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Trauma-Related Pain, Reexperiencing Symptoms, and Treatment of Posttraumatic Stress Disorder: A Longitudinal Study of Veterans

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Research has demonstrated a strong positive association between posttraumatic stress disorder (PTSD) symptoms and physical pain. However, few studies have explored the impact of pain problems on the symptoms and treatment of PTSD, and results remain inconsistent. This longitudinal study examined whether trauma-related and trauma-unrelated pain differentially and uniquely predicted reexperiencing symptoms. We also examined whether levels of reexperiencing symptoms mediated the relationship between pain intensity and posttreatment symptoms of avoidance, numbing, and hyperarousal (ANH). Analyses were conducted using archival data from 99 treatment-seeking veterans who reported the etiology and intensity of their pain and severity of PTSD symptoms pre- and posttreatment. Among veterans with trauma-related pain, pain intensity (a) uniquely corresponded to greater posttreatment reexperiencing symptoms ($b = 1.09$), and (b) was indirectly predictive of ANH symptoms via the reexperiencing symptoms ($b = 1.93$). However, veterans with trauma-unrelated pain evidenced no associations between pain intensity and reexperiencing ($b = 0.04$) or ANH symptoms ($b = 0.06$). We thus found that trauma-related pain was indirectly related to poor PTSD treatment outcomes via reexperiencing symptoms. These findings offer additional insight into factors that may influence PTSD treatment outcomes for pain-suffering trauma survivors.

Posttraumatic stress disorder (PTSD) is a psychological disorder that develops in response to a traumatic event such as direct or indirect exposure to actual or threatened death, serious injury, or sexual violence (American Psychiatric Association [APA], 2013). The characteristic symptoms of PTSD include persistent reexperiencing of the traumatic event (e.g., intrusive and recurrent thoughts, traumatic nightmares, dissociative reactions), persistent avoidance of stimuli associated with the trauma, negative changes in cognitions and mood that began or worsened after the traumatic event, and persistent symptoms of increased arousal and reactivity.

Posttraumatic stress disorder is frequently comorbid with other psychopathologies, such as mood and substance use disorders (Kessler, Chiu, Demler, & Walters, 2005). Research exploring the impact of co-occurring psychopathologies on outcomes of trauma-focused therapy is abundant (e.g., Kaysen et al., 2014; van Minnen, Zoellner, Harned, & Mills, 2015). By contrast, research involving the impact of physical health comorbidities, such as physical pain, on PTSD treatment outcomes is lacking (Beck & Clapp, 2011). This is surprising given the high rate of PTSD and physical pain comorbidity (e.g., McFarlane, Atchison, Rafalowicz, & Papay, 1994; Sharp & Harvey, 2001; Shipherd et al., 2007). In fact, as much as 80.0% of Vietnam veterans presenting for PTSD treatment report the presence of comorbid pain (Beckham et al., 1997). Furthermore, the severity of PTSD symptoms has been shown to be positively associated with the intensity of physical pain in both cross-sectional (e.g., Bryant, Marosszeky, Crooks, Baguley, & Gurka, 1999) and longitudinal (e.g., Stratton et al., 2014) studies.

The nature of the relationship between physical pain and PTSD symptomatology requires further exploration, as it has implications for treatment. If one condition leads to or exacerbates the other, but not vice versa, then a sequential treatment (i.e., treating one disorder at a time) approach may be appropriate. However, in the case of mutual influence of physical pain and PTSD, sequential treatment may not be optimal. That is, the condition that is not initially treated may hinder treatment of the other condition and thus prolong suffering of an afflicted individual, raise socioeconomic costs due to reduced occupational functioning (e.g., Bolton et al., 2004; Gaskin, & Richard,

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2012), and increase health care utilization (e.g., Tuerk et al., 2013). Consequently, integrative treatment protocols for both pain and PTSD have recently been developed (Otis, Keane, Kerns, Monson, & Scioli, 2009; Plagge, Lu, Lovejoy, Karl, & Dobscha, 2013). However, the limited understanding of comorbidity between physical pain and PTSD continues to complicate conceptualization of these two health problems, and more research is needed to better understand how the two conditions interact with each other to optimize their treatment.

Sharp and Harvey (2001) were first to formally summarize possible mechanisms contributing to the mutual maintenance of PTSD and pain problems over time. Although some of these mechanisms (e.g., attentional biases, anxiety sensitivity, depression-related reductions in activity levels) received empirical considerations, other mechanisms (e.g., reasoning biases, trauma reminders, cognitive demand) remain largely unexplored, especially in longitudinal studies (Beck & Clapp, 2011). There have been two longitudinal studies conducted that examined the relationship of pain and PTSD at three timepoints over a 1-year period (Jenewein, Wittmann, Moergeli, Creutzig, & Schnyder, 2009; Stratton et al., 2014). Although initial PTSD symptom severity (Time 1) predicted pain intensity at later timepoints (Time 2 and Time 3), initial pain intensity did not consistently predict future severity of PTSD. Specifically, initial pain intensity was predictive of PTSD severity at Time 2, but not at Time 3.

It is important to note that these studies focused on overall levels of PTSD. Yet it has been demonstrated that treating all symptoms of a particular disorder as equal, by summing them into a total score, may not be optimal and may lead to a loss of useful information (Fried & Nesse, 2015). Correspondingly, some studies examined the effects of individual PTSD symptom clusters on levels of pain (although designs of these studies were cross-sectional in nature; Cyders, Burris, & Carlson, 2011; Powell et al., 2015). One study found that across reexperiencing, avoidance, and hyperarousal symptoms, only reexperiencing symptoms were significantly associated with ratings of pain (Powell et al., 2015). Conversely, another study suggested that reexperiencing symptoms were not associated with pain intensity (Cyders et al., 2011).

Reexperiencing symptoms appear to be particularly influential in the development and maintenance of PTSD. Michael (2000) found that characteristics of intrusive memories (e.g., distress caused by such memories) predicted subsequent presence or absence of PTSD, as well as PTSD severity. Additionally, Ehlers, Hackmann, and Michael (2004) theorized that reexperiencing symptoms are causally related to other PTSD symptoms. Reexperiencing symptoms lack contextual information (e.g., "this is happening in the past; this is not happening now") and thus may have a priming effect on other PTSD symptoms. For example, celebratory fireworks may trigger disturbing memories, feelings, and physiological reactions (i.e., reexperiencing symptoms) related to the traumatic event. Such negative experiences could (a) easily interfere with one's ability to concentrate, sleep, or stay calm (hyperarousal symptoms); and (b) naturally lead to avoidance of the situation (avoidance symptoms). Avoidance of (celebratory) activities may, over time, distance a person from other people and diminish his or her interest in any (triggering) activities (numbing symptoms).

In sum, studies revealed inconsistencies: (a) in the effects of pain on overall PTSD severity in longitudinal studies (Jenewein et al., 2009; Stratton et al., 2014), and (b) in the links between pain and reexperiencing symptoms in cross-sectional studies (Cyders et al., 2011; Powell et al., 2015). Such inconsistencies are concerning, as they continue to limit our understanding of how pain may exert its effect on PTSD symptomatology in general and reexperiencing symptoms in particular over time. Equally limited is our understanding of the mechanism through which pain may interfere with treatment of PTSD and the role of reexperiencing symptoms in this process.

One suggested possibility is that experiences of pain serve as reminders of the traumatic event and thus exacerbate reexperiencing symptoms (Sharp & Harvey, 2001). A recent qualitative study of torture survivors corroborates this idea in that pain was reported to be a trigger for intrusive memories of the torture (Taylor, Carswell, & Williams, 2013). However, among the nine interviewed torture survivors, only one provided such a report. That the remaining eight interviewees did not report triggering effects of pain is in line with the studies showing inconsistent effects of pain on reexperiencing symptoms or overall PTSD symptoms. Such findings suggest the existence of moderating variables. Although it is reasonable to expect that pain sensations could trigger memories of a traumatic event, it is equally conceivable that such effects of pain on reexperiencing symptoms would be evident primarily for pain that resulted from the traumatic event itself. That is, the effects of trauma-unrelated pain (e.g., arthritis) could have little or no effect on reexperiencing symptoms. To our knowledge, there are no published studies that have examined the possibility that the etiology of pain (trauma-related vs. trauma-unrelated) moderates the effects of pain on reexperiencing symptoms of PTSD.

The goal of this longitudinal study of veterans was to further examine, in a moderated mediation model, the effects of initial pain intensity (predictor) on posttreatment reexperiencing symptoms (mediator) and posttreatment avoidance, numbing, and hyperarousal (ANH) symptoms (outcome; see Asmundson, Stapleton, & Taylor, 2004, for empirical support on separating the numbing and avoidance clusters). Given the above considerations, we hypothesized that pain etiology would moderate the relationship between pain intensity and posttreatment reexperiencing symptoms. That is, we expected that pain intensity would be predictive of reexperiencing symptoms only among veterans who reported trauma-related pain, but not among those with trauma-unrelated pain. Furthermore, because reexperiencing symptoms are theorized to be causally related to the other clusters of PTSD symptomatology, we hypothesized that veterans' severity of reexperiencing symptoms would mediate the relationship between pain intensity and PTSD symptoms of ANH. In sum, we hypothesized that only trauma-related pain would lead to persistent posttreatment

Table 1
Means (and Standard Deviations) of Variables Tested in the Moderated Mediation Model and Pretreatment Severity of Self-Reported Posttraumatic Stress Disorder (PTSD) Symptoms

	Overall pretreatment PTSD symptoms		Pain intensity (predictor)		Posttreatment reexperiencing symptoms (mediator)		Posttreatment ANH symptoms (outcome)	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Total sample	60.31	12.08	5.26	2.71	13.56	5.63	32.12	12.34
Treatment								
CPT	59.88	11.53	4.97	2.68	13.68	5.34	32.43	11.94
PE	61.30	13.42	5.93	2.68	13.27	6.34	31.40	13.55
Era								
Pre-Vietnam/Vietnam	60.07	11.01	5.37	2.78	13.52	5.83	31.62	12.63
Post-Vietnam	60.05	13.59	5.10	2.61	13.61	5.41	32.83	12.16
Gender								
Male	60.72	12.00	5.34	2.62	13.77	5.54	32.44	12.50
Female	56.70	12.82	4.60	3.47	11.60	6.31	29.30	11.49
Ethnicity								
Non-Hispanic White	58.03*	11.65	4.76*	2.71	12.86	5.42	29.88*	11.55
Ethnic minority	63.68*	12.05	6.00*	2.55	14.57	5.84	35.43*	12.97

Note. ANH = avoidance, numbing, hyperarousal; CPT = cognitive processing therapy; PE = prolonged exposure; PTSD = posttraumatic stress disorder.
*$p < .05$.

reexperiencing symptoms, which in turn would be associated with greater posttreatment levels of ANH symptoms.

Method

Participants

The current study is based on archival data of 99 U.S. military veterans who both reported pain problems and engaged in either cognitive processing therapy (CPT; $n = 69$) or prolonged exposure therapy (PE; $n = 30$) in a PTSD outpatient clinic at a midwestern Veterans Affairs hospital.

Of the 99 veterans (10 of whom were women), 2 reported service during the Korean war era, 56 during the Vietnam war era, 5 during the post-Vietnam war era (1975–1990), 9 during the Persian Gulf war era (1990–1991), and 27 during years 2001 through 2014). Traumatic events were defined as directly experiencing or witnessing a life-threatening or personally violating incident that generated feelings of intense fear, helplessness, or horror, in accordance with criteria outlined in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM-IV-TR*; APA, 2000). Considering that many veterans reported experiencing multiple potentially traumatic events, index traumatic events were defined as the most distressing traumatic event reported by the veteran. Of participants, 62 veterans reported combat trauma as their index trauma, 8 reported military sexual trauma, 1 reported childhood physical assault, 4 reported childhood sexual assault, 2 reported motor vehicle accidents, and 17 reported "other" Criterion A stressors. Index trauma data were not available for five veterans. Moreover, 41 veterans reported experiencing pain resulting from injuries received during traumatic experiences (trauma-related pain) and the remaining 58 veterans indicated that their pain was not related to traumatic experiences (trauma-unrelated pain). With regard to ethnic background, 59 veterans self-identified as White, 24 as Black, 12 as Hispanic, 1 as Asian, and 3 did not report their ethnicity.

We examined the effects of the sample characteristics on the veterans' self-reported levels of (a) pretreatment PTSD symptoms, (b) average pain intensity, (c) posttreatment reexperiencing symptoms, and (d) the aggregate of posttreatment avoidance, numbing, and hyperarousal (ANH) symptoms (Table 1). Veterans did not significantly differ on any of these variables regardless of whether the treatment type was CPT or PE: (a) $t(97) = 0.53$, $p = .598$, $d = 0.11$; (b) $t(97) = 1.64$, $p = .104$, $d = 0.36$; (c) $t(97) = 0.32$, $p = .747$, $d = 0.07$; (d) $t(97) = 0.37$, $p = .713$, $d = 0.08$; era of service—pre-Vietnam/Vietnam versus post-Vietnam: (a) $t(97) = 0.25$, $p = .801$, $d = 0.05$; (b) $t(97) = 0.51$, $p = .612$, $d = 0.10$; (c) $t(97) = 0.10$, $p = .924$, $d = 0.02$; (d) $t(97) = 0.49$, $p = .625$, $d = 0.10$; or gender—men versus women: (a) $t(97) = 1.00$, $p = .322$, $d = 0.32$; (b) $t(97) = 0.82$, $p = .417$, $d = 0.24$; (c) $t(97) = 1.15$, $p = .251$, $d = 0.36$; (d) $t(97) = 0.75$, $p = .454$, $d = 0.26$. However, when compared with non-Hispanic White veterans, ethnic minority veterans reported elevated levels of pretreatment PTSD symptoms: $t(97) = 2.33$, $p = .022$, $d = 0.48$; pain intensity: $t(97) = 2.28$, $p = .025$, $d = 0.47$; and posttreatment ANH symptoms,

Table 2
Zero-Order Correlations Among Variables Tested in the Moderated Mediation

Variable	1	2	3	4	5	6
1. Pain intensity	–					
2. Pain etiology	.33**	–				
3. Reexperiencing symptoms	.22*	.24*	–			
4. ANH symptoms	.25*	.23*	.85***	–		
5. Number of sessions	−.02	−.05	.37***	.36***	–	
6. Gender	−.08	−.15	−.12	−.08	−.14	–
7. Ethnicity	.23*	.19	.15	.22*	−.11	.07

Note. ANH = avoidance, numbing, hyperarousal. The binary variables were coded as follows: pain etiology, 0 = *trauma-unrelated pain*, 1 = *trauma-related pain*; Gender, 0 = *man*, 1 = *woman*; Ethnicity, 0 = *non-Hispanic White veterans*, 1 = *ethnic minority veterans*.
*$p < .05$. **$p < .01$. ***$p < .001$.

$t(97) = 2.23$, $p = .028$, $d = 0.45$ (cf., Brewin, Andrews, & Valentine, 2000). Ethnic groups did not differ in posttreatment reexperiencing symptoms ($p = .135$). Moreover, the total number of attended sessions ($M = 11.23$, $SD = 2.00$, range = 6 to 18) was significantly correlated with posttreatment reexperiencing and ANH symptoms (Table 2).

Procedure

First, during routine clinical care, veterans attended a 2-session psychoeducation class within a PTSD clinic at a Veterans Affairs hospital. During the first session, veterans completed multiple self-report measures (including the General Information form) and learned about common emotional and behavioral responses to trauma, including a discussion of the development and maintenance of PTSD symptoms. During the second session, veterans learned about treatment options available to them in the PTSD clinic, and if they wished to engage in treatment, chose one of the options. Administration of both CPT and PE followed respective manuals (CPT: Resick, Monson, & Chard, 2014; PE: Foa, Hembree, & Rothbaum, 2007). Reports of PTSD symptoms (i.e., PCL-S scores) were collected immediately prior to therapy sessions.

Institutional review board approval for this project was obtained through the local research and development committee. Because this project involved only a review of deidentified archival data collected during routine clinical care, the informed consent requirement was waived. Clinical data on both physical pain and pre- and posttreatment PCL-S scores were available from 122 veterans. Of these, we excluded 21 veterans who reported no pain problems and 2 who completed fewer than six sessions of trauma-focused therapy. By Session 6 of PE (Foa et al., 2007) or CPT (Resick et al., 2014), participants would have started to practice skills (e.g., exposures, cognitive restructuring) that are believed to facilitate therapeutic change.

Measures

PTSD symptoms were measured using the 17-item self-report PTSD Checklist-Specific Stressor Version for *DSM-IV* (PCL-S; Weathers, Litz, Herman, Huska, & Keane, 1993). This measure has demonstrated good psychometric properties in trauma populations with a score of ≥ 50 indicating a likely PTSD diagnosis (e.g., Forbes, Creamer, & Biddle, 2001; Wilkins, Lang, & Norman, 2011). Veterans indicated on a 5-point Likert scale (from 1 = *not at all* to 5 = *extremely*) the extent to which a prior traumatic event bothered them in the past week. To create posttreatment reexperiencing symptoms scores (our mediator variable), we summed veterans' responses to the first five items of the PCL-S that was administered immediately prior to their final therapy session. These five items assess the reexperiencing symptoms, including intrusive thoughts and memories, flashbacks, nightmares, and physical reactions in relation to the traumatic event. Our outcome (i.e., dependent) variable represents the aggregate of ANH symptoms of PTSD. This outcome variable was computed by summing veterans' final-session responses to PCL-S Items 6 through 17 (we excluded the first 5 items related to reexperiencing symptoms so as not to artificially inflate the correlation between our outcome and mediator variables). Both the 5- and 12-item subscales of the PCL-S had good internal consistencies ($\alpha = .95$ for both).

All veterans also completed a General Information form, which assessed demographic information and information related to military service history and traumatic experiences (e.g., era of service, trauma type). Veterans indicated on this form how severe their pain was on average in the past week (0 = *no pain* to 10 = *worst pain imaginable*). We used this self-reported pain rating as an assessment of pain intensity, which served as our predictor variable. Veterans also indicated on this form whether their "pain problem [was] due to an injury [they] received during the traumatic experience" (*No*, *Yes*, or *No pain problem*). This variable (trauma-related pain, i.e., yes vs. no) was used as our moderator variable in subsequent analyses.

Statistical Analyses

Hypotheses were tested via a moderated mediation analysis using the PROCESS Model 7 macro for SPSS version 21 (Hayes, 2013). Using bootstrapped sampling, PROCESS constructed 10,000 samples yielding 95% confidence intervals (CIs) estimating the size of effects. Pain intensity was entered as the predictor variable, the total sum of posttreatment reexperiencing symptoms as the mediator variable, pain etiology (trauma-related vs. trauma-unrelated) as the moderator variable, and the total sum of ANH symptoms as the outcome variable.

We conducted the analyses with the following covariates. To account for ethnic differences (see the Participants section), we controlled for ethnicity in our analyses. Moreover, although male and female veterans did not significantly differ on pretreatment PTSD symptoms, $t(97) = 1.00$, $p = .321$, $d = 0.32$, considering the extensive literature on gender differences in initial levels of PTSD symptoms (e.g., Tolin & Foa, 2006) and

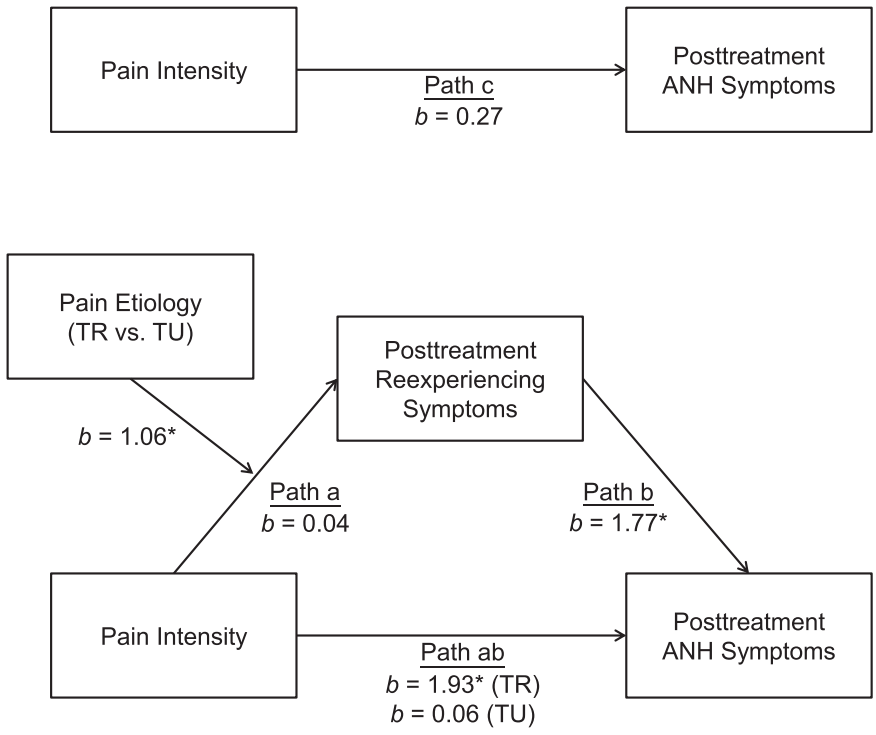

Figure 1. Moderated mediation model examining posttreatment reexperiencing symptoms as a mediator between pain intensity and posttreatment avoidance, numbing, and hyperarousal (ANH) symptoms, with the etiology of pain (trauma-related [TR] vs. trauma-unrelated [TU]) as a moderator between pain intensity and posttreatment reexperiencing symptoms. Error terms and covariates are not depicted. *$p < .05$.

the small-to-medium effect size ($d = 0.32$) in our sample, we also controlled for gender. Finally, the total number of attended trauma-focused therapy sessions was used as a third covariate because of its correlations with posttreatment reexperiencing and ANH symptoms. When indicated, the results of main analyses without covariates are presented parenthetically.

Results

Veterans' self-reports on the overall pretreatment PTSD symptoms and on the main variables tested in the moderated mediation model—average pain intensity, posttreatment reexperiencing symptoms, and posttreatment ANH symptoms—are reported in Table 1. Correlations among the main variables are reported in Table 2. All variables were normally distributed, and an insignificant amount (0.20%) of PCL-S data was missing.

Figure 1 and Table 3 present the summary of the moderated mediation analyses. First, we examined the moderating effects of pain etiology (trauma-related vs. trauma-unrelated) on the association between pain intensity and posttreatment reexperiencing symptoms; the overall model was significant, $R^2 = .28$, $F(6, 92) = 5.88$, $p < .001$; without covariates: $R^2 = .11$, $F(3, 95) = 4.03$, $p = .010$. Although the relationship between pain intensity and posttreatment reexperiencing symptoms was not significant, Path a: $b = 0.04$, 95% CI [−0.41, 0.48], $t = 0.16$, $p = .872$, the etiology of pain moderated that relationship, $b = 1.06$, 95% CI [0.13, 1.99], $t = 2.26$, $p = .026$. Crucially, as depicted in Figure 2, when pain had resulted from a traumatic event, higher pain intensity was predictive of more severe posttreatment reexperiencing symptoms, $b = 1.09$, 95% CI [0.27, 1.91], $t = 2.65$, $p = .009$. However, when pain was unrelated to a traumatic event, pain intensity was not associated with posttreatment reexperiencing symptoms, $b = 0.04$, 95% CI [−0.41, 0.48], $t = 0.16$, $p = .872$.

The overall model predicting posttreatment ANH symptoms from pain intensity and posttreatment reexperiencing symptoms was also significant, $R^2 = .75$, $F(5, 93) = 55.50$, $p < .001$; without covariates: $R^2 = .74$, $F(2, 96) = 133.89$, $p < .001$. Greater posttreatment reexperiencing symptoms were significantly associated with more severe posttreatment ANH symptoms, Path b: $b = -1.77$, 95% CI [1.51, 2.02], $t = 13.76$, $p < .001$; whereas the direct effect of pain intensity on ANH symptoms was not significant, Path c: $b = 0.27$, 95% CI [−0.23, 0.77], $t = 1.08$, $p = .285$. However—and more importantly—the indirect effect of pain intensity on posttreatment ANH symptomatology via posttreatment reexperiencing symptoms (Path ab) was significant for trauma-related pain, $b = 1.93$, standard error (SE) = 0.82, 95% CI [0.55, 3.81], but not significant for trauma-unrelated pain, $b = 0.06$, $SE = 0.41$, 95% CI [−0.76, 0.87]. Accordingly, of those whose PCL-S scores were above the clinical cutoff of 50 at the initial treatment sessions, 45.90% of veterans with trauma-related pain were below the cutoff at the end of the treatment compared to 53.50% of veterans with trauma-unrelated pain.

Table 3
Moderated Mediation Analysis With Pain Intensity as the Predictor; Posttreatment Reexperiencing Symptoms as the Mediator; Avoidance, Numbing, and Hyperarousal Symptoms as the Outcome Variable; and Pain Etiology (Trauma-Related vs. Trauma-Unrelated) as the Moderator Between Pain Intensity and Posttreatment Reexperiencing Symptoms

Outcome	Variable	*b*	*SE*	*t*	*p*	LLCI	ULCI
Posttreatment reexperiencing symptoms	Pain intensity	0.04	0.23	0.16	.872	−0.41	0.48
	Interaction	1.06	0.47	2.26	.026	0.13	1.99
	Pain intensity: TU pain	0.04	0.23	0.16	.872	−0.41	0.48
	Pain intensity: TR pain	1.09	0.41	2.65	.009	0.27	1.91
Posttreatment avoidance, numbing, and hyperarousal symptoms	Pain intensity (DE)	0.27	0.25	1.08	.285	−0.23	0.77
	Reexperiencing Symptoms	1.77	0.13	13.76	<.001	1.51	2.02
	Pain intensity: TU pain (IE)	0.06	0.42	–	–	−0.76	0.89
	Pain intensity: TR pain (IE)	1.93	0.83	–	–	0.53	3.85

Note. DE = direct effect; IE = indirect effect; Interaction = Pain Intensity × Pain Etiology; TU = trauma-unrelated; TR = trauma-related.

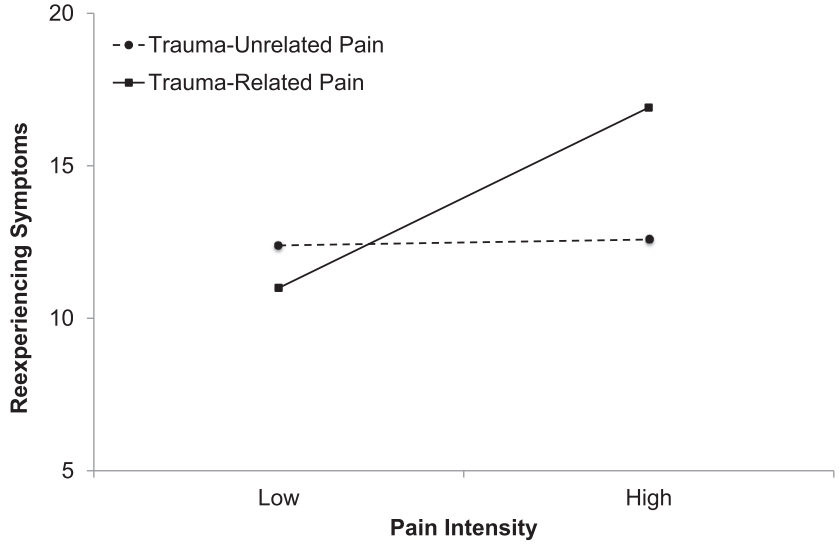

Figure 2. Simple slopes between reexperiencing symptoms and pain intensity for trauma-related and trauma-unrelated pain.

Finally, we investigated whether our findings are specific to reexperiencing symptoms of PTSD. To this end, we examined the effects of pain on the other PTSD symptom clusters by subjecting them to moderated mediation analyses. The pain etiology did not moderate the relationship between pain intensity and posttreatment avoidance symptoms, $b = 0.44$, 95% CI [−0.08, 0.95], $t = 1.69$, $p = .095$; numbing symptoms, $b = 0.76$, 95% CI [−0.04, 1.56], $t = 1.88$, $p = .063$; or hyperarousal symptoms, $b = 0.43$, 95% CI [−0.48, 1.35], $t = 0.94$, $p = .349$. These results further indicate that the differential effects of trauma-related and trauma-unrelated pain are unique to reexperiencing symptoms.

Discussion

This study examined the relationships among physical pain and symptoms of PTSD. The longitudinal design was advantageous in examining these relationships over time. In addition to findings of previous longitudinal studies (Jenewein et al., 2009; Stratton et al., 2014), the moderated mediation analysis revealed three unique aspects of the pain-PTSD relationship: the effects of pain on posttreatment reexperiencing symptoms, the indirect effects of pain on ANH symptoms, and the role of pain etiology (trauma-related vs. trauma-unrelated) in moderating these effects. Specifically, among veterans who reported trauma-related pain, the intensity of pain before treatment was associated with greater posttreatment reexperiencing symptoms. These reexperiencing symptoms mediated the effects of trauma-related pain on the other PTSD symptoms such that higher pain intensity was indirectly linked to more severe ANH symptoms. Conversely, pain that was unrelated to veterans' traumatic experiences was not associated with posttreatment reexperiencing or other symptoms of PTSD. In other words, in contrast to trauma-unrelated pain, trauma-related pain was indirectly predictive of posttreatment ANH symptoms via reexperiencing symptoms.

Trauma-related and trauma-unrelated pain did not differentially predict PTSD symptoms of avoidance, numbing, or hyperarousal. Instead, the moderating effects of pain etiology were unique to reexperiencing symptoms. These findings highlight reexperiencing symptoms as key in linking pain sensations and PTSD symptomatology over time. Thus these results are consistent with Sharp and Harvey's (2001) hypothesis that sensations of pain may serve as a reminder of traumatic events, with the caveat that only pain caused by traumatic events triggered reexperiencing symptoms in our sample. The fact that trauma-unrelated pain was not associated with severity of reexperiencing symptoms may provide an explanation for the mixed results of previous studies examining the association between pain and reexperiencing symptoms (cf. Cyders et al., 2011; Powell et al., 2015). Indeed, when considering our combined sample of veterans with both trauma-related and trauma-unrelated pain, we found no relationship between pain intensity and reexperiencing symptoms. Thus, our findings indicate that samples that include even a subset of trauma-survivors with trauma-unrelated pain may evidence weak or no relationship between pain and PTSD symptomatology.

Trauma-related pain, as assessed prior to the start of trauma-focused therapy, continued to be positively associated with greater PTSD symptoms at the conclusion of therapy. This finding allows researchers and clinicians to identify, among trauma survivors with physical pain, a subgroup that may be resistant to the treatment of PTSD. It further highlights the need for pain management in parallel or prior to treatment of PTSD and the importance of integrative treatment approaches for both PTSD and physical pain. It is conceivable that physical pain may not only maintain symptoms of PTSD over time, but also constitute a vulnerability factor for the development of PTSD after experiencing a traumatic event. For example, Sharp and Harvey (2001) noted that individuals with pain overestimate the probability of reinjury. Such a pain-related cognitive bias may be indicative of the onset of negative schemas found in PTSD. Thus, it may be critical to screen for and treat pain soon after the occurrence of trauma. Indeed, studies show that initial levels of pain predict PTSD over time (Jenewein et al., 2009; Stratton et al., 2014).

The current study is not without limitations. First, the sample consisted solely of veterans; as a result women were underrepresented and reported traumatic events were mainly combat related. Future studies should utilize more diverse samples and examine whether the effects of pain on treatment of PTSD are similar across gender and types of trauma. Second, although the results suggest detrimental effects of initial pain intensity on PTSD symptoms, it is possible that the pain-PTSD relationship is bidirectional. Examining whether initial PTSD severity interferes with the treatment of physical pain could provide additional insights into optimal treatment sequencing. Third, both the mediator and outcome variables were assessed at the same timepoint during the final treatment sessions. However, our moderated mediation model did involve a temporal association by inclusion of the predictor assessed prior to the start of treatment, and thus was superior to a fully cross-sectional design (Winer et al., 2016). Fourth, due to the archival nature of the current dataset, we were also unable to specify whether two pain groups (trauma-related vs. trauma-unrelated) differed in the type of pain reported (e.g., musculoskeletal, gastrointestinal). Similarly, it is unclear how well veterans were able to differentiate trauma-related pain from trauma-unrelated pain although in many cases (e.g., arthritis pain vs. neuropathic pain) such differentiation could be relatively straightforward. Fifth, the predictor (i.e., pain intensity) and moderator (i.e., etiology of pain) variables were operationalized using single-item questions. Although there is precedent for using single items when assessing psychopathology (Stuart et al., 2014), future studies could benefit from incorporating multi-item scales assessing physical pain. Nonetheless, the single items differentially predicted and moderated the association between pain intensity and posttreatment reexperiencing symptoms.

In summary, the results of the current study indicated that among veterans who reported trauma-related pain (vs. trauma-unrelated pain), pain intensity assessed before the treatment was indirectly predictive of greater posttreatment symptoms of avoidance, numbing, and hyperarousal through reexperiencing symptoms. These unique findings suggest how experiences of pain may hinder psychotherapy progress and provide a means for identifying pain-suffering trauma survivors who may be treatment resistant. The results also highlight the importance of integrative treatment approaches for both PTSD and pain.

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