Assessing Psychological Pain Among Individuals With Chronic Pain: Invariant Factor Structure
of the Psychache Scale

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Abstract

Psychological pain is a significant risk factor for suicidality across several populations, including among undergraduates and individuals with depression. Chronic physical pain is also associated with increased risk for suicidality, and neuroimaging research suggests that psychologically painful experiences (i.e., rejection, loss) activate neural systems that are involved in physical pain. Despite these overlaps, psychological pain has not previously been examined among individuals with chronic pain. In the current study, we performed a psychometric analysis of a commonly used assessment of psychological pain in a sample of individuals with and without chronic pain. Confirmatory factor analyses and tests of measurement invariance were used to assess the hypothesized one-factor structure of the Psychache Scale and its relationship to depression and pain catastrophizing across individuals with and without chronic pain. Undergraduates ($N = 724$) completed the study, 100 of whom reported chronic pain lasting for at least 3 months (*Mean duration of pain* = 37 months). Results indicated that psychological pain, depression, and pain catastrophizing each loaded onto unique factors that were significantly positively correlated with one another. The model was invariant across the two groups, and individuals with chronic pain reported elevated levels of psychological pain, depression, and pain catastrophizing. These results indicate that the Psychache Scale may be a valuable tool for assessing the psychological component of chronic pain syndromes.

Keywords: Psychological pain; Chronic pain; Depression; Pain catastrophizing; Factor analysis

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Chronic pain is commonly conceptualized as a biopsychosocial phenomenon: biological mechanisms interact with psychological and social factors to exacerbate or attenuate pain (Gatchel, Peng, Peters, Fuchs, & Turk, 2007). In addition to modulating pain, psychological experiences may have the capacity to evoke pain, a phenomenon referred to as *psychological pain*, or *psychache*. In healthy individuals, social rejection and reminders of romantic rejection are associated with activation in the dorsal anterior cingulate cortex and anterior insula. These regions are also implicated in the perception of painful sensory stimuli (Eisenberger & Lieberman, 2004; Fisher, Brown, Aron, Strong, & Mashek, 2010; Kross, Berman, Mischel, Smith, & Wager, 2011). This neural overlap suggests important similarities in the perception of physical and psychological experiences as painful.

Physical and psychological events may share the capacity to evoke *pain affect*. In contrast to the sensory component of pain (i.e., location, quality, and intensity of the physical experience), *pain affect* is defined as the unpleasant feelings associated with a painful experience (MacDonald & Leary, 2005). It is this affective component that is conceptualized as the neural alarm system that motivates rapid withdrawal from pain-inducing stimuli (i.e., a hot stove) and fosters subsequent avoidance of similar stimuli (Price, 2000). An experience that causes bodily harm but does not evoke unpleasant feelings will not be interpreted as painful. Conversely, *pain affect* may occur in absence of physically harmful stimuli, such as in response to loss or social rejection. In this way, psychological experiences may have the capacity to evoke feelings that are perceived as painful. It has been suggested that the neural alarm system evolved to respond to these types of threats because social connections help us avoid threats of a physical nature and

are themselves protective against many diseases and life-threatening experiences (MacDonald & Leary, 2005).

Psychological pain has been primarily studied as a predictor of suicide. Through years of observations of suicidal individuals, Shneidman (1993) coined the term psychological pain, or psychache, to describe the intense affective experience of hurt, anguish, or ache that takes hold of the mind. The construct has been shown to correlate significantly with suicidal ideation and action in samples of undergraduates (DeLisle & Holden, 2009; Flamenbaum & Holden, 2007; Lam, Bond, Chen, & Wu, 2010; Troister & Holden, 2010), depressed patients (Li et al., 2014; Meerwijk, Ford, & Weiss, 2015), bereaved individuals (Robinaugh, LeBlanc, Vuletich, & McNally, 2014), offenders (Pereira, Kroner, Holden, & Flamenbaum, 2010), and homeless men (Patterson & Holden, 2012). Importantly, psychological pain has been found to be more strongly associated with suicidal thoughts and action than well-established suicide risk factors including depression, hopelessness, and perfectionism (Flamenbaum & Holden, 2007; Holden, Mehta, Cunningham, & McLeod, 2001; Troister & Holden, 2010). In a 5-month longitudinal study, change in psychache was significantly associated with change in suicidal ideation, even after including depression and hopelessness as competing predictors (Troister, Davis, Lowndes, & Holden, 2013).

The construct of psychological pain has not previously been examined among individuals with chronic pain. This is surprising, given that the neuroimaging findings discussed above suggest important overlaps in the perception of physical and psychological experiences as painful. Furthermore, risk for suicide is at least doubled among individuals with chronic pain, and the relationship between psychological pain and suicidality is well-established (Hooley, Franklin, & Nock, 2014; Tang & Crane, 2006). Thus, we propose that individuals with chronic

physical pain may also be prone to experience high levels of psychological pain, or *pain affect* associated with psychological experiences.

The goal of the current study was to assess whether the Psychache Scale (PAS; Holden et al., 2001) was suitable for use among individuals with chronic pain, as well as to test the hypothesis that psychological pain would be elevated in this group. Our first aim was to determine whether psychological pain was a unifactorial construct distinct from the constructs of depression and pain catastrophizing, two established correlates of chronic pain and suicidality. We hypothesized that psychache, depression, and pain catastrophizing would represent distinct, yet positively correlated latent constructs in a combined sample of individuals with and without chronic pain. Prior to conducting comparisons across these two groups, we then tested measurement invariance to confirm that the scales were measuring similar constructs across individuals with and without chronic pain. If the measures were invariant across the two groups, we hypothesized that psychological pain, depression, and pain catastrophizing would be elevated among individuals with chronic pain.

Materials and Methods

Participants

Participants were undergraduate students at a U.S. Midwestern university. Students enrolled in Psychology courses were invited to complete surveys online in exchange for course credit. To be included in the current sample, individuals were required to at least partially complete all study measures and be over the age of 18.

Procedure

All research activities were approved by the Washington University in St. Louis Institutional Review Board. Participants completed all study measures online via Experimetricx.

After completion, participants viewed an online debriefing form explaining the nature of the study and providing referrals for psychological resources.

Measures

Physical pain. Physical pain was assessed using the following question, “Are you experiencing recurrent or chronic pain (any pain lasting longer than 3 months from any source)?” Participants who responded “yes” also reported their average pain severity in the past week, worst pain severity in the past week, and current pain severity on a Likert scale of 1 (*Had no pain*) to 5 (*Very severe*).

Psychological pain. The Psychache Scale (PAS; Holden et al., 2001) was used to assess levels of psychological pain. The PAS uses a 5-point Likert scale ranging from 1 (*Never*) to 5 (*Always*). An example item is, “I seem to ache inside.” The PAS has primarily been used to assess psychache in individuals experiencing suicidality (Ducasse et al., 2017), with psychometric testing indicating excellent internal consistency and convergent validity with measures of suicidality and self-injury (Holden et al., 2001). However, to our knowledge, an item-level factor analysis has never been conducted. As such, a 1-factor structure was tested in the current study. The internal consistency of the PAS in this study was excellent ($\alpha = .96$).

Pain catastrophizing. The Pain Catastrophizing Scale (PCS; Sullivan, Bishop, & Pivik, 1995) was used to assess the nature of thoughts about physical pain. The PCS was developed to assess three related components of catastrophizing: rumination (*I can't stop thinking about how much it hurts*), magnification (*I worry that something serious may happen*), and helplessness (*There is nothing I can do to reduce the intensity of the pain*). The PCS uses a 5-point Likert scale ranging from 0 (*Not at all*) to 4 (*All the time*) and an example item is, “I worry all the time about whether the pain will end.” The PCS has been used in several studies assessing perceptions

of pain and has demonstrated excellent internal consistency in community and pain outpatient samples (Osman et al., 2000), as well as undergraduate samples (Osman et al., 1997). The internal consistency of the PCS in this study was excellent ($\alpha = .94$).

Depression. A modified version of the Beck Depression Inventory – II (BDI-II; Beck, Steer, & Brown, 1996) assessed depressive symptom severity. The suicidality item (item 9) was not administered due to concerns about participant safety given that the study was administered online without experimenter oversight. The BDI-II uses a scale from 0 to 3 to assess level of depressive symptoms. The original BDI-II is often considered one of the gold standard assessment tools for measuring depressive symptoms and has demonstrated good to excellent internal consistency (Beck et al., 1996; Steer & Clark, 1997) and good convergent validity (Steer & Clark, 1997). The internal consistency of the version of the BDI-II used in this study was excellent ($\alpha = .94$).

Data Analytic Procedure

Confirmatory factor analysis (CFA) conducted in Mplus 8.1 (Muthén & Muthén, 2017) was used to assess how well the data fit the hypothesized model. The robust weighted least squares estimator (referred to as WLSMV in Mplus) was used as all items were treated as categorical. Additionally, the use of WLSMV helped to improve computational speed (Muthén & Muthén, 2017).

First, Model 1 (Figure 1) was constructed in the full sample to test the hypothesized factor structure. Model 1 was comprised of three latent factors: (a) A psychache factor indicated by all items of the PAS, (b) A catastrophizing factor indicated by three latent sub-factors of rumination, magnification, and helplessness, each indicated by items of the PCS (Van Damme, Crombez, Bijttebier, Goubert, & Van Houdenhove, 2002), and (c) A general depression factor

indicated by all items of the BDI-II, as well as somatic and cognitive factors indicated by a subset of BDI-II items (Ward, 2006). The general depression, somatic, and cognitive factors using the BDI-II items were not allowed to correlate with one another. All factor variances were constrained at 1 for model identification.

Model fit was evaluated using (a) the Tucker–Lewis incremental fit index (TLI; Tucker & Lewis, 1973); (b) the comparative fit index (CFI; Bentler, 1990); and (c) the root mean square error of approximation (RMSEA). The magnitudes of these indices were evaluated with the aid of recommendations by Hu and Bentler (1999), Muthén and Muthén (1998-2017), and Yuan (2000).

Once the factor structure was determined to be appropriate for the full sample, multiple-group CFA was used to test measurement invariance across the non-chronic pain and chronic pain groups of our sample. Tests of measurement invariance (MI; Vandenberg & Lance, 2007) examined whether the PAS, PCS, and BDI-II items functioned similarly in individuals with and without chronic pain. This is a critical step before comparing levels of psychological pain between the two groups, as scales can measure different constructs in different groups (Millsap & Kwok, 2004).

[Figure 1 about here.]

We tested MI by specifying four nested models, each examining a different facet of invariance: (a) In Model 2, we tested configural invariance (i.e., whether the groups had the same general factor structure). This was the least restrictive MI model, wherein loadings and thresholds were allowed to vary between groups. Item residual variances were fixed to 1 in both groups, factor means were fixed to 0, and factor variances were fixed to 1 for model identification. (b) In Model 3, we tested metric invariance (i.e., invariance of item functioning).

We constrained factor loadings to be equal across groups. Thresholds were freely estimated, and item residual variances and factor means remained fixed. Factor variances continued to be held at 1 in the reference (non-chronic pain) group, but were freely estimated in the chronic pain group (Yoon & Millsap, 2007). (c) In Model 4, we tested scalar invariance (i.e., whether the groups had the same thresholds). Both loadings and thresholds were held equal across groups. Item residual variances remained fixed; however, both factor variances and factor means were now freely estimated in the chronic pain group. (d) Finally, we tested residual variance invariance in two nested models. In Model 5a, item residual variances were fixed to 1 in the reference group and freely estimated in the chronic pain group. This model was then compared to Model 5b, in which the item residual variances were held equal across groups.

To evaluate MI, a less constrained model is compared to a more constrained model to evaluate whether the added constraints produce a decrement in model fit. Configural invariance, metric invariance, and scalar invariance were iteratively assessed by comparing model fit to that of the previous model. Residual variance invariance was tested by comparing fit statistics for the freed residuals model to those of the fixed residuals model. According to Chen (2007), noninvariance is indicated by a change of $\leq -.005$ in CFI and $\geq .010$ in RMSEA; that is, a lower CFI and higher RMSEA would indicate worse model fit with added constraints and thus a lack of MI. The chi-square difference statistic was also reported; however, this measure has been shown to be more sensitive to sample size and violation of the normality assumption than CFI and RMSEA (Chen, 2007). As such, only CFI and RMSEA values were considered when determining MI.

After establishing MI, tests of structural invariance (SI) were used to assess differences in how the latent traits are distributed and related across the chronic pain and non-chronic pain

groups. We tested two components of SI: (a) In the factor variance invariance model (Model 6), all factor variances were fixed to 1 in both groups, and model fit was compared to the final residual variance invariance model from the MI procedure (Model 5b). (b) In the factor covariance invariance model (Model 7), the correlations between latent factors were held equal across groups, and model fit was compared to the factor variance invariance model (Model 6). As in tests of MI, a change of $\leq -.005$ in CFI and $\geq .010$ in RMSEA would indicate a lack of structural invariance.

Finally, latent mean differences were assessed to determine whether individuals with and without chronic pain reported different levels of the latent constructs. This was tested by constraining factor means to zero in the non-chronic pain group and allowing means to be freely estimated in the chronic pain group. The difference from zero on the latent means in the chronic pain group provides effect sizes (Cohen's d) for the differences between latent means.

Missing data. The primary source of missing data in the current study was an item of the PAS that was accidentally omitted during initial data collection. Item 5 ("My pain makes my life seem dark") was collected in 48.7% of the full sample (49% of the non-chronic pain group and 44% of the chronic pain group). The WLSMV estimator handled missing data, but other estimators are generally thought to provide better estimation of missing data (e.g., maximum likelihood estimators). Thus, to account for missing data, two models were run with a maximum likelihood estimator that is robust to violations of normality (i.e., the MLR in Mplus; Yuan & Bentler, 2000): (a) the least constrained multi-group model (Model 2) and (b) the most constrained multi-group model (Model 7). To test which model exhibited better fit, we evaluated comparative model fit using the Information Criterion proposed by Akaike (AIC; Akaike, 1974), and the Bayesian Information Criterion (BIC; Schwarz, 1978). Lower values indicated better fit.

Results

Participants

The initial sample consisted of 746 individuals who completed the online surveys. Of this initial sample, 14 individuals were removed for not at least partially completing each of the primary measures. We also included seven validity questions (e.g., *Answer 4 for this question*) in the survey to ensure the accuracy of participant responses. Participants who failed 4 or more of the validity questions ($n = 8$) were excluded from study analyses on the assumption that they were not completing the survey in good faith. The demographics of the final study sample ($N = 724$) are presented in Table 1.

Of the final sample, 100 individuals (13.8%) reported chronic pain lasting longer than three months. Duration of chronic pain ranged from three months to 11 years ($M = 37$ months, $SD = 32$ months). The most commonly reported pain locations were head or neck (39%), back (35%), and joints (22%). Additionally, 41% of individuals with chronic pain reported pain in more than one area. Pain severity questions were added partway through data collection. Thus, these data were available for 87% of the chronic pain sample. Most individuals reported mild (54%, $n = 47$) or moderate (33%, $n = 29$) pain severity in the past week.

[Table 1 about here.]

Observed Mean Differences

Descriptive statistics comparing mean levels of psychache, depression, and pain catastrophizing between individuals with and without chronic pain are displayed in Table 1. The chronic pain group scored significantly higher on all study measures based on observed means (p 's $< .01$). However, as noted above, group differences in observed means can be due to a lack of MI.

Hypothesized Model Fit

We first examined fit of the hypothesized model (Model 1) in the full sample. Model 1 showed excellent fit (CFI = .976, TLI = .974, RMSEA = .040). As hypothesized, psychache, depression, and catastrophizing appeared to represent distinct latent factors, although factors were moderately to highly correlated with one another (psychache and depression $r = .79$, depression and catastrophizing $r = .53$, psychache and catastrophizing $r = .56$). In subsequent analyses, the hypothesized factor structure was used as a baseline model for testing MI across the non-chronic pain and chronic pain groups of our sample.

Measurement Invariance

The results of MI are displayed in Table 2. The hypothesized model was used in all multi-group analyses. Three alterations were undertaken to allow convergence, including rescaling of items between groups, estimating a single factor (with no subfactors) for the PCS, and, in the case of Model 2 only, constraining a correlation due to initial nonconvergence. Details on these decisions are available from the first author.

As can be seen in Table 2, the successive model constraints did not produce decrements in model fit above the cutoffs suggested by Chen (2007). Constraints on factor structure (Model 2), factor loadings (Model 3), item thresholds (Model 4), and residual variances (Model 5) did not substantively worsen model fit. Thus, the model was found to exhibit full MI between the non-chronic pain and chronic pain groups of our sample.

[Table 2 about here.]

Structural Invariance

The model also demonstrated structural invariance; that is, constraints on factor variance (Model 6) and covariance (Model 7) did not worsen model fit. In the final model (Model 7;

Figure 2), psychache, depression, and catastrophizing continued to represent distinct latent factors that were moderately to highly correlated with one another (psychache and depression $r = .79$, depression and catastrophizing $r = .50$, psychache and catastrophizing $r = .53$).

[Figure 2 about here.]

Latent Mean Differences

Based on the results described above, the PAS, BDI-II, and PCS demonstrated full metric invariance between the non-chronic pain and chronic pain groups of our sample. As such, latent factor means can be compared for differences across the two samples on levels of each symptom. These differences were assessed using Model 7, in which latent means were set to zero in the non-chronic pain group and freely estimated in the chronic pain group. In the chronic pain group, latent mean values were significantly different from zero for psychache, catastrophizing, depression (general), and somatic symptoms (all p 's $< .005$). Results showed higher latent mean values of somatic symptoms ($d = 0.54$), catastrophizing ($d = 0.53$), depression ($d = 0.46$), and psychache ($d = 0.35$) for the chronic pain group as compared to the non-chronic pain group.

Demographic Differences

As can be seen in Table 1, the chronic pain and non-chronic pain groups significantly differed in terms of race and gender. Thus, we conducted a post-hoc test of whether chronic pain is uniquely related to observed mean differences, above and beyond these demographic differences. Chronic pain status, gender, and race were included in regression analyses predicting total scores on the PAS, BDI-II, and PCS. Chronic pain remained a significant predictor of PAS, BDI-II, and PCS scores above and beyond gender and race.

Missing Data

Finally, to determine whether model results might be different if a better missing data estimator were used, we examined model fit across the MI sequence of models. Using an MLR estimator, Model 7 (AIC = 52183.53; BIC = 53123.41) exhibited better fit than Model 2 (AIC = 52275.38; BIC = 54054.28). Thus, we concluded that missing data did not substantively impact the findings above in terms of preference for Model 7.

Discussion

Psychological pain is commonly associated with risk for suicidality, which is elevated among individuals with chronic pain. The current study confirms the one-factor structure of Holden's (2001) measure of psychological pain and demonstrates the scale functions similarly in chronic pain and non-chronic pain samples. Furthermore, individuals with chronic pain reported higher levels of psychological pain than individuals without chronic pain. This study is a critical first step in examining the role of psychological pain in predicting adverse psychological outcomes among individuals with chronic pain. Specifically, increased psychological pain may help explain the complex relationship between chronic pain and suicidality.

We also sought to determine whether psychological pain represents a construct that is related to, yet distinct from other psychological components of chronic pain syndromes. In the current study, psychological pain was positively correlated with both pain catastrophizing ($r = .56$) and depression ($r = .79$). Although the correlation between depression and psychological pain was high, it remains below the .85 benchmark indicating poor discriminant validity of latent variables (Brown, 2006). Furthermore, when factor covariances were allowed to differ between the two groups, the chronic pain group had a lower correlation between depression and psychological pain ($r = .62$) than the non-chronic pain group ($r = .81$). Thus, psychological pain does appear to be a construct unique from depression and pain catastrophizing. This suggests that

psychological pain may be an important component of chronic pain syndromes, above and beyond other well-established psychological risk factors.

A strength of the current study was the use of tests of measurement invariance to assess whether the PAS functions similarly across undergraduates with and without chronic pain. If the scale did not measure the same construct across the two groups, it would be difficult to make meaningful comparisons between individuals with and without chronic pain. However, given that the measure was invariant across these two groups, we are able to conclude that psychological pain is elevated among our sample of undergraduates with chronic pain. These findings suggest that the PAS is a suitable tool for assessing psychological pain among individuals with chronic pain.

In addition to its strengths, the current study had several limitations. A primary limitation was that our sample is comprised of undergraduates. As such, our participants were younger and experienced a shorter duration and lesser severity of chronic pain than many community-dwelling individuals with pain disorders. Given that pain severity is positively associated with depressive symptom severity (Bair, Wu, Damush, Sutherland, & Kroenke, 2008; Gore et al., 2005; Von Korff, Ormel, Keefe, & Dworkin, 1992), and depression and psychological pain were positively correlated in the current study, we would expect psychological pain to be even higher among a community sample with more severe chronic pain.

Although we collected a large sample overall, our chronic pain sample was also relatively small for the analyses performed. Future research is needed using data from larger samples of community-dwelling patients with chronic pain. Additionally, the current study did not assess adverse psychological outcomes, such as suicidality, due to the unmonitored nature of the online

surveys. Thus, future research should assess whether psychological pain is predictive of suicidality among individuals with chronic pain, as it is in other populations.

Further research is also needed to examine whether physical pain and psychological pain are indeed distinct constructs among individuals with chronic pain. Initially, Shneidman (1993) coined the term *psychological pain* to refer generally to emotional states that are so negative and distressing that they become painful. More recently, the neuroimaging literature has suggested that psychological or emotional pain refers to *pain affect* caused by non-physical stimuli. What remains to be tested is whether individuals with chronic pain distinguish *pain affect* stemming from physical vs. emotional stimuli. If these experiences are reported as distinct symptoms, one could then examine how the two experiences relate to one another over time (i.e., does psychological pain predict physical pain, or visa versa?). If they are largely overlapping constructs, such a finding may also elucidate an important pathway through which chronic pain becomes comorbid with depression and suicidality.

In summary, we performed a psychometric analysis of the PAS among individuals with and without chronic pain. Results from the current study confirm the one-factor structure of the PAS and demonstrate that this factor structure is invariant across individuals with and without chronic pain. Furthermore, psychological pain appears to be elevated among individuals with chronic pain, and positively correlated with two previously established risk factors for suicidality in this population: depression and pain catastrophizing. Given these findings, future research should examine psychological pain as a predictor of suicidality among individuals with chronic pain. Further research is also necessary to examine the nature of psychological pain as it is related to and distinct from the experience of chronic physical pain.

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Table 1

Demographics and Descriptive Statistics of Study Measures

Characteristic	No chronic pain (<i>n</i> = 624)	Chronic pain (<i>n</i> = 100)	χ^2 or <i>t</i>
Age (M, <i>SD</i>)	19.2 (1.2)	19.2 (1.3)	.67
Gender (N, %)			10.78*
Male	228 (36.6%)	26 (26%)	
Female	395 (63.3%)	72 (72%)	
Other	1 (<1%)	2 (2%)	
Race (N, %)			13.69*
White	369 (59.1%)	68 (68%)	
Black or African-American	54 (8.7%)	16 (16%)	
Asian	189 (30.3%)	15 (15%)	
Native Hawaiian or Other Pacific Islander	3 (<1%)	0 (0%)	
Unknown/other	9 (1.4%)	1 (1%)	
Ethnicity (N, %)			.49
Not Hispanic or Latino	564 (90.4%)	91 (91%)	
Hispanic or Latino	57 (9.1%)	9 (9%)	
Unknown/other	3 (<1%)	0 (0%)	
PAS (M, <i>SD</i>)	19.7 (8.7)	22.7 (10.5)	2.72*
BDI-II (M, <i>SD</i>)	9.1 (7.7)	12.9 (9.1)	3.93*
PCS (M, <i>SD</i>)	13.1 (10.4)	18.7 (11.4)	4.67*

Note. BDI-II = Beck Depression Inventory-II; PAS = Psychache Scale; PCS = Pain

Catastrophizing Scale. Item 9 (suicidality item) of the BDI-II was not administered due to the unmonitored nature of online surveys. Item 5 of the PAS (“My pain makes my life seem dark”) was also added partway through data collection. As such, this table reflects a 20-item version of the BDI-II and a 12-item version of the PAS.

* $p < .01$

Table 2

Results of Confirmatory Factor Analyses (CFA)

Model	CFI	TLI	RMSEA	χ^2 (df)	<i>p</i>
1. Full sample CFA	.976	.974	.040		
2. Configural invariance	.976	.975	.039		
3. Metric invariance	.981	.980	.034	71.35 (51)	.03
4. Scalar invariance	.981	.981	.033	156.40 (123)	.02
5. Residual variance invariance					
a. Variances freed	.979	.979	.035		
b. Variances fixed	.981	.981	.033	76.87 (46)	.003
6. Factor variance invariance	.983	.984	.031	2.22 (5)	.82
7. Factor covariance invariance	.987	.987	.027	11.06 (7)	.14

Note. CFI = Comparative fit index; TLI = Tucker–Lewis incremental fit index; RMSEA = Root mean square error of approximation; df = Degrees of freedom.

Figure 1

Simplified depiction of Model 1 fit for the full sample. This model served as a baseline model for testing Measurement Invariance. All paths displayed were significant ($p < .01$)^a.

Note. BDI-II = Beck Depression Inventory-II; PAS = Psychache Scale; PCS = Pain Catastrophizing Scale. Two significant paths are not displayed here for simplicity: the BDI-II Cognitive factor was significantly correlated with Pain Catastrophizing, and the BDI-II Somatic factor was significantly correlated with Psychological Pain (p 's < .01).

^aBDI-II items 6 and 8 did not load significantly onto the BDI-II Cognitive factor.

Figure 2

Simplified depiction of Model 7 (full metric invariance with factor covariance and variance also held equal between groups). Latent means were held at zero in the non-chronic pain group.

Effect sizes indicate the difference from zero in the chronic pain group. All paths displayed were significant ($p < .05$)^a.

Note. BDI-II = Beck Depression Inventory-II; PAS = Psychache Scale; PCS = Pain

Catastrophizing Scale. Two significant paths are not displayed here for simplicity: the BDI-II Cognitive factor was significantly correlated with Pain Catastrophizing, and the BDI-II Somatic factor was significantly correlated with Psychological Pain (p 's $< .01$).

^aBDI-II item 6 did not load significantly onto the BDI-II Cognitive factor.