Pain Catastrophizing Mediates and Moderates the Link Between Acute Pain and Working Memory

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Abstract

The bidirectional relationship between pain and working memory (WM) deficits is well-documented but poorly understood. Pain catastrophizing — exaggerated, negative cognitive and emotional responses toward pain — may contribute to WM deficits by occupying finite, shared cognitive resources. The present study assessed the role of pain catastrophizing as both a state-level process and trait-level disposition in the link between acute pain and WM. Healthy, young adults were randomized to an experimentally-induced ischemic pain or control task, during which they completed verbal and non-verbal WM tests. Participants also completed measures of state- and trait-level pain catastrophizing. Simple mediation analyses indicated that participants in the pain group (vs. control) engaged in more state-level catastrophizing about pain, which led to worse verbal and non-verbal WM. Moderated mediation analyses indicated that the indirect (mediation) effect of state-level pain catastrophizing was moderated by trait-level pain catastrophizing for both verbal and non-verbal WM. Participants in the pain group who reported a greater trait-level tendency to catastrophize about pain experienced greater state-level catastrophizing about pain during the ischemic task, which led to worse verbal and non-verbal WM performance. These results provide evidence for pain catastrophizing as an important mechanism and moderating factor of WM deficits in acute pain. Future research should replicate these results in chronic pain samples, investigate other potential mechanisms (e.g., sleep disturbances), and determine if interventions that target pain catastrophizing directly can ameliorate cognitive deficits in people with pain.

Perspective: This article presents a laboratory study examining the relationships among pain, pain catastrophizing, and working memory in healthy participants. The results shed new light on

these relationships and raise the possibility that interventions that reduce catastrophizing may lead to improved cognitive function among people with pain.

Introduction

People with chronic pain frequently experience deficits in working memory (WM) and other cognitive domains [6,33,36,48,57,65,77]. Similar cognitive deficits have been observed in healthy individuals undergoing experimental pain tasks [9,54]. While the pain sensation itself is an important contributor to these deficits, the cognitive and affective correlates are also critical, but poorly understood [17,33,75]. In particular, pain catastrophizing[1]—a cognitive and emotional process that frequently involves ruminating, magnifying, and feeling helpless about pain—may contribute to WM deficits by competing for the same finite pool of cognitive resources, but this has not been systematically tested [63,70].

Pain catastrophizing can be conceptualized as both a state-level process of catastrophizing about pain in the moment and a trait-level tendency to catastrophize about pain in general [12,24,61,68]. Experimental research with healthy individuals has indicated that both types of catastrophizing are correlated with more intense pain [12,24,71,72]. Clinical studies have found that trait-level pain catastrophizing is associated with greater disability and reduced occupational functioning [43,71,72]. State-level pain catastrophizing is a demonstrated mediator between pain and relevant clinical outcomes, predicting greater depressive symptoms and negative affect, as well as lower positive affect [68]. Moreover, trait-level pain catastrophizing moderated each of these mediations, amplifying their effects for people who reported higher general tendency to catastrophize about pain [68].

The evidence linking catastrophizing, pain, and affect notwithstanding, relatively little is known about the roles of state-level and trait-level pain catastrophizing in other important

[1] The authors would like to acknowledge the growing dissatisfaction with the term “catastrophizing” and the ongoing efforts to find a new patient-centered term. However, given an alternative term has not yet been established in the literature, we use the term “catastrophizing” for this paper.

domains of functioning, particularly as mediators and moderators in the pain—WM nexus [26,43,65]. Moreover, the few studies that did examine the relationship between pain catastrophizing and WM analyzed them as parallel predictors of pain outcomes, used correlational designs that do not allow for drawing strong causal inferences, used various measures that differ in their conceptualization of pain catastrophizing (e.g., Pain Catastrophizing Scale vs. Coping Strategies Questionnaire), and failed to differentiate between state- and trait-level pain catastrophizing [26,43,65]. Not surprisingly, these methodological limitations have yielded conflicting results [3,21,26,37,43,51,65].

To resolve these issues, the current study utilized an experimental design to investigate the mediating and moderating roles of state- and trait-level pain catastrophizing in pain-related WM deficits. Healthy, pain-free adults were randomized to an experimentally-induced ischemic pain group or a no-pain control group. Participants also completed well-established measures of verbal and non-verbal WM and state- and trait-level pain catastrophizing. First, based on prior studies [6,9,12,24,33,36,48,53,57,65,71,72,77], we hypothesized that the pain group would experience greater state-level catastrophizing, but worse verbal and non-verbal WM, compared to the control group. Second, we hypothesized that the relationships between group (pain vs. control) and both verbal and non-verbal WM would be mediated by state-level pain catastrophizing, such that the pain group's greater catastrophizing during the ischemic task would lead to worse WM performance. This hypothesis is derived from prior research and theory implicating catastrophizing as a candidate mechanism linking pain to cognitive deficits [26,43,65]. Third, we hypothesized that trait-level pain catastrophizing would moderate the mediation pathways for both verbal and non-verbal WM, with stronger mediation effects for participants in the pain group who reported greater trait-level pain catastrophizing. This

hypothesis is based on results of a similar moderated mediation model in which state- and trait-level catastrophizing predicted mood-based outcomes in patients with chronic pain [68]. It is also grounded in response style theory linking dispositional emotional vulnerabilities to in-the-moment emotional reactions to stressors [55].

Methods

This study was approved by the Indiana University-Purdue University Indianapolis (IUPUI) institutional review board (IRB).

Participants

Participants were 102 healthy adults (52 female, 50 male) enrolled at a large, urban, Midwestern university in the U.S. in Spring 2019. Exclusion criteria were (a) current chronic pain, circulatory system problems, hypertension or high blood pressure, diabetes, or pregnancy; (b) serious injuries to the non-dominant hand within the prior year; and (c) history of serious mental illness (i.e., autism spectrum disorders, schizophrenia or other psychotic disorder, bipolar disorder, or major depressive disorder featuring suicidal ideation) or serious heart/cardiovascular disease. Eligible participants were instructed to avoid analgesic medications within 24 hours and alcohol, tobacco, nicotine, and caffeine products within two hours of their scheduled appointments.

Measures

All measures were completed in the research laboratory on a desktop computer through the Qualtrics online survey software [60].

Demographic Information

Participants reported their age, sex, race, ethnicity, marital status, annual income, work status, student status, college major, current cumulative grade point average (GPA), and current

major GPA. They were also asked to “Rate your level of personal experience (i.e., your own prior experiences) with chronic pain (i.e., lasting longer than 3 months)” on a 4-point Likert-type scale (none, minimal, some, much).

Pain Catastrophizing

Trait-level pain catastrophizing was assessed using the Pain Catastrophizing Scale (PCS), a 13-item scale of commonly experienced thoughts or feelings related to pain [69,70]. The PCS is composed of 13 items, each rated on a 5-point scale, ranging from “not at all (0)” to “all the time (4).” These item scores are summed to produce a total score ranging from 0 to 52, with higher scores indicating greater degrees of trait-level pain catastrophizing. The PCS has demonstrated high internal consistency in both community (Cronbach’s alpha = 0.95) and outpatient chronic pain (Cronbach’s alpha = 0.92) samples [58]. The total score was analyzed in the current study and had high internal consistency (Cronbach’s alpha = 0.92).

State-level pain catastrophizing was assessed using the Situational Catastrophizing Questionnaire (SCQ). The SCQ was adapted from the PCS for use in experimental pain research [12,24]. It consists of 6 items to assess state-level pain catastrophizing during or immediately following experimentally-induced pain. The SCQ uses the same 5-point scale of the PCS and the items are summed to yield scores ranging from 0 to 24, with higher scores indicating greater degrees of state-level pain catastrophizing. The SCQ has demonstrated high internal consistency for healthy (Cronbach’s alpha = 0.87) and chronic pain samples (Cronbach’s alpha = 0.81-0.94) and also in the current sample (Cronbach’s alpha = 0.96) [12].

Mood

The GAD-7 asks respondents to rate how often they have been bothered by seven anxiety-related problems over the prior two weeks. The GAD-7 was developed for the assessment of generalized anxiety, for which it has high sensitivity (89%), specificity (82%), and internal consistency (Cronbach's alpha = 0.92) [67]. The GAD-7 has been shown to reliably predict functional impairments and disability [44,67]. The GAD-7 demonstrated high internal consistency (Cronbach's alpha = 0.83) in the current study.

The Patient Health Questionnaire-8 (PHQ-8) was used to assess recent (e.g., two weeks) depressive symptoms. The PHQ-8 is identical to the widely-used PHQ-9, except for the exclusion of the single item about suicidal ideation and self-harm [39,40]. The PHQ-8 has demonstrated similar internal consistency and utility to the PHQ-9 in screening for depression (Cronbach's alpha = 0.89 & 0.88, respectively) [15,39,40,64]. The PHQ-8 demonstrated high internal consistency (Cronbach's alpha = 0.82) in the current sample.

Participants responded to items on the GAD-7 and PHQ-8 using the same scale ranging from "Not at all (0)" to "Nearly every day (3)." Scores were summed, yielding a range from 0 to 24 and 0 to 21, respectively, with higher scores indicating greater symptom severity.

Task Demands

The NASA Task Load Index (NASA-TLX), which evaluates the workload of a current or recently completed task, was used to assess the demands of the WM task [29,30]. Workload is composed of six dimensions: mental, physical, and temporal demands; performance; effort; and frustration. All six dimensions were rated on 7-point scales with 21 gradations, ranging from "very low" to "very high," except performance, which was rated from "perfect" to "failure."

Pain Intensity

A 100-point visual analogue scale (VAS) was located on a 10-inch strip of paper below the computer monitor in front of participants. Participants used this VAS as a reference to verbally indicate their pain intensity throughout the pain task.

Working Memory Tasks

Verbal Working Memory

A backwards digit span task was used to measure participants' verbal WM [6,48,65]. For each trial, a researcher read aloud sequences of numbers at a pace of one digit per second. After the last digit of each trial, participants attempted to verbally recall the digits in reverse order. Trials were grouped into pairs with equal digit-lengths, beginning at two digits. Participants attempted both trials of a given pair, but only proceeded to the next pair of trials if they successfully recalled all digits backwards for at least one trial of the pair. Each subsequent pair was one digit longer than the previous pair, up to a maximum of nine digits. The task ended when participants were unable to complete both trials of a pair or when they completed the final nine-digit pair. Each successful trial was scored at one point, the number of trials correctly recalled were summed (maximum of 16), and this sum was multiplied by the number of digits in the longest correct sequence (maximum of 9) to compute a final product score (maximum of 144), with higher scores indicating better verbal WM. Product scoring emphasizes consistently correct responses, is more statistically reliable, and provides a greater range of scoring compared to either the sum of total correct trials or the longest correct sequence individually [56]. Between each trial, participants were asked to verbally rate their pain using the VAS.

Non-Verbal Working Memory

A computerized version of the Corsi block-tapping task was used to measure non-verbal, visuospatial WM [38]. A desktop computer displayed nine boxes, which individually illuminated in a pre-set order. After viewing a given sequence of flashing boxes, participants used a mouse to click on the boxes in the same order that they were presented. Participants clicked “Done” to confirm their response or “Reset” to change their response, but the original stimulus was not re-presented. After clicking “Done” to finish each trial, but before the next trial began, a message appeared on the screen prompting participants to verbally rate their pain using the VAS.

Similar to the backwards digit span, the trials were grouped into pairs of sequences with equal numbers of illuminated boxes, beginning with two boxes per sequence. Participants attempted both trials of a given pair and proceeded to the next pair only if they successfully completed at least one trial of each pair. Each subsequent pair of trials had one additional box than the previous pair, up to a nine-box maximum, after which the task ended. If participants did not correctly complete either trial for a given pair, the test was discontinued. Each successful trial was scored at one point, the number of trials correctly recalled were summed (maximum of 16), and this sum was multiplied by the number of boxes in the longest correct sequence (maximum of 9) to compute a final product score (maximum of 144), with higher scores indicating better non-verbal WM. Similar to the approach used for the backwards digit span, product scoring for the Corsi block-tapping task is more statistically reliable and provides a greater range of scoring than approaches using the total correct trials or the longest correct sequence individually [38,56]. Participants verbally rated their pain using the VAS between trials.

Procedures

A flow chart of study procedures is depicted in Figure 1. All procedures were completed in a single session. We collected data on participants' total time in task (see below); however, we did not collect data on the amount of time participants spent completing each separate task.

Baseline

After providing informed consent and affirming that they avoided analgesic medications, alcohol, tobacco, nicotine, and caffeine as instructed during screening, participants completed the demographic questionnaire, PCS, GAD-7, and PHQ-8. Then, participants were randomly assigned to either a pain group or a no-pain control group, equally balanced by sex, using a block randomization paradigm. Participants were not explicitly told their group assignment. Each group was instructed in their respective pain/control tasks, the two WM tasks, and the use of the VAS for pain ratings.

Ischemic Pain Induction

Based on the protocol described by Dannecker & George (2009), a submaximal effort tourniquet test (SETT) was used to induce ischemic pain with a Medline Standard Handheld Aneroid Sphygmomanometer (BP cuff) and CAMRY Digital Hand Dynamometer (handgrip dynamometer) [19]. Participants' non-dominant hands were used throughout, and any jewelry, wristwatches, or other accessories were removed from that arm. To begin, a handgrip dynamometer was used to determine participants' maximum grip strength. Next, an uninflated BP cuff was placed on their bicep above the elbow. Participants were then instructed to lift their arm directly above their head for 30 seconds to desanguinate venous blood.

For participants in the pain group, after 30 seconds elapsed, the BP cuff was inflated to 260 mmHg, and participants slowly lowered their arm to a resting position on the armrest of their

chair. Timing began the moment their arm came to rest. Then, while the BP cuff was still inflated, participants performed 20 gripping repetitions (reps) with the handgrip dynamometer while the researcher maintained the BP cuff pressure to 230-250 mmHg. Participants gripped and held the dynamometer at 50% of their maximum grip strength for two seconds, and then released and relaxed for two seconds before gripping again. After completing the 20 gripping reps, the dynamometer was removed and participants were instructed to say “pain” upon first feeling pain in their arm (i.e., pain threshold) and verbally rate their pain using the VAS. The inflated BP cuff was adjusted as necessary to maintain pressure at 230-250 mmHg. Participants were asked to remain in the task for as long as possible, and to say “stop” when they could no longer endure the pain, at which time they were asked for a final VAS pain rating. At this point, timing was stopped (recorded as “total time in task”), and the BP cuff was deflated and removed. A maximum time cut-off of 15 minutes was used (but not communicated to participants), upon which the BP cuff was removed, and participants gave a final VAS pain rating.

Participants in the control group underwent identical procedures (including VAS ratings), with two exceptions: (1) the BP cuff remained on their arm but was not inflated, and (2) they did not perform any gripping exercises beyond the initial measurement of maximum grip strength. As in the pain group, “total time in task” was determined by the moment participants’ lowered their arm until they said “stop” or 15 minutes had elapsed.

Working Memory Assessment

After all gripping reps were completed and while the BP cuff was still inflated, participants in the pain group completed the two WM tasks, which were counterbalanced. For participants in the control group, these counterbalanced WM tasks were initiated while the deflated BP cuff was still on the arm. If participants said “stop” at any point during the WM

tasks, the BP cuff was removed as previously indicated, and the WM tasks continued. For the remaining participants, after completing both WM tasks, they were asked to sit quietly for as long as they could endure (or until the 15-minute limit was reached), and were reminded that they could discontinue at any time by saying "stop." At the conclusion of the task, the BP cuff was removed.

Post-Task

Following the aforementioned tasks, all participants completed the SCQ and two versions of the NASA-TLX, one for each WM task. Finally, participants were granted course credit for completing the study and the session was ended.

Statistical Analyses

Chi-square and independent samples t-tests were used to examine differences between the pain and control groups on demographic variables (randomization check), final pain VAS ratings, PCS and SCQ total scores, NASA-TLX dimensions, and WM performance. An alpha level of .01 was used to adjust for the multiple between-group comparisons. Bootstrapping mediation procedures with 10,000 resamples and Hayes' PROCESS macro with model 4 for SPSS were used to examine the degree to which state-level pain catastrophizing (SCQ scores) mediated the relationships between pain group and performance on each WM task [14,31]. Significant mediation was inferred when the 95% confidence interval (CI) did not include zero. Cohen effect size benchmarks were used to assess the mediation effects [13,41]. We then repeated these mediation analyses with the addition of anxiety (GAD-7) and depression (PHQ-8) as parallel mediators alongside state-level pain catastrophizing (SCQ). Neither anxiety nor depression were significant in the model. Moreover, results of the two mediation models (with

and without depression and anxiety) were equivalent. As such, in the interest of parsimony, only state-level pain catastrophizing was included as a mediator in the subsequent moderated mediation analyses.

Next, the procedure described in Preacher, Rucker, & Hayes (2007) and Hayes' PROCESS macro with model 7 for SPSS tested whether trait-level pain catastrophizing (PCS scores) moderated the aforementioned mediating effect of state-level pain catastrophizing (SCQ scores) on the relationship between pain group and performance on each WM task [31,59]. Both raw and transformed z-scores were calculated, as standardized coefficients are not available in PROCESS model 7 [31,59]. Significant moderated-mediation was inferred by p-values less than .05 and/or 95% CIs that did not include zero. In the event of significant effects, the Johnson-Neyman method was used to determine at what level of the moderator (PCS), if any, the interaction between pain group and PCS transitioned to significance or non-significance [31]. In the event of no identified transition points – i.e., the interaction was significant at all levels of the moderator – we computed the conditional indirect effect (i.e., conditional mediation) with regression coefficients and 95% CIs at the mean of the moderator and one standard deviation above and below it. Cohen effect size benchmarks were also used to assess the moderated mediation effects [13,41].

Results

Sample Characteristics

Three participants from the pain group discontinued the ischemic task (i.e., said "stop" before 15 minutes had elapsed) prior to finishing the WM tasks. All of the planned analyses were conducted twice – once with these three participants included and once without them included.

The results of all paired analyses were equivalent. Therefore, to retain the most amount of data, we included these three participants in our final sample.

The final sample consisted of 102 participants (52 females and 50 males), with a mean age of 20.1 (SD = 3.7) years. Participants most frequently identified as White/Caucasian (n = 68, 66.7%), Black/African American (n = 10, 9.8%), or Asian/Pacific Islander (n = 7, 6.9%), and most (n = 80, 78.4%) were not of "Hispanic or Latino or Spanish origin." Further sample characteristics are reported in Table 1.

Group Differences

The results of chi-square and t-tests indicated that the pain and control groups did not significantly differ on any of the demographic variables (all p values > .05; Table 1). There were no significant group differences in PCS (trait-level catastrophizing), GAD-7, PHQ-8, backwards digit span, or Corsi block-tapping scores (all p values > .05; Table 2). As expected, the pain group reported significantly higher pain ratings ($t(100) = -21.27$, $p < .001$) and SCQ scores (state-level catastrophizing; $t(100) = -10.79$, $p < .001$) than the control group (Table 2). The pain group also rated the backwards digit span and Corsi block tapping tasks as significantly more demanding across most, but not all, of the NASA-TLX domains than did the control group (Supplementary Table 1).

Mediation Analyses

We first tested the parsimonious model that only included state-level pain catastrophizing (SCQ) as a mediator (Figure 2, Panel A). These analyses identified a significant effect of group (pain vs. control) on state-level pain catastrophizing (path a; $\beta = 1.46$, $p < .001$) and of state-level pain catastrophizing on verbal WM (path b; $\beta = -.35$, $p = .018$). There was also a significant indirect effect of group on verbal WM through state-level pain catastrophizing (path ab; $\beta = -.51$,

95% CI: [-.90, -.18]). The pain group reported greater state-level pain catastrophizing than the control group, which was associated with worse performance on the verbal WM task. Detailed results are presented in Supplementary Table 2.

Next, we added anxiety (GAD-7) and depression (PHQ-8) to the model as parallel mediators alongside state-level pain catastrophizing (SCQ) (Figure 3, Panel A). These analyses identified a significant effect of group (pain vs. control) on state-level pain catastrophizing (path a; β = 1.46, p < .001) but not on anxiety (path a; β = .14, p = .487) or depression (path a; β = -.03, p = .890). Moreover, there was a significant effect of state-level pain catastrophizing on verbal WM (path b; β = -.33, p = .022), whereas neither anxiety (path b; β = .27, p = .052) nor depression (path b; β = -.26, p = .069) had significant effects on verbal WM. The total indirect effect (β = -.44, 95% CI: [-.87, -.08]) and the indirect effect of state-level pain catastrophizing (β = -.49, 95% CI: [-.89, -.16]) were both significant, but the indirect effects of anxiety (β = .04, 95% CI: [-.08, .19]) and depression (β = .01, 95% CI: [-.12, .14]) were not. The pain group reported greater state-level pain catastrophizing than the control group (controlling for anxiety and depression), which was associated with worse verbal WM performance. By contrast, there were not similar mediating effects for anxiety or depression. Detailed results are presented in Supplementary Table 3.

Similarly, when state-level pain catastrophizing was included as the sole mediator in the model (Figure 2, Panel B), mediation analyses found significant effects of pain group on state-level pain catastrophizing (path a; β = 1.46, p < .001) and of state-level pain catastrophizing on non-verbal WM (path b; β = -.38, p = .008), as well as a significant indirect effect of pain group on non-verbal WM through state-level pain catastrophizing (path ab; β = -.56, 95% CI: [-1.00, -.16]). The pain group reported greater state-level pain catastrophizing than the control group,

which was associated with worse non-verbal WM performance. Detailed results are presented in Supplementary Table 4.

When anxiety (GAD-7) and depression (PHQ-8) were added to the model as parallel mediators (Figure 3, Panel B), there was a significant effect of group (pain vs. control) on state-level pain catastrophizing (path a; $\beta = 1.46$, $p < .001$), but not on anxiety (path a; $\beta = .14$, $p = .487$) or depression (path a; $\beta = -.03$, $p = .890$). Moreover, there was a significant effect of state-level pain catastrophizing on non-verbal WM (path b; $\beta = -.41$, $p = .005$), but neither anxiety (path b; $\beta = .14$, $p = .307$) nor depression (path b; $\beta = -.01$, $p = .959$) had significant effects on non-verbal WM. The total indirect effect ($\beta = -.58$, 95% CI: [-1.01, -.18]) and the indirect effect of state-level pain catastrophizing ($\beta = -.60$, 95% CI: [-1.01, -.22]) were both significant, but the indirect effects of anxiety ($\beta = .02$, 95% CI: [-.05, .13]) and depression ($\beta = .00$, 95% CI: [-.08, .07]) were not. The pain group reported greater state-level pain catastrophizing than the control group (controlling for anxiety and depression), which was associated with worse non-verbal WM performance. Anxiety and depression did not show similar mediating effects. Detailed results are presented in Supplementary Table 5.

Moderated Mediation Analyses

A significant index of moderated mediation was found for verbal WM (standardized index = -.10, 95% CI: [-.24, -.01]), indicating that the indirect effect of state-level pain catastrophizing was moderated by trait-level pain catastrophizing (Figure 4, Panel A). The results of a Johnson-Neyman analysis showed that this conditional indirect effect was significant at all levels of the moderator, including at the mean (PCS = 11.14, standardized conditional indirect effect= -.50, 95% CI: [-.93, -.16]) and one standard deviation above (PCS = 19.50, standardized conditional indirect effect = -.60, 95% CI: [-1.13, -.19]) and below (PCS = 2.78, standardized

conditional indirect effect = -.40, 95% CI: [-.78, -.12]) the mean (Table 3). Participants in the pain group who reported higher (vs. lower) trait-level pain catastrophizing experienced greater state-level pain catastrophizing during the ischemic task, which led to worse verbal WM performance.

Similarly, a significant index of moderated mediation was found for non-verbal WM (standardized index = -.11, 95% CI: [-.24, -.02]), again indicating that the indirect effect of state-level pain catastrophizing was moderated by trait-level pain catastrophizing (Figure 4, Panel B). A Johnson-Neyman analysis indicated that this conditional indirect effect was significant at all levels of the moderator, including at the mean (PCS = 11.14, standardized conditional indirect effect = -.56, 95% CI: [-1.00, -.15]) and one standard deviation above (PCS = 19.50, standardized conditional indirect effect = -.67, 95% CI: [-1.16, -.18]) and below (PCS = 2.78, standardized conditional indirect effect = -.45, 95% CI: [-.88, -.11]) the mean (Table 4). Similar to verbal WM, participants in the pain group who reported higher (vs. lower) trait-level pain catastrophizing experienced greater state-level pain catastrophizing during the ischemic task, which led to worse non-verbal WM performance.

Discussion

We investigated pain catastrophizing in the pain—WM nexus. Participants in the pain group reported greater state-level catastrophizing than participants in the control group, which was associated with poorer verbal and non-verbal WM. Moderated-mediation analyses indicated that the mediating effect of state-level catastrophizing was potentiated among participants in the pain group who reported higher (vs. lower) trait-level catastrophizing.

Hypothesis 1 (pain group would report greater state-level catastrophizing and demonstrate worse verbal and non-verbal WM) was partially supported, such that the pain group reported greater state-level catastrophizing than the control group, which aligns with previous research [12,24,68]. The pain and control groups performed similarly on WM tasks, which conflicts with much of the experimental literature [9,34,53,73]. Although these non-significant total effects (group → WM) are initially surprising, they are better understood when considered alongside the significant mediation. The indirect effects were negative, whereas the direct effects were positive. As such, they offset each other when summed to calculate the total effects [31]. That is to say, state-level catastrophizing suppressed the effect of pain on WM when it was not explicitly modeled in the analyses [45].

Another explanation for the non-significant total effects may be found in a recent paper that also reported that laboratory-induced pain did not significantly impact cognitive performance [1]. The authors discussed the idea of pain having detrimental or facilitating effects on cognitive performance depending on its intensity [1]. Specifically, minor pain is distracting, but may not promote increased effort on cognitive tasks to compensate, leading to net performance decrements. Pain that exceeds minor levels may initiate greater effort, paradoxically leading to better cognitive performance. However, at some point along the pain intensity continuum this pattern may reverse, such that further increases in pain override any compensatory processes, thereby degrading cognitive performance. This idea is supported in the cognitive science literature wherein disfluency and other forms of “desirable difficulties” can, at times, enhance – as opposed to degrade – cognitive performance [8,23]. Interpreting the current results through this (speculative) lens, the ischemic task may have been painful enough to initiate increased effort among the pain group, thereby leading to better performance compared to the

control group (i.e., positive direct effects, controlling for catastrophizing). However, when pain catastrophizing is explicitly modeled in the analyses, its deleterious effect on WM performance (negative path b coefficients) offsets the positive effect of pain (positive path c' coefficients), thereby yielding insignificant total effects. Although the ischemic task appeared to elicit considerable pain (final pain rating = 73/100), it is possible that a more intense stimulus (that exceeds a presumed inflection point) would yield a different pattern of results [1].

Hypothesis 2 (relationships between group and WM would be mediated by state-level catastrophizing) was also supported – the pain group experienced more state-level catastrophizing than the control group, leading to worse WM performance. These results persisted even when anxiety and depression symptoms were included in the model; thus, despite research supporting associations between mood and cognitive impairment, our findings were not driven by mood [11,50,68,76]. The mediating effects of state-level catastrophizing were medium to large [13,41], suggesting that verbal and non-verbal WM deficits among people with pain are due, in part, to in-the-moment catastrophizing about their pain. These findings expand upon the existing literature by providing stronger evidence for state-level catastrophizing as a causal mechanism in the pain—WM nexus [3,26,43,65].

Although we did not directly test the resource depletion model, these results appear to align with it – that is, pain catastrophizing may have consumed finite cognitive resources, leaving less available for other activities requiring WM [66]. Emotional, cognitive, and behavioral self-regulation requires executive functions and related processes like WM [4,5,54,66]. In a zero-sum manner, each self-regulatory process consumes cognitive resources, thereby leaving less available for other functions. For example, a person engaging in pain-related rumination – a feature of pain catastrophizing – and/or attempting to suppress such thoughts has

fewer available cognitive resources to devote to activities that demand WM [27,54,66]. Relatedly, much like pain itself consumes attentional resources, catastrophizing may also draw on these finite resources, particularly when pain sufferers are wrestling to control their catastrophic thoughts. Indeed, this perspective lies at the heart of mindfulness and acceptance-based approaches to pain [20,28,35,62].

Hypothesis 3 (trait-level catastrophizing would moderate the mediation pathways for WM) was supported such that participants in the pain group who reported higher trait-level catastrophizing engaged in greater state-level catastrophizing, leading to worse WM performance. In other words, the mediation effect (group→state-level catastrophizing→WM) was stronger among participants with a greater trait-level tendency to catastrophize about pain. Although these moderated mediation effects were relatively small in magnitude, they suggest that one's tendency to catastrophize about pain in general may increase the likelihood and intensity of catastrophizing about pain while it is occurring, thereby causing greater decrements in WM. The experimental design and focus on WM deficits in the current study expands on the correlational work of Sturgeon and Zautra (2013), who used a similar moderated mediation model to predict mood-based outcomes among patients with rheumatoid arthritis [68].

Our results may also help resolve the inconsistent and sometimes contradictory findings of the few studies examining pain catastrophizing and cognitive function. A study of patients with fibromyalgia assessed catastrophizing with the trait-level Coping Strategies Questionnaire but interpreted the results as if it were a state-level process. Specifically, catastrophizing was correlated with worse performance on components of the Trail Making Test (a measure of attention, psychomotor speed, and executive function), but not with performance on a test of verbal learning and memory [26]. These results were interpreted as evidence that catastrophizing

actively disrupted cognitive functions, implying that participants were engaging in state-level catastrophizing. Although possible, this assumption is unwarranted given that catastrophizing was measured at the trait level. In another correlational study of a heterogeneous pain sample, catastrophizing (measured with the trait-level PCS) was associated with poorer learning and recall on tests of verbal learning and memory, but not with the Trail Making Test [43]. As with the fibromyalgia study, these results were discussed at the state level – i.e., catastrophizing as a "process" that interrupted cognitive performance – even though catastrophizing was measured at the trait level [26,43]. By using an experimental design, differentiating between state- and trait-level measures, and conducting moderated mediation analyses, the current study yields a clearer, more nuanced understanding of the relationships among pain, catastrophizing, and cognition.

Several study limitations should be noted. It is possible that the cross-sectional nature of our data may yield biased parameter estimates, although the experimental design (i.e., randomization to pain and control groups) and immediate process under study (i.e., acute pain and WM) mitigate against this possibility [25,47]. Another potential concern pertains to measurement. Although we used gold standard measures of state (SCQ) and trait (PCS) catastrophizing, a recent study raises questions about these and other measures of pain catastrophizing. Crombez and colleagues (2020) suggest that common measures, including the PCS, may fail to distinctly capture the construct of "pain catastrophizing" as defined in the cognitive-behavioral literature [16]. Moreover, the SCQ has not been psychometrically vetted as thoroughly as trait measures. As such, caution is warranted when considering our findings, especially in regard to pain catastrophizing per se.

Participants were relatively young, healthy, and mostly White students from a Midwestern university. Individuals with different levels of education may have more or less

cognitive reserve, which could affect the degree to which catastrophizing impacts WM [42,46]. Relatedly, the results may not generalize to people with chronic pain. The ischemic task induced a temporary pain that lacked the persistent and often disabling characteristics of chronic pain, possibly contributing to different experiences of pain catastrophizing and ramifications for WM. Moreover, the sample scored on the lower end of the catastrophizing scales. Although we still were able to detect statistically reliable mediation and moderation effects, our pain-free sample may underestimate the magnitude of these associations among patients with chronic pain. Another potential constraint to generalizability is our focus on WM. The literature is mixed concerning the relationships between pain catastrophizing and various aspects of cognitive function [26,43,65]. Extending our methodological paradigm to other cognitive domains is a prudent next step. There is reason to suspect that executive functions would be prime candidates for this work, as they are impacted by chronic pain and are related to, yet distinct from, WM [2,7,32,48,52].

Future research should also examine sleep in this context. Sleep disturbances are associated with subjective complaints of WM deficits in chronic pain populations, though their relationship with objective tests of WM are less consistent [10,22,49,69]. These inconsistencies may be the consequence of different methods of assessing sleep disturbance (e.g., sleep efficiency vs. total hours slept) [28]. Together with the current findings, this body of research indicates that future studies could benefit from examining sleep disturbance as an additional mediating variable in the pain—WM nexus, possibly in a parallel mediation alongside state-level pain catastrophizing.

These replications and extensions of the current study might inform intervention research to ameliorate the cognitive consequences of pain. Cognitive and acceptance-based therapies hold

particular promise for improving pain-related WM deficits, as they have demonstrated effectiveness in treating pain catastrophizing and pain itself [20,35,62]. Furthermore, as these therapies are effective for insomnia and other sleep problems, future research could investigate the mechanisms (i.e., state-level catastrophizing vs. sleep disturbances vs. a dynamic relationship between them) by which they improve WM function in chronic pain [18,74].

Overall, findings of the current study implicate pain catastrophizing as a potential causative and magnifying factor in the relationship between acute pain and WM deficits. Future research should examine whether a similar putative causal relationship holds in patients with chronic pain, which could then inform the development and/or adaptation of interventions to reduce catastrophizing, ameliorate WM deficits, and improve the quality of life in patients with pain.

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Figures

Figure 1. Flow chart of study procedures.

Figure 2. Simple mediation model results for verbal (Panel A) and non-verbal (Panel B) working memory. For both verbal and non-verbal working memory, a significant indirect effect indicated that participants in the pain group engaged in more state-level pain catastrophizing than the control group, which was then associated with worse verbal and non-verbal working memory performance.

Figure 3. Parallel mediation model results for verbal (Panel A) and non-verbal (Panel B) working memory. For both verbal and non-verbal working memory, there was a significant indirect effect for state-level pain catastrophizing but not for anxiety or depression symptoms. Participants in the pain group engaged more state-level pain catastrophizing than the control group, which was then associated with worse verbal and non-verbal working memory performance.

Figure 4. Moderated mediation model results for verbal (Panel A) and non-verbal (Panel B) working memory. For both verbal and non-verbal working memory, a significant conditional indirect effect was found at all levels of the moderator (trait-level pain catastrophizing). Participants in the pain group who reported a greater trait-level tendency to catastrophize about pain engaged in greater state-level catastrophizing about pain during the ischemic task, which led to worse verbal and non-verbal working memory performance.

Tables

Supplementary Tables

Supplementary Table 1: NASA Task Load Index

Supplementary Table 2: Results of simple mediation for verbal working memory

Supplementary Table 3: Results of parallel mediation for verbal working memory

Supplementary Table 4: Results of simple mediation for non-verbal working memory

Supplementary Table 5: Results of parallel mediation for non-verbal working memory

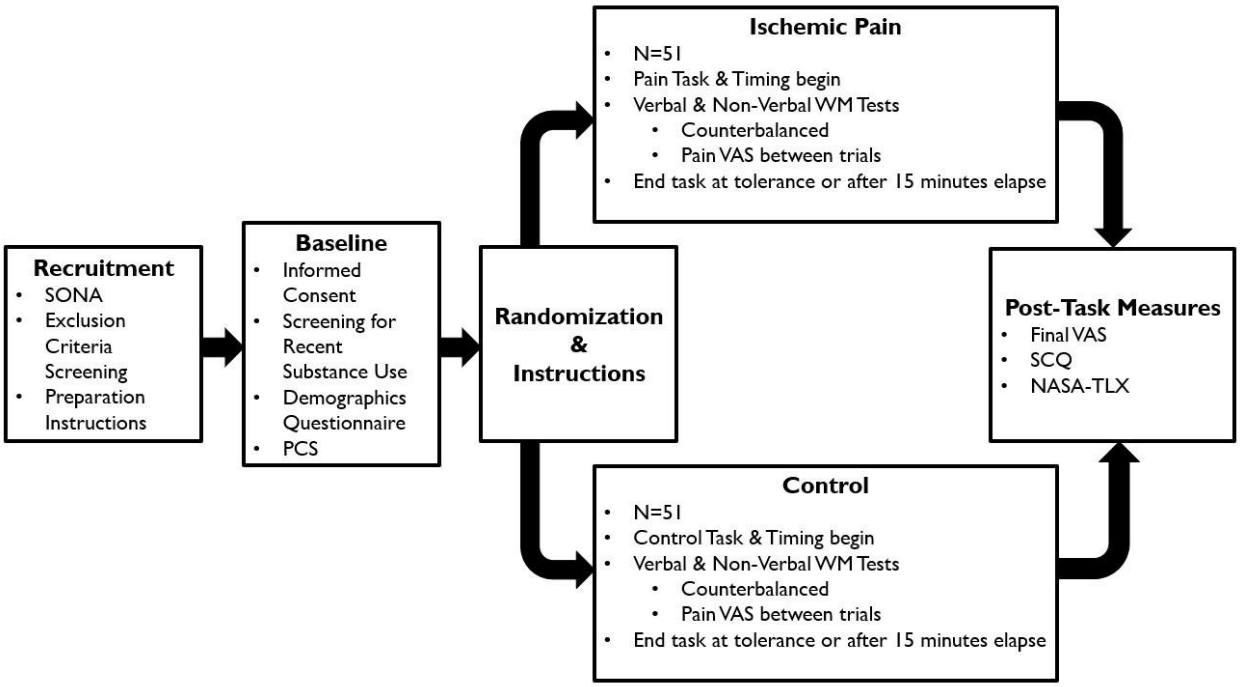
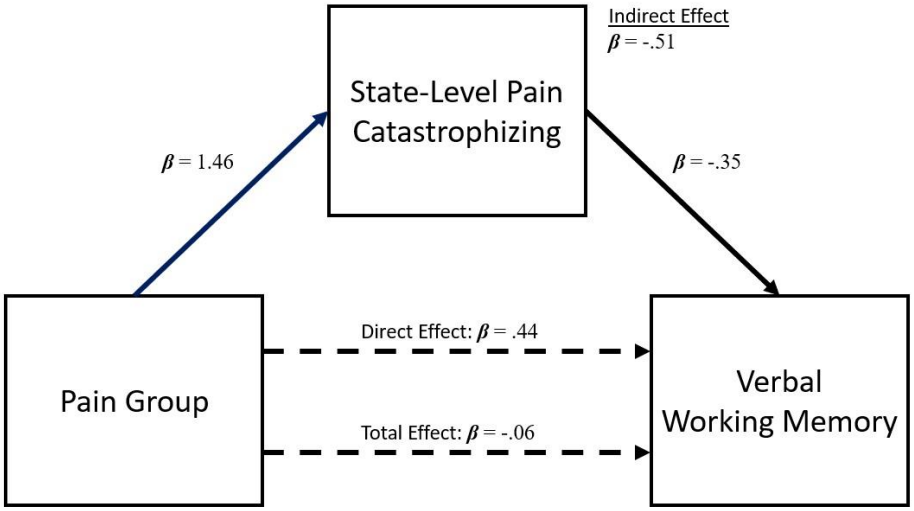
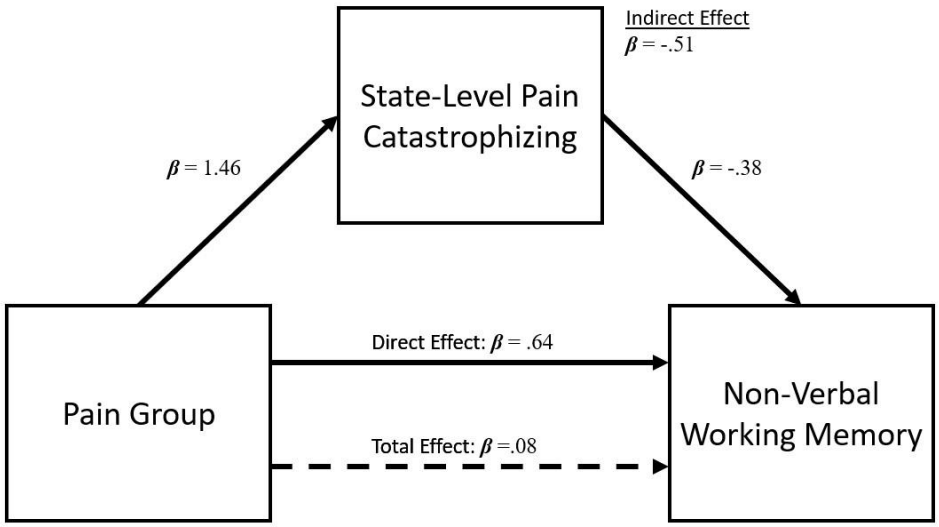


Figure 1. Flow chart of study procedures.





Solid lines ⟶ Significant paths

Dashed lines ⇢ Non-significant paths

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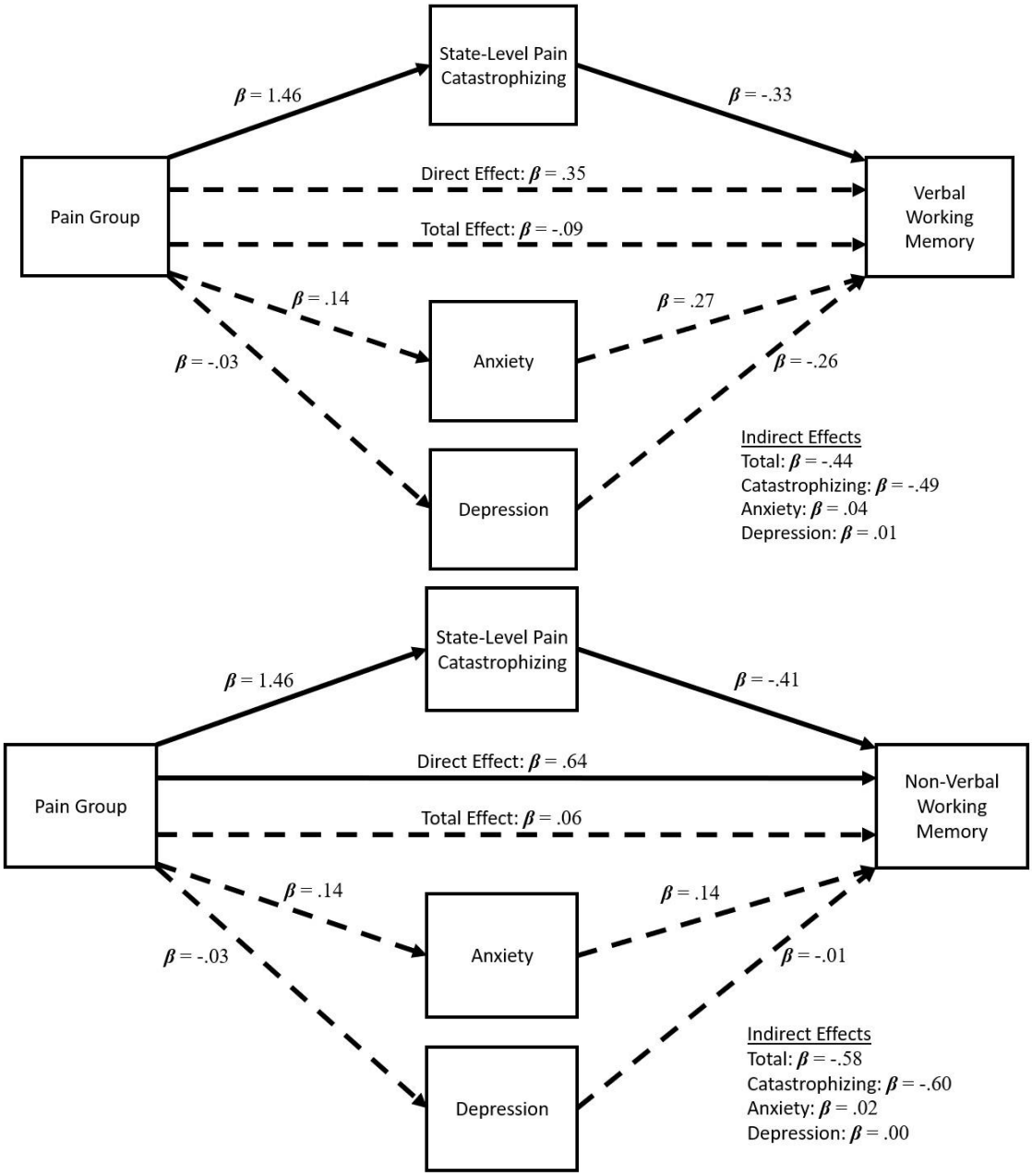
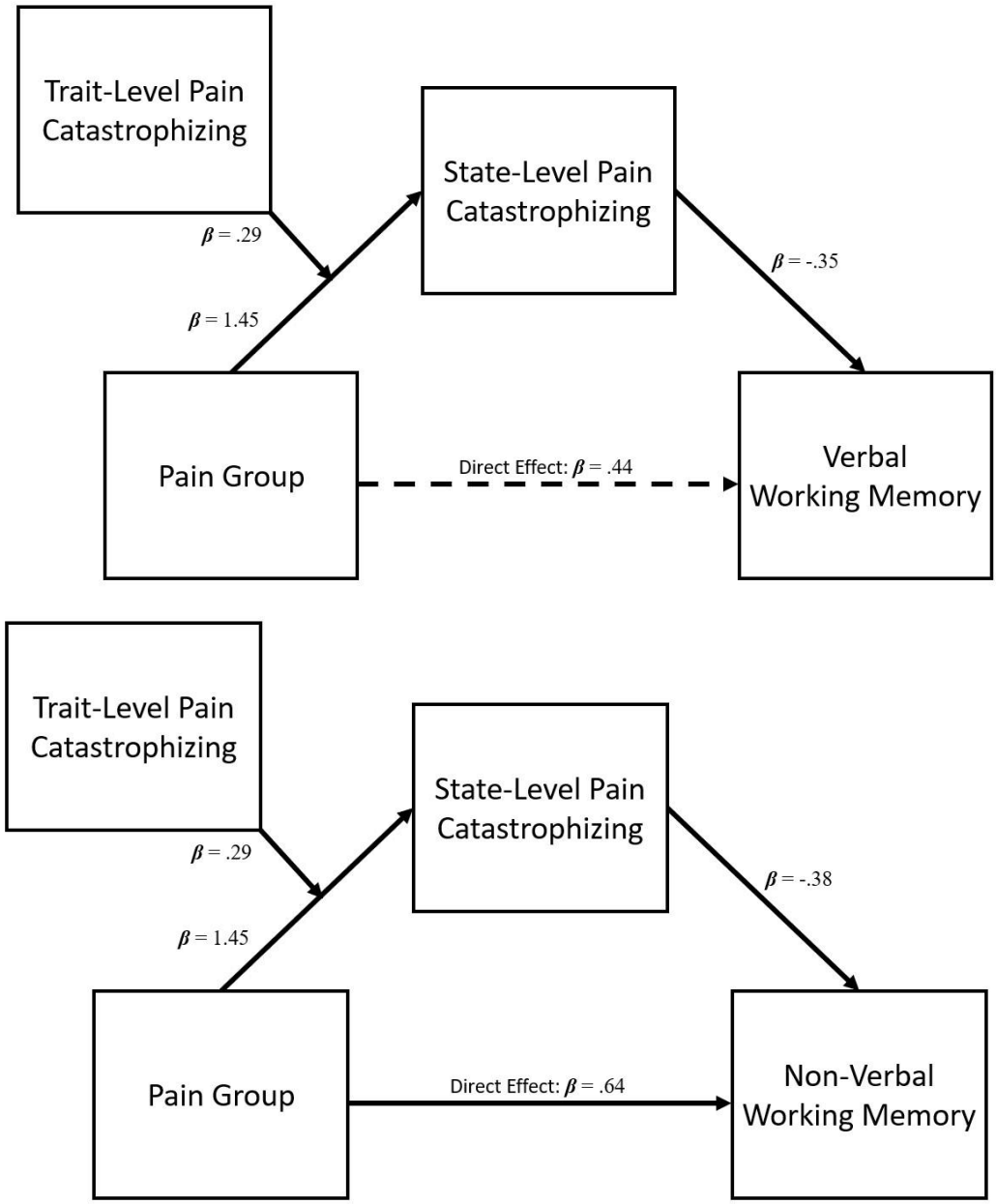


Solid lines ⟶ Significant paths

Dashed lines ⇢ Non-significant paths

Figure 3. Parallel mediation model results for verbal (Panel A) and non-verbal (Panel B) working memory. For both verbal and non-verbal working memory, there was a significant indirect effect for state-level pain catastrophizing but not for anxiety or depression symptoms. Participants in the pain group engaged in more state-level pain catastrophizing than the control group, which was then associated with worse verbal and non-verbal working memory performance.



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		All Participants (N = 102)	Pain Group (N = 51)	Control Group (N = 51)	t/χ^2	*p*
Age	Mean (SD)	20.12 (3.73)	20.57 (4.42)	19.67 (2.85)	1.22	.224
Sex	Female	52 (51%)	26 (51%)	26 (51%)	.00	1.00
	Male	50 (49%)	25 (49%)	25 (49%)		
Race	White/Caucasian	68 (66.7%)	35 (68.6%)	33 (64.7%)	3.74	.588
	Black/African American	10 (9.8%)	4 (7.8%)	6 (11.8%)		
	Asian/Pacific Islander	7 (6.9%)	4 (7.8%)	3 (5.9%)		
	Native American/Inuit/Aleut	1 (1%)	1 (2%)	0 (0%)		
	Multiple Racial Backgrounds	1 (1%)	1 (2%)	0 (0%)		
	Other	14 (13.7%)	6 (11.8%)	9 (17.6%)		
Ethnicity	Not Hispanic or Latinx	80 (78.4%)	37 (72.5%)	43 (84.3%)	2.09	.149
	Hispanic or Latinx	22 (21.6%)	14 (27.5%)	8 (15.7%)		
Personal Experience with Chronic Pain	None	64 (62.7%)	36 (70.6%)	28 (54.9%)	2.76	.430
	Minimal	20 (19.6%)	8 (15.7%)	12 (23.5%)		
	Some	11 (10.8%)	4 (7.8%)	7 (13.7%)		
	Much	7 (6.9%)	3 (5.9%)	4 (7.8%)		

Table 1. Sample Demographic Characteristics

	All Participants (N = 102)		Pain Group (N = 51)		Control Group (N = 51)			
	M	SD	M	SD	M	SD	*t*	*p*
Pre-task measures								
PCS	11.14	8.36	11.31	8.94	10.96	7.83	-.21	.832
GAD-7	4.96	3.97	5.25	4.34	4.67	3.59	-.75	.457
PHQ-8	4.74	4.09	4.69	4.04	4.80	4.17	.14	.890
Post-task measures								
Final Pain Rating	37.76	39.50	73.33	23.30	2.19	5.29	-21.27	<.001
Total Time in Task (minutes)	11.39	3.86	9.11	3.18	13.66	3.08	7.33	<.001
Digit Span Total Score	33.51	18.07	32.94	19.77	34.08	16.38	.32	.752
Corsi Block Total Score	62.16	22.13	63.18	21.02	61.16	23.33	-.42	.648
SCQ	5.46	6.53	10.10	5.98	.82	1.38	-10.79	<.001

Note: PCS = Pain Catastrophizing Scale; GAD-7 = Generalized Anxiety Disorder 7-item Scale; PHQ-8 = Patient Health Questionnaire 8; Digit Span = Backwards Digit Span; Corsi Block = Corsi Block Tapping Task; SCQ = Situational Catastrophizing Questionnaire

Table 2. Pre-task and Post-task Measures

Path	*b*	SE	*β*	*t*	*p*	95% CI
Pain Group ➔ SCQ	6.81	1.36	1.45	5.00	<.001	4.11, 9.52
PCS ➔ SCQ	.03	.07	.04	.36	.716	-.12, .17
Pain Group x PCS ➔ SCQ	.22	.10	.29	2.20	.030	.02, .41
SCQ ➔ Digit (b)	-.99	.41	-.35	-2.41	.018	-1.80, -.18
Pain Group ➔ Digit (c' = Direct Effect)	8.01	5.17	.44	1.55	.124	-2.24, 18.26

Index of Moderated Mediation	Index	SE	Standardized Index	Standardized 95% CI	95% CI
PCS	-.21	0.13	-.10	-.24, -.01	-.51, -.03

Moderator Level (PCS)	Conditional Indirect Effect	SE	Standardized Conditional Indirect Effect	Standardized 95% CI	95% CI
-1SD (2.78)	-7.31	3.07	-.40	-.78, -.12	-14.38, -2.37
Mean (11.14)	-9.10	3.55	-.50	-.93, -.16	-17.06, -3.04
+1SD (19.50)	-10.89	4.24	-.60	-1.13, -.19	-20.30, -3.56

Note: SCQ = Situational Catastrophizing Questionnaire; PCS = Pain Catastrophizing Scale; Digit = Backwards Digit Span

Table 3. Results of moderated mediation for verbal working memory

Path	*b*	SE	*β*	*T*	*p*	95% CI
Pain Group ➔ SCQ	6.81	1.36	1.45	5.00	<.001	4.11, 9.52
PCS ➔ SCQ	.03	.07	.04	.36	.716	-.12, .17
Pain Group x PCS ➔ SCQ	.22	.10	.29	2.20	.030	.02, .41
SCQ ➔ Digit (b)	-1.33	.49	-.38	-2.69	.008	-2.31, -.35
Pain Group ➔ Digit (c' = Direct Effect)	14.19	6.25	.64	2.27	.026	1.78, 26.60

Index of Moderated Mediation	Index	SE	Standardized Index	Standardized 95% CI	95% CI
PCS	-.29	.15	-.11	-.24, -.02	-.66, -.05

Moderator Level (PCS)	Conditional Indirect Effect	SE	Standardized Conditional Indirect Effect	Standardized 95% CI	95% CI
-1SD (2.78)	-9.87	4.34	-.45	-.88, -.11	-19.15, -2.16
Mean (11.14)	-12.28	4.78	-.56	-1.00, -.15	-21.93, -2.95
+1SD (19.50)	-14.69	5.47	-.67	-1.16, -.18	-25.47, -3.79

Note: SCQ = Situational Catastrophizing Questionnaire; PCS = Pain Catastrophizing Scale; Corsi = Corsi Block Tapping Task

Table 4. Results of moderated mediation for non-verbal working memory

	All Participants (N = 102)		Pain Group (N = 51)		Control Group (N = 51)			
	M	SD	M	SD	M	SD	*t*	*p*
NASA-TLX Digit Span								
Mental Demand	11.77	5.43	12.18	4.99	11.37	5.85	-.75	.457
Physical Demand	4.26	5.40	7.16	5.80	1.37	2.86	-6.39	<.001
Temporal Demand	6.79	4.79	8.00	4.65	5.59	4.67	-2.61	.010
Performance	9.45	4.50	10.10	4.88	8.80	4.03	-1.46	.147
Effort	12.14	5.32	13.20	4.68	11.08	5.74	-2.04	.044
Frustration	6.56	6.03	8.08	6.04	5.04	5.68	-2.62	.010
NASA-TLX Corsi Block								
Mental Demand	9.23	4.84	9.76	4.70	8.69	4.96	-1.13	.262
Physical Demand	4.17	5.15	6.98	5.73	1.35	2.14	-6.57	<.001
Temporal Demand	7.36	5.14	8.92	4.84	5.80	5.00	-3.20	.002
Performance	8.39	5.15	9.53	5.27	7.25	4.80	-2.28	.025
Effort	11.01	5.50	12.82	4.63	9.20	5.74	-3.51	.001
Frustration	5.02	5.25	6.31	5.25	3.73	4.98	-2.56	.012

Note: Digit Span = Backwards Digit Span; Corsi Block = Corsi Block Tapping Task

Supplementary Table 1: NASA Task Load Index

Path	***b***	**SE**	***β***	***t***	***p***	**95% CI**
Pain Group ➔ SCQ (a)	9.27	.86	1.46	10.79	<.001	7.57, 10.98
SCQ ➔ Digit (b)	-.99	.41	-.35	-.24	.018	-1.80, -.18
Pain Group ➔ Digit (c' = Direct Effect)	8.01	5.17	.44	1.55	.124	-2.24, 18.26
Pain Group ➔ Digit (c = Total Effect)	-1.14	3.59	-.06	-.32	.752	-8.27, 5.99
Path	***b***	**SE**	***β***		**Standardized 95% CI**	**95% CI**
Indirect Effect (ab)	-9.15	3.57	-.51		-.90, -.18	-17.15, -2.91

Note: SCQ = Situational Catastrophizing Questionnaire; Digit = Backwards Digit Span

Supplementary Table 2: Results of simple mediation for verbal working memory

Path	*b*	SE	*β*	*t*	*p*	95% CI
Pain Group ➔ SCQ (a)	9.26	.87	1.46	10.67	<.001	7.54, 10.98
Pain Group ➔ GAD-7 (a)	.55	.80	.14	.70	.487	-1.02, 2.13
Pain Group ➔ PHQ-8 (a)	-.11	.82	-.03	-.14	.890	-1.74, 1.51
SCQ ➔ Digit (b)	-.95	.41	-.33	-2.32	.022	-1.76, -.14
GAD-7 ➔ Digit (b)	1.24	.63	.27	1.97	.052	-.01, 2.49
PHQ-8 ➔ Digit (b)	-1.14	.62	-.26	-1.84	.069	-2.37, .09
Pain Group ➔ Digit						
(c' = Direct Effect)	6.31	5.15	.35	1.23	.223	-3.91, 16.54
Pain Group ➔ Digit						
(c = Total Effect)	-1.64	3.60	-.09	-.46	.650	-8.77, 5.50

Path	*b*	SE	Partially Standardized Indirect Effect	Partially Standardized 95% CI	95% CI
Indirect Effect Total (ab)	-7.95	3.83	-.44	-.87. -.08	-16.71, -1.27
Indirect Effect of SCQ (ab)	-8.77	3.61	-.49	-.89, -.16	-17.13, -2.68
Indirect Effect of GAD-7 (ab)	.69	1.15	.04	-.08, .19	-1.45, 3.29
Indirect Effect of PHQ-8 (ab)	.13	1.09	.01	-.12, .14	-2.15, 2.45

Note: SCQ = Situational Catastrophizing Questionnaire; GAD-7 = Generalized Anxiety Disorder Scale; PHQ-8 = Patient Health Questionnaire; Digit = Backwards Digit Span

Supplementary Table 3: Results of Parallel mediation for verbal working memory

Path	*b*	SE	*β*	*t*	*p*	95% CI
Pain Group ➔ SCQ (a)	9.27	.86	1.46	10.79	<.001	7.57, 10.98
SCQ ➔ Digit (b)	-1.33	.49	-.38	-2.69	.008	-2.31, -.35
Pain Group ➔ Digit (c' = Direct Effect)	14.19	6.25	.64	2.27	.026	1.78, 26.60
Pain Group ➔ Digit (c = Total Effect)	1.84	4.38	.08	.42	.675	-6.85, 10.54
Path	***b***	**SE**	***β***		**Standardized 95% CI**	**95% CI**
Indirect Effect (ab)	-12.34	4.83	-.56		-1.00, -.16	-22.15, -3.29

Note: SCQ = Situational Catastrophizing Questionnaire; Corsi = Corsi Block Tapping Task

Supplementary Table 4: Results of simple mediation for non-verbal working memory

Path	*b*	SE	*β*	*t*	*p*	95% CI
Pain Group ➔ SCQ (a)	9.26	.87	1.46	10.67	<.001	7.54, 10.98
Pain Group ➔ GAD-7 (a)	.55	.80	.14	.70	.487	-1.02, 2.13
Pain Group ➔ PHQ-8 (a)	-.11	.82	-.03	-.14	.890	-1.74, 1.51
SCQ ➔ Corsi (b)	-1.43	.50	-.41	-2.85	.005	-2.53, -.43
GAD-7 ➔ Corsi (b)	.79	.77	.14	1.03	.307	-.74, 2.33
PHQ-8 ➔ Corsi (b)	-.04	.76	-.01	-.05	.959	-1.55, 1.47
Pain Group ➔ Corsi (c' = Direct Effect)	14.08	6.33	.64	2.23	.028	1.52, 26.64
Pain Group ➔ Corsi (c = Total Effect)	1.32	4.39	.06	.30	.765	-7.40, 10.04
Path	***b***	**SE**	**Partially Standardized Indirect Effect**		**Partially Standardized 95% CI**	**95% CI**
Indirect Effect Total (ab)	-12.76	4.85	-.58		-1.01, -.18	-22.80, -3.69
Indirect Effect of SCQ (ab)	-13.20	4.59	-.60		-1.01, -.22	-22.77, -4.62
Indirect Effect of GAD-7 (ab)	.44	.94	.02		-.05, .13	-.98, 2.83
Indirect Effect of PHQ-8 (ab)	.00	.70	.00		-.08, .07	-1.68, 1.46

Note: SCQ = Situational Catastrophizing Questionnaire; GAD-7 = Generalized Anxiety Disorder Scale; PHQ-8 = Patient Health Questionnaire; Corsi = Corsi Block Tapping Task

Supplementary Table 5: Results of parallel mediation for non-verbal working memory