PSYCHOLOGY, PSYCHIATRY, IMAGING & BRAIN NEUROSCIENCE SECTION

Bidirectionality of Pain Interference and PTSD Symptoms in Military Veterans: Does Injury Status Moderate Effects?

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Abstract

Objective. Pain and post-traumatic stress disorder (PTSD) symptoms are strongly correlated in veteran populations. Arguments for which one condition predicts or worsens the other condition have gone in both directions. However, research addressing this issue has been primarily limited to cross-sectional studies rather than examinations of a potential bidirectional relationship between pain interference and PTSD symptoms over time. In addition, no studies have examined deployment injury status as potentially moderating this bidirectional effect in veterans. To address these gaps in the literature, the present longitudinal study examined whether there is a bidirectional relationship between pain interference and PTSD symptoms in a sample of male and female veterans returning from Operation Iraqi Freedom, Operation Enduring Freedom, or Operation New Dawn ($N=729$) and whether deployment injury status moderates this relationship. **Methods.** Participants completed phone interviews regarding pain interference and PTSD symptoms at three time points, each three months apart. **Results.** Pain interference at Time 1 predicted worse PTSD symptoms at Time 2 for the subset of veterans who sustained injuries during deployment ($n=381$) but not for veterans with pain interference who did not sustain injuries ($n=338$). From Time 1 to Time 3, elevations in PTSD symptoms were mediated by pain interference for injured veterans; in contrast, PTSD symptoms did not appear to drive changes in pain interference in either group. **Conclusions.** These results indicate that physical symptom management should be a crucial target of psychological intervention for returning veterans with PTSD symptoms and deployment-related injuries.

Key Words: Pain Interference; Posttraumatic Stress Disorder; Injury; Veterans; Mutual Maintenance

Introduction

Recent veterans experience high rates of co-occurring pain and post-traumatic stress disorder (PTSD) symptoms. Studies have found that up to 66% of veterans with PTSD report chronic pain [1]. Individuals with comorbid chronic pain and PTSD report greater psychological distress and interference with functioning due to pain compared with individuals with chronic pain but without PTSD [2–3]. Similarly, the co-occurrence of pain and

PTSD symptoms predicts decreased life satisfaction and role functioning above and beyond the effects of PTSD symptoms alone [4]. Clearly, the comorbidity of pain and PTSD symptoms has worse implications for well-being than does either condition individually.

The prevailing model that explains the high comorbidity between pain and PTSD symptoms is the Mutual Maintenance Model, in which the properties of one condition contribute to the escalation of symptoms for the other due to underlying cognitive, affective, and

behavioral mechanisms [5]. Due to the challenges of capturing variation in these symptoms longitudinally, just four studies to date have tested this model of a bidirectional relationship in which pain and PTSD symptoms contribute to and maintain one another over time. However, the literature is inconsistent about whether PTSD symptoms have a stronger effect on pain [6–8] or vice versa [9].

Moderating Role of Injury Status

For veterans, the strength of the relationship between PTSD symptoms and pain may differ between individuals depending on deployment-related injury status. In fact, the role of injury as the underlying cause of both PTSD symptoms and pain has been well documented. Medical records of recent veterans reveal that 35.8% of chronic pain conditions are attributable to identifiable injuries sustained during military service [10]. Experiences of pain likely serve as reminders of the event in which the injury was originally inflicted. For veterans who experienced injuries during deployment, pain may be attributable to the injurious traumatic experience and thus exacerbate psychological symptoms associated with memories of this event. Research conducted with motor vehicle accident survivors has suggested that motor vehicle accident-related pain is likely to exacerbate symptoms of PTSD following the event [11]. Injury is also a major risk factor for PTSD symptom development among veterans, such that injured veterans face higher odds than do noninjured veterans [12]. However, no studies have compared groups of injured and noninjured veterans to examine how the associations between pain and PTSD symptoms may be *strengthened* for injured veterans.

Pain Interference

Studies of the bidirectional relationship between pain and PTSD symptoms have measured pain intensity alone [7–8] or a composite of the different facets of pain [6,9]; however, none have isolated the effects of pain interference. Pain interference is the degree to which an individual's pain limits or interferes with daily activities related to physical, psychological, and social functioning. Although pain interference is one facet of the multidimensional construct of pain, it is distinctively valuable in capturing the functional consequences of an individual's experience with pain.

The Present Study

Few studies have examined the bidirectional relationship between pain and PTSD symptoms by measuring pain interference specifically, and none have examined injury status as a moderator. To address these gaps in the literature, the present study examined the bidirectional relationship between pain interference and PTSD symptoms in both injured and noninjured veterans. We tested three hypotheses: 1) Pain interference and PTSD symptoms share a

bidirectional relationship, in that changes in each predict changes in the other over time for both injured and noninjured veterans. 2) Sustaining a deployment-related injury moderates the relationship between pain interference and PTSD symptoms, such that the bidirectional relationship is stronger for injured veterans than for noninjured veterans. 3) PTSD symptoms more strongly predict pain interference than pain interference predicts PTSD symptoms in the mutual maintenance of pain and PTSD symptoms for both injured and noninjured veterans.

Methods

Participants and Procedures

The sample is part of a larger study called the Survey of the Experiences of Returning Veterans study ($N = 729$), which examines postdeployment experiences of post-9/11 military veterans. Institutional review board (IRB) approval was acquired, and study procedures were followed in accordance with the IRB and the Helsinki Declaration of the World Medical Association. Veterans who served in Iraq, Afghanistan, or surrounding areas in support of Operation Iraqi Freedom, Operation Enduring Freedom, or Operation New Dawn were eligible to participate. Veterans were recruited through various outlets including Veterans Affairs (VA) resources and facilities (e.g., listservs), the Internet (e.g., Facebook), and word of mouth. Eligible participants consented and completed a 60–80-minute structured telephone survey with VA staff members at baseline, with the option to complete subsequent follow-up surveys of similar duration. The current study examined three time points: Time 1 (baseline), Time 2 (three-month follow-up), and Time 3 (six-month follow-up). To make use of all available data for the current study, we included the 719 veterans for whom we had data on deployment-related injuries and the amount of time lapsed between their end of service and the Time 1 survey.

Measures

Demographic Variables

Participants self-reported demographic variables including gender, age, race, marital status, highest level of education, household income, and combat exposure. Combat exposure was assessed using the Combat Experiences subscale of the Deployment Risk and Resilience Inventory (DRRI) [13]. The subscale consists of 17 items (e.g., “I went on combat patrols or mission”) that are rated by frequency of occurrence on a scale of 0 (never) to 5 (daily or almost daily). The subscale score was computed as the sum of the items, with higher scores indicating greater combat exposure (Cronbach's $\alpha = 0.92$).

End of Service

The number of days between the end of active service and the Time 1 survey was computed for every participant as a

covariate in the cross-lagged panel analyses. In accordance with best statistical practice for cross-lagged panel analyses [14], we controlled for this lag to account for the effects of time since injury and time since trauma exposure.

Injury from Deployment

Injury from deployment, which was used as the grouping variable, was assessed using one item from the Brief Traumatic Brain Injury Screen (BTBIS) [15], a screening tool by the Department of Veterans Affairs. The item asks “Did you have any injury(ies) during your deployment from a . . .” with the following response options: fragment, bullet, vehicular (any type including airplane), fall, explosion (improvised explosive device, rocket-propelled grenade, land mine, grenade, etc.), other. Although the BTBIS is intended for screening traumatic brain injuries, this item was intentionally designed without specific reference to head injuries to encourage wide reporting of deployment-related injuries [16]. Participants endorsed yes or no for each response option; individuals who endorsed no or at least one injury were classified as noninjured and injured, respectively.

Pain Interference

General pain interference was measured using one item (e.g., “During the past month, how much did pain interfere with your normal work including both work outside the home and housework?”) from the 12-Item Short Form Health Survey (SF-12) [17]. The item is rated on a scale of 1 (not at all) to 5 (extremely).

PTSD Symptoms

Symptoms of PTSD were assessed in relation to participants’ most stressful military experiences using the PTSD Checklist (PCL–Civilian) [18]. Because the PCL is a screening tool not intended for diagnostic purposes, the phrase “PTSD symptoms” will be used in reference to symptoms of PTSD, which do not definitively indicate diagnostic status. The PCL consists of 17 items (e.g., “Feeling jumpy or easily startled”) that correspond to the symptoms of PTSD from the Diagnostic and Statistical Manual IV, Text Revision diagnostic criteria [19]. Items are rated by frequency of occurrence in the past month on a scale from 1 (not at all) to 5 (extremely). The scale score was computed as the sum of items, with higher scores indicating more PTSD symptoms (Time 1: Cronbach’s $\alpha = 0.96$; Time 2: Cronbach’s $\alpha = 0.96$; Time 3: Cronbach’s $\alpha = 0.97$).

Analytic Plan

To characterize our sample, we calculated descriptive statistics (percentages, means), tested differences between the injured and noninjured groups on study variables (chi-square tests, t tests), and examined the distribution of our dependent variables (Kolmogorov-Smirnov test, skewness, kurtosis) using IBM SPSS 23.0 (IBM SPSS,

Inc., Chicago, IL, USA). Injury status was used as a grouping variable to create two groups: noninjured and injured veterans. Cases without responses for the injury item were removed from analyses ($n = 2$), and cases without responses for the end of service variable were removed from analyses ($n = 8$), resulting in a final sample size of 719 at baseline. To diagnose missing data, we used Little’s Missing Completely At Random (MCAR) test, which demonstrated nonsignificance, indicating that completers and noncompleters did not significantly differ in levels of PTSD symptoms and pain interference across time points. To handle missing data, we used maximum likelihood estimation, which estimates the parameters that would have most likely resulted in the observed data [20].

We confirmed that the assumptions for structural equation modeling (SEM) were met [21]. Regressions demonstrated linear relationships between pain interference and PTSD symptoms at each time point. Visual inspection of P-P plots of standardized residuals showed multivariate normality with no outliers for pain interference and PTSD symptoms at each time point. An acceptable range was demonstrated for both skewness (PTSD symptoms: 0.09 to 0.11; pain interference: 0.31 to 0.53) and kurtosis (PTSD symptoms: -0.94 to -1.09 ; pain interference: -0.76 to -0.99) [22]. To test cross-sectional and prospective associations between pain interference and PTSD symptoms for noninjured and injured veterans, we ran cross-lagged panel analyses via SEM using MPlus (version 7.3) [23]. First, we conducted invariance testing to examine whether the relationship between pain interference and PTSD symptoms was different for noninjured vs injured veterans. Then, we ran two cross-lagged panel analyses using maximum likelihood estimation [24]. The following paths were estimated: cross-lagged paths (i.e., relationships between one variable at baseline and another variable at a subsequent time point), autoregressive paths (i.e., relative stability of a variable over time), and concurrent paths (i.e., relationships between variables at baseline, correlated change at three months and six months).

One indication of model fit is the chi-square goodness of fit statistic, which assumes that nonsignificant chi-square values generally indicate a good model fit. However, the chi-square statistic assesses how well the model *exactly* fits the data and is almost always significant in larger samples [25]. Therefore, alternative fit indices that assess how well the model estimates the data were used, including the root mean square error of approximation (RMSEA), the 90% confidence interval (CI) of the RMSEA, the Comparative Fit Index (CFI), and the Tucker-Lewis Index (TLI). Models with an RMSEA ≤ 0.05 have good fit, ≤ 0.08 have adequate fit, and ≥ 0.10 have poor fit [26–27]. A confidence interval with a lower bound near 0 (and less than 0.05) and an upper bound < 0.08 demonstrates a more precise estimate of the RMSEA [14]. Models with CFI and TLI ≥ 0.90 have

Table 1. Sample descriptive statistics

Variable	Noninjured (N = 381)	Injured (N = 338)	χ^2 or t
Male gender, %	57.3	75.1	$\chi^2(1) = 25.71^{***}$
Age, M (SD), y	34.73 (8.57)	35.12 (8.93)	$t(725) = -0.60$
Race, %	—	—	
American Indian or Alaska Native	5.0	6.0	$\chi^2(1) = 0.70$
White	77.5	79.0	$\chi^2(1) = 2.58$
Black or African American	14.9	15.1	$\chi^2(1) = 0.09$
Native Hawaiian or other Pacific Islander	2.0	0.3	$\chi^2(1) = 4.81^*$
Asian	4.1	2.9	$\chi^2(1) = 0.50$
Marital status, %	—	—	$\chi^2(3) = 1.69$
Married or living with someone	50.9	54.0	
Divorced, widowed, or separated	19.0	19.6	
Never married	29.8	25.7	
Education, %	—	—	$\chi^2(3) = 3.85$
Some high school, GED, high school graduate	10.3	12.9	
Vocational, technical, Associate's degree	11.7	12.7	
Some college, college graduate (Bachelor's)	61.4	61.9	
Graduate (Master's) or advanced graduate degree (MD, PhD, DMD)	16.7	12.2	
Household income, %	—	—	$\chi^2(6) = 7.04$
<\$20,000	14.3	17.4	
\$20,000–\$35,000	21.3	23.1	
\$35,000–\$50,000	17.8	19.7	
\$50,000–\$75,000	22.5	16.1	
\$75,000–\$100,000	12.6	11.4	
>\$100,000	10.2	11.2	
Combat exposure (range [0–85])	13.49 (12.44)	25.38 (17.56)	$t(692) = -10.63^{***}$

* $p < 0.05$;*** $p < 0.001$.

acceptable fit and ≥ 0.95 have very good fit [14,26]. All parameters were freely estimated, with an alpha level < 0.05 indicating significance.

Results

Descriptive Characteristics

Demographics

Descriptive statistics for the sample are presented in Table 1. The overall sample ($N = 729$) was mostly male (66.7%), with a mean age of 34.93 years. Most participants were White (78.3%), were married or living with someone (52.5%), had attended or graduated from college (85.5%), and earned an annual income $< \$50,000$. On average, participants experienced combat between once/twice and several times during their deployment ($M = 1.15$, $SD = 0.97$). Just over half of participants endorsed injuries (52.3%), with 338 noninjured veterans and 381 injured veterans at Time 1. The noninjured and injured veteran groups differed on gender, Native Hawaiian or Pacific Islander race, and combat exposure, such that the injured veteran group had a higher percentage of males, a lower percentage of Native Hawaiians or Pacific Islanders, and greater combat exposure.

PTSD Symptoms and Pain Interference

Means, standard deviations, and ranges for PTSD symptoms and pain interference are presented in Table 2 for noninjured and injured veterans. Using a cutoff score of

≥ 36 on the PCL to indicate probable PTSD in VA primary care settings [28], 55.6% of the noninjured group and 78.7% of the injured group were found to have probable PTSD at Time 1. Both groups continued to have a substantial percentage of veterans with probable PTSD at Time 2 (noninjured: 40.2%; injured: 56.2%) and Time 3 (noninjured: 31.1%; injured: 49.1%). For pain interference, both the noninjured and injured groups reported average pain interference levels that were between a little bit and moderate for each time point. Results from t tests comparing noninjured and injured veterans on PTSD symptoms and pain interference, also presented in Table 2, indicate that injured veterans reported greater PTSD symptoms and pain interference for every time point.

Attrition

From Time 1 to Time 2, the sample size decreased from $n = 719$ to $n = 582$, resulting in a sample attrition rate of 19.1% (21.8% for injured, 16.0% for noninjured). From Time 2 to Time 3, the sample size decreased again from $n = 582$ to $n = 531$, resulting in a sample attrition rate of 8.8% (6.3% for injured, 9.5% for noninjured). From Time 1 to Time 3, the total attrition rate was 26.1% (28.0% for injured, 24.0% for noninjured). Despite attrition, rules of thumb for recommended sample size when conducting SEM were met, including at least five to 10 observations per parameter [29] and at least 10 cases per variable [30].

Table 2. Group differences for pain interference and PTSD symptoms

Variable	Range	<i>t</i>	Noninjured		Injured	
			<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
1. Pain (Time 1)	1–5	$t(717) = -8.19^{***}$	2.20	1.17	2.93	1.21
2. Pain (Time 2)	1–5	$t(578) = -6.84^{***}$	2.05	1.08	2.70	1.20
3. Pain (Time 3)	1–5	$t(529) = -6.88^{***}$	2.02	1.10	2.70	1.20
4. PTSD (Time 1)	17–85	$t(716) = -8.42^{***}$	41.40	17.75	52.42	17.27
5. PTSD (Time 2)	17–85	$t(580) = -6.93^{***}$	37.69	17.35	47.76	17.70
6. PTSD (Time 3)	17–85	$t(529) = -6.99^{***}$	36.59	17.05	47.29	18.17

PTSD = post-traumatic stress disorder symptoms.

*** $p < 0.001$.

Table 3. Model fit statistics for noninjured and injured groups

Model	χ^2	df	Model Comparison	$\Delta\chi^2$	Δdf	p	RMSEA	RMSEA 90% CI	CFI	TLI	Better Fit?
Noninjured											
1. Autoregressive	18.86	6	–	–	–	**	0.08	0.04–0.12	0.99	0.96	–
2. Pain to PTSD	14.13	3	1	4.73	3	**	0.11	0.05–0.16	0.99	0.94	N
3. PTSD to pain	4.31	3	1	14.55	3	NS	0.04	0.00–0.11	1.00	0.99	N
4. Mutual	0.00	0	1	18.86	6	***	0.00	0.00–0.00	1.00	1.00	Y
			2	14.13	3	Y					
			3	4.31	3	Y					
Injured											
1. Autoregressive	39.44	6	–	–	–	***	0.12	0.09–0.16	0.99	0.92	–
2. Pain to PTSD	17.12	3	1	22.32	3	***	0.11	0.06–0.17	0.99	0.93	Y
3. PTSD to pain	18.68	3	1	18.56	3	***	0.12	0.07–0.17	0.99	0.92	Y
4. Mutual	0.00	0	1	39.42	6	***	0.00	0.00–0.00	1.00	1.00	Y
			2	17.12	3	Y					
			3	18.68	3	Y					

CFI = Comparative Fit Index; CI = confidence interval; NS = nonsignificant; PTSD = post-traumatic stress disorder symptoms; RMSEA = root mean square error of approximation; TLI = Tucker-Lewis Index.

** $p < 0.01$; *** $p < 0.001$.

Invariance Testing

To test for invariance between the noninjured and injured groups, we first ran the base model with all paths freely estimated. When autoregressive and cross-lagged paths were added to the model and constrained to be equal across groups, there was still no significant difference in the PTSD symptoms–pain interference relationships between the noninjured and injured groups. When intercepts were added and constrained, significant differences emerged between the noninjured and injured groups. The final model, with error variances added and constrained, indicated significant differences between groups, thereby warranting separate cross-lagged panel analyses for the noninjured and injured groups.

Cross-Lagged Panel Analyses

Model fit statistics and comparisons for both the noninjured and injured groups are presented in Table 3. The following were entered as covariates at every time point due to differences between the injured and noninjured groups: race, combat exposure, and end of service. However, only end of service was a significant predictor of PTSD symptoms, and only at Time 1, such that those with a greater lag since end of service had greater PTSD symptoms at Time 1; therefore, gender,

race, and combat exposure were removed from the models. Only end of service was included as a covariate in the models. Model 4, the complete model (autoregressive paths, cross-lagged paths, and mutual causation paths), demonstrated the best fit for both the noninjured and injured groups. For both groups, Model 4 demonstrated better fit than Model 3 (autoregressive paths, cross-lagged paths of pain interference regressed on PTSD symptoms), Model 2 (autoregressive paths, cross-lagged paths of PTSD symptoms regressed on pain interference), and Model 1 (autoregressive paths only). Only significant estimates for the paths in Model 4 for noninjured and injured veterans are presented in Figures 1 and 2, respectively.

Autoregressive Paths

For both noninjured and injured veterans, all autoregressive paths for PTSD symptoms and pain interference were significant. PTSD symptoms had moderate to strong relationships between consecutive time points ($\beta = 0.62$ – 0.88 , $SE = 0.02$ – 0.06 , $p < 0.001$) and weak relationships between nonconsecutive time points ($\beta = 0.21$ – 0.28 , $SE = 0.05$ – 0.06 , $p < 0.001$). Pain interference had weak to moderate relationships between consecutive time points ($\beta = 0.44$ – 0.61 , $SE = 0.04$ – 0.06 , $p < 0.001$)

Figure 1. Complete autoregressive cross-lagged model for the noninjured group. Only significant paths are illustrated. ** $p < 0.01$; *** $p < 0.001$. PTSD = post-traumatic stress disorder symptoms.

Figure 2. Complete autoregressive cross-lagged model for the injured group. Only significant paths are illustrated. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$. PTSD = post-traumatic stress disorder symptoms.

and between nonconsecutive time points ($\beta = 0.28$ – 0.32 , $SE = 0.06$, $p < 0.001$).

Concurrent Paths

PTSD symptoms were associated with pain interference at all three time points for injured veterans (Time 1: $\beta = 0.46$, $SE = 0.04$, $p < 0.001$; Time 2: $\beta = 0.19$, $SE = 0.06$, $p < 0.01$; Time 3: $\beta = 0.21$, $SE = 0.06$, $p < 0.001$), but not for noninjured veterans. For noninjured veterans,

PTSD symptoms were associated with pain interference at Time 1 ($\beta = 0.35$, $SE = 0.05$, $p < 0.001$) and Time 3 ($\beta = 0.28$, $SE = 0.06$, $p < 0.001$) but not associated with pain interference at Time 2.

Cross-Lagged Paths

From Time 1 to Time 2, PTSD symptoms predicted pain interference for both noninjured and injured veterans. However, pain interference at Time 1 predicted PTSD

symptoms at Time 2 for injured veterans only ($\beta = 0.10$, $SE = 0.03$, $p < 0.01$), not for noninjured veterans. For both injured and noninjured veterans, the effect of PTSD symptoms at Time 1 on pain interference at Time 2 was stronger than the effect of pain interference at Time 1 on PTSD symptoms at Time 2. From Time 2 to Time 3, pain interference predicted PTSD symptoms for injured veterans only ($\beta = 0.10$, $SE = 0.04$, $p < 0.05$), not for noninjured veterans. PTSD symptoms at Time 2 did not significantly predict pain interference at Time 3 for either the noninjured or injured veterans. From Time 1 to Time 3, pain interference and PTSD symptoms did not predict each other in either direction for either noninjured or injured veterans.

Reciprocal Relationships

For the injured group only, PTSD symptoms and pain interference shared a reciprocal relationship from Time 1 to Time 2 for the injured group only. Specifically, PTSD symptoms at Time 1 predicted pain interference at Time 2 ($\beta = 0.15$, $SE = 0.05$, $p < 0.01$), and pain interference at Time 1 predicted PTSD symptoms at Time 2 ($\beta = 0.10$, $SE = 0.03$, $p < 0.01$). Furthermore, pain interference at Time 2 predicted PTSD symptoms at Time 3 for injured veterans ($\beta = 0.10$, $SE = 0.04$, $p < 0.05$), demonstrating a mediating effect of pain interference on elevations in PTSD symptoms over time. However, injured veterans demonstrated no reciprocal relationship between pain interference and PTSD symptoms from Time 2 to Time 3. For the noninjured group, no reciprocal relationships were found.

Discussion

Bidirectionality of pain interference and PTSD symptoms was found among injured veterans from Time 1 to Time 2, providing partial support for our first hypothesis. However, there was no reciprocal relationship for the injured veterans from Time 2 to Time 3, and there were no reciprocal relationships for noninjured veterans at any time point. Thus, these results provide preliminary evidence that the Mutual Maintenance Model is particularly relevant for trauma-exposed, injured veterans who experience pain. Although our results demonstrated that greater PTSD symptoms at baseline predicted increased pain interference three months later for noninjured veterans, the lack of bidirectional relationships in this group suggests that the Mutual Maintenance Model may not be applicable for veterans whose pain is not associated with deployment-related injury.

Our ability to distinguish between these two groups of veterans based on their experience of deployment injury status represents a significant contribution to the existing literature on the mutual maintenance of pain interference and PTSD symptoms. To date, all of the studies demonstrating bidirectional relationships between

pain interference and PTSD symptoms used samples in which all participants either experienced a deployment-related injury [6–8] or had PTSD [9]. By including both injured and noninjured veterans in our sample, as well as a range of PTSD symptom severity, we were able to detect a moderating effect of injury on the bidirectionality of pain interference and PTSD symptoms. More broadly, our study supported our second hypothesis and demonstrated the importance of testing moderators in theoretical models, such as the Mutual Maintenance Model, to identify the specific subsets of a population (e.g., injured veterans) for whom these theoretical pathways may be most applicable, rather than generalizing across all members of a population (e.g., all veterans).

With regard to the relative contributions of pain interference vs PTSD symptoms in maintaining these bidirectional relationships, our findings also showed that pain interference consistently predicted subsequent increases in PTSD symptoms in injured veterans. Thus, pain interference may drive the mutual maintenance between pain interference and PTSD for this subset of the sample. Importantly, PTSD symptoms also demonstrated significant effects on the mutual maintenance model; among the injured veterans, greater PTSD symptoms at baseline contributed to increased pain interference three months later, which in turn contributed to further increases in PTSD symptoms in the subsequent three months. However, there was no evidence of PTSD symptoms driving elevations in pain interference for either the injured or noninjured veterans. Specifically, greater pain interference at baseline contributed to greater PTSD symptoms in three months, which in turn contributed to further pain interference in another three months. This finding suggests that pain interference, rather than PTSD symptoms, may play a larger role in driving the Mutual Maintenance Model for veterans who sustained injury during deployment. This finding does not support our final hypothesis but is consistent with the findings from one of the few studies that examined bidirectional relationships between pain interference and PTSD symptoms [9].

Although not part of our original hypotheses, additional findings were yielded from our study. Injured and noninjured veterans in our sample had average levels of baseline PTSD symptoms that were comparable to the average levels found in veterans with and without symptoms of traumatic brain injury following blast exposure [6]. Consistent with previous studies that used an autoregressive cross-lagged design to test bidirectionality between pain interference and PTSD symptoms [6–9], the severity of pain interference and PTSD symptoms remained fairly stable between consecutive time points in our sample. This finding suggests that levels of pain interference and PTSD symptoms remain fairly stable over a span of three months. The weak associations between Time 1 and Time 3 measurements for both pain interference and PTSD symptoms may indicate regression to the

mean, which is expected in longitudinal studies [31]. Furthermore, pain interference and PTSD symptoms at Time 1 did not predict one another at Time 3, suggesting that the cross-lagged relationships unfold in the short term (three months) but not in the long term (six months).

Overall, our study provides support for pain interference as a mechanism by which PTSD symptoms are maintained for veterans who sustained injuries during their deployment. We outlined a potentially causal process of changes in psychiatric symptoms affecting changes in somatic symptoms, and vice versa, which developed over a relatively short amount of time. Our findings regarding the relationship between pain interference and PTSD symptoms build upon the body of literature on the Mutual Maintenance Model by demonstrating that the model may better fit trauma-exposed veterans who were injured during deployment and who also experience pain. For injured veterans, PTSD symptoms and pain interference may be inextricably linked if the injury causes pain interference and was incurred from the same traumatic event that gave rise to the PTSD symptoms. It is also possible that our results demonstrate that the bidirectionality of PTSD symptoms and pain interference is only evident in subgroups with high levels of both, considering that the injured veterans in our sample had greater PTSD symptoms and pain interference compared with the noninjured veterans at all time points.

Strengths and Limitations

The present study has several limitations. Although our data were longitudinal, the duration of time that elapsed between Time 1 and Time 3 was a total of six months, which is fairly short. As such, our results cannot speak to how the relationships between pain interference and PTSD symptoms change over extended periods of time. Moreover, for the injured veterans, we did not directly assess whether pain interference and PTSD symptoms were experienced in relation to the event in which the injury was sustained. We also did not have data on what types of traumas participants were referring to when completing the PCL, nor on when the injury-causing and/or traumatic events occurred, the latter of which was best estimated by the end of service date. Because PTSD symptoms were assessed using the PCL, a screening measure, a formal diagnosis of PTSD cannot be made. Finally, because the present study used secondary data, pain interference was captured by a single item from the SF-12 that was originally developed for the chronic pain population. However, our study was unable to characterize the pain experienced by veterans in our sample by other dimensions (e.g., intensity), locations (e.g., back), types (e.g., nociceptive, neuropathic), and durations (e.g., chronic, acute). Therefore, it is unclear how our results generalize to other aspects of pain (e.g., pain-related

disability) or specific pain conditions (e.g., chronic lower back pain).

Despite these limitations, our study makes several contributions to the literature about pain and PTSD symptoms. Unlike most previous studies, ours uses longitudinal data from three time points, which allows for short-term temporal conclusions and for causal inferences. Furthermore, we tested for the effects of covariates and controlled for the time lag between end of service and Time 1 as a proxy for time since injury and time since trauma exposure in order to obtain a more accurate picture of the ways in which pain interference and PTSD symptomology evolve over time, independent of one's time since deployment. Because our sample size was fairly large, our analyses were sufficiently powered to detect the small effect sizes typically observed in observational research on psychological and somatic symptoms. Additionally, our sample size allowed for group comparisons between injured and noninjured veterans, which builds upon previous work that used injury status as a covariate [6]. With respect to our measurement of pain, the use of pain interference circumvents subjective ratings of pain intensity that are difficult to interpret across individuals, provides a more functional measure of pain, and informs the association between higher PTSD symptoms and greater disability at work and home [32].

Clinical Implications

Our findings suggest that assessment of deployment injury status is crucial for veteran populations with comorbid pain and PTSD symptoms. Relationships between pain and PTSD symptoms were observed in our study; future studies addressing our limitations are needed to form more conclusive implications for clinical practice. However, treatments simultaneously targeting pain and PTSD symptoms should continue to be developed and optimized; for instance, research has demonstrated the efficacy of an integrated cognitive behavioral therapy approach for addressing the common mechanisms underlying pain and PTSD symptoms [33]. It is also important to consider that targeting pain may be an indirect way of treating PTSD symptoms among those who are hesitant about psychotherapy for PTSD, especially in the case of injured veterans. Veterans, in particular, may be amenable to a pain-focused approach, given that many feel that seeking mental health support is stigmatized in military culture [34]. Offering treatments that target pain but also reduce PTSD symptoms may provide a less stigmatized and more comprehensive approach to treatment.

Future Directions

In considering future directions for research, additional studies about the Mutual Maintenance Model between pain and PTSD symptoms could explore potential modifiers and mechanisms of the relationship. In particular, the relationship between pain and PTSD symptoms can

be examined in the context of different *types* of injuries, such as comparing bidirectionality between veterans with and without traumatic brain injuries. Beyond injury status, other variables such as personality traits may alter the strength of the bidirectional relationships between pain interference and PTSD symptoms; neuroticism is a known risk factor for both pain and PTSD symptoms [35–36]. Other emotional and affective variables may also explain the relationship between pain and PTSD symptoms. For example, anxiety sensitivity, and avoidant coping styles have been hypothesized to reinforce both pain and PTSD symptoms [37]. Understanding the mechanisms by which pain and PTSD symptoms maintain each other and identifying factors that modify their mutual maintenance are needed to more fully understand and effectively treat both conditions.

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