely. SEM simultaneously estimates the relationships between observed and latent variables (the measurement model) and among latent variables (theoretical model), providing estimates for both direct and indirect effects. Maximum likelihood (ML) using MPLUS[®] was used for data analysis. Assumptions of multicollinearity were met. A few variables had distributions that deviated from normal; however, assumptions of normal distribution were met by the ML model, as indicated by similarities between bootstrapped estimates of the standard errors and those obtained by the ML model [42]. Scaling for each of the latent variables was achieved by assigning a metric to one of the indicators (reference indicator). Because a listwise deletion of cases containing missing data would have reduced the sample size by about 16%, the proposed model was first estimated using a listwise deletion method and then reestimated using a full information, ML (FIML) technique. The parameter estimates from both these approaches were almost identical, both in magnitude and level of significance; therefore, the results of the FIML are used in this report in order to include the full sample.

Results

Descriptive statistics for the measures of the latent variables are in Table 1 and the correlation matrix for relationships among these measures is in Table 2. Factor loadings for all measures were statistically significant and of substantial magnitude (0.58–0.90) (see Figure 2), providing support for the measurement model. Parameter estimates were reasonable and appeared to fall in the expected range of values. The hypothesized model adequately fit the data, as indicated by acceptable goodness of fit indices: Comparative Fit Index (CFI) = 0.97, root mean square error of approximation (RMSEA) = 0.049, and Tucker–Lewis Index (TLI) = 0.95. The generally agreed upon critical values for assessing model fit are CFI \geq 0.90, TLI \geq 0.95, and RMSEA $<$ 0.05 [42]. There were no modification indices above the minimum value, supporting the decision to retain the model. The standardized parameter coefficients are in Figure 2 and Table 3.

The model accounted for 37.8% of the variance in chronic pain severity. The direct and indirect effects of child abuse severity on chronic pain severity were significant and of approximate equal magnitude ($\beta = 0.17$), and the indirect effects through PTSD symptom severity were more than twice as large as those through lifetime abuse-related injury ($\beta = 0.13$ vs $\beta = 0.05$). The direct effect of psychological IPV severity on chronic pain severity was significant ($\beta = 0.20$) while the indirect effects through PTSD symptom severity were not. There was no direct effect of assaultive IPV severity on chronic pain; however, the total indirect effects ($\beta = 0.12$) were

Table 1 Descriptive statistics of measures of latent variables (N = 309)

Measure	Mean	SD	Range	N
CTQ_emotional	14.80	9.87	5–25	304
CTQ_psychological	10.05	5.73	5–25	304
CTQ_sexual	11.88	7.20	5–25	304
ISA_physical	48.58	23.47	7.18–100	303
ISA_nonphysical	65.38	18.63	18.54–100	290
Hospitalized	0.54	1.14	0–4	306
Emergency room visits	1.08	1.43	0–4	306
Broken bones	0.43	1.02	0–4	304
Head injury	0.64	1.21	0–4	304
DTS_avoidance	17.71	13.83	0–56	285
DTS_hyperarousal	16.94	10.85	0–40	286
DTS_intrusion	13.07	10.07	0–40	285
CPG_pain disability	37.28	32.11	0–100	303
CPG_pain intensity	49.01	25.84	0–100	303
CPG_days lost	23.92	44.66	0–180	292

CPG = Chronic Pain Grade; CTQ = Childhood Trauma Questionnaire; DTS = Davidson Trauma Scale; ISA = Index of Spouse Abuse; SD = standard deviation.

Table 2 Pearson's correlations among measured variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. CTQ_emotional	1.00															
2. CTQ_physical	0.65	1.00														
3. CTQ_sexual	0.48	0.44	1.00													
4. ISA_physical	0.23	0.17	0.19	1.00												
5. ISA_nonphysical	0.25	0.20	0.20	0.57	1.00											
6. Hospitalized	0.26	0.28	0.31	0.27	0.08	1.00										
7. Emergency	0.33	0.30	0.32	0.43	0.18	0.64	1.00									
8. Broken bones	0.14	0.18	0.11	0.37	0.16	0.54	0.59	1.00								
9. Head injury	0.17	0.22	0.23	0.39	0.17	0.51	0.57	0.56	1.00							
10. DTS_avoidance	0.34	0.25	0.22	0.24	0.20	0.19	0.26	0.18	0.16	1.00						
11. DTS_hyperarousal	0.32	0.24	0.21	0.27	0.24	0.12	0.26	0.18	0.10	0.75	1.00					
12. DTS_intrusion	0.36	0.29	0.18	0.28	0.22	0.22	0.28	0.11	0.19	0.64	0.60	1.00				
13. CPG_pain disability	0.36	0.30	0.21	0.26	0.30	0.20	0.31	0.23	0.15	0.34	0.36	0.38	1.00			
14. CPG_pain intensity	0.30	0.30	0.23	0.32	0.31	0.22	0.29	0.22	0.23	0.35	0.38	0.38	0.71	1.00		
15. CPG_days lost	0.21	0.13	0.06	0.15	0.19	0.17	0.21	0.13	0.80	0.27	0.30	0.37	0.60	0.49	1.00	
16. Age	0.02	0.02	0.03	-0.20	-0.14	-0.03	-0.06	0.00	-0.10	-0.31	-0.02	0.64	0.07	-0.01	0.13	1.00

CPG = Chronic Pain Grade; CTQ = Childhood Trauma Questionnaire; DTS = Davidson Trauma Scale; ISA = Index of Spouse Abuse.

significant and equally divided between PTSD symptom severity and abuse-related injury. Thus, PTSD symptom severity has considerably larger mediating effects (beta = 0.13 child abuse; beta = 0.06 assaultive IPV) on the relationship between abuse severity and chronic pain severity than did abuse-related injury (beta = 0.05 child abuse; beta 0.06 assaultive IPV). Similarly, the significant direct effect of PTSD symptom severity on chronic pain severity was approximately twice the magnitude of that of abuse-related injury (beta = 0.33 vs beta = 0.16) when the effects of abuse severity were controlled.

The model accounted for 24.4% of the variance in PTSD symptom severity, with both child abuse severity and assaultive IPV severity having significant direct effects but that of child abuse being twice the magnitude (beta = 0.38 vs beta = 0.19). The direct effect of psychological abuse severity on PTSD symptom severity was not significant. With respect to abuse-related injury, the model accounted for 32.6% of the variance, with the significant direct effect of assaultive IPV severity (beta = 0.41) being greater than that of child abuse severity (beta = 0.31).

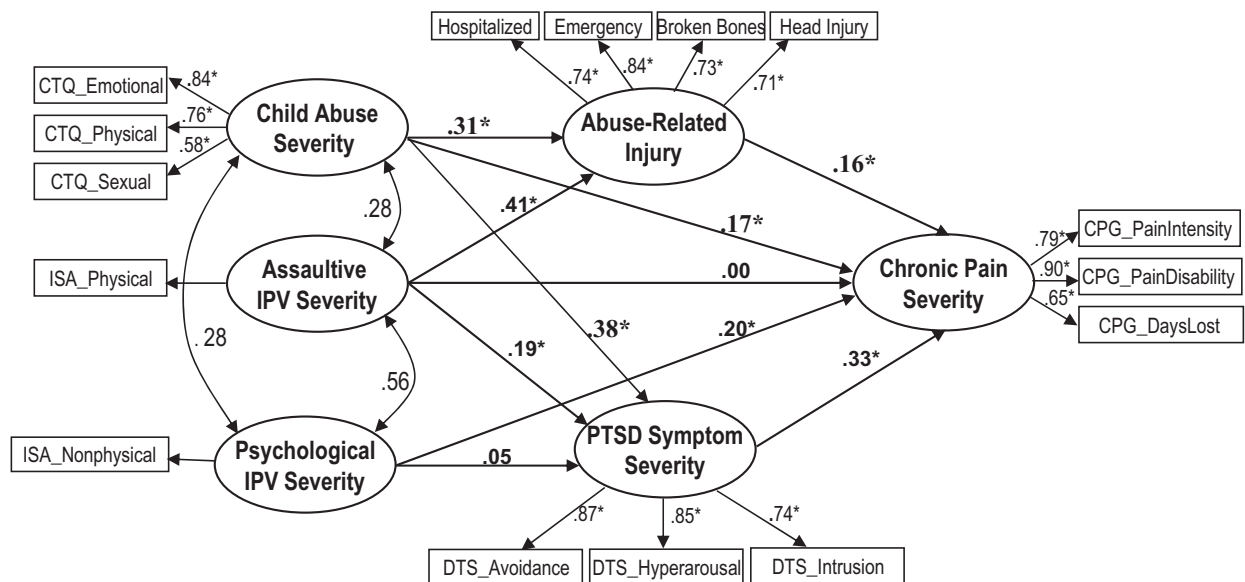


Figure 2 Structural equation model with latent variables, measures, and standardized path coefficients. * $P < 0.05$. CPG = Chronic Pain Grade; CTQ = Childhood Trauma Questionnaire; DTS = Davidson Trauma Scale; IPV = intimate partner violence; ISA = Index of Spouse Abuse; PTSD = posttraumatic stress disorder.

Table 3 Standardized coefficients for tested model

Outcome and Determinants	Causal Effects		Total
	Direct	Indirect	
PTSD symptom severity ($R^2 = 0.244$)			
Child abuse severity	0.38*		
Psychological IPV severity	0.05		
Assaultive IPV severity	0.19*		
Abuse-related injury ($R^2 = 0.326$)			
Child abuse severity	0.31*		
Assaultive IPV severity	0.41*		
Chronic pain severity ($R^2 = 0.378$)			
PTSD symptom severity	0.33*		
Abuse-related injury	0.16*		
Child abuse severity	0.17*	0.13* (PTSD) 0.05* (Injury)	0.35*
Psychological IPV severity	0.20*	0.02 (PTSD)	0.21*
Assaultive IPV severity	0.00	0.06* (PTSD) 0.06* (Injury)	0.12

* Statistically significant as t -value > 1.9 .

IPV = intimate partner violence; PTSD = posttraumatic stress disorder.

Discussion

Our findings provide the first evidence through multivariate analysis that both lifetime abuse-related injury and PTSD symptom severity are significant mediators of the effects of both assaultive IPV severity and past child abuse severity on chronic pain severity in women survivors of IPV. These findings highlight the importance of appropriate treatment and follow-up for acute abuse-related injuries in both childhood and adulthood in order to reduce the likelihood of developing chronic pain. Currently, fewer than half of women injured in IPV seek health care for the injury [43]. Limited implementation of health professional protocols for assessing IPV [44] may increase women's vulnerability for repeat assaults and contribute to missed opportunities for appropriate intervention for those who do seek help. Many abused women present to the health care system with complaints of chronic pain but without routine assessment for IPV, they may not obtain appropriate treatment that addresses current safety and their traumatic mental health response as well as treatment for their pain.

This analysis provides further support for the assertion that PTSD mediates the relationship between trauma and physical health outcome found in other studies [20]. Our findings reinforce the need for clinicians working with women with chronic pain to assess for lifetime abuse and PTSD. In the present sample, only 7.1% of women survivors of IPV reported having received

a clinical diagnosis of PTSD from a health provider, although almost 50% met the Diagnostic and Statistical Manual of Mental Disorders-IV criteria for at least mild PTSD according to the DTS screening test [45]. Given the efficacy of therapies now available to help women manage and lessen the severity of PTSD symptoms [46], our findings suggest that assessment and appropriate referrals for PTSD symptoms could be helpful in the management of chronic pain.

The findings that child abuse severity had larger total effects on chronic pain severity than either psychological IPV severity or assaultive IPV severity enhance our understanding of the effects of lifetime abuse on chronic pain. While the enduring impact of child abuse on adult health is well established [47], these results underscore its continuing effects even in the presence of more recent trauma in adulthood. This highlights the importance for clinicians working with this population to consider the need for counseling to address issues specific to child abuse in addition to other treatment modalities [48]. The identification of child abuse history provides an opportunity to affirm the woman's experience and explore the significance of past abuse in the context of her current health challenges and social environment.

With respect to significant direct effects of abuse severity on chronic pain severity, psychological IPV severity had the largest effect, followed by child abuse severity, with assaultive IPV severity having no direct effect. This finding highlights the recent acknowledgement of the linkage between psychological abuse and physical health outcomes [49] and reinforces the inseparability of mind and body. Our results show that multiple types of abuse across the life span may be linked to physical health consequences.

The finding that child abuse severity has a direct significant effect on PTSD symptom severity in women survivors of IPV offers support for the contention that women who have experienced previous trauma in childhood may be more likely to experience PTSD following IPV [22,24]. Interestingly, psychological IPV severity did not have significant direct effects on PTSD symptom severity, a finding contrary to the assertion that PTSD is associated not only with specific assaults but also with the chronic traumatization of living in a threatening environment [14]. Our finding that assaultive IPV severity had significant direct effects on PTSD symptom severity supports the notion of PTSD being linked to specific traumatic

events, especially those resulting in injury or fear of death [15]. Collectively, these findings support the viewpoint that PTSD is linked with specific traumatizing events that may be more likely to occur in assaultive IPV than psychological IPV. At the same time, it is important to acknowledge that assaultive and psychological IPV are linked and these experiences are often tied to the same events. Thus, it is also possible that these findings reflect the problem inherent in attempting to artificially separate and measure these exposures or, alternatively, failing to include salient mediators of the relationship between psychological IPV and chronic pain in the model.

While our findings add to our understanding of the effects of lifetime abuse-related injury on physical health outcomes, a future direction for our research is consideration of the effects of depression. In two multivariate studies exploring the mediating effects of recent (past 6 months) injury and mental health indicators on the relationship between recent IPV and physical health outcomes including pain, findings were mixed [50,51]. In one study, only depression and anxiety, not abuse-related injury, influenced physical health outcomes [50]. In the other, recent injury (not necessarily abuse related) had a small mediating effect between recent abuse and physical health outcomes, but stress and depression had larger mediating effects [51]. Neither study considered the role of past child abuse severity.

One strength of this analysis is that data were collected from a community sample of IPV survivors who had been living separately from their abusive partners on average of 20 months. Existing research on the health consequences of abuse on women who have left their abusive partners concentrates on women living in shelters who have been separated from their partners for a short period. Another strength is that the measure of injury was associated with lifetime abuse. Although reports of lifetime injury may be underestimated or overestimated due to poor recall, accounting for abuse-related injury in childhood as well as adulthood is important for understanding chronic pain severity in survivors of women abuse. Furthermore, the comprehensiveness of the measure for chronic pain, which includes pain intensity, pain disability, and days lost from usual activities in past 6 months, tapped into various dimensions of the pain experience.

A consistent challenge for research involving retrospective recall of child maltreatment is distortion of recall due to passage of time, intervening

events [34], or current psychological status [52,53]. People in poor health have been found to have better recall of early negative events or be more inclined to attribute ill health to early abuse than healthy people [53]. Similarly, negative mood, such as depression, may result in recall bias [53]. Recall of child abuse severity by our sample may also be affected by the women's mental and physical health. However, both retrospective recall and use of documented reports of child maltreatment may underestimate child maltreatment. In one study of the relationship between adult pain and child maltreatment, pain symptoms were no more likely in adults with documented child abuse history than those without such history, but the reverse was found in the same sample when standardized retrospective measures of child maltreatment were used; in the sample, 73% of those with documented abuse and 49% of those without documented abuse reported child maltreatment [52]. Standardized measures of child maltreatment are thought to be more accurate than broad screening questions [34]. In our study, we found that 69% of women responded positively to a screening question asking whether she had been abused as a child, whereas 81.2% experienced child abuse based on the CTQ. A major criticism of research focusing on health effects of IPV is that lifetime abuse history, particularly child abuse, is not considered. Therefore, despite possible recall bias, inclusion of child abuse severity in this model is important.

Another limitation of the research is our use of a self-report measure of PTSD rather than a clinical diagnostic work-up, but the DTS compares well with clinical diagnoses of PTSD. The analysis was limited by the measures of IPV only taking into account abuse from the woman's most recent ex-partner and not lifetime IPV. Further research is needed to understand the mediating effects of abuse-related injury and PTSD symptom severity on the relationships between other forms of violence such as lifetime IPV, sexual assault by someone other than an abusive partner, workplace violence and community violence, and chronic pain severity.

Conclusion

This study has provided an important test of a model of the relationships of child abuse and IPV with chronic pain. The significant mediating effects of both PTSD and abuse-related injury in that model demonstrate the importance of

considering both physical and mental health effects as well as lifetime experiences of violence in the health responses of women to trauma. The results also support the importance of routine assessment for IPV and child abuse in the health care system.

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