Posttraumatic Stress Symptoms and Chronic Pelvic Pain in Women Veterans

By
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B.A., Tufts University, 2016

Submitted to the graduate degree program in Psychology and the Graduate Faculty of the University of Kansas in partial fulfillment of the requirements for the degree of Master of Arts.

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Date Defended: 1 November 2021

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Date Approved: 18 November 2021

Abstract

Chronic Pelvic Pain (CPP), a common pain disorder, is associated with high levels of psychological distress and healthcare service utilization, as well as a complex and at times indeterminate etiology. A history of trauma and Posttraumatic Stress Disorder (PTSD) symptoms commonly co-occur with chronic pain conditions and have negative implications for pain-related outcomes. This study used a latent variable analytic approach to investigate the relationships among PTSD and depressive symptoms and their influence on pain and quality of life in a cohort of patients with CPP. The sample was drawn from records of women veterans within the Veterans Health Administration Corporate Data Warehouse. The DSM-5 model represented the best fit to the data. Hyperarousal and Negative Alterations in Mood and Cognition were significant predictors of lower Quality of Life, while Re-experiencing symptoms were a significant predictor of increased pain severity.

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Introduction

Chronic pelvic pain (CPP) is defined by the American College of Obstetricians as "pain symptoms perceived to originate from pelvic organs/structures typically lasting more than six months… often associated with negative cognitive, behavioral, sexual and emotional consequences as well as with symptoms suggestive of lower urinary tract, sexual, bowel, pelvic floor, myofascial, or gynecological dysfunction" (ACOG, 2020). A recent US survey found a 20% prevalence of CPP among women ages 30-64 (Choung et al., 2010), though this research was geographically and ethnically restricted (89% white participants). Studies have found a high rate of trauma and post-traumatic stress symptoms among patients with CPP, although the extant literature is small compared to other chronic pain conditions. Although the relationship between CPP and posttraumatic stress symptoms has been explored, there are questions that remain unexamined. In particular, the relationship between posttraumatic stress symptoms in relation to common chronic pain complications, such as depression and sleep disturbances, has yet to be explored in a CPP sample.

Chronic Pelvic Pain

CPP can be attributed to a range of diseases, or it can be considered a standalone condition without clear etiology. Cyclic pain disorders (e.g., dysmenorrhea) and pain associated with sexual intercourse (e.g., dyspareunia), while often long-lasting, are typically excluded from the definition of CPP. Factors influencing CPP in women may be gynecological in nature, such as endometriosis or adenomyosis, pelvic congestion syndrome, uterine fibroids, ovarian tumors, pelvic inflammatory disease, and post-operative or post-inflammatory adhesions. Extra-gynecological factors can include surgery, urological syndromes, gastrointestinal syndromes, ortho-neuromuscular conditions, psychosomatic influences, and neurological factors (Wozniak,

2016). Though pain may be linked to a specific condition, one study of over 3,000 women in South Korea reported normal diagnostic laparoscopy findings in 21% of the sample (Kang et al., 2007). These data indicate that the varied etiology and presentation of CPP make it difficult to generalize research from one study population to another.

Heterogeneity in diagnostic assessment and research methodology have led to inconsistent estimates of prevalence rates for CPP (Ahangari, 2014; Latthe et al., 2006; Zondervan et al., 1998). A 1996 random-digit dialing US survey found a 3-month prevalence of 14.7% (±1.0%) among women ages 18-50, with 61% of the sample reporting an unknown source of the pain (Mathias et al., 1996). Zondervan and colleagues (Zondervan et al., 1999) reported an annual prevalence of 38.3% (ages 15-73) and monthly average incidence rate of 1.58% (ages 12-70) in a UK medical record system, and, later, a 3-month community prevalence of 24% (Zondervan et al., 2001). More recently, a UK community survey found a 3-month period prevalence of 14.8%, using the same criteria in women ages 25 and older (Ayorinde et al., 2017). Further complicating research is the substantial overlap with cyclical and sexual disorders, which are typically excluded from the "chronic pain" label if they occur without additional CPP (Ayorinde et al., 2015), though self-report measures may not consistently clarify the differences. Despite variability in exact estimates, CPP is clearly a common pain syndrome that carries a high burden.

Patients with CPP perceive their health as poor, and present with increased rates of distress compared to pain-free controls. Patients with CPP have been shown to rate their mental health and physical functioning as worse than the norm of the US female population (Leserman et al., 2006) and their general health as worse than patients with dysmenorrhea alone (Zondervan et al., 2001). Patients with CPP presenting for laparoscopy were more likely to have a current or

lifetime mood disorder, phobic disorder, sexual dysfunction, or unexplained somatic symptoms, and were more likely to meet somatization disorder criteria (per DSM-III) than patients undergoing diagnostic laparoscopy for non-CPP issues (Walker et al., 1995). Not surprisingly, when compared with healthy controls, male and female participants with urologic chronic pelvic pain syndromes not only had more pain, but also had elevated levels of anxiety and depression, perceived stress, pain catastrophizing, functional symptoms, fatigue, and sleep disturbance (Schrepf et al., 2018). Distress related to pain itself is also not uncommon. Psychological disorder may represent a vulnerability to CPP or may be the consequence of pain; bidirectional pathways are likely.

Given that CPP often has an undetermined biological etiology, it is important to note that some early theories of chronic pain classified psychogenic pain as different from pain with a clear etiology. While researchers and clinicians may have become more cognizant of blatantly biased nosology, such as the early categorization of pelvic pain as 'hysteria' (Trimble et al., 2016), more subtle influences on management of female pain persist. Pain medicine has not been immune to biases that influence provider-patient relationships to the detriment of women with chronic pain, and gendered assumptions are also pervasive in the research literature (Samulowitz et al., 2018). Furthermore, early studies positing a causal role of personality or psychopathology in chronic pain used unstandardized interviews or measures with questionable validity in pain populations, and more recent methodology produced inconsistent results (Savidge & Slade, 1997). The longitudinal relationship between mental disorder and chronic pain is indeterminate. Cross-sectional research, which constitutes much of the extant literature on CPP, cannot be used to identify temporal precedence in the relationship between pain and psychological disorder.

Trauma and Chronic Pain.

Although much attention has been paid to the role of trauma exposure in chronic pain, mostly cross-sectional data have obfuscated a causal trajectory. Researchers have found that women with CPP have more trauma exposure than pain-free controls (Leserman et al., 2006; Reiter & Gambone, 1990), although other studies have noted equivalent trauma exposure (Osório et al., 2016). Childhood emotional neglect (Poli-Neto et al., 2017) and childhood sexual abuse are more frequently reported in women with CPP compared to pain-free women (Walker et al., 1995) and in women with interstitial cystitis/painful bladder syndrome (Curtis Nickel et al., 2011), though at least one study failed to find significant differences between CPP patients and healthy controls when examining reports of lifetime sexual abuse (Lampe et al., 2000). A community study in New Zealand also did not find a significant relationship between childhood sexual abuse and pelvic pain (Romans et al., 2002). In general, the literature suggests that some types of trauma may be more frequent in patients with CPP, but results are inconsistent.

Beyond questions of causal trajectory, the extent to which childhood trauma exposure influences pain severity and symptomatology in adulthood is also unclear (Chiu et al., 2017; Curtis Nickel et al., 2011). In urologic chronic pelvic pain syndrome patients, Adverse Childhood Experiences (ACE) were not predictive of illness trajectory. However, the indirect relationships between ACE score and physical health appraisal, as well as physical health appraisal and illness trajectory, were significant, with worse health appraisal linked to worsening symptoms over a period of 1 year (Schrepf et al., 2018). A longitudinal study of abused and neglected children with matched controls in the US found no prospective relationship between court-documented childhood abuse and reported pain symptoms after 20 years (Raphael et al., 2001). While extremely controversial after publication, one meta-analysis found only a weak effect of childhood abuse on psychopathology in college students (Rind et al., 1998). Null or

equivalent findings do not negate the potential deleterious consequences of childhood abuse but suggest that the relationship is not easily captured through retrospective reporting. Children with trauma and chronic stress exposure are vulnerable to higher risk of health problems in adulthood (e.g., (Felitti et al., 1998), but the relationship is non-specific to CPP and is hard to capture in the absence of intensive longitudinal data incorporating the complex pathways to illness.

In female veteran samples, research has incorporated measurement of lifetime trauma exposure but also attended to the issue of military sexual trauma (MST) in adulthood. One study of half a million female veterans' medical record data found significant associations between MST and many chronic pain conditions after accounting for age, body mass index, smoking status, ethnicity, and mental health differences in PTSD, anxiety and depression (Cichowski et al., 2017). Another trial at an outpatient clinic found a strong association between sexual trauma, sexual harassment, and pain, with abuse and harassment also linked to psychological and physical comorbidity (Haskell et al., 2008). While assessing trauma exposure is integral to understanding pain, the direct causal relationship posited by some early researchers is perhaps reductive, and additional contextual information is required when incorporating models that analyze the trauma-CPP link.

PTSD and Chronic Pain

In addition to measuring trauma exposure, research has also focused on rates of PTSD in chronic pain. A 2017 systematic review found a 9.8% prevalence of PTSD across chronic pain populations, though only two studies were published on CPP (Fishbain et al., 2017). A similar meta-analytic protocol found a PTSD prevalence of 9.7% in the chronic pain population, with rates varying by study methodology and pain location (Siqveland et al., 2017). In a German protocol, 40% of patients with CPP without organic pathology met DSM-III criteria for PTSD,

and 10% of CPP patients with adhesions met criteria (Ehlert et al., 1999). More recently in the US, a large study of 713 women (primarily ages 18-45) from a pelvic pain referral clinic found that 31.3% of the sample screened positive for PTSD (Meltzer-Brody et al., 2007) and a study of interstitial cystitis/bladder pain syndrome found a provisional PTSD diagnosis rate of 42% (McKernan et al., 2019). The wide range of possible PTSD comorbidity in CPP (9.5 – 42%) suggests that rates of PTSD may be elevated in CPP compared to the non-CPP population, but additional research is needed to resolve inconsistencies. Furthermore, there is an open question of whether PTSD rates may be elevated in a CPP population when compared with non-CPP adults who also have verified trauma exposure, as in the veteran population. The rate of PTSD in veterans varies widely by deployment location, time of assessment and screening methodology. One meta-analysis found a prevalence estimate of 23% (with a standard error of 8.4%) of Operation Enduring Freedom/Operation Iraqi Freedom veterans (Fulton et al., 2015). Given the ambiguous relationship between earlier life trauma and later physical illness, it may be beneficial to investigate this disorder in a population where trauma exposure is known by virtue of occupation.

The majority of research investigating the relationship between PTSD diagnosis and posttraumatic stress symptoms on chronic pain has demonstrated that individuals with comorbid chronic pain and PTSD have worse pain outcomes. PTSD intrusion symptoms self-reported by Vietnam veterans (n = 129 males) were significantly related to pain disability, overall pain index via the McGill Pain Questionnaire – Short Form, and current VAS pain rating (Beckham et al., 1997). Comparing veterans with chronic pain by probable PTSD status, Outcalt et al. found that those with PTSD had higher pain severity, pain-related disability and pain interference, as well as more maladaptive pain cognitions and higher levels of distress (Outcalt et al., 2015), and

additional research found that re-experiencing symptoms were most influential in pain (Powell et al., 2015). Furthermore, even subsyndromal PTSD was linked with higher pain intensity, more pain-related functional disability, and greater pain-related interference (Langford et al., 2018). In one of the few studies that focused exclusively on CPP, a study of over 700 women, a positive PTSD screen was associated with lower healthy physical functioning and functioning without pain, more nonpelvic medical symptoms, a higher number of lifetime surgeries, and a higher number of bed days due to illness (Meltzer-Brody et al., 2007). Perhaps because of difficulty with defining the CPP population, or due to the diffuse treatment settings that patients may visit, the influence of PTSD has been studied less often in CPP than other types of chronic pain.

PTSD and CPP: Mechanisms and models

The extant literature consistently demonstrates a relationship between pain and PTSD, and a few theories have been proposed to explain the association. The shared vulnerability theory (Asmundson et al., 2002) proposed common biologic and psychological underpinnings to both disorders. The mutual maintenance theory (Sharp & Harvey, 2001) posited that cognitive, behavioral and affective components of both chronic pain and PTSD maintain and exacerbate symptoms in each disorder. Although the mutual maintenance model enjoys widespread support (Brennstuhl et al., 2015), there is a relative paucity of empirical longitudinal studies designed to support specific mechanisms and evidence has demonstrated that only some pain-related constructs may be influenced by PTSD symptoms (Ravn et al., 2018). In addition to methodological limitations, lack of specificity in both the shared vulnerability and mutual maintenance models may be the result of analyses that include total scores rather than latent variables that point towards mechanisms of comorbidity and symptom exacerbation. A final model, the Perpetual Avoidance model (Liedl & Knaevelsrud, 2008), describes intrusive

symptoms as the primary driver of PTSD and pain-related avoidance, hyperarousal, and catastrophizing, which in turn drive additional intrusive symptoms. Given the complexity involved in obtaining longitudinal data of populations with both PTSD and chronic pain, the Pain Avoidance model has relatively modest support and the Mutual Maintenance model is the most referenced theory in the literature.

Sleep disruption may represent a common symptom linking PTSD to pain. Trauma-related nightmares and comorbid sleep disorders are common in patients with PTSD (Miller et al., 2017). In self-report research, veterans with PTSD and Traumatic Brain Injury (TBI) or PTSD alone reported worse sleep than those with TBI alone (Balba et al., 2018), though objective studies have revealed some evidence of sleep misperception (Slightam et al., 2018). Moreover, there is substantial support for a bidirectional relationship between sleep and pain. Chronic pain patients have been found to have both objectively and subjectively poor sleep (Finan et al., 2013). More specifically, sleep was found to mediate the effect between posttraumatic stress symptoms and pain in a community sample of women (Aaron et al., 2019), as well as between PTSD symptoms and the longitudinal development of chronic pain in youth (Noel et al., 2018). Conversely, sleep disturbance over time was predicted by both baseline and worsening PTSD symptoms in a veteran sample with comorbid TBI (King et al., 2017). These data clearly suggest that sleep disruption is a candidate mechanism linking PTSD with more severe pain symptoms.

It is important to note that chronic pain is comorbid with other types of mental health problems. However, research in the CPP population has not typically delineated the role of depressive symptoms, despite the high correlation between depression and chronic pain (IsHak et al., 2018). In data from the National Health and Resilience in Veterans Study, the prevalence of

probable comorbid PTSD and Major Depressive Disorder (MDD) was estimated at 3.4% in a sample of over 2,000 individuals (Nichter et al., 2019). Furthermore, chronic comorbid PTSD/MDD has been associated with bodily pain and high levels of somatic symptoms (Armenta et al., 2019). Though PTSD and MDD are conceptualized as separate disorders and occur independently, there are also likely shared vulnerabilities, such as neuroticism (Post et al., 2016) and negative affect (Brown et al., 1998). Therefore, it is important to identify and account for the influence of depression on samples with posttraumatic stress symptoms.

Factor analysis allows for the reduction of many items with shared variance. The extant literature of chronic pain and PTSD has most often used total scores on PTSD symptom checklists to determine probable diagnoses, and latent variables have not been examined in the CPP population. Not only does factor analysis allow for dimension reduction, but latent variables can point to clusters that are particularly influential on pain. Previous research has focused on DSM categorical symptom classifications (e.g., (Beckham et al., 1997), rather than investigating underlying attributes. Given the overlap between PTSS, depressive symptoms and sleep disruption within CPP, a factor analytic method can account for symptoms that frequently co-occur. Furthermore, alternative dimensional models of psychiatric classification (e.g., (Kotov et al., 2017) may provide a superior model fit that does not cluster around the categories of DSM-5 symptoms.

Factor analyses of PTSD symptoms have been widely debated, with structures of between one and seven factors. A five-factor Dysphoric Arousal model (Re-experiencing, Avoidance, Negative Alterations of Mood and Cognition, Dysphoric Arousal, and Anxious Arousal) has shown superiority over previous four-factor structure (Armour, Mullerová, et al., 2016). However, almost no research has accounted for depression symptoms or sleep disruption

together with PTSD symptoms, although one study found a four-factor model for both the PTSD Checklist-Military version (for DSM-IV) and the PHQ-9 when used together (Tsai et al., 2011): Reexperiencing/Avoidance, Detached/Numbness, Hopelessness/Depression, and Bodily Disturbance. The structure of PTSD and depressive symptoms in conjunction with sleep problems has not been delineated previously. Thus, additional factor analyses were warranted to determine whether sleep and depressive symptoms represented distinct constructs or substantial overlap with PTSD symptoms.

The Current Study

Because of the complex etiology and difficulty in categorizing CPP, few studies have investigated the relationships between PTSD and CPP. Furthermore, the literature is largely silent on the relationships between latent variables measured by a common PTSD questionnaire (the PCL-5), depressive symptoms (PHQ-9), and sleep disturbance (the Insomnia Severity Index) within the veteran population, a group at high risk for trauma exposure. Therefore, the current study sought to replicate previous findings of the rate of probable PTSD in a chronic pelvic pain population and investigate the factor structure between posttraumatic stress symptoms, depressive symptoms, and sleep disturbance, and their effect on pain and quality of life. Confirmatory factor analysis was used to reduce items on the PTSD Checklist for DSM (PCL), Patient Health Questionnaire (PHQ), and the Insomnia Severity Index (ISI) to latent variables.

Although much of the extant literature has used total PCL scores, only a few studies provided guidance on factors influential on outcomes of pain and quality of life. Specifically, prior findings suggested that pain outcomes would be most likely to be predicted by intrusion symptoms (indicated in the re-experiencing factor) and depression/dysphoria symptoms. In a

study of Vietnam veterans, intrusion symptoms were related to pain and pain disability (Beckham et al., 1997) and there is a plethora of research documenting the relationship between depressed mood and pain. Moreover, one study found that depression symptoms mediated the relationship between PTSD and pain (Morasco et al., 2013). Sleep disturbance is also likely to be related to depressive symptoms as well as physiologic arousal. Physiological arousal is captured by pre-sleep arousal, which has been associated with poorer perceived sleep and nightmares (Tang & Harvey, 2004). Poor quality of life is also likely to have multiple predictors. For instance, depressive symptoms have shown a strong relationship with quality of life in chronic pain (e.g. (Elliott et al., 2003)), and thus factors capturing dysphoria would be likely to predict lower quality of life. Additionally, one study of PTSD and quality of life in veterans without chronic pain found a relationship between numbing symptoms and all domains of life quality (Schnurr & Lunney, 2008), suggesting that the proposed negative alterations in mood and cognition factor would demonstrate a relationship with quality of life. As the literature investigating PTSD factor structure in chronic pain samples is limited, these studies provided potential directions for hypothesized relationships in this analysis.

Hypotheses were as follows:

1. The rate of probable PTSD in the CPP population would be consistent with previous literature at ~30%.
2. In an exploratory factor analysis, a 7-factor model (Figure 1; Re-experiencing, Avoidance, Negative Alterations of Mood and Cognition, Dysphoric Arousal, Anxious Arousal, Depression and Sleep) model would represent the best fit to PTSD and depressive symptomatology. This

model is consistent with the Dysphoric Arousal model specified by Armour and colleagues (Armour, Jana, et al., 2016) with an additional factor emerging for depression as in Tsai and colleagues (Tsai et al., 2011) .

3a. Consistent with prior work (Beckham et al., 1997; Morasco et al., 2013) on PTSD and depression, and the body of research on sleep and pain (Finan et al., 2013), factors related to depression, re-experiencing and sleep disturbance would have a stronger effect on pain severity and interference than factors related to avoidance, negative alterations of mood and cognition, and dysphoric and anxious arousal.

3b. Consistent with previous work (Elliott et al., 2003; Schnurr & Lunney, 2008), factors related to dysphoria and negative alterations of mood and cognition would show the strongest effect on quality of life.

Methods

Participants

Participant data from patients 18 and older was sampled from the Veteran's Affairs Corporate Data Warehouse (CDW). To create the patient cohort, any female patient who carried a diagnostic code for CPP during two or more outpatient visits scheduled at least 6 months apart. Because administration of specific mental health measures did not begin until 2013, only patients who met criteria on or after January 1, 2013 were included. International Classification of Disease codes to define CPP were adapted from researchers utilizing ICD-9 codes to examine chronic pain in a sample of female veterans (Table 1; Cichowski et al., 2018). The cohort was anticipated to be majority non-Hispanic white, based on reports of patient demographics at the VHA (*Profile of Veterans: 2016 NCVAS National Center for Veterans Analysis and Statistics*, 2018).

Measures

The following demographic variables were extracted from the record: age, body mass index (BMI), comorbid medical conditions, Disability Rating, Race, Ethnicity, and patient-identified gender.

Depression

The Patient Health Questionnaire-9 (PHQ-9) is a self-report measure of depressive symptoms over the previous 2 weeks. Items are scored from 0-3, with a maximum score of 27. Scores of 5, 10, 15, and 20 correspond to mild, moderate, moderately severe, and severe depressive symptoms (Kroenke et al., 2001). Coefficient α (Cronbach, 1951) in this sample was 0.87.

Pain

The Pain Outcome Questionnaire (PQ) – Short Form is a pain treatment outcome measure developed cooperatively by the VA and pain management professional societies. It assesses pain intensity and interference, as well as satisfaction with treatment, medication and care services use and history, disability status, and employment status; scores range from 0-190 (Clark et al., 2003). Coefficient α was 0.83.

Posttraumatic Stress Symptoms

Primary Care PTSD (PC-PTSD questionnaire). The PC-PTSD screen is a measure to detect possible PTSD. Scores range from 0-5; a score of 4 has been found to be the optimally efficient screening threshold (sensitivity 0.83, specificity 0.91), while a score of 3 maximized sensitivity (Prins et al., 2016). Coefficient α in the sample was 0.89.

PTSD Checklist for DSM-5 (PCL-5). The PCL-5 is a 20-item self-report measure assessing symptom clusters in PTSD as dictated by DSM-5. The scale is 0-4 for each item; a total score can be calculated (range 0-80) or symptom clusters can be scored (Weathers et al., 2013). Psychometric and diagnostic validation studies are ongoing but preliminary research suggested a sensitivity of 0.78 and specificity of 0.98 for the PCL-5 with a cutoff of 38 in an undergraduate sample (Cohen et al., 2014). At the time of this study studies with a cutoff score of 38 had not been published in veteran samples. Coefficient α in the sample was 0.95.

Sleep

Insomnia Severity Index (ISI). The ISI is a 7-item self-report scale that assesses problems in sleep onset, maintenance, and wake, as well as sleep quality and daytime impairment. The score range is 0-28, with a score of 15 used as a marker of Insomnia Disorder (Bastien et al., 2001). Coefficient α in the sample was 0.89.

Quality of Life

Veterans Rand 12 Item Health Survey (VR-12). The VR-12 is a brief self-report measure of health-related quality of life in 8 domains: general health perception, physical functioning, role limitation due to physical and emotional problems, bodily pain, energy-fatigue, social functioning and mental health. The scale produces Physical Health and Mental Health component scores (*PCS* and *MCS*, respectively). The VR-12 was developed from the Veterans RAND 36 Item Health Survey, which was developed from the Medical Outcomes Trust RAND SF-36 Version 1.0 (Jones et al., 2001). PCS and MCS scores are standardized and have a mean

of 50 and a standard deviation of 10, with lower scores indicative of lower quality of life (higher burden of physical or mental health problems). Coefficient α was 0.89.

Data Analysis

Analyses were conducted within the VA Informatics and Computing Workspace (*VINCI*, 2021) using Rstudio (RStudio Team, 2021). Structural equation modeling was conducted in the lavaan package(Rosseel, 2012). Duplicate responses were removed and data were screened for out-of-range answers. When participants had multiple questionnaire administrations, the most recent data were included. Item-level data were checked for missing or skipped responses; analyses utilizing total scores were conducted with complete questionnaires only when item-level information could be verified.

Results

Participants

A total of 18,928 individuals met criteria for CPP in this study. The sample was composed of midlife women veterans (Table 2). In the data, gender identifiers were “physician-identified”, and only a small portion of participant data extracted included a self-identified gender. Of the self-identified group, the majority (96%) of patients were identified as female. The majority of veterans self-identified as White (53%) or Black or African American (38%), and most were not Hispanic or Latino (88%). Average BMI was 30.47 and average disability was rated as 28.09%. The most commonly comorbid physical conditions were low back pain, essential hypertension, and gastroesophageal reflux disease. 1524 individuals (8% of the sample) had at least one outpatient visit coded for dysmenorrhea.

Of these individuals, 14,056 completed the PHQ-9, 680 completed the ISI, 5,283 completed the PCL, and 10,026 completed the PCPTSD. Psychological symptoms, on average, indicated a moderate level of depressive symptoms, clinical insomnia symptoms of moderate severity, and a sample at risk of probable PTSD as measured by the PCL-5. VR-12 MCS and PCS scores were reflective of a higher burden on mental and physical health. The average PQ-Pain score is in the 25th percentile of intake Outpatient Data.

A subset of the sample completed the pain and quality of life outcomes. Only 357 individuals had a verified POQ scale, and 220 individuals had item-level VR-12 data (18 completed both). There were 239 PCL-5 administrations and 456 PHQ-9 administrations. Missing data were shown not to be Missing Completely At Random through Little's Test (Little, 1988). Age was a predictor of missingness on the pain questionnaire as well as the PCL-5, thus it was included in structural models. Self-Identified Gender ("male") was also a significant predictor, but given the gender identity distribution this variable was not included in the model. Demographics for the reduced sample (N = 559) are available in Table 2. The Physical Component Score was significantly lower in individuals who had completed the pain measure than those without pain measurements, such that individuals who completed quality of life surveys had a mean PCS of 37.30, whereas individuals who also completed pain surveys had a mean PCS of 30.00, indicating differences in physical health-related quality of life. Other psychological questionnaires did not statistically differ between participants who completed pain or quality of life measures alone.

Hypothesis 1: PTSD Prevalence

It was hypothesized that 30% of the sample would score in the range of probable PTSD. The screening cutoff for this study was 38, given the elevation of posttraumatic stress symptoms (PT SS) in the VA system (Weathers et al., 2013). PC-PTSD scores were summed. A total of 2,048 women exceeded the cutoff score of 3 (Bovin et al., 2021; Prins et al., 2016) and had a score of 4 or 5, meaning that 19.76% of the sample screened positive for PTSD using the optimal cutoff (95% confidence interval: 19.01%, 20.55%). In comparison, a much higher percentage of patients screened positive for probable PTSD using the PCL-5. A total of 3,794 (69.2%) of the sample screened positive for PTSD (95% confidence interval: 67.95%, 70.42%). Using the PC-PTSD, the rate of probable PTSD was lower than hypothesized, whereas the PCL-5 rate of probable PTSD far exceeded the hypothesized rate.

Hypothesis 2: Factor Analysis

To reduce collinearity in measures of PTSD symptoms and depression symptoms, confirmatory factor analysis was used to determine the optimal latent structure of the data. A seven-factor model was proposed for the data (see Figure 1). However, prior to fitting structural equation models, item-level and variable-level descriptive data were examined. Tests of univariate normality indicated skew in many variables, thus a robust maximum likelihood estimator was used to account for non-normality (Satorra & Bentler, 1994). Missing data were accounted for by using Full Information Maximum Likelihood estimation. Seven cases were removed as multivariate outliers by Mahalanobi's distance. A full information maximum likelihood correlation table is available in Table 3 and can be compared with the scatterplot matrix of correlations in Figure 2. All variables had near-zero correlations with the Physical Component Score of the VR-12, but correlated in the expected direction with the PQ total score.

Contrary to expectation, insomnia scores had a very weak correlation with the pain outcomes score. Because this was an unexpected outcome, the correlation between individual ISI, PQ, and PHQ-9 items were inspected to determine whether there were obvious coding errors and to provide evidence of construct validity. ISI items were generally found to have weak or negligible correlations with items representing pain interference and severity. Moreover, ISI items had low to negligible correlations (ranged from -0.02 to 0.25) with the PHQ-9 item that represents poor sleep (Figure 3), indicating poor convergent validity.

Collinearity diagnostics were assessed and when all psychological items from the ISI, PCL and PHQ-9 were included in the model, Variance Inflation Factor (VIF) was high (>10) for many variables, indicating multicollinearity among items. When the ISI items were examined without the PCL-5 or PHQ-9, the 7 ISI items formed a non-positive definite matrix. Given both the weak correlations with expected outcomes (pain, depression) in addition to substantial redundancy when considering all ISI items in a model, the ISI was removed from further analyses.

Although many of the expected relationships were not present, the proposed seven-factor model was tested (Figure 1). The model did not converge, indicative of poor fit. Additional models were considered after using Horn's parallel analysis (Horn, 1965) using the psych package (Revelle, 2021). Parallel analysis determined six factors should be extracted from the matrix, though the analysis does not specify factor loadings. Given the proliferation of models in the literature, several models with six factors were tested. Models were considered based on commonly accepted robust fit statistics, including CFI and TLI > 0.9, RMSEA < 0.05 for good fit and <0.08 for acceptable fit, and SRMR <0.08 (Hu & Bentler, 1999).

The parallel analysis indicated six factors for the PHQ-9 and PCL-5 combined. Three models indicated by the literature were fit to the data: Dysphoric Arousal, DSM-5, and Dysphoria models of PTSD. The DSM-5 and Dysphoria models consist of four factors, and in those models a two-factor solution for the PHQ-9 previously tested in veterans was fit to the data (Elhai et al., 2012). Model fit indices are listed in Table 4. The Dysphoria and DSM-5 models were virtually indistinguishable in fit. However, the dysphoria model had a slightly smaller AIC value. The DSM-5 model was selected for structural regression in Hypothesis 3 given that the model mapped onto clinical criteria. Modification indices included correlation of residual error between indicators of the same latent factor (e.g., PHQ item 3 and PHQ item 4, measuring sleep disturbance and tiredness, respectively; and PCL item 17 and item 18, feeling "superalert" or watchful and feeling jumpy, respectively). Because these items loaded on the same factors and measure similar constructs, modification indices were reasonable to implement. Figure 4 shows the final factor structure of the model and Table 5 lists factor loadings. The final structure included factors of Re-experiencing, Avoidance, Negative Alterations in Mood and Cognition (NAMC), Hyperarousal, Depression – Non-Somatic, and Depression – Somatic.

Hypothesis 3: Outcome Measurement and Regression

To examine the relationship among PTSD and depression symptoms and their relationship to pain and quality of life, a structural equation model was estimated with paths drawn from psychological symptom factors to pain and quality of life factors. It was hypothesized that latent factors related to depression, re-experiencing and sleep disturbance would have a larger effect (path coefficient) on pain severity and interference, and that factors related to dysphoria and NAMC would have larger effects on quality of life. As such, the model

used three observed outcomes: total PQ score, VR-12 MCS, and VR-12 PCS. The exogenous variables were latent scores derived from the CFA. The model did not converge, demonstrative of misspecification. Because models with total PQ and VR-12 did not converge, the model was respecified using previously researched subscales for the PQ and VR-12. Measurement models assessed acceptability of respecified outcome variables given the failure of the first model.

For pain outcomes, the PQ was decomposed into subscales: Pain Intensity, Mobility, and Activities of Daily Living, as these subscales best represent the constructs of pain intensity and interference (Clark et al., 2003). The Mobility and Activities of Daily Living scales were represented by an additional latent factor of Impairment given their factor intercorrelation. The model provided a good fit by metrics of TLI, CFI and SRMR, and an acceptable RMSEA value (Robust TLI = 0.98, Robust CFI = 0.99, SRMR = 0.05, Robust RMSEA = 0.05 [0.02, 0.08]; see Tables 6 and 7 for parameter estimates and robust fit statistics).

The VR-12 was decomposed into two factors: mental health and physical heath (Selim et al., 2009). This factor structure demonstrated good fit (Robust CFI = .96, Robust TLI = 0.95, Robust RMSEA = 0.07 [0.045, 0.087], SRMR = 0.06). Finally, a model with both the respecified pain and quality of life outcomes was generated. However, when considered together in a measurement model, pain and quality of life outcomes did not converge. Given the focus on predicting health-related outcomes, models that predicted pain and QOL were considered separately. Parameter estimates are reported in Table 6, and model fit statistics are listed in Table 7.

Quality of Life. To assess quality of life, a new structural model (Figure 5) was estimated with paths from NAMC, Depression – Somatic, and Depression – Non-Somatic factors to factors representing physical and mental health-related quality of life while all other paths

from psychological latent factors to endogenous variables were constrained to zero. The model demonstrated acceptable fit (SRMR = 0.07, RMSEA 0.04 [0.038 0.045], CFI = 0.90, TLI = 0.90). Respecifying the model to include non-zero paths from PCL-5 latent factors of Re-experiencing and Avoidance did not significantly improve the model (Likelihood Ratio Tests $p > .05$). When a non-zero path was allowed from Hyperarousal to quality of life factors, it was a significant addition to the model ($p < .05$), however, Hyperarousal and NAMC appeared to suppress each others' effects. Therefore, two models were estimated that contained paths from Hyperarousal (path to mental health quality of life: standardized $\beta = -0.24$) and NAMC (path to mental health quality of life: standardized $\beta = -0.26$). Non-somatic depressive symptoms (path to mental health quality of life: standardized $\beta = -0.46$) and Age were also significantly associated with quality of life. Only Age was a significant predictor of physical health-related quality of life (standardized $\beta = -0.25$).

Pain. In the structural model with pain outcomes (Figure 6), factors related to depression, re-experiencing and sleep disturbance were assigned non-zero paths, while other factors were constrained to zero. Avoidance and NAMC did not appreciably improve the model (Likelihood Ratio Tests $p > 0.5$). When Hyperarousal symptoms were not constrained to zero, the model produced improbable coefficients given the observed correlations during data screening. Similarly to the quality of life models, two models were estimated separately. Re-experiencing symptoms were a significant predictor of pain severity (standardized $\beta = 0.29$), while only Age was a significant predictor of Pain-Related Interference (standardized $\beta = 0.19$). When Hyperarousal symptoms were not constrained, the factor was not a significant predictor of pain severity or interference ($p > 0.5$).

Discussion

The present study of women veterans with CPP showed that patients with moderate pain had rates of probable PTSD either slightly lower than the general veteran population or at significantly higher rates than expected, depending on screening instrument. The study also demonstrated that several factor structures of PTSD and depressive symptoms fit the data adequately, with DSM-5 delineation of latent factors representing a good fit from several comparable models. Finally, structural regression models showed that Hyperarousal symptoms and NAMC were related to lower levels of mental health-related quality of life, whereas Re-experiencing symptoms were related to higher pain severity.

Hypothesis 1: PTSD Prevalence

CPP has been relatively understudied in comparison to other chronic pain conditions, particularly in relation to posttrauma symptoms. Only two studies have assessed the prevalence of PTSD in a pelvic pain population and none has done so in a veteran population. This study first investigated rates of probable PTSD and compared rates by screening instrument. Administrations of the PC-PTSD resulted in 19.76% individuals above the screening threshold for probable PTSD, whereas 69% of PCL-5 administrations resulted in a score above the screening cutoff. Neither survey resulted in rates of probable PTSD close to the hypothesized value (30%) based on prior literature, though the rate as determined by the PC-PTSD was close to a general veteran population estimate of 23% (Fulton et al., 2015). The cutoff of 38 on the PCL-5 had been used in undergraduate samples only although recommended for veteran samples as well; had the traditional cutoff of 33 been used an even higher proportion of PCL-5 administrations would have considered positive for probable PTSD. The discrepancy of the two screening instruments may in part be explained by context of assessment. The PC-PTSD was

used as a screening tool in primary care settings, and thus given to all patients in the VHA system. The PCL-5 was used mostly in the mental health clinics within the VHA. Thus, individuals who were already being treated for PTSD or other mental health concerns were more likely to complete this measure, which likely explains the much higher prevalence of probable PTSD. It may be that in primary care clinics in the VA system, assessment of PTSD in pelvic pain is akin to rates seen in community samples. By the time patients have been referred to mental health clinics, there may be a reasonable expectation of exceeding screening cutoffs on self-report psychological questionnaires. Accurate estimates of the prevalence of PTSD among CPP patients is important as the role of traumatic stress symptoms in health outcomes continues to be of interest through both behavioral and physiological pathways, and ascertaining accuracy of associations is critical to guide future research.

These data demonstrated a rate of probable PTSD in a CPP population commensurate with estimates from a trauma-exposed population (VHA patients), and similar to a recent community survey (Choung et al., 2010). However, the prevalence rate diverged from earlier work in chronic pelvic pain (Meltzer-Brody et al., 2007) but the sampling paradigm also differed – earlier researchers surveyed women in a pain referral clinic, whereas VA patients completing the PC-PTSD did so in primary care and the PCL-5 typically in the context of mental health clinics. Nonetheless, additional research is necessary to confirm whether PTSD is associated with this specific population beyond the expected population rate. Inconsistency in research settings lead to widely divergent prevalence estimates, as demonstrated by these data.

Hypothesis 2: Measurement Models

The factor structure of PTSD has been a subject of much attention and a variety of valid structures have been proposed. Because of substantial comorbidity between PTSD and depressive symptoms in addition to sleep disturbance in the CPP population, a factor analysis of these data was warranted to assess the existence of latent transdiagnostic variables. It was hypothesized that seven factors (Figure 1) would best capture overlapping variance. However, the hypothesized model was not an adequate fit and was respecified. Additionally, because of multicollinearity evident within the ISI, the survey was eliminated from further analyses.

Because the proposed seven factor model did not fit the data, alternate measurement models were tested and many fit this dataset reasonably well. Rather than explore models with numerous two-indicator factors, a confirmatory model was selected that aligned with subscales measured on the PCL while permitting correlations among factors. In this sample, depression and PTSD symptoms were best fit onto separate but correlated factors. It is possible that with different instruments that do not map directly onto DSM-5 criteria (e.g., the Impact of Events Scale) that other factor structures would emerge or that one model would outperform the others. Nonetheless, in terms of maximizing predictive utility, the DSM-5 model with two depression factors represented an appropriate model for these data.

Although several models demonstrated acceptable fit, the substantial correlation between latent factors also complicated the subsequent structural regression. In a prior factor analytic comparison study using PCL-5 data from veterans, correlations between factors of the DSM-5 model had a range of 0.62 – 0.85 with the majority of correlations between 0.62 – 0.71 (Lee et al., 2019), much lower on average than in the data presented in this paper. One possibility is a method effect such that participants' self-report on the PCL-5 conflated symptom structure, though Lee and colleagues demonstrated measurement invariance between the PCL-5 and the

Clinician-Administered PTSD Scale for DSM-5, a structured interview. An additional possibility was that the sample was too broad in terms of inclusion of PTSD and non-PTSD caseness. One study found differences in fit measures between groups of Canadian veterans with and without PTSD, though the researchers used DSM-IV criteria. Counterintuitively, the results of their analyses found that there was more residual error associated with the PTSD group than the non-PTSD group (Biehn et al., 2012). Thus, excluding cases without probable PTSD may not have improved model estimates in this sample. A final possibility is low construct validity of the PCL-5 subscales such that they do not truly define unique constructs within this sample, or that they are reflective of higher-order distress. Perhaps additional measurement methods or physiological correlates of symptom clusters (e.g., hemodynamics associated with hyperarousal) may reveal more distinct latent constructs.

Unusually, the insomnia surveys showed a low correlation with pain variables. Examination of item-level data revealed moderate associations with some pain indicators but near-zero with others. This is surprising considering the wealth of literature exploring the link between sleep and pain (Finan et al., 2013) and putative biological mechanisms connecting the two events (Haack et al., 2020). Additional dimensions of sleep disturbance not captured by the Insomnia Severity Index may have accounted for more of the relationship between sleep and chronic pain in the CPP population. This may have included such constructs as symptoms of Restless Leg Syndrome or Sleep-Disordered Breathing, or behavioral factors that facilitated delayed sleep onset or nighttime awakenings, e.g., lengthy daytime naps or avoidance of bed. Future research may expand on factors that are associated with poor sleep in the CPP population, given that this sample reported moderate insomnia symptoms overall.

Hypothesis 3: Structural Regression

Structural equation models were estimated to determine the influence of PTSD and depressive symptoms on pain and quality of life factors. Separate models for pain and quality of life were estimated because of poor fit in a model when they were considered together. Based on prior findings, sleep disturbance, depression, and re-experiencing were hypothesized as significant predictors in the pain regression model. Consistent with predictions, re-experiencing was a significant predictor of pain intensity, though depression factors were not significant and sleep problems did not emerge as a separate factor as proposed in Hypothesis 2. This result was partially in line with one prior study that found re-experiencing symptoms and sleep quality were the most influential symptom clusters in pain (Powell et al., 2015). While ratio tests indicated that other latent factors of PTSD were important to include in the model, the release of path constraints on those factors also introduced significant suppressor effects. When considered separately, hyperarousal did not emerge as a significant predictor for pain-related variables. In this sample, re-experiencing which included both daytime and nighttime intrusion symptoms was the strongest predictor of greater pain intensity, while no predictors of pain-related interference were found to be significant.

Identifying the PTSD symptom clusters most closely associated with pain symptoms could offer treatment implications. After nonpharmacological treatment for PTSD, residual symptoms of sleep disturbance including both insomnia symptoms and nightmares have been shown to persist in active duty military personnel (Pruiksma et al., 2016). Pharmacological options such as Prazosin may be used to treat nightmares, but may not be suitable for all patients. Therefore, additional treatment for nightmares, such as Exposure, Relaxation and Rescripting

Therapies or Image Rehearsal Therapy may be indicated particularly for individuals with CPP to help lower or maintain pain severity levels by reducing intrusive dreams.

In the pain regression model, some support was provided for the Perpetual Avoidance Model (Liedl & Knaevelsrud, 2008). This model proposed that intrusive symptoms (re-experiencing) are the primary driver of additional PTSD symptoms and pain. Intrusive thoughts and nightmares (re-experiencing symptoms) are proposed to trigger physiological arousal, which in turn may lower pain perception thresholds (Dunne-Proctor et al., 2016). Night-time arousal may also precipitate conscious awakening, leading to sleep loss and subsequently altered pain response (Finan et al., 2013). This is also consistent with recent longitudinal data in a chronic pain sample (de Vries et al., 2021). In this sample, re-experiencing symptoms were the strongest predictor of pain intensity, followed by hyperarousal. Future research might consider more immediate assessment of arousal in response to intrusive symptoms and their subsequent impact on pain intensity as well as the dynamic fluctuation of PTSD symptoms in relation to pain.

CPP carries a substantial burden, such that individuals with intense pain and psychological symptoms are likely to experience lower quality of life. In the present study, depressive symptoms and NAMC significantly predicted mental health-related quality of life. Total PTSD symptoms had a stronger correlation with the mental health component (Pearson correlation $r = 0.36$, with higher scores representing worse mental health) scores than physical health component scores which were close to zero ($r = -0.03$). In the structural models, greater NAMC and Hyperarousal symptoms predicted lower mental health-related quality of life. Depressive symptoms have been linked to lower quality of life through participation in social, occupational and leisure activities (Muller et al., 2017). It's possible that NAMC may be related to lower quality of life because of a lack of social or occupational engagement, or withdrawal

from social support. Although NAMC and depressive symptoms acted as distinct factors in this model, they may share similar mechanisms to quality of life. Additional research on the mechanisms between dysphoria and negative cognitions and quality of life variables is necessary to elucidate these relationships.

In quality of life models, hyperarousal also emerged as a significant predictor of quality of life. The link between hyperarousal and quality of life was consistent with prior research on individuals with war-related PTSD (Giacco et al., 2013). Future research may wish to assess potential mediators of hyperarousal symptoms, such as cognitive impairment due to sleep deprivation, or interpersonal conflict related to hypervigilance, that may explain some of the associations between quality of life and the specific hyperarousal cluster. These data were not adequate to test pain and quality of life variables simultaneously, but it is possible that pain severity or pain-related impairment could have had an indirect effect in the relationships between PTSD symptoms and lower quality of life.

A common thread throughout each structural regression models was that Avoidance was not identified as a significant predictor of pain or quality of life (with the caveat of evident multicollinearity). One possibility is that the Avoidance factor was under-identified given that it had only two indicators (Rasmussen et al., 2019). It's also possible that self-reported avoidance served as a coping strategy that was perceived as effective by pain patients when assessing role limitations or social functioning impairment. Nonetheless, the latent factor representing avoidance was positively correlated with other factors as expected. Additional study of perceived impairment related to avoidance may be beneficial in chronic pain to elucidate motivations for maintaining avoidance behavior.

Limitations

This study was limited by its use of data sampled across different timepoints, though elapsed time between surveys did not significantly correlate with relationships between surveys. While the equations here captured associations and covariance, they also did not permit causal inference. Some recent work has focused on depressive symptoms as a mediator to PTSD symptoms but it was not feasible to test this structure in the model given different temporal associations between patients' mental health administrations. Additionally, sample size was also reduced drastically due to unverified item-level data. It is possible that these data differed in some dimensions from the rest of the dataset. Future work could replicate these models in a different sampling frame, e.g. across all chronic pain patients at the VA, though it is suggested that biopsychosocial models remain disease-specific (Bolton & Gillett, 2019) and therefore models of PTSD and pain symptoms may differ across different sites of pain.

An ideal structural regression would have modeled both pain and quality of life together, though there appeared to be multicollinearity. There are a few solutions to this problem, such as averaging variables, performing a principal components analysis, or deleting redundant variables (Tarka, 2018), yet these solutions would have compromised the research questions at hand, namely, predicting health and quality of life outcomes. Pain and quality of life are tightly linked, and additional measurement models may appropriately assess these constructs jointly.

It is important to consider the generalizability of this sample. In terms of demographics, although self-identified gender was only available for a fraction of the sample, the data suggest a mainly cisgender female population, and so these estimates may differ across the gender identity spectrum. Results of this study may be most applicable to midlife women. Menopausal status was not retrieved with these data but is likely relevant given the overlap between chronic pelvic pain and cyclical menstrual pain. Conversely, in terms of generalizability across race and

ethnicity, 30% of this sample of women identified as Black or African-American, a proportion that exceeds population representation. Thus, these data captured psychological symptom and pain relationships that are most generalizable to Black or African-American and White women veterans. On the other hand, generalizability to other patients across the spectrum of racial and ethnic identities was limited and may be a topic of future study given cultural differences in the psychology of pain.

Conclusions

In a sample of women veterans with CPP, rates of probable PTSD were commensurate with a recent community sample as well as likely expected prevalence in the VA system. Screening environment drastically changed the rate of probable disorder, thus additional estimations of the rate of disorder in the population should consider characteristics of treatment-seeking patients. Common factor structures of depression and PTSD were replicated in this population, and the DSM-5 model of PTSD represented an adequate fit to these data along with somatic and non-somatic depressive symptoms. The latent factor of Re-experiencing symptoms was the strongest predictor of higher pain severity, providing evidence for the Perpetual Avoidance model and indicating directions for future research into the cycle of posttrauma symptoms and pain.

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Appendix A: Figures

Figure 1

Hypothesized factor structure

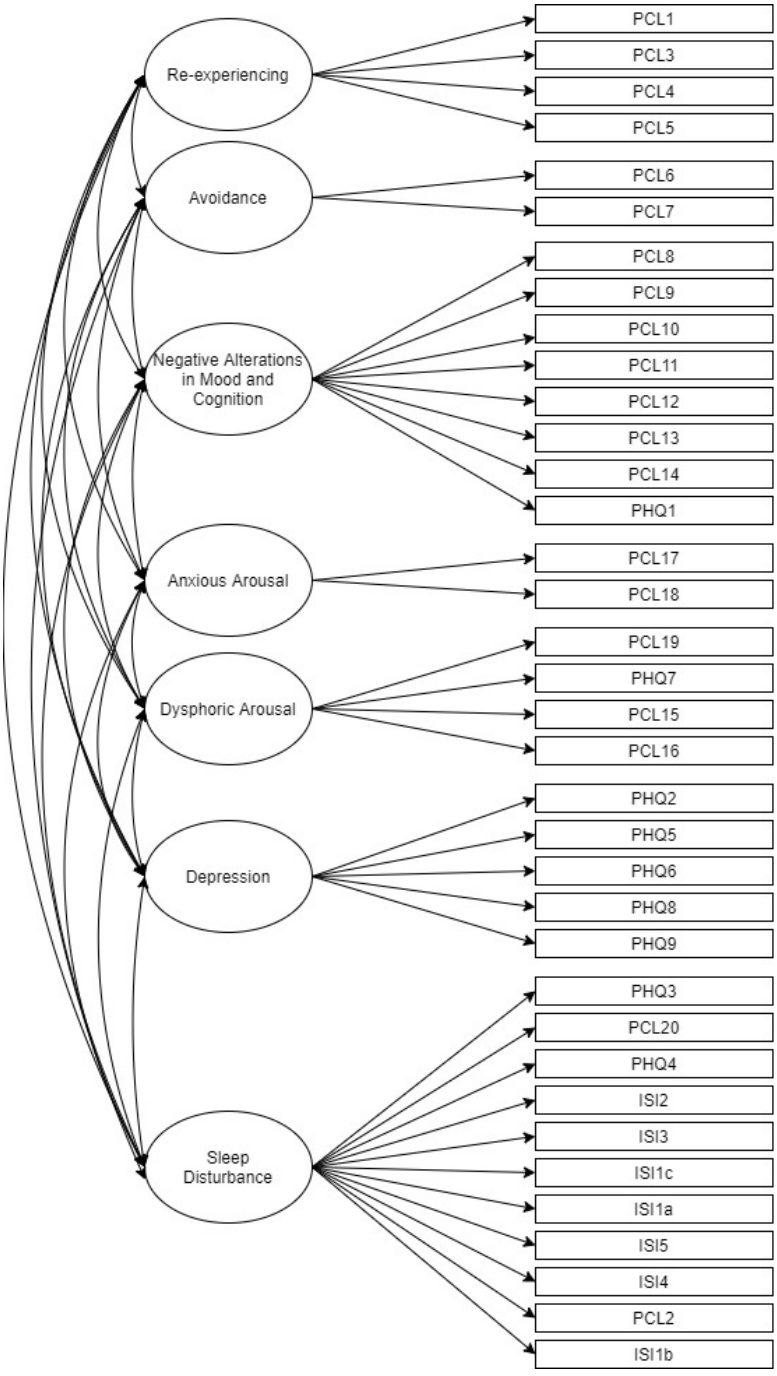
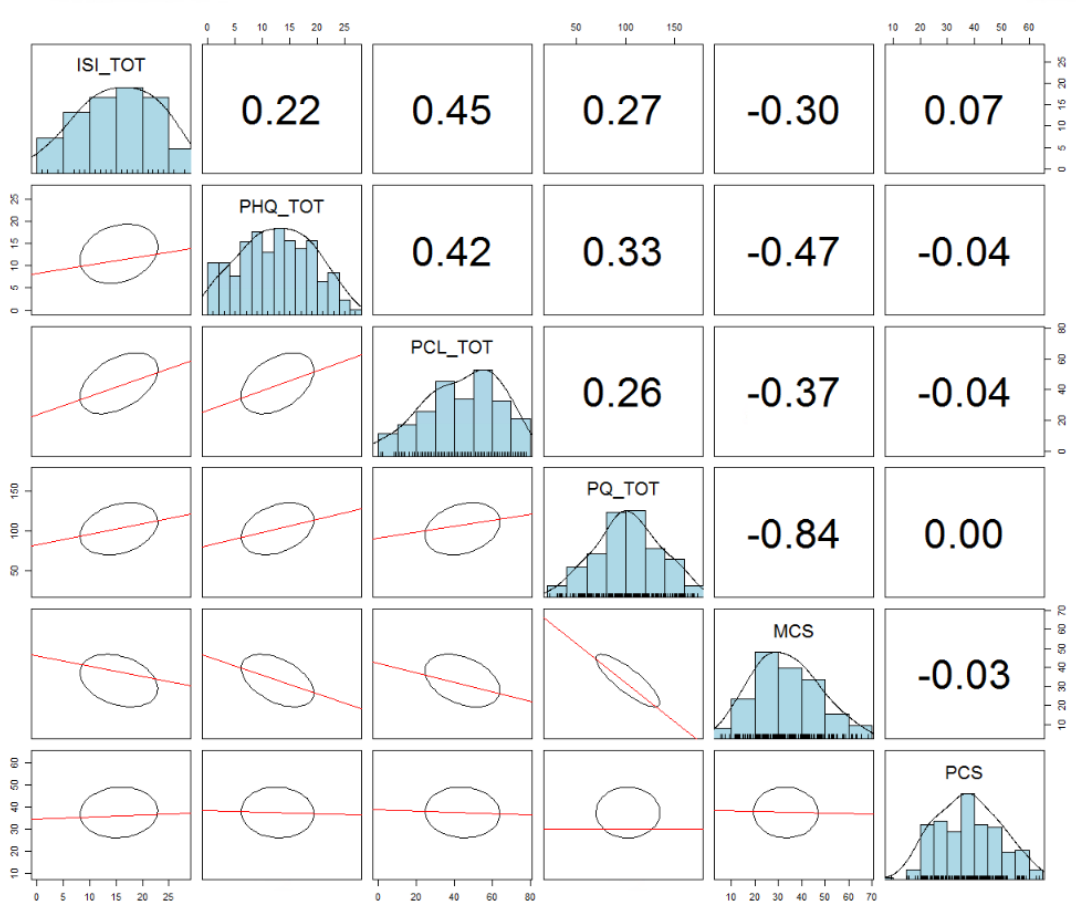


Figure 2

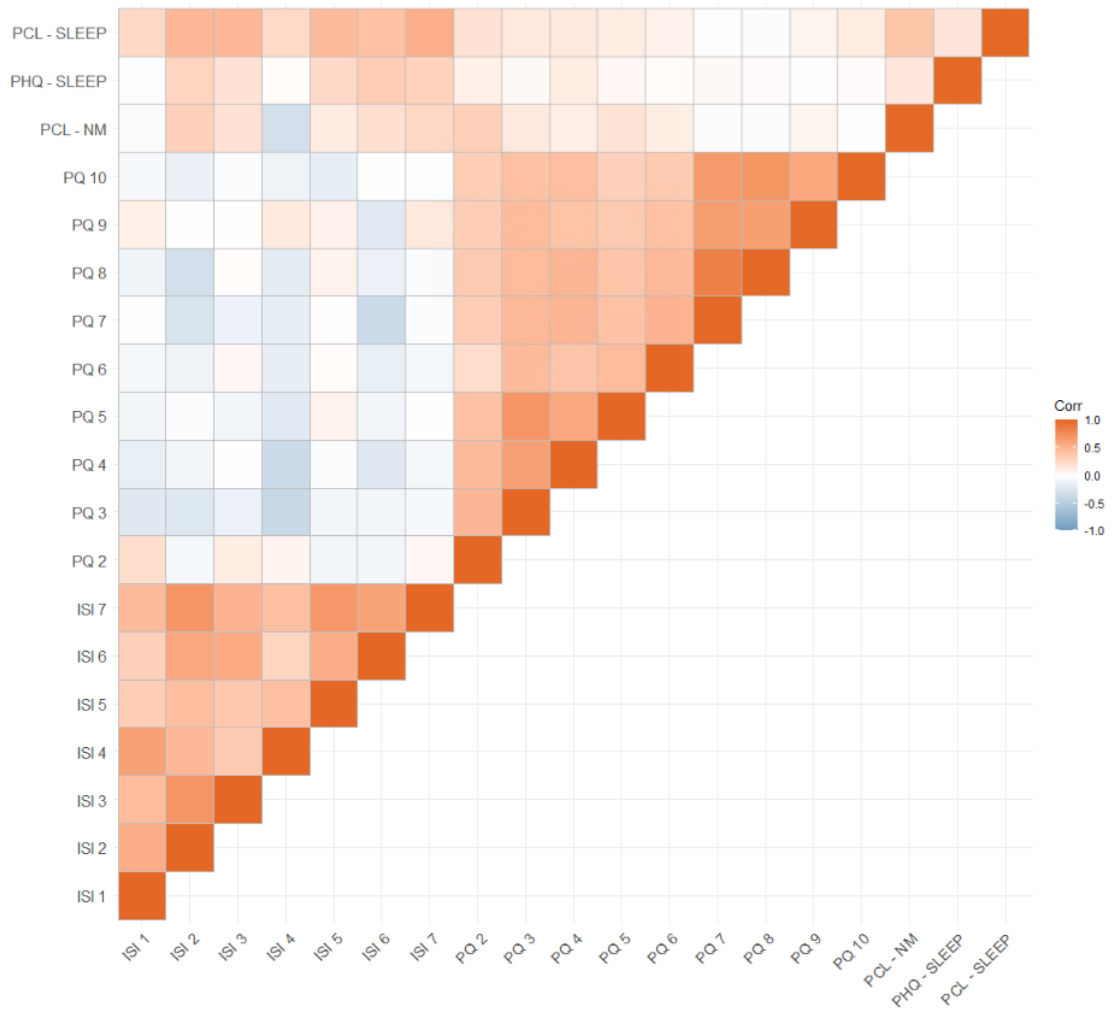
Scatter Plot Matrix of total survey scores



ISI_TOT = Insomnia Severity Index. PHQ_TOT = Patient Health Questionnaire-9. PCL_TOT = PTSD Checklist for DSM-5. PQ_TOT = Pain Questionnaire, Short Form. MCS = Mental Component Score. PCS = Physical Component Score.

Figure 3

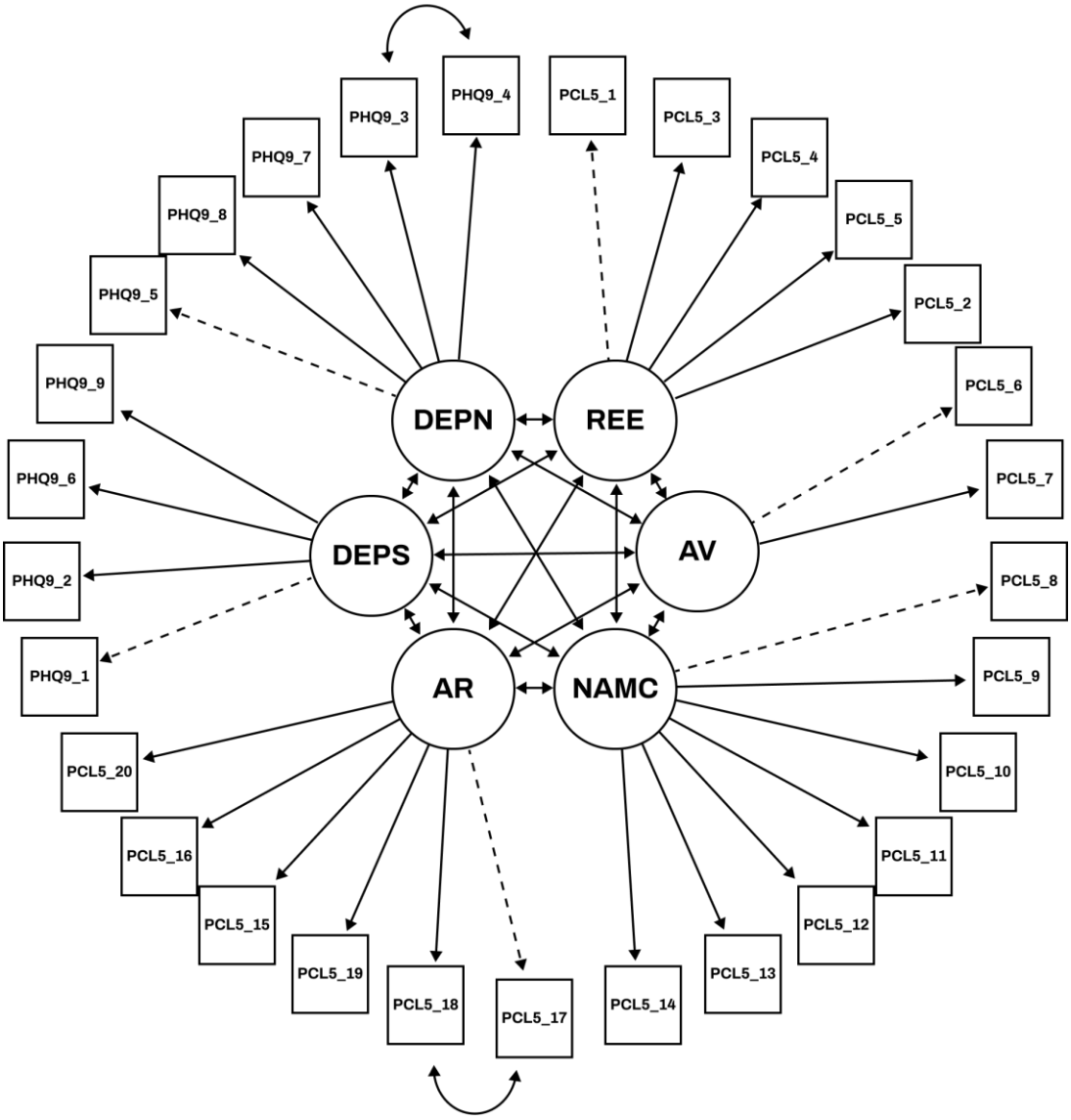
Full Information Maximum Likelihood Correlation of items related to sleep and pain



Stronger hues indicate stronger correlations. ISI = Insomnia Severity Index. PQ = Pain Outcomes Questionnaire. PCL – SLEEP = PCL-5 Sleep Item. PHQ – SLEEP = PHQ-9 Sleep Item. PCL-NM = PCL-5 Nightmare Item.

Figure 4

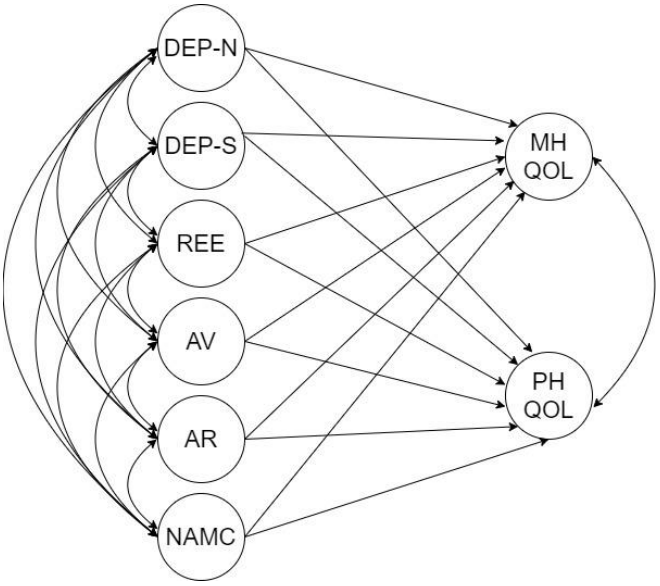
Factor Structure of PCL-5 and PHQ-9



DEPN = Depression, Non-Somatic Symptom. DEPS = Depression, Somatic Symptoms. REE = Re-experiencing. AV = Avoidance. AR = Hyperarousal. NAMC = Negative Alterations in Mood and Cognition.

Figure 5

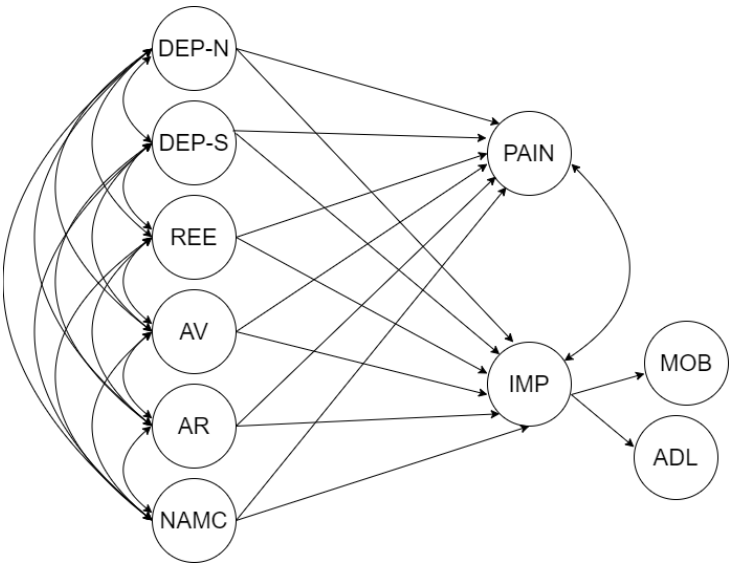
Structural Regression: Quality of Life



DEP-N = Depression, Non-Somatic Symptom. DEP-S = Depression, Somatic Symptoms. REE = Re-experiencing. AV = Avoidance. AR = Hyperarousal. NAMC = Negative Alterations in Mood and Cognition. MH QOL = Mental Health Quality of Life. PH QOL = Physical Health Quality of Life.

Figure 6

Structural Regression: Pain



DEP-N = Depression, Non-Somatic Symptom. DEP-S = Depression, Somatic Symptoms. REE = Re-experiencing. AV = Avoidance. AR = Hyperarousal. NAMC = Negative Alterations in Mood and Cognition. IMP = Pain Impairment. MOB = Mobility. ADL = Activities of Daily Living.

Appendix B: Tables

Table 1

International Classification of Disease Codes for Pelvic Pain

Disorder	ICD-9 Code	ICD-10 Code
Chronic Pelvic Pain	625.9	R10.2
Pelvic Congestion Syndrome	625.5	N94.89
Abdominal pain RLQ	789.03	R10.31
Abdominal Pain LLQ	789.04	R10.32
Abdominal Tenderness LLQ	789.64	R10.814
Abdominal Tenderness RLQ	789.63	R10.813
Female Menstrual-Related Pain		
Dysmenorrhea	625.3	N94.6
Adenomyosis	617.0	N80.0 – N80.9
Endometriosis	All 617. Codes	
Mittelschmerz	625.2	N94.0
IC Bladder Pain		
Interstitial cystitis	595.1	N30.10

Table 2

Demographics

Characteristic	Full Sample		SEM Sample	
	Mean	Standard Deviation	Mean	Standard Deviation
Body Mass Index (n = 18314)[a]	30.47	6.21	30.78	6.4
Age (years)	46.85	11.76	48.42	11.34
Average Disability Rating[b]	28.09	17.24	29.10	16.18
Patient Health Questionnaire-9	11.41	7.12	12.74	6.60
Primary Care PTSD Screen for DSM-5	1.36	1.86	1.62	1.92
Pain Outcomes Questionnaire-Short Form	101.25	33.20	101.25	33.40
PTSD Checklist for DSM-5	45.66	18.44	44.20	19.69
Insomnia Severity Index	16.81	7.34	15.30	7.44
Mental Component Score	33.27	13.67	33.63	13.92
Physical Component Score	39.26	11.36	37.49	11.46
	n	%	*n*	%
Race (Self-Identified)	18995	100		
AIAN[c]	316	1.66	12	2.17
Asian	248	1.31	7	1.27
Black or African American	7257	38.20	176	31.88
NHOPI[d]	278	1.46	6	1.09
White	10160	53.49	340	61.59
Unknown/Declined	1261	6.64	11	1.99
Ethnicity (Self-Identified)	18874	100		
Hispanic or Latino	2111	11.18	64	11.23
Not Hispanic or Latino	16763	88.82	506	88.77
Self-Identified Gender	4228	100		
Declined	68	1.61	6	4.54
Female	4088	96.69	125	94.70
Male	67	1.58	1	0.76
Other	1	0.02	0	0
Transgender Male	4	0.09	0	0

[a]Outliers removed outside of interquartile range times 1.5. [b]Range 1-100%, with 100% indicating higher disability and impairment. [c]American Indian or Alaska Native. [d]Native Hawaiian or Other Pacific Islander.

Table 3

Correlations of Total Scores (Full Information Maximum Likelihood)

	Insomnia	Depression	PTSD	Pain	MCS	PCS
Insomnia	1.00					
Depression	0.29	1.00				
PTSD	0.48	0.43	1.00			
Pain	0.01	0.33	0.29	1.00		
MCS	-0.34	-0.46	-0.37	-0.67	1.00	
PCS	0.05	-0.02	-0.03	-0.10	-0.04	1.00

MCS = Mental Component Score. PCS = Physical Component Score.

Table 4

Confirmatory Factor Analysis Fit Statistics

	One-Factor Model	Dysphoric Arousal Model	DSM-5 Model	Dysphoria Model	Modified DSM-5 Model
Robust Chi-Square	1850.67	836.13	791.68	778.16	715.01
Degrees of freedom	377	362	362	362	360
Robust Comparative Fit Index	0.70	0.90	0.91	0.91	.93
Robust Tucker-Lewis Index	0.67	0.89	0.90	0.90	0.92
Robust RMSEA [90% CI]	0.09 [0.094, 0.098]	0.05 [0.054, 0.059]	0.05 [0.047, 0.057]	0.05 [0.046, 0.056]	0.05 [0.042, 0.052]
SRMR	0.23	0.05	0.05	0.05	0.05
AIC	26213.83	25115.70	25069.28	25055.95	5601.14

RMSEA = Root Mean Square Error of Approximation. SRMR = Standardized Root Mean Square Residual. AIC = Akaike's Information Criterion.

Table 5

Standardized factor Loadings of DSM-5 Model with Two Depression Factors

	Reexperiencing	Avoidance	NAMC	Hyperarousal	Depression-Somatic	Depression-Non-Somatic
PCL1	0.81					
PCL3	0.75					
PCL4	0.86					
PCL5	0.83					
PCL2	0.70					
PCL6		0.90				
PCL7		0.90				
PCL8			0.46			
PCL9			0.78			
PCL10			0.67			
PCL11			0.80			
PCL12			0.81			
PCL13			0.79			
PCL14			0.80			
PCL17				0.75		
PCL18				0.77		
PCL19				0.77		
PCL15				0.69		
PCL16				0.51		
PCL20				0.64		
PHQ1					0.85	
PHQ2					0.85	
PHQ6					0.72	
PHQ9					0.48	
PHQ5						0.63
PHQ8						0.60
PHQ7						0.70
PHQ3						0.60
PHQ4						0.71

PCL = PTSD Checklist for DSM-5 (PCL-5). PHQ = Patient Health Questionnaire (PHQ-9).

Table 6

Standardized Parameter Estimates of Quality of Life and Pain Models

Model	Outcome	REE	AV	NAMC	HYP	DEP-N	DEP-S	AGE
HYP → QOL	Physical QOL	0[a]	0[a]	0[a]	-0.15	0.158	-0.331	0.02
	Mental QOL				-0.24*	-0.46*	-0.03	0.02*
NAMC → QOL	Physical QOL	0[a]	0[a]	-0.15	0[a]	0.27	-0.44	-0.25*
	Mental QOL			-0.26*		-0.26	-0.22	0.02
RXP → PAIN	Pain	0.29*	0[a]	0[a]	0[a]	0.02	0.13	0.03
	Interference	0.3	0[a]	0[a]	0[a]	-0.14	0.3	0.19*
HYP → PAIN	Pain	0[a]	0[a]	0[a]	0.16	0.09	0.09	0.03
	Interference	0[a]	0[a]	0[a]	0.21	-0.06	0.23	0.18

Cells are populated with standardized parameter estimates for each latent factor (rownames) to each outcome. *p <0.05. [a]Constrained to 0. REE = Reexperiencing. AV = Avoidance. NAMC = Negative Alterations in Mood and Cognition. HYP =Hyperarousal. DEP-N = Depression, Non-Somatic. DEP-S = Depression, Somatic.

Table 7

Robust Fit Statistics of Structural Models

	HYP → QOL	NAMC → QOL	REE → PAIN	HYP → PAIN
Robust Chi-Square	1433.303	1432.430	1264.48	1269.194
Degrees of freedom	797	797	681	681
Robust Comparative Fit Index	0.90	0.90	0.91	0.91
Robust Tucker-Lewis Index	0.90	0.89	0.90	0.90
Robust RMSEA	0.041	0.04	0.043	0.04
SRMR	0.07	0.07	0.07	0.07
AIC	31845.243	31844.67	36488.404	36493.558

REE = Reexperiencing. AV = Avoidance. NAMC = Negative Alterations in Mood and Cognition. HYP =Hyperarousal.

Appendix C: Measures

PTSD Checklist for DSM-5 (PCL-5)

Instructions: Below is a list of problems that people sometimes have in response to a very stressful experience. Please read each problem carefully and then circle one of the numbers to the right to indicate how much you have been bothered by that problem in the past month.

In the past month, how much were you bothered by:	**Not at all**	**A little bit**	**Moderately**	**Quite a bit**	**Extremely**
1. Repeated, disturbing and unwanted memories of the stressful experience?	0	1	2	3	4
2. Repeated, disturbing dreams of the stressful experience?	0	1	2	3	4
3. Suddenly feeling or acting as if the stressful experience were actually happening again (as if you were actually back there reliving it)?	0	1	2	3	4
4. Feeling very upset when something reminded you of the stressful experience?	0	1	2	3	4
5. Having strong physical reactions when something reminded you of the stressful experience (for example, heart pounding, trouble breathing, sweating)?	0	1	2	3	4
6. Avoiding memories, thoughts, or feelings related to the stressful experience?	0	1	2	3	4
7. Avoiding external reminders of the stressful experience (for example, people, places, conversations, activities, objects, or situations)?	0	1	2	3	4
8. Trouble remembering important parts of the stressful experience?	0	1	2	3	4
9. Having strong negative beliefs about yourself, other people, or the world (for example, having thoughts such as: I am bad, there is something seriously wrong with me, no one can be trusted, the world is completely dangerous)?	0	1	2	3	4
10. Blaming yourself or someone else for the stressful experience or what happened after it?	0	1	2	3	4
11. Having strong negative feelings such as fear, horror, anger, guilt, or shame?	0	1	2	3	4
12. Loss of interest in activities that you used to enjoy?	0	1	2	3	4
13. Feeling distant or cut off from other people?	0	1	2	3	4
14. Trouble experiencing positive feelings (for example, being unable to feel happiness or have loving feelings for people close to you)?	0	1	2	3	4
15. Irritable behavior, angry outbursts, or acting aggressively?	0	1	2	3	4
16. Taking too many risks or doing things that could cause you harm?	0	1	2	3	4
17. Being “superalert” or watchful or on guard?	0	1	2	3	4
18. Feeling jumpy or easily startled?	0	1	2	3	4
19. Having difficulty concentrating?	0	1	2	3	4
20. Trouble falling or staying asleep?	0	1	2	3	4

Primary Care PTSD Screen for DSM-5 (PC-PTSD-5)

Sometimes things happen to people that are unusually or especially frightening, horrible, or traumatic. For example:

- a serious accident or fire
- a physical or sexual assault or abuse
- an earthquake or flood
- a war
- seeing someone be killed or seriously injured
- having a loved one die through homicide or suicide.

Have you ever experienced this kind of event?
YES / NO

If no, screen total = 0. Please stop here.

If yes, please answer the questions below.
In the past month, have you...

1. Had nightmares about the event(s) or thought about the event(s) when you did not want to?
   YES / NO
2. Tried hard not to think about the event(s) or went out of your way to avoid situations that reminded you of the event(s)?
   YES / NO
3. Been constantly on guard, watchful, or easily startled?
   YES / NO
4. Felt numb or detached from people, activities, or your surroundings?
   YES / NO
5. Felt guilty or unable to stop blaming yourself or others for the event(s) or any problems the event(s) may have caused?
   YES / NO