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Fear of Pain as a Predictor of Concurrent and Downstream PTSD Symptoms

Anna C. Barbano, B.S.[a,*], **Matthew T. Tull, Ph.D.**[a], **Nicole Christ, M.A.**[a], **Hong Xie, MD, Ph.D.**[b], **Brian Kaminski, D.O.**[c], **Xin Wang, MD, Ph.D.**[b]

[a]Department of Psychology, University of Toledo

[b]Department of Neurosciences, University of Toledo

[c]Department of Emergency Medicine, ProMedica Hospital

Abstract

Background: Pain anxiety has been associated with more severe posttraumatic stress disorder (PTSD) symptoms. However, the unique role of individual domains of pain anxiety has yet to be explored in the prediction of PTSD severity. This study examined whether specific pain anxiety domains (i.e., cognitive anxiety, escape/avoidance, fear of pain, and physiological anxiety) predict both concurrent and downstream PTSD symptoms above and beyond other PTSD risk factors.

Method: Participants were 63 survivors of traumatic events with moderate to high baseline pain treated in the emergency department and assessed for PTSD symptoms and pain anxiety at 3- and 12-months.

Results: Three-month pain anxiety domains of fear of pain and physiological anxiety (inversely related) significantly predicted concurrent 3-month PTSD symptoms above and beyond other established PTSD risk factors (i.e., sex, age, pain, and trauma type). However, only 3-month fear of pain significantly predicted 12-month PTSD symptoms.

Conclusions: Findings highlight the relevance of specific pain anxiety domains in concurrent and future PTSD symptoms and suggest the importance of evaluating pain anxiety among patients with PTSD. Interventions focused on increasing willingness to experience and tolerate fear of pain may help mitigate this risk, thereby improving outcomes for individuals with acute PTSD symptoms.

Keywords

Posttraumatic stress disorder; pain; anxiety; prediction

[*]corresponding author: anna.barbano@rockets.utoledo.edu, 419-530-4681, Department of Psychology, University of Toledo, 2801 West Bancroft St., Toledo, Ohio 43606-3390.

Declarations of Interest

None.

1. Introduction

Posttraumatic stress disorder (PTSD) is a chronic and debilitating disorder that occurs in a significant proportion of individuals exposed to a traumatic event (Liu et al., 2017). Approximately 3.9% of individuals worldwide (Koenen et al., 2017) and 6.4% of individuals in the United States (Kessler & Wang, 2008) exposed to a traumatic event develop PTSD in their lifetimes, a disorder characterized by intrusive recollections of the traumatic event, attempts to avoid reminders of the event, negative alterations in cognitions and mood, and alterations in physiological arousal (American Psychiatric Association, 2013). Despite its relatively high incidence, however, most individuals exposed to a traumatic event do not develop PTSD (Galatzer-Levy, Huang, & Bonanno, 2018). Therefore, identification of predictors of PTSD development is pertinent to the timely diagnosis and treatment of PTSD. To date, several known static risk predictors have been identified, such as sex (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Koenen et al., 2017), interpersonal traumatic exposure (Liu et al., 2017; Shalev et al., 2019), and prior exposure to traumatic events (Brewin, Andrews, & Valentine, 2000; Shalev et al., 2019). However, because modifiable predictors present viable targets for intervention, these variables are of particular interest in the prevention and treatment of PTSD.

Pain anxiety, defined as elevated negative psychological reactivity to actual or anticipated pain (Sullivan et al., 2001), has been proposed as one such modifiable predictor based on its theorized association with PTSD (Asmundson, Coons, Taylor, & Katz, 2002). Pain anxiety is considered a multidimensional construct consisting of the following components: (1) fear of pain; (2) escape/avoidance; (3) cognitive anxiety (i.e., anxious thoughts); and (4) physiological anxiety (i.e., bodily sensations associated with heightened anxiety, such as nausea or sweating; Asmundson, Norton, & Norton, 1999; McCracken, Zayfert, & Gross, 1992). Pain and PTSD are highly co-occurring (Asmundson et al., 2002; Otis, Keane, & Kerns, 2003) and together result in significant disability (Giummarra et al., 2017; Sareen et al., 2007). Compared to the 3.9% global prevalence of PTSD (Koenen et al., 2017), it is estimated that approximately 10 to 50% of patients involved in chronic pain settings also meet criteria for PTSD (Asmundson et al., 2002), demonstrating the elevated vulnerability for this disorder among individuals with pain. Some individuals who experience pain are particularly susceptible to experiencing elevated anxiety about their pain (Asmundson, Bonin, Frombach, & Norton, 2000), and, among individuals exposed to a traumatic event, pain anxiety may influence the course of PTSD. Specifically, pain anxiety putatively worsens PTSD symptoms by increasing arousal, negative affect, and avoidance behaviors (Martin, Halket, Asmundson, Flora, & Katz, 2010; Norton & Asmundson, 2004). Thus, elevated pain anxiety following traumatic exposure is expected to increase risk for the development of PTSD and contribute to more severe PTSD symptoms. However, research to date has not yet explored specific pain anxiety domains in PTSD or the extent to which these domains predict future PTSD severity.

Some researchers have conceptualized pain anxiety as an essential component of a higher order anxiety sensitivity (Greenberg & Burns, 2003), whereas others have argued its importance as a distinct construct (Carleton & Asmundson, 2009). Extant studies of psychological reactivity to pain have used several different measures, such as the Pain

Anxiety Symptoms Scale (PASS-20; McCracken & Dhingra, 2002), the Anxiety Sensitivity Index (ASI; Reiss, Peterson, Gursky, & McNally, 1986), the Distress Tolerance Scale (DTS; Rogers, Bakhshaie, Mayorga, Ditre, & Zvolensky, 2018; Simons & Gaher, 2005), and the Pain Catastrophizing Scale (PCS; Sullivan, Bishop, & Pivik, 1995), limiting the ability to compare results across studies. However, studies have found the constructs as assessed by these measures to be highly related, indicating overlap across these constructs (Hadjistavropoulos, Asmundson, & Kowalyk, 2004; Keogh & Asmundson, 2004; Kleiman, Clarke, & Katz, 2011; McCracken, Gross, Aikens, & Carnrike, 1996). Further, items on several of these measures have been found to collectively load onto one factor that is highly correlated with posttraumatic stress symptoms (Kleiman et al., 2011). Given these findings, results from studies using the PASS-20, ASI, and PCS may be commeasurable.

Previous studies of pain anxiety among individuals with PTSD have mainly examined pain or pain reactivity as the outcome variable. Nonetheless, they have consistently found that pain anxiety is higher among participants with PTSD compared to controls. For example, Tsur, Defrin, Lahav, and Solomon (2018) found that former prisoners of war exhibited significantly higher pain catastrophizing (which is consistent with the cognitive anxiety component of pain anxiety) than veterans who had not experienced captivity. In addition, participants on a chronic PTSD trajectory exhibited significantly higher pain catastrophizing than participants without PTSD. Likewise, Defrin, Schreiber, and Ginzburg (2015) found that participants with PTSD exhibited significantly higher levels of anxiety, anxiety sensitivity, and pain catastrophizing than healthy participants. Multiple studies have also shown that PTSD is associated with lower levels of distress tolerance (for a review, see Vujanovic & Zegel, 2020), which has been theorized and empirically demonstrated to be relevant to pain intensity and pain-related problems (Rogers et al., 2018). Moreover, the tolerance of pain in particular has been found to be reduced among individuals with PTSD (Vaegter, Anersen, Harvold, Andersen, & Graven-Nielsen, 2018).

Among patients with musculoskeletal pain, López-Martínez, Ramirez-Maestre, and Esteve (2014) found that all DSM-IV PTSD symptom clusters (i.e., intrusions, avoidance, numbing, and hyperarousal; American Psychiatric Association, 2004) were significantly positively correlated with pain anxiety, pain catastrophizing, and anxiety sensitivity. Paré et al. (2019) similarly found that pain catastrophizing among survivors of whiplash injury was highly and significantly correlated with posttraumatic stress symptoms. Furthermore, the authors found that elevated levels of pain catastrophizing were highly predictive of depressive or posttraumatic psychopathology. Within this sample, pain catastrophizing accounted for a significant proportion of the unique variance contributing to the prediction of downstream psychopathology.

1.1. Present Study Aims and Hypotheses

Thus, evidence consistently suggests that pain anxiety is associated with elevated PTSD risk among survivors of traumatic events. However, previous studies have primarily focused on a higher order domain of pain anxiety, pain anxiety in general, or distinct components of pain anxiety, such as pain catastrophizing. Given evidence that pain anxiety is a multidimensional construct, knowledge can be gained on the unique roles of individual domains of pain

anxiety in the prediction of PTSD severity. Pain anxiety exists across multiple domains, including anxiety cognitions (e.g., catastrophizing), avoidance, physiological arousal, and fear of pain (McCracken & Dhingra, 2002), each of which may have differential relevance as predictors of PTSD. For example, fear of pain, anxiety cognitions about pain, and pain-related physiological arousal may exacerbate trauma-related anxious arousal, placing individuals at greater risk for more frequent and severe intrusive thoughts and hyperarousal symptoms (e.g., anger, sleep difficulties). Fear of pain and anxiety cognitions about pain may also contribute to increased inactivity or isolation, increasing the potential for anhedonia symptoms of PTSD (e.g., loss of interest in pleasurable activities, detachment from others). Finally, pain avoidance may interfere with functional exposure to trauma-related cues (especially if pain is related to an experienced traumatic event), increasing risk for PTSD development. Cognitive-behavioral treatments for pain (Edhe, Dillworth, & Turner, 2014) and pain anxiety (Bailey, Carleton, Vlaeyen, & Asmundson, 2010) currently exist, as do treatments for pain in the context of PTSD (e.g., Plagge, Lu, Lovejoy, Karl, & Dobscha, 2013); however, these treatments consist of multiple components, which could interfere with their feasibility for preventing the development of pain anxiety and PTSD when time may be limited following a traumatic event. Research focused on the unique relevance of specific components of pain anxiety for PTSD symptoms may aid in the development of brief, targeted interventions for pain anxiety among individuals with recent exposure to a traumatic event.

Thus, the present study sought to examine the influence of these four distinct pain anxiety domains on concurrent and downstream PTSD symptoms within a sample of traumatic injury survivors recruited from an emergency department (ED) with moderate to high baseline pain intensity levels. Specifically, we tested whether these four pain anxiety domains at 3-months post-traumatic exposure predicted concurrent (3-month) and downstream (12-month) PTSD symptoms above and beyond other established PTSD risk factors. Given previous evidence for the role of avoidance behavior (Gil & Weinberg, 2015) and physiological reactivity (Shalev et al., 1998) in the development of PTSD, we hypothesized that avoidance and physiological arousal pain anxiety domains assessed 3-months following the experience of a traumatic event would significantly predict concurrent and downstream (12-months post-traumatic exposure) PTSD.

2. Method

2.1. Participants

Participants were from a larger sample of adults admitted to two Midwestern emergency departments (EDs) of affiliated medical centers following exposure to traumatic events. As part of a larger, ongoing longitudinal study of neurobiological and environmental correlates of PTSD development and recovery, participants were recruited within 48 hours of presentation to the ED. Participants subsequently completed diagnostic interviews, surveys, and fMRI scans over the course of 12 months. Exclusion criteria included current pregnancy, drug or alcohol intoxication at the time of the traumatic event, moderate to severe traumatic brain injury, or any other significant medical illness precluding participation in the study.

Other studies published from these data include: (1) an investigation of the role of dispositional optimism in the relation between childhood maltreatment and PTSD symptom severity (Chen et al., 2021); (2) an examination of the role of rumination in the association between PTSD symptoms and anger reactions (Christ, Contractor, Wang, & Elhai, 2020); (3) an investigation of emotion dysregulation following traumatic exposure as a predictor of 3-month PTSD symptom severity (Forbes, Tull, Rapport, Xie, Kaminski, & Wang, 2020); (4) a study of the combined role of emotional avoidance and social support following traumatic exposure in depression symptom severity one-year later (Forbes, Tull, Xie, Christ, Brickman, Mattin, & Wang, 2020); and (5) an examination of the mediating role of 2-week post-trauma acute stress in the association between post-concussion symptoms (also assessed 2-weeks post-trauma) and PTSD symptoms 3-months later, as well as the moderating role of mild traumatic brain injury in these associations (Shih et al., 2020). There was no overlap in the aims of the present study and previously published studies from these data.

The participants for this study (n = 63; 63.5% male, 36.5% female) represent a subsample of participants enrolled on the basis of the above inclusion criteria who also had complete PTSD diagnostic and pain anxiety data at the 3-month and 12-month assessment points (79.7% of individuals from the larger study sample who had completed 3-month and 12-month interviews). Sixteen participants (20.3%) from the larger study sample were excluded on the basis of missing pain anxiety data.

Participants for this study were between the ages of 18 and 59 (M = 34.57, SD = 11.21). The majority identified as either Black/African-American (52.4%) or White/European-American (31.7%), with a minority identifying as Asian-American (1.6%), Biracial (1.6%), or other (1.6%). Seven participants (11.1%) did not report race or ethnicity. Approximately half (50.8%) of the participants had experienced interpersonal traumatic events (physical and/or sexual assault), while the remainder had experienced non-interpersonal traumatic events (motor vehicle accidents [44.4%], dog attacks [3.2%], and falls [1.6%]).

2.2. Procedure

All procedures were approved by the University of Toledo Institutional Review Board. Participants who were enrolled in the study provided their subjective pain intensity rating within two weeks of ED admission. At three months post-trauma, participants completed packets of questionnaires, including the measures described below, which they either completed in their homes or in a laboratory space prior to their 3-month diagnostic interviews. At this time, participants were administered the Clinician-Administered PTSD Scale for DSM-5 (CAPS-5; Weathers et al., 2013) by trained, doctoral-level students in clinical psychology to assess for 3-month PTSD symptoms. Participants were again administered the CAPS-5 at 12 months post-trauma.

Pain anxiety data were examined at three months to compare their relationship to concurrent PTSD symptoms, which would be expected to have developed and be sustained by three months post-traumatic exposure (American Psychiatric Association, 2013). Although pain anxiety data were also collected within two weeks of the traumatic event, analyses for this study focused on 3-month pain anxiety. It was expected that pain anxiety would develop

over time following a traumatic event. Consequently, it was assumed that 3-month pain anxiety would better capture post-trauma pain anxiety (as opposed to pain anxiety soon after a traumatic event which may instead reflect pre-existing pain anxiety and not necessarily pain anxiety prompted by the experience of a traumatic event).

2.3. Measures

The Life Events Checklist for DSM-5 (LEC-5; Weathers et al., 2013) is a 17-item measure used to assess lifetime exposure to traumatic events. Participants may indicate whether particular traumatic events were directly experienced, witnessed, learned about, experienced as part of a job (e.g., firefighter, emergency medical personnel), or do not apply. In this study, participants were asked to indicate which endorsed event had been most emotionally bothersome during the past month to determine the "worst" traumatic event. In cases in which the worst traumatic event was different from the index traumatic event (i.e., the event precipitating the ED visit), each event was separately evaluated using the Clinician-Administered PTSD Scale for DSM-5 to determine PTSD symptoms that were unique to each event. In these cases, a composite score was also calculated based on the participant's most severe symptoms from both events, capturing the participant's lived experience with PTSD symptoms (as opposed to event-specific symptoms). This score was used for analyses in this study.

The Clinician-Administered PTSD Scale for DSM-5 (CAPS-5; Weathers et al., 2013) is a widely used, semi-structured diagnostic interview for PTSD. Considered the "gold standard" for assessing PTSD, the CAPS-5 yields both dichotomous PTSD diagnoses and dimensional symptom severity scores ranging from 0 to 80. The CAPS-5 has demonstrated strong psychometric properties, including good to excellent interrater reliability ($\kappa = 0.78 – 0.93$; Marmar et al., 2015; Weathers et al., 2018), good internal consistency (full scale alpha = 0.88; Weathers et al., 2018), and good test-retest reliability ($\kappa = 0.83$; Weathers et al., 2018). In this study, the CAPS-5 was administered by two clinical psychology doctoral candidates trained to reliability.

The Pain Anxiety Symptoms Scale—Short Form (PASS-20; McCracken & Dhingra, 2002) is an abbreviated, 20-item version of the original Pain Anxiety Symptoms Scale (PASS; McCracken et al., 1992). The PASS-20 is a self-report instrument used to measure heightened negative psychological reactivity to actual or anticipated pain. It contains four five-item subscales measuring lower order domains of pain anxiety: cognitive anxiety, physiological anxiety, escape/avoidance, and fear of pain (McCracken & Dhingra, 2002). This four-factor structure has been supported as superior to a one-factor higher order structure of pain anxiety in patients with pain and non-clinical samples (Abrams, Carleton, & Asmundson, 2007; Coons, Hadjistavropoulos, & Asmundson, 2004). The PASS-20 has demonstrated good internal consistency across subscales (alpha = 0.70 – 0.86) and as a whole (alpha = 0.83 – 0.91; Coons et al., 2004; McCracken & Dhingra, 2002) in studies of patients with pain and non-clinical participants. In this sample, the PASS-20 demonstrated overall excellent internal consistency (alpha = 0.95) and good consistency across subscales (alpha = 0.80 – 0.89).

2.4. Data Analysis

A power analysis determined that a sample size of 85 would be needed to detect a medium effect ($f = .15$) with power at .80 and alpha at .05 (Faul, Erdfelder, Lang, & Buchner, 2007). As such, with a sample size of 63, our study was under-powered. However, a sample size of 63 provided us with sufficient power (.81) to detect an effect of $f = .21$ with alpha at .05 (Faul et al., 2007). Data were analyzed using SPSS version 26. Pain intensity was measured on a subjective 0-10-point scale within 48 hours of ED admission. Consistent with standard procedures for assessing subjective pain in medical settings, participants were asked to respond to the following question: "On a scale of 0 to 10, where '0' means no pain at all and '10' equals pain as severe as it could possibly be, how would you rate your overall pain intensity due to your injury?" Participants' scores on this measure were used as an approximate baseline measure of subjective pain intensity. Three participants were missing subjective pain intensity ratings. For these participants, missing data were handled via mean imputation respective to traumatic exposure type, where the mean subjective pain intensity rating for interpersonal traumatic events was 6.24 and the mean subjective pain intensity rating for non-interpersonal traumatic events was 7.05. Due to the differential conditional risk of PTSD development associated with interpersonal versus non-interpersonal traumatic events (Liu et al., 2017; Shalev et al., 2019), traumatic exposure type was dichotomized as interpersonal and non-interpersonal to include traumatic events involving perpetration (e.g., assault, rape) and traumatic events not involving perpetration (e.g., motor vehicle accidents, occupational accidents), respectively. Additionally, two participants were each missing one item on the PASS-20; these missing data were also handled via within-subject mean imputation.

PASS-20 subscale items were summed to create subscale scores for each participant. Two hierarchical linear regressions were performed to assess the predictive value of PASS-20 subscales to concurrent (3-month) and downstream (12-month) PTSD symptoms, respectively, while controlling for covariates associated with increased PTSD risk, including female sex (Koenen et al., 2017; Shalev et al., 2019), older age (Kessler et al., 2017), interpersonal traumatic exposure (Liu et al., 2017; Shalev et al., 2019), and pain intensity (Norman, Stein, Dimsdale, & Hoyt, 2008). For both hierarchical linear regressions, participants' sex, age, traumatic exposure type (interpersonal vs. non-interpersonal), and subjective pain intensity rating comprised the first step of the model. The four PASS-20 subscales comprised the second step: cognitive anxiety, escape/avoidance, fear of pain, and physiological anxiety. Only overall PTSD symptom severity at 3-months and 12-months were examined as dependent variables. Due to the small sample size, an associated need to limit the number of analyses conducted, and concerns related to multicollinearity, PTSD symptom cluster data were not examined; however, zero-order associations between PASS-20 subscales and PTSD symptom clusters were calculated. As a sensitivity analysis, an additional regression was performed for the 12-months CAPS score prediction in which 3-months CAPS scores were included as a covariate in the first step of the model.

3. Results

3.1. Preliminary Analyses

At 3-months post-trauma, 31.7% (n = 20) of participants met PTSD diagnostic criteria based on their composite CAPS-5 scores. At 12-months post-trauma, 28.6% (n = 18) of participants met PTSD criteria.

A correlation matrix was formulated to assess the relationships between primary variables of interest (i.e., age, sex, traumatic event type, pain intensity, and PASS subscales). These results, as well as descriptive statistics, are presented in Table 1. All variables were normally distributed (skew = −1.05 – 1.08, all SE > 0.301; kurtosis = −1.19 – 1.36, all SE > 0.594).

3.2. Primary Analyses

To confirm previous findings that pain anxiety contributes to PTSD symptoms (e.g., López-Martínez et al., 2014; Paré et al., 2019), hierarchical linear regressions were used to examine the contribution of PASS-20 total scores to 3-month and 12-month CAPS scores above and beyond relevant covariates. PASS-20 total scores significantly predicted 3-month CAPS scores (b = 0.21, SE = 0.08, p = 0.012) and 12-month CAPS scores (b = 0.16, SE = 0.08, p = 0.047) above and beyond relevant covariates. Age, sex, trauma type, and subjective pain intensity rating did not significantly predict CAPS scores at either assessment point.

In the first hierarchical linear regression examining the contribution of concurrent 3-months pain anxiety to concurrent 3-months PTSD symptom severity, 3-month PASS-20 subscale scores together accounted for significant additional variance in 3-month PTSD symptom severity above and beyond the covariates entered in the first step (ΔR^2 = 0.53, ΔF (4, 51) = 17.16, p < .001; Table 2). Cohen's f was 1.07, indicating a large effect size. The PASS-20 subscales of physiological reactivity (b = −0.77, SE = 0.32, p = 0.020) and fear of pain (b = 1.90, SE = 0.59, p = 0.002) emerged as significant unique predictors of 3-month PTSD symptom severity.

In the second hierarchical linear regression examining the contribution of 3-month pain anxiety to downstream (12-month) PTSD symptom severity, the inclusion of 3-month PASS-20 subscale scores also explained significant additional variance above and beyond the covariates in the first step (ΔR^2 = 0.43, ΔF (4, 51) = 11.26, p < .001; Table 2). Cohen's f was 0.86, indicating a large effect size. Unlike the first regression model, only the PASS-20 subscale of fear of pain emerged as a significant predictor of 12-month PTSD symptom severity (b = 1.69, SE = 0.67, p = 0.015).[1]

Of note, little change in results was observed when using the index CAPS scores (PTSD symptoms specifically related to traumatic events that brought participants to the ED) as opposed to the composite CAPS scores. In predicting 3-month index CAPS scores, subjective pain intensity was a unique predictor (b = −1.55, SE = 0.63, p = 0.018) in addition to fear of pain (b = 1.95, SE = 0.58, p = 0.002) and physiological pain anxiety (b = −0.69, p = 0.035). In predicting 12-month index CAPS scores, fear of pain was again the only unique predictor (b = 1.61, SE = 0.32, p = 0.026).

3.3. Sensitivity Analyses

As a sensitivity analysis for the prediction of 12-month CAPS scores, a follow-up hierarchical linear regression was performed, this time including 3-month CAPS scores in the first step of the model as a covariate. With this addition, the inclusion of PASS-20 subscales in the second step of the model no longer significantly improved the model ($\Delta R^2 = 0.06$, $\Delta F(4, 50) = 2.04$, $p = 0.103$); however, PASS-20 subscales together did explain an additional 6% of the variance in 12-month PTSD symptom severity, and this change in variance was associated with a medium effect size (Cohen's $f = 0.28$). Three-month PTSD symptom severity emerged as the only significant predictor ($b = 0.73$, SE = 0.10, $p < 0.001$ in step 1) of 12-month PTSD symptom severity.

3.4. Follow-up Analyses

Follow-up analyses were also run to explore associations between pain anxiety dimensions 2-weeks post-trauma and 3-month and 12-month PTSD symptom severity. Consistent with analyses involving 3-month pain anxiety, PASS-20 subscale scores explained significant additional variance in 3-month ($\Delta R^2 = 0.15$, $\Delta F (4, 51) = 2.60$, $p < .047$, Cohen's $f = 0.34$) and 12-month ($\Delta R^2 = 0.17$, $\Delta F (4, 51) = 2.60$, $p < .029$, Cohen's $f = 0.37$) PTSD symptom severity above and beyond covariates. In both models, fear of pain (3-month PTSD symptom severity: $b = 1.15$, SE = 0.47, $p = 0.018$; 12-month PTSD symptom severity: $b = 1.15$, SE = 0.49, $p = 0.023$) and pain avoidance (3-month PTSD symptom severity: $b = -1.18$, SE = 0.57, $p = 0.045$; 12-month PTSD symptom severity: $b = -1.45$, SE = 0.59, $p = 0.018$) emerged as significant predictors. Although the associations between fear of pain and 3-month and 12-month PTSD symptom severity were in the expected direction and consistent with findings focused on 3-month pain anxiety, pain avoidance was inversely associated with PTSD symptom severity outcomes. Initial reduced avoidance behavior may increase the likelihood that individuals come into contact with trauma-related cues (especially if pain was associated with the experience of a traumatic event), increasing the severity of PTSD symptoms over time.

4. Discussion

This study aimed to examine the predictive value of four domains of pain anxiety (cognitive anxiety, physiological anxiety, escape/avoidance, and fear of pain), measured three months post-traumatic exposure, to concurrent 3-month PTSD symptoms and future 12-month PTSD symptoms. It was hypothesized that escape/avoidance and physiological domains of pain anxiety would uniquely predict concurrent and downstream PTSD symptoms due to the established roles of avoidance behavior (Gil & Weinberg, 2015) and physiological reactivity (Shalev et al., 1998) in the development and exacerbation of PTSD. Moreover, it was expected that pain anxiety would contribute unique variance to 3-month and 12-month PTSD symptoms above and beyond established PTSD risk factors, including sex, age, traumatic exposure type, and subjective pain intensity ratings at the time of traumatic exposure.

Hypotheses were counter to expectations. Although the physiological domain of pain anxiety (i.e., physiological reactivity in response to pain, such as trembling, nausea, or

dizziness) demonstrated a significant positive zero-order correlation with 3-month PTSD symptom severity, this domain had a significant unique negative association with 3-month PTSD symptom severity when relevant covariates and other pain anxiety domains were taken into account. Other domains of pain anxiety (e.g., fear of pain, cognitive anxiety about pain) likely overlap with the physiological domain of pain anxiety. Therefore, by including all other pain anxiety domains in the model, the unique aspect of the physiological pain anxiety domain that remained exhibited a negative association with 3-month PTSD symptom severity. This finding may suggest that diminished pain-related physiological anxiety (separate from the influence of all other pain anxiety domains) may be capturing the experience of more severe emotional numbing or dissociation symptoms of PTSD. Also contrary to hypotheses, fear of pain (i.e., fearful or catastrophic reactions to pain sensations) emerged as a significant unique positive predictor of 3-month PTSD symptom severity. In the prediction of 12-month PTSD symptom severity, fear of pain again emerged as a significant unique predictor. However, this association was no longer significant when controlling for 3-month PTSD symptom severity. Nonetheless, 3-month PASS-20 subscales together explained a moderate amount of variance in 12-month PTSD symptom severity. Increased subjective fear reactions to pain may increase general attention to or awareness of threat-related cues, thus motivating avoidance of trauma-related stimuli and contributing to the maintenance and worsening of PTSD symptoms. Negative affect stemming from subjective fear reactions to pain may also increase susceptibility to experience more severe PTSD symptoms (see Vujanovic et al., 2013).

Overall, findings highlight the relevance of pain anxiety to PTSD symptoms, consistent with previous literature (e.g., Defrin et al., 2015; López-Martínez et al., 2014; Paré et al., 2019). In addition, findings offer a significant contribution to this literature by demonstrating the importance of specific pain anxiety domains in PTSD symptom severity, as well as providing evidence that pain anxiety may play a potential role in the worsening of PTSD symptoms over time. However, several limitations warrant consideration. First, this study relied on a modest sample, limiting power to detect significant effects and the ability to generalize findings to the larger population of individuals with PTSD. Consequently, results should be considered preliminary and replication of findings is needed in larger samples. Larger samples may also allow a more extensive examination into whether specific pain anxiety domains contribute to the worsening of specific PTSD symptom clusters. This study also relied on self-report methods to evaluate pain intensity and pain anxiety. Though pain intensity and pain anxiety are inherently subjective and thus potentially well-suited for self-report measurement, the use of self-report measures is associated with potential limitations (e.g., social desirability). Therefore, laboratory-based procedures designed to assess pain thresholds and tolerance may be useful to incorporate into future studies examining the relationship between pain anxiety and PTSD. Additionally, pain intensity and pain anxiety were not assessed before the experience of the traumatic event. Thus, we are unable to determine to what extent these factors, including chronic pain, served as pre-traumatic versus post-traumatic exposure vulnerabilities for the development of PTSD. We also did not assess psychopathology other than PTSD, precluding the examination of the role of other psychiatric symptoms in PTSD development.

Our measures of pain intensity and pain anxiety also did not evaluate whether participants' reported pain was related to the experience of their traumatic event(s), which may have implications for understanding their relationship to PTSD symptoms. Specifically, pain related to a traumatic event may be associated with greater physiological arousal, subjective fear, and avoidance behaviors, and therefore, be more likely to contribute to the worsening of PTSD symptoms. Future studies would benefit from examining type of pain, source of pain, as well as pain-related disability and functional impairment. Moreover, data analyzed at the 3-month time point were cross-sectional in nature, restricting our ability to draw conclusions regarding the directionality of the relationship between pain anxiety and PTSD symptoms. Additionally, the majority of participants who experienced non-interpersonal traumatic events were motor vehicle accident survivors, potentially limiting the generalizability of findings to sequelae of other non-interpersonal traumatic events. Finally, we did not conduct an extensive evaluation of past traumatic exposure, as well as the potential experience of pain associated with those events; thus, it is not possible to evaluate the influence of trauma load or the experience of childhood abuse on the relationship between pain anxiety and PTSD symptoms. Future studies would benefit from exploring these variables, especially with regard to childhood abuse which has been found to increase vulnerability for chronic pain (Davis, Luecken, & Zautra, 2005) and the development of PTSD (Twaite & Rodriguez-Srednicki, 2004).

Despite limitations, results provide preliminary evidence for the concurrent and prospective role of pain anxiety in PTSD symptoms. With replication, results suggest the importance of assessing for pain and pain anxiety and incorporating such data into case conceptualizations of and treatment plans for patients with PTSD (McGeary, Moore, Vriend, Peterson, & Gatchel, 2011; Shipherd et al., 2007). Treatment programs that integrate empirically-supported interventions for PTSD and pain have been shown to be beneficial for patients with PTSD and elevated levels of pain (Otis, Keane, Kerns, Monson, & Scioli, 2009; Plagge et al., 2013); however, studies evaluating these integrated treatment programs have not specifically examined the effect of these interventions on pain anxiety. Our findings suggest that pain anxiety is associated with more severe PTSD symptoms above and beyond pain intensity. Consequently, interventions that specifically focus on reducing negative reactions to pain may be particularly beneficial for patients with PTSD and elevated levels of pain anxiety. Previous research has demonstrated the efficacy of cognitive-behavioral and acceptance-based behavioral interventions for reducing pain-related fear (e.g., Bailey et al., 2010). These interventions include interoceptive exposure to pain-related stimuli and sensations, which increase tolerance of and reduce reactivity to the experience of pain, and acceptance-based approaches that aid in modifying the ways in which individuals relate to internal experiences, including pain (e.g., acceptance and commitment therapy; see Hann and McCracken, 2014). Moreover, consistent with recommendations by Ehde et al. (2014) to identify the active components of pain interventions, our results suggest that interventions which specifically focus on the fear of pain may be most beneficial for influencing concurrent and later PTSD symptoms. For example, instead of targeting multiple components of pain anxiety through different interventions (e.g., cognitive restructuring, graded activity, coping skills, relaxation training, etc.), briefer exposure-based interventions

may be sufficient. Replication of findings and further examination of this suggestion are warranted.

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Highlights

- Examined role of pain anxiety dimensions in concurrent and later PTSD symptoms.
- Pain anxiety dimensions assessed 3-months post-traumatic exposure.
- PTSD assessed 3- and 12-months post-traumatic exposure.
- Physiological reactivity and fear of pain predicted concurrent PTSD symptoms.
- Fear of pain prospectively associated with PTSD symptoms at 12-months.

Table 1

Bivariate Correlations Between Primary Variables of Interest and Descriptive Statistics

		1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.
1.	Age	-																	
2.	Sex	0.04	-																
3.	Trauma type	−0.10	−0.24	-															
4.	Pain	0.15	0.17	−0.19	-														
5.	PASS cognitive	0.17	−0.14	0.19	−0.001	-													
6.	PASS fear of pain	0.15	−0.12	0.20	0.02	0.89***	-												
7.	PASS avoidance	0.26*	−0.18	0.12	0.02	0.48***	0.43**	-											
8.	PASS physiological	0.26*	−0.08	0.14	0.01	0.65***	0.63***	0.62***	-										
9.	3-months CAPS	0.15	−0.10	0.16	−0.17	0.66***	0.70***	0.24	0.31*	-									
10.	12-months CAPS	0.11	−0.13	0.21	−0.15	0.61***	0.65***	0.10	0.33**	0.69***	-								
11.	3-months CAPS B	0.15	−0.09	0.09	−0.17	0.56***	0.61***	0.25	0.26*	0.92***	0.60***	-							
12.	3-months CAPS C	0.18	−0.15	0.04	−0.22	0.62***	0.56***	0.27*	0.35**	0.81***	0.60***	0.84***	-						
13.	3-months CAPS D	0.11	−0.12	0.24	−0.15	0.65***	0.73***	0.23	0.31*	0.94***	0.69***	0.78***	0.65***	-					
14.	3-months CAPS E	0.15	−0.03	0.10	−0.12	0.58***	0.57***	0.16	0.23	0.92***	0.57***	0.77***	0.66***	0.80***	-				
15.	12-months CAPS B	0.09	−0.10	0.17	−0.11	0.56***	0.64***	0.19	0.36**	0.58***	0.91***	0.54***	0.47***	0.59***	0.47***	-			
16.	12-months CAPS C	0.21	−0.05	0.17	−0.15	0.50***	0.50***	0.25*	0.42***	0.58***	0.76***	0.59***	0.63***	0.50***	0.48***	0.70***	-		
17.	12-months CAPS D	0.06	−0.16	0.25	−0.12	0.57***	0.62***	0.06	0.27*	0.65***	0.95***	0.53***	0.56***	0.69***	0.52***	0.80***	0.64***	-	
18.	12-months CAPS E	0.11	−0.11	0.13	−0.16	0.54***	0.55***	−0.03	0.23	0.66***	0.92***	0.58***	0.56***	0.62***	0.60***	0.77***	0.62***	0.84***	-
	Mean	34.57	-	-	6.68	10.10	8.13	11.73	9.68	18.21	19.29	5.34	2.92	5.75	5.08	4.79	2.22	5.29	5.90
	Standard deviation	11.21	-	-	2.31	5.82	5.26	6.85	6.81	16.75	15.88	4.09	2.00	6.45	4.88	4.42	2.19	6.65	5.14

Note.

*$p < 0.05$;

**$p < 0.01$;

***$p < 0.001$;

CAPS B = re-experiencing; CAPS C = avoidance; CAPS D = negative alterations in mood and cognition; CAPS E = alterations in arousal; PASS = Pain Anxiety Symptoms Scale.

Table 2

Contribution of Pain Anxiety Symptoms Scales-20 Subscale Scores to Concurrent and Downstream PTSD Symptom Severity via Hierarchical Linear Regression

	3-month PTSD symptom severity					12-month PTSD symptom severity				
	R^2	ΔR^2	b	SE	p	R^2	ΔR^2	b	SE	p
Step 1	0.08	0.08[a]				.09	.09[c]			
Pain			−0.97	0.93	0.302			−0.86	0.96	0.375
Age			0.22	0.19	0.251			0.25	0.19	0.201
Sex			−2.26	4.37	0.607			−2.30	4.48	0.611
Traumatic Event Type			5.21	4.28	0.229			5.88	4.39	0.186
Step 2	0.61	0.53[b]				0.52	0.43[d]			
Pain			−1.23	0.64	0.059			−1.10	0.73	0.139
Age			0.10	0.13	0.453			0.17	0.15	0.275
Sex			−0.25	3.02	0.935			−1.34	3.45	0.700
Traumatic Event Type			1.28	2.99	0.672			2.05	3.42	0.552
PASS Cognitive			0.83	0.55	0.139			0.73	0.63	0.251
PASS Fear of Pain			1.90	0.59	0.002			1.69	0.67	0.015
PASS Avoidance/Escape			0.01	0.27	0.981			−0.54	0.31	0.081
PASS Physiological			−0.77	0.32	0.020			−0.23	0.37	0.540

Note. PASS = Pain Anxiety Symptoms Scale;

a = $\Delta F(4, 55) = 1.26$, $p = 0.297$, $f = 0.13$.

b = $\Delta F(4, 51) = 17.16$, $p < 0.001$, $f = 1.07$.

c = $\Delta F(4, 55) = 1.33$, $p = 0.270$, $f = 0.15$.

d = $\Delta F(4, 51) = 11.26$, $p < 0.001$, $f = 0.86$.