AN EXPERIMENTAL INVESTIGATION AND CONDITIONAL PROCESS ANALYSIS OF THE ROLE OF CATASTROPHIZING IN THE PAIN—WORKING MEMORY NEXUS

By

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ABSTRACT

There is a well-documented bidirectional relationship between pain and cognitive dysfunction, especially working memory. Despite this extensive body of research, the pain–working memory relationship is poorly understood. Pain catastrophizing – exaggerated negative cognitive and emotional responses towards pain – may contribute to working memory deficits by occupying finite, shared cognitive resources, but this has yet to be investigated. The present study sought to clarify the role of pain catastrophizing (assessed as both a trait-level disposition and state-level process) in working memory dysfunction. Healthy undergraduate participants were randomized to an ischemic pain or control task, during which they completed verbal and non-verbal working memory tests. They also completed measures of state- and trait-level pain catastrophizing. Mediation analyses indicated that state-level pain catastrophizing mediated the relationships of pain group to both verbal and non-verbal working memory, such that participants in the pain group (vs. the control group) catastrophized more about their pain, which then resulted in worse verbal and non-verbal working memory performance. In moderated mediation analyses, trait-level pain catastrophizing moderated this mediation effect for both verbal and non-verbal working memory. Those participants in the pain group who reported greater tendency to catastrophize about pain in general exhibited greater catastrophizing in-the-moment during the pain task, thereby leading to worse verbal and non-verbal working memory performance. These results provide evidence for pain catastrophizing as a putative mechanism and moderating factor of working memory dysfunction in pain. Future research should replicate these results in chronic pain samples, investigate other potential mechanisms (e.g., sleep), and develop interventions to ameliorate cognitive dysfunction by targeting pain catastrophizing.

1. INTRODUCTION

Chronic pain, defined as lasting beyond the normal healing period and/or for longer than three to six months, is a serious public health concern (Merskey & Bogduk, 1994; Treede et al., 2015). Chronic pain is estimated to occur in over 30% of the US population, is a leading cause of disability, and is responsible for $635 billion in annual costs (direct and indirect) (Johannes, Le, Zhou, Johnston, & Dworkin, 2010; Murray et al., 2013). Although the severity of the pain sensation itself is an important contributor to functional impairment in chronic pain, the cognitive and affective correlates (e.g., depression, pain-related fear) are also critical and may be the strongest predictors of pain-related impairment (Crombez, Vlaeyen, Heuts, & Lysens, 1999; Higgins, Martin, Baker, Vasterling, & Risbrough, 2018; Turner, Jensen, Warms, & Cardenas, 2002).

People with chronic pain frequently report cognitive problems coinciding with the course and onset of their pain, and these have also been quantitatively demonstrated through neurocognitive testing (Berryman et al., 2013; Higgins et al., 2018; Jongsma et al., 2011; Mazza, Frot, & Rey, 2018; Oosterman, Derksen, van Wijck, Veldhuijzen, & Kessels, 2011). Similar cognitive problems have been found in healthy participants during laboratory-based pain induction tasks (Buhle & Wager, 2010; Moore, Eccleston, & Keogh, 2017). In particular, significant deficits in working memory performance have been found in people with chronic pain compared to healthy controls, as well as in healthy subjects during experimental pain tasks (Berryman et al., 2013; Mazza et al., 2018; Moore et al., 2017). Working memory is the neurocognitive system for temporarily storing and manipulating verbal and visuospatial information in service of other cognitive functions (e.g., logical reasoning and problem solving)

(Baddeley, 1992, 2010). There are competing theories as to the underlying causes of these decrements in working memory performance. For example, differences in brain morphology amongst chronic pain patients may contribute to neurocognitive dysfunction (Luerding, Weigand, Bogdahn, & Schmidt-Wilcke, 2008). Alternatively, coping with pain is distracting and consumes cognitive resources shared with other functions, which may leave fewer resources available to devote to working memory and other cognitive tasks (Park, Glass, Minear, & Crofford, 2001). Clarifying the etiology, mechanisms, and moderators of these disparities in working memory functioning is crucial, as cognitive impairment that is comorbid with chronic pain has been shown to be significantly more disabling than either problem individually (Shega et al., 2010).

One approach to elucidating the pain—working memory relationship is to focus on the content of thoughts that frequently occur with pain, as these thoughts may compete with working memory for cognitive resources. Pain catastrophizing—a cognitive and emotional process of ruminating, magnifying, and feeling helpless about pain—affects how people experience pain, and it predicts distress and perceived disability (Severeijns, Vlaeyen, van den Hout, & Weber, 2001; Sullivan, Lynch, & Clark, 2005). Pain catastrophizing can be conceptualized as both a state-level process and a trait-level tendency (Campbell et al., 2010; Quartana, Campbell, & Edwards, 2009; Sturgeon & Zautra, 2012). State-level pain catastrophizing is a situational response to specific instances of pain (i.e., currently or recently experienced pain, typically in laboratory contexts). Trait-level pain catastrophizing is a general disposition to catastrophize about pain. Individuals higher in trait-level pain catastrophizing are more likely to engage in state-level pain catastrophizing (Campbell et al., 2010).

In experimental pain research with healthy controls, state-level pain catastrophizing predicted pain thresholds and post-induction pain sensations, whereas trait-level pain catastrophizing was positively correlated with depressive symptoms (Campbell et al., 2010). Trait-level pain catastrophizing was also positively correlated with perceiving pain as more intense and more disabling in terms of daily activity and occupational functioning (Legarreta, Bueler, DiMuzio, McGlade, & Yurgelun-Todd, 2016; Sullivan et al., 2005; Talaei-Khoei et al., 2017). Similarly, greater state-level pain catastrophizing significantly predicted higher daily negative affect and depressive symptoms and lower positive affect, mediating the relationship between daily pain ratings and these outcomes (Sturgeon & Zautra, 2012). Moreover, and particularly relevant to the current study, trait-level pain catastrophizing acted as a moderator, such that the relationships among state-level pain catastrophizing, depressive symptoms, and positive and negative affect were amplified in people high in trait-level pain catastrophizing (Sturgeon & Zautra, 2012).

Despite this extensive body of research on the physical and psychological consequences of state-level and trait-level pain catastrophizing, relatively little is known about their connections to cognitive function. Some research has found negative correlations between pain catastrophizing and cognitive function in people with chronic pain (with higher scores on measures of trait-level pain catastrophizing associated with worse cognitive function), but these relationships have not been fully explained and are, at times, conflicting (Galvez-Sánchez, Reyes del Paso, & Duschek, 2018; Legarreta et al., 2016). For example, Galvez-Sánchez et al. (2018) found a significant relationship between trait-level pain catastrophizing and completion time on the Trail Making Test (TMT) – a measure of attention, processing speed, and executive function – but Legarreta et al. (2016) failed to find statistically significant associations between pain

catastrophizing and the TMT. These conflicting findings are further complicated by inconsistent measurement of pain catasphtophizing, with the former study utilizing the 6-item version of the Coping Strategies Questionnaire and the latter study using the Pain Catastrophizing Scale (Galvez-Sánchez et al., 2018; Legarreta et al., 2016).

The pain—working memory literature is plagued with similar issues. Pain catasrophizing is conspicuously absent from many of these studies (Berryman et al., 2013; Jongsma et al., 2011; Luerding et al., 2008; Mazza et al., 2018; Oosterman et al., 2011). Of the few that did assess both, the analyses were done in parallel and did not differentiate between or independently measure both state and trait pain catastrophizing (Dick & Rashiq, 2007). The few studies that did examine their relationship used correlational designs that do not allow for strong causal inferences (Baker, Gibson, Georgiou-Karistianis, Roth, & Giummarra, 2016; Galvez-Sánchez et al., 2018; Jorge, Gerard, & Revel, 2009; Legarreta et al., 2016; Melkumova, Podchufarova, & Yakhno, 2011). Experimental designs are needed for stronger conclusions about the relationships among pain, pain catastrophizing, and working memory, which will lead to better understanding of the mechanisms underlying the pain—emotion—cognition nexus. (Berryman et al., 2013; Dick & Rashiq, 2007; Galvez-Sánchez et al., 2018; Jongsma et al., 2011; Jorge et al., 2009; Legarreta et al., 2016; Luerding et al., 2008; Mazza et al., 2018; Melkumova et al., 2011; Oosterman et al., 2011)

2. CURRENT STUDY

The current study aimed to achieve increased understanding of the relationships among pain, catastrophizing, and working memory. I recruited healthy, pain-free participants and randomized them to an experimentally-induced pain condition or a no-pain control condition. Participants in both conditions completed well-established measures of verbal and non-verbal working memory and were assessed for both trait- and state-level pain catastrophizing. First, I hypothesized that participants randomized to the pain group would report significantly higher pain and state-level catastrophizing than those in the control group (H1). Second, I hypothesized that participants in the pain group would demonstrate significantly worse verbal and non-verbal working memory performance than participants in the control group (H2). Third, I hypothesized that the differences in verbal (Figure 1) and non-verbal (Figure 2) working memory performance between the pain and control groups would be mediated by state-level catastrophizing (H3). Finally, this mediation effect (group → state catastrophizing → verbal working memory, Figure 1; group → state catastrophizing → non-verbal working memory, Figure 2) would be moderated (Figures 3 & 4, respectively) by trait-level catastrophizing, such that the mediation effect would be stronger among participants who scored high on trait-level catastrophizing compared to participants who scored low on trait-level catastrophizing (H4).

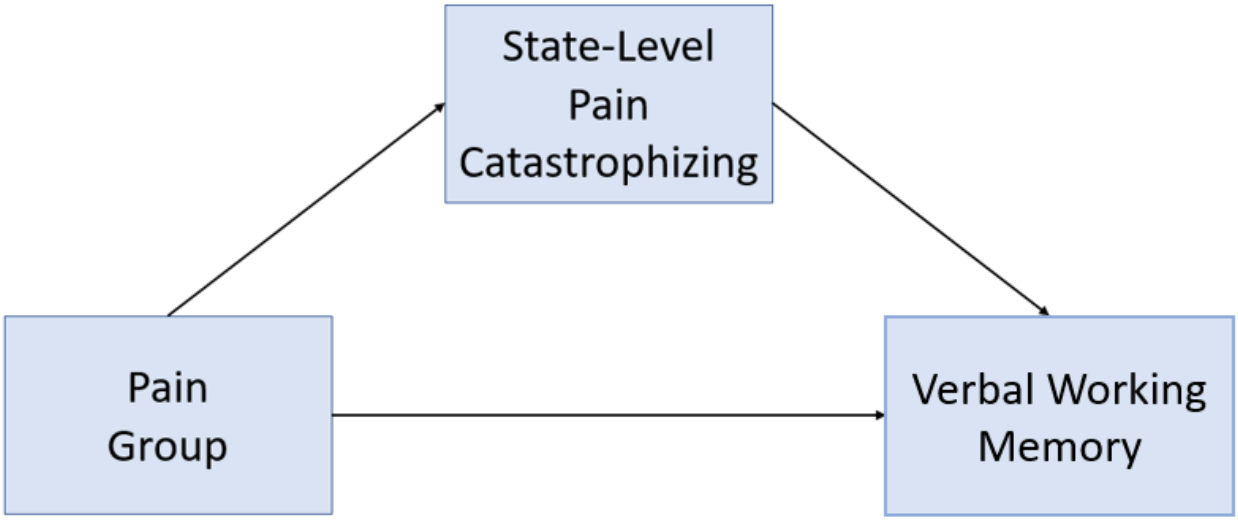

Figure 1. Proposed mediation model for verbal working memory.

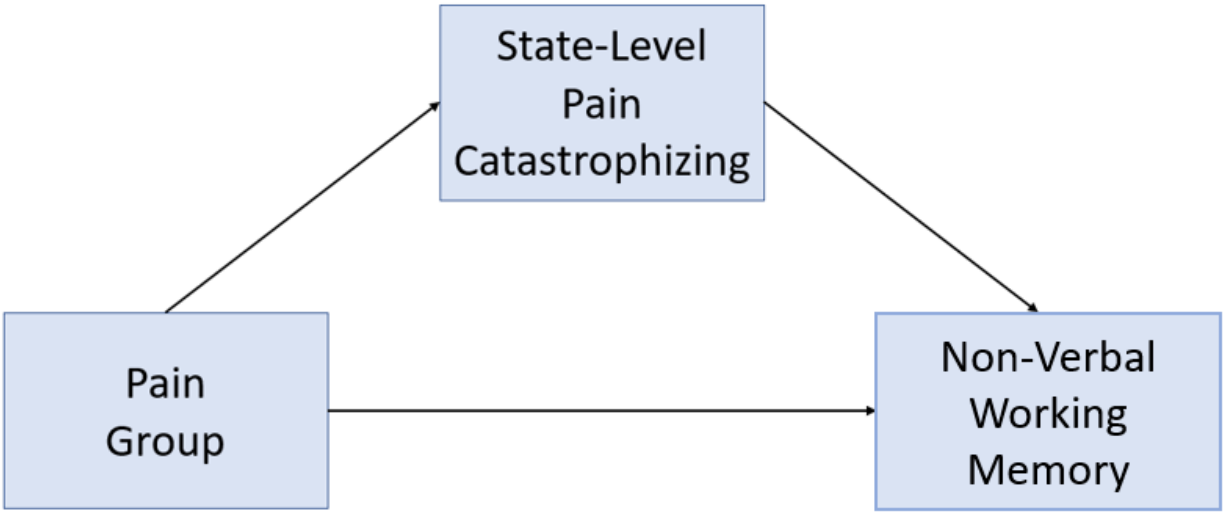

Figure 2. Proposed mediation model for non-verbal working memory.

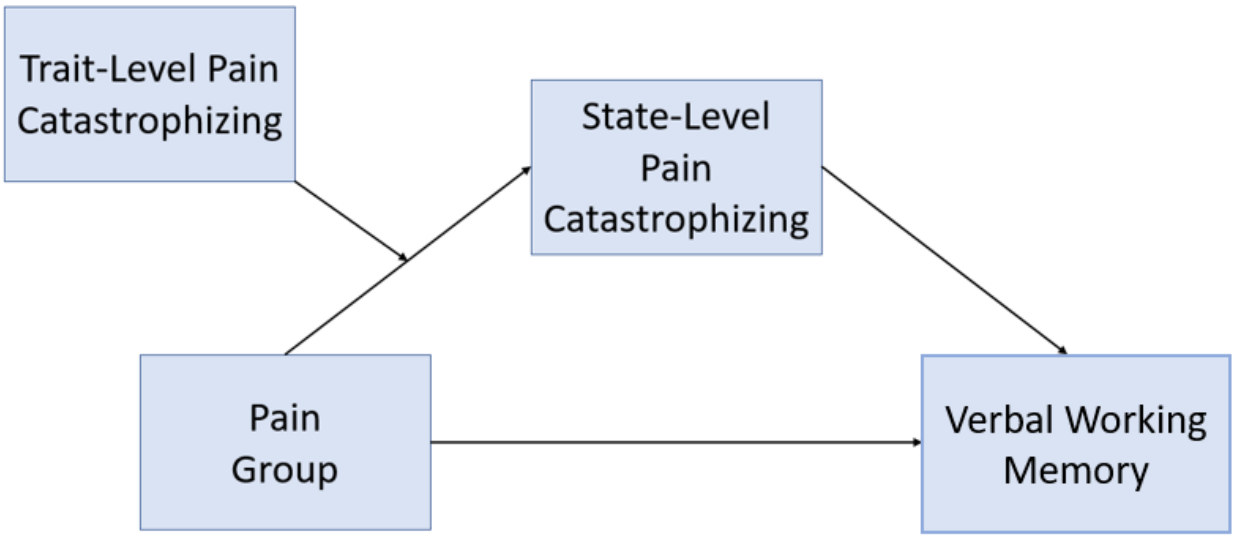

Figure 3. Proposed moderated mediation model for verbal working memory.

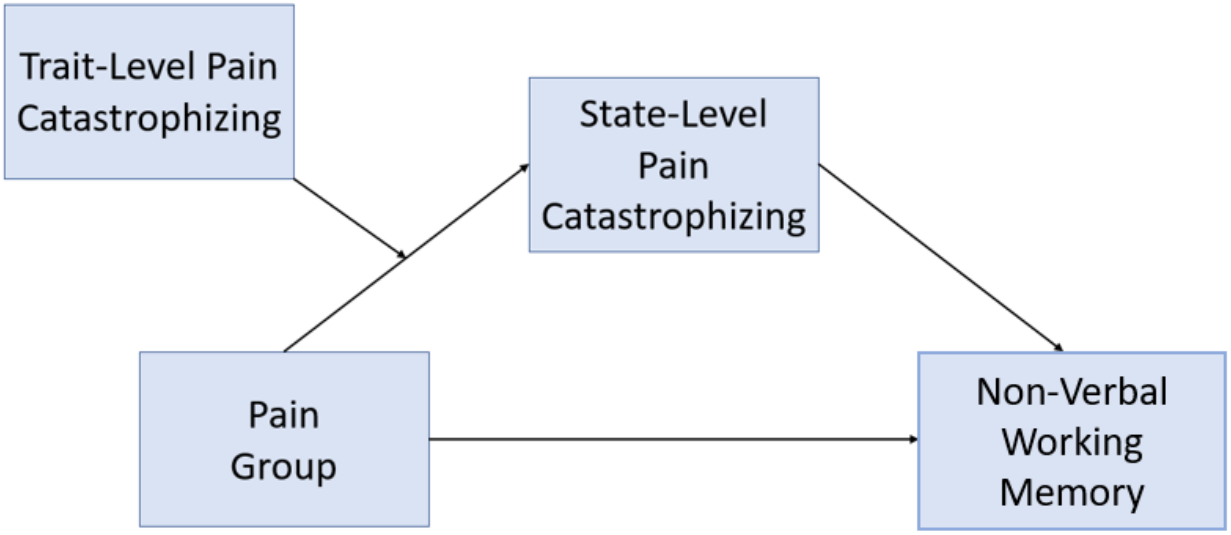

Figure 4. Proposed moderated mediation model for non-verbal working memory.

3. METHODS

3.1 Participants

The participants for this study were healthy adults enrolled in an introductory psychology course at Indiana University-Purdue University Indianapolis (IUPUI). Participants selected a specific date and time to participate via the Sona online management system and were screened by telephone for the exclusion criteria listed in Table 1. These exclusion criteria were determined to either pose unnecessary risks or confound the results of the working memory tasks. Eligible participants were advised to avoid analgesic medications within 24 hours and alcohol, tobacco, nicotine, and caffeine products within two hours of their scheduled appointments given that these substances may affect cognitive function and/or pain perception (Derry, Derry, & Moore, 2014; Fillmore, Carscadden, & Vogel-Sprott, 1998; Jamner, Girdler, Shapiro, & Jarvik, 1998; Rezvani & Levin, 2001; Ruxton, 2008; Woodrow & Eltherington, 1988).

Table 1. Exclusion criteria

1. Current pain lasting longer than three months
2. Circulatory system problems
3. Hypertension or high blood pressure
4. History of heart or cardiovascular diseases or problems
5. Diabetes
6. Pregnancy[a]
7. Serious mental illness diagnosis[b]
8. Serious injuries to the non-dominant hand or arm within prior year

a Current pregnancy (or planning to become pregnant in the next month)

b Autism spectrum disorders, schizophrenia or other psychotic disorder, bipolar disorder, or major depressive disorder that included thoughts of suicide

3.2. Measures

All measures were completed on a desktop computer through the Qualtrics online survey software (Qualtrics, 2014).

3.2.1. Demographic Information

Participants reported their sex/gender, race, ethnicity, marital status, annual income, work status, student status, college major, current cumulative grade point average (GPA), current major GPA, and personal experience with chronic pain.

3.2.2. Mood

As mood disorders are associated with cognitive impairment, depressive and anxious symptomatology were assessed with the Patient Health Questionnaire 8 (PHQ-8) and Generalized Anxiety Disorder 7-item Scale (GAD-7), respectively (Castaneda, Tuulio-Henriksson, Marttunen, Suvisaari, & Lönnqvist, 2008; McDermott & Ebmeier, 2009; Vytal, Cornwell, Arkin, Letkiewicz, & Grillon, 2013).

The Patient Health Questionnaire 9 (PHQ-9) is a nine-item measure used to assess recent (e.g., two weeks) depressive symptoms, with high sensitivity (88%) and specificity (88%) for major depression (Kroenke, Spitzer, & Williams, 2001). The PHQ-8 includes all but one item from the PHQ-9, excluding the final item inquiring about suicidal ideation and self-harm (Kroenke et al., 2009). 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) (Corson, Gerrity, & Dobscha, 2004; Kroenke et al., 2009; Shin, Lee, Han, Yoon, & Han, 2019).

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 disorder, for which it has high sensitivity (89%), specificity (82%), and internal consistency (Cronbach's alpha = 0.92) (Spitzer, Kroenke, Williams, & Löwe, 2006). The GAD-7 has been shown to reliably predict functional impairments and disability (Löwe et al., 2008; Spitzer et al., 2006).

Participants rated items on both the PHQ-8 and GAD-7 with the same four-point scale ranging from "Not at all (0)" to "Nearly every day (3)." These scores were then summed, with total scores ranging from 0 to 24 and 0 to 21, respectively, with higher scores indicating greater symptom severity. Then, participants indicated the degree to which these problems have interfered with functioning ("Not difficult at all" to "Extremely difficult").

3.2.3. Sleep

Due to the deleterious effects of sleep deprivation on cognitive function, sleep duration and quality for the previous month was assessed with the Pittsburgh Sleep Quality Index (PSQI), a clinical scale with high sensitivity (98.7) and specificity (84.4) in differentiating sleep disturbances in patients with primary insomnia vs normal-sleeping controls(Backhaus, Junghanns, Broocks, Riemann, & Hohagen, 2002). It is composed of 19 items concerning sleep length and quality and any disturbances over the prior month. These yield seven subscales concerning subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medications, and daytime dysfunction (Buysse, Reynolds III, Monk, Berman, & Kupfer, 1989). Sleep duration and quality the night before participating in this study was assessed using the Recent Sleep Quality Questionnaire (RSQQ), a four-item measure

created for this study. Participants estimated how long they slept, compared it to their "usual" sleep in terms of duration ("less than usual" to "more than usual") and quality ("worse than usual" to "better than usual"), and reported if this sleep was sufficient ("slept too little" to "slept too much").

3.2.4. Pain Catastrophizing and Coping

Trait-level pain catastrophizing was assessed using the Pain Catastrophizing Scale (PCS), a 13-item scale of different thoughts or feelings people tend to have when experiencing pain (Sullivan, 2009). The PCS instructs participants to evaluate the degree to which they have experienced these thoughts and feeling on a five-point scale, ranging from "not at all (0)" to "all the time (4)." Scores on these items are added together to yield a total sum score of pain catastrophizing ranging from 0 to 52, with higher scores indicating greater degrees of trait pain catastrophizing. The PCS also has three subscales – rumination, magnification, and helplessness – each composed of non-overlapping subsets of the overall 13 items; however, only the total score was analyzed in the current study.

State-level pain catastrophizing was assessed using the Situational Catastrophizing Questionnaire (SCQ), which was adapted from the trait-level PCS to be used in experimental pain research (Campbell et al., 2010). It consists of six items from the PCS to assess state-level pain catastrophizing immediately after the cessation of induced pain. The SCQ includes the same five-point scale used by the PCS and the items are summed to yield scores ranging from 0 to 24, with higher scores indicating greater degrees of state pain catastrophizing.

Pain-related coping was measured with the Coping Strategies Questionnaire-Revised (CSQ-R), a 27-item measure of cognitive and behavioral strategies for managing chronic pain

derived from the longer (42-item) Coping Strategies Questionnaire (CSQ) (Riley III & Robinson, 1997). Participants rate each item on two domains, the frequency in which they tend to use the strategy and its effectiveness in coping with pain, using a seven-point Likert scale of "Never (0) to "Always (6). These items load onto six factors of cognitive and behavioral coping, including distraction, catastrophizing, ignoring pain, distancing from the pain, coping self-statements, and praying (Riley III & Robinson, 1997).

3.2.5. Task Demands

The cognitive demands of the working memory tasks were assessed using the NASA Task Load Index (NASA-TLX). Originally developed for use in aviation, but subsequently used in real-world and laboratory-based research for myriad activities and contexts, the NASA-TLX assesses the workload of a given task either while it is in progress or immediately afterwards (Hart, 2006; Hart & Staveland, 1988). This "workload" is composed of seven component dimensions, including mental, physical, and temporal (e.g., how "rushed" the pacing of the task felt) demands; performance (to what degree the respondent felt they were successful in their task); effort (how difficult it was for them to attain their achieved performance); and frustration. All seven dimensions were rated on seven-point scales with 21 gradations ranging from "very low" to "very high," except performance, which was rated from "perfect" to "failure."

3.2.6. Visual Analogue Scale

A 100-point visual analogue scale (VAS) located at the bottom of the computer monitor was used throughout the study procedures to indicate pain intensity. This VAS was a 10-inch horizontal line with 11 smaller vertical hashmarks along its length, with ratings ranging from 0

for “no pain at all” to 100 for the “worst pain imaginable.” Participants verbally indicated their VAS ratings.

3.3. Working Memory Tasks

3.3.1. Verbal Working Memory

Digit span tasks, which require the verbal repetition of number sequences of varying digit-lengths (typically two to nine digits) immediately after being heard, are well-established working memory assessments in clinical and research contexts (Berryman et al., 2013; Mazza et al., 2018; Simon et al., 2016). They measure a person’s ability to attend to, process, maintain, and manipulate verbal information (Oosterman et al., 2011). Backwards digit span tasks, where digits are recalled in reverse order of presentation, are commonly used in pain research and have been found to reliably detect significant differences in verbal working memory performance between chronic pain patients and healthy controls (Berryman et al., 2013; Mazza et al., 2018; Oosterman et al., 2011; Simon et al., 2016). A backwards digit span task was used for this study.

The administrator read participants sequences of numbers at a pace of one digit per second. After the last digit of a sequence, participants attempted to verbally recall the digits in reverse order. These trials were grouped into pairs of sequences with equal digit-lengths, beginning with two digits per pair. Participants attempted both trials of a given pair and proceeded to the next pair of trials only if they successfully recalled all digits backwards for at least one trial of a pair. Each subsequent pair of trials were one digit longer than the previous pair, up to a nine-digit maximum, after which the task ended. If participants did not correctly recall the digits backwards in either trial of a given pair, the task was discontinued. Between each trial, the administrator asked participants to verbally rate their pain using a visual analogue scale

(VAS) (see below). Each successful trial was scored for one point, up to a maximum total score of 16. Participants' scores were then multiplied by the number of digits in the last correctly completed sequence (a maximum of 9) to compute a final score (a maximum of 144). Higher scores indicated better verbal working memory performance.

3.3.2. Non-Verbal Working Memory

The Corsi block-tapping task, an assessment of non-verbal, visuospatial working memory, involves an administrator presenting nine blocks distributed between them and a participant (Kessels, Van Zandvoort, Postma, Kappelle, & De Haan, 2000). The administrator touches the blocks in a particular order and the participant must replicate the administrator's sequence in the same order demonstrated. The number of target blocks in a given sequence can range from two to nine. The Corsi block-tapping task has been used to assess working memory in chronic pain populations (e.g., Luerding et al., 2008).

The current study employed Inquisit's Corsi block tapping task, which is a computerized version of the original task (Millisecond Software, 2015). A desktop computer monitor displayed nine boxes, illuminated in a pre-set order, identical for every participant. After viewing a 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 to prompt participants to verbally rate their pain using the VAS.

Similar to the backwards digit span task, 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 of a given pair, the test was discontinued. Each successful trial was scored for one point, up to a maximum score of 16. Participants' scores were then multiplied by the number of boxes in the last correctly completed sequence (a maximum of 9) to compute a final score (a maximum of 144). Higher scores indicated better non-verbal working memory performance.

3.4. Apparatuses

3.4.1. Sphygmomanometer

A Medline Standard Handheld Aneroid Sphygmomanometer was used to monitor the participants' blood pressure. This device will henceforth be referred to as a BP cuff.

3.4.2. Handgrip Dynamometer

Maximum grip strength in the non-dominant hand was determined using a CAMRY Digital Hand Dynamometer. This device was later used by participants in the pain group as part of the ischemic pain induction task.

3.5. Procedures

The IUPUI Institutional Review Board approved this study (IRB # 1806830022).

3.5.1. Initial Procedures

Upon arriving at the IUPUI Pain Research Laboratory, participants completed the informed consent process and were screened for recent usage of alcohol, tobacco, nicotine, caffeine, and analgesic medications, as per instructions when they were previously screened for exclusion criteria via telephone. Participants who violated the substance use instructions were deemed temporarily ineligible to participate, but allowed to reenroll at a later date. Next, participants completed the demographics questionnaire, PCS, CSQ-R, GAD-7, PHQ-8, PSQI, and RSQQ, presented in random order. Then, participants were randomized to either a pain task or a no-pain control condition, equally balanced by sex. Each group was instructed in their respective tasks (though without being explicitly told whether they were in the pain or control group), the two working memory tasks, and the use of the VAS for pain ratings.

3.5.2. Ischemic Pain Induction

Participants in the pain condition underwent a submaximal effort tourniquet test (SETT) procedure to induce ischemic pain, using a protocol described by Dannecker & George (2009). The administrator ascertained participants' non-dominant hand, which was then used in the remaining procedures. Participants were asked to remove any jewelry, wristwatches, or other accessories from their arm. A handgrip dynamometer was used to determine participants' maximum grip strength and an uninflated BP cuff was placed on their bicep above the elbow. Then, participants were instructed to lift their arm directly above their head for 30 seconds to desanguinate venous blood. After 30 seconds elapsed, the BP cuff was inflated to 260 mmHg by

the administrator, and participants slowly lowered their arm to a resting position on the armrest of their chair. Then, participants performed 20 gripping repetitions (reps) with the handgrip dynamometer while the administrator maintained the BP cuff pressure to 230-250 mmHg. The reps involved the participant gripping and holding the dynamometer at 50% of their maximum grip strength for two seconds and then releasing and relaxing for two seconds before gripping again. After completing the gripping exercises, participants were instructed to say "pain" upon first feeling pain in their arm and to verbally rate their pain level on the VAS. From this point, the administrator monitored the BP cuff to maintain pressure at 230-250 mmHg, adjusting pressure when necessary. Participants were asked to remain in the pain task for as long as possible, and to say "stop" when they could no longer endure the pain, at which time they were again asked to verbally rate their pain. The administrator then deflated the BP cuff and removed it from their arm. Barring the participants stopping the pain procedure themselves in this manner, the administrator deflated and removed the BP cuff after 15 minutes elapsed, at which time the participants also gave a final pain rating.

Participants in the control group underwent similar procedures (including VAS ratings), but did not have their BP cuff inflated, nor did they perform any gripping exercises with the handgrip dynamometer beyond the initial measurement of their maximum grip strength before the deflated BP cuff was placed on their arm.

3.5.3. Working Memory Assessment

During the pain and control procedures (e.g., after all gripping exercises were completed in the pain group and after the BP cuff was placed in the control grip), participants completed the two working memory tasks, which were counterbalanced. If the participants said "stop" at any

point during the working memory tasks, their BP cuff was deflated and removed as indicated, and the working memory tasks continued, with the administrator recording which participants failed to complete both working memory tasks while in their respective conditions. For the remaining participants, after completing both working memory tasks, they were asked to sit quietly in their pain or control condition 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."

3.5.4. Post-Task Procedures

After completing the working memory tasks and their pain or control condition, all participants completed the SCQ and two versions of the NASA-TLX in counterbalanced order, one for each working memory task.

3.6. Analyses

3.6.1. Power Analysis

To estimate the sample size needed to obtain adequate power for the primary analyses, I conducted a Monte Carlo power analysis simulation for indirect effects using an application for RStudio developed by Schoemann, Boulton, & Short (2017) (RStudio Team, 2015). This power analysis simulation requires parameters for the predictor, mediator, and outcome variables, including the correlation coefficients for paths a, b, and c', and variances for each of the three variables (Schoemann, Boulton, & Short, 2017). For this analysis, a correlation coefficient corresponding to a moderate effect size (0.3) was chosen for path a (group → state-level pain catastrophizing) due to the lack of relevant available literature from which to draw a precise estimate for this path. Campbell et al. (2010) found correlations of at least 0.3 between non-ischemic pain tasks and the SCQ, while Dannecker & George (2009) found a correlation of -

0.477 between the duration of an ischemic pain task and the PCS. Therefore, this correlation coefficient was chosen as a conservative estimate of the relationship between ischemic pain group and state-level pain catastrophizing.

The variances in ischemic pain intensity (3.61) and the SCQ (3.89) were also obtained from the aforementioned two studies (Campbell et al., 2010; Dannecker & George, 2009). The correlation coefficient (-0.554) for path b (state-level pain catastrophizing → working memory) was obtained from Legarreta et al. (2016). The correlation coefficient (-0.226) for path c' (group → working memory) and the variance (2.800) in working memory performance were obtained from Berryman et al. (2013). After inputting the parameters, I incrementally adjusted the sample size until a minimum power of 0.80 was achieved at a confidence level of 0.95 with 10,000 bootstrapped replications. The results indicated that a sample size of 83 was necessary to satisfy these benchmarks. To facilitate equal distribution of random assignment across the experimental conditions and counterbalancing of the working memory tasks, I increased the total minimum sample size to 88 for the current study.

3.6.2. Preliminary Analyses and Statistical Assumptions

Before performing any other analyses on the collected data, I computed the descriptive statistics for the overall sample's demographics. I then analyzed the data for normality and other statistical assumptions of the parametric tests originally proposed for hypothesis testing. As the data for all measures other than the NASA-TLX for both working memory tasks were not normally distributed, I used the appropriate non-parametric tests to assess any differences between the pain and control groups. Subsequently, I used the corresponding parametric tests (i.e., chi square tests of independence, independent samples t-tests) to analyze the same between-group differences, as they are robust even with deviations from normality in large sample sizes,

as in that of this study (N = 102). The non-parametric and parametric tests yielded similar results; therefore, for simplicity, only the parametric test results will be reported here.

3.6.3. Hypothesis 1

Participants' final pain scores (reported when participants stopped the pain task or after 15 minutes elapsed) and SCQ total scores were analyzed using independent samples t-tests to compare pain and state-level pain catastrophizing, respectively, between the pain and control groups.

3.6.4. Hypothesis 2

Participants' backwards digit span and Corsi block-tapping task scores were also analyzed using independent samples t-tests to compare verbal and non-verbal working memory, respectively, between the pain and control groups.

3.6.5. Hypothesis 3

To test my third hypothesis that state-level pain catastrophizing would mediate the relationships between pain and verbal (Figure 1) and non-verbal working memory (Figure 2), I used a bootstrapping procedure with 10,000 resamples, as described in Preacher & Hayes (2008), and Hayes' PROCESS macro with model 4 for SPSS (Corp., 2017; Hayes, 2017). While SCQ and working memory task scores were non-normally distributed, bootstrapping through PROCESS is robust to violations of normality assumptions (Hayes, 2017). This procedure generated regression coefficients, p-values, and 95% confidence intervals (CIs) for group membership (i.e., pain vs control) predicting state-level pain catastrophizing scores (path a) and state-level pain catastrophizing scores predicting verbal [non-verbal] working memory scores

(path b). These analyses also provided estimates of the regression coefficients and 95% CIs of the overall mediating effect (group → state-level pain catastrophizing → verbal [non-verbal] working memory; path ab). Significant results for the mediating effect were indicated by a CI that did not include zero.

3.6.6. Hypothesis 4

To test my fourth hypothesis that trait-level pain catastrophizing would moderate the mediating effect of state-level pain catastrophizing for both verbal (Figure 3) and non-verbal (Figure 4) working memory, I used the procedure described in Preacher, Rucker, & Hayes (2007) and Hayes' PROCESS macro with model 7 for SPSS (Corp., 2017; Hayes, 2017). Again, while the distributions for the PCS, SCQ, and working memory task scores were not normal, this bootstrapping test of moderated mediation is robust to violations of normality assumptions. This procedure produced indices of moderated mediation with 95% CIs, and regression coefficients, p-values, and 95% CIs for the moderated a path (with separate statistics for group, PCS, and their interaction) and the same b and c' paths as in the simple mediation used for hypothesis 3. Significant results were indicated by a p-value less than .05 and/or CIs that did not include zero. In the event of a significant effect, I used the Johnson-Neyman method to determine at what level of the moderator (PCS), if any, the interaction between group and PCS transitioned to significance or non-significance (Hayes, 2017). In the event that there were no transition points, meaning that the interaction was significant at all levels of the moderator, I computed the conditional indirect effect (i.e., conditional mediation) with regression coefficients and 95% confidence intervals at average, low, and high levels of the moderator (the mean and one standard deviation below and above it, respectively).

4. RESULTS

4.1. Sample Demographic Characteristics

Of the 103 participants recruited for the study, one participant was excluded from analyses due to voluntarily withdrawing before completing the protocol, leaving a final sample of 102 participants (51 participants per group). The sample consisted of 52 females (51%) and 50 males (49%), with a mean age of 20.12 (SD = 3.73) years. In terms of race, the three most frequently endorsed identifications were White/Caucasian (n = 68, 66.7%), Black/African American (n = 10, 9.8%), and Asian/Pacific Islander (n = 7, 6.9%). For ethnicity, 22 (21.6%) participants identified as “Hispanic or Latinx or Spanish origin.” Most participants (n = 64, 62.7%) reported having no personal experience with chronic pain. Further details of the sample characteristics are reported in Table 2.

Table 2. Sample demographic statistics

		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%)		

4.2. Randomization Check

There were no statistically significant differences between the pain and control groups on demographic characteristics (Table 2) or baseline scores (i.e., prior to random assignment) on the measures (Table 3).

Table 3. Baseline measures

	All Participants (N = 102)		Pain Group (N = 51)		Control Group (N = 51)			
	M	SD	M	SD	M	SD	*t*	*p*
PCS	11.14	8.36	11.31	8.94	10.96	7.83	-0.21	0.832
PHQ-8	4.74	4.09	4.69	4.04	4.80	4.17	0.14	0.890
GAD-7	4.96	3.97	5.25	4.34	4.67	3.59	-0.75	0.457
PSQI - Average Sleep	6.83	1.40	6.69	1.27	6.97	1.51	1.03	0.307
Recent Sleep	6.87	1.53	6.74	1.37	7.01	1.68	0.91	0.367
CSQ-R Subscales								
Distraction	3.09	1.45	3.19	1.46	3.00	1.45	-0.65	0.515
Catastrophizing	1.33	1.20	1.23	1.16	1.43	1.25	0.85	0.398
Ignoring	3.06	1.28	3.04	1.12	3.08	1.43	0.07	0.878
Distancing	1.51	1.53	1.31	1.36	1.71	1.67	1.32	0.191
Self-Statements	4.35	1.21	4.32	1.14	4.37	1.28	0.18	0.855
Praying	2.42	2.09	2.24	2.18	2.59	2.00	0.85	0.396

Note: PCS = Pain Catastrophizing Scale; PHQ-8 = Patient Health Questionnaire 8; GAD-7 = Generalized Anxiety Disorder 7-item Scale; PSQI = Pittsburgh Sleep Quality Index; RSQQ = Recent Sleep Quality Questionnaire; CSQ-R = Coping Strategies Questionnaire-Revised

4.3. Hypothesis 1

The results of an independent samples t-test indicated that participants in the pain group (M = 73.33, SD = 23.30) reported higher pain ratings, on average, than those in the control group (M = 2.19, SD = 5.29), $t(100) = -21.27$, $p < .001$. The ischemic pain group (M = 10.10, SD = 5.98) also reported more state-level pain catastrophizing (SCQ) than the control group (M = .82, SD = 1.38), $t(100) = -10.79$, $p < .001$. See Table 4.

Table 4. Post-task measures

	All Participants (N = 102)		Pain Group (N = 51)		Control Group (N = 51)			
	M	SD	M	SD	M	SD	*t*	*p*
Final Pain Rating	37.76	39.50	73.33	23.30	2.19	5.29	-21.27	<.001
Total Time in Task	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	-.46	.648
SCQ	5.46	6.53	10.10	5.98	.82	1.38	-10.79	<.001
NASA-TLX Digit Span	50.98	21.56	58.71	19.60	43.25	20.81	-3.86	<.001
NASA-TLX Corsi Block	45.18	21.39	54.33	19.66	36.02	19.15	-4.77	<.001

Note: Digit = Backwards Digit Span; Corsi = Corsi Block Tapping; SCQ = Situational Catastrophizing Questionnaire; NASA-TLX = NASA Task Load Index

4.4. Hypothesis 2

The results of independent samples t-tests on backwards digit span and Corsi block-tapping task total scores indicated that, on average, there were no significant differences in verbal working memory performance between the ischemic pain (M = 32.94, SD = 19.77) and control groups (M = 34.08, SD = 16.38), $t(100) = .32$, $p = .752$, nor were there significant group differences (pain: M = 63.18, SD = 21.02; control: M = 61.16, SD = 23.33) in non-verbal working memory performance, $t(100) = -.46$, $p = .648$.

4.5. Hypothesis 3

A simple mediation analysis was conducted to determine if state-level pain catastrophizing mediated the relationship between group and verbal working memory performance. There was a significant effect of group (pain vs. control) on state-level pain catastrophizing (path a; $b = 9.27$, $p < .001$) and of catastrophizing on verbal working memory (path b; $b = -.99$, $p = .018$). There was also a significant indirect effect of group on verbal

working memory through state-level pain catastrophizing (path ab; *b* = -9.15, 95% CI: [-17.37, -2.96]). Neither the direct effect (path c'; *b* = 8.01, *p* = .124) nor the total effect (path c; *b* = -1.14, *p* = .752) of pain group on verbal working memory were significant. See Table 5 for detailed results.

Table 5. Results of simple mediation for verbal working memory.

Path	***b***	**SE**	***β***	***t***	***p***	**95% CI**
Pain Group ➔ SCQ (a)	9.27	0.86	1.46	10.79	<.001	7.57, 10.98
SCQ ➔ Digit (b)	-0.99	0.41	-.35	-.241	.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	.750	-8.27, 5.99
Indirect Effect (ab)	-9.15	3.55				-17.12, -3.17

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

Similarly, I conducted a simple mediation analysis to determine if state-level pain catastrophizing mediated the relationship between pain group and non-verbal working memory. I found a significant effect of group on state-level pain catastrophizing (path a; *b* = 9.22, *p* < .001) and of catastrophizing on non-verbal working memory (path b; *b* = -1.32, *p* = .009). There was also a significant indirect effect of group on non-verbal working memory through state-level pain catastrophizing (path ab; *b* = -12.19, 95% CI: [-22.19, -2.93]). The direct effect of pain group on non-verbal working memory was significant (path c'; *b* = 14.22, *p* = .026), but the total effect was not (path c; *b* = 2.02; *p* = .648). See Table 6 for detailed results.

Table 6. Results of simple mediation for non-verbal working memory.

Path	***b***	**SE**	***β***	***t***	***p***	**95% CI**
a	9.22	0.87	1.45	10.64	<.001	7.50, 10.94
b	-1.32	0.5	-.38	-2.66	0.009	-2.31, -.33
c'	14.22	6.28	.64	2.26	.026	1.74, 26.69
c	2.02	4.42		.46	.648	-6.75, 10.80
ab	-12.19	4.79				-21.94, -3.24

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

4.6. Hypothesis 4

I conducted a moderated mediation analysis to determine if trait-level pain catastrophizing moderated the mediated effect of group → state-level pain catastrophizing → verbal working memory in hypothesis 3 (Figure 3). The index of moderated mediation was significant (index = -.21, 95% CI: [-.51, -.03]), indicating that the indirect effect of state-level pain catastrophizing was moderated by trait-level pain catastrophizing. The results of the Johnson-Neyman analysis showed that this conditional indirect effect was significant at all levels of the moderator, including at the mean (PCS = 11.14, *b* = -9.10, 95% CI: [-17.01, -3.02]) and one standard deviation above (PCS = 19.50, *b* = -10.89, 95% CI: [-20.27, -3.53]) and below (PCS = 2.78, *b* = -7.31, 95% CI: [-14.33, -2.34]) the mean (Table 7).

Table 7. Results of moderated mediation for verbal working memory.

Path		***b***	**SE**	***t***	***p***	**95% CI**
Pain Group ➔ SCQ		6.81	1.37	5.00	<.001	4.11, 9.52
PCS ➔ SCQ		.03	.07	.36	.716	-.12, .17
Pain Group x PCS ➔ SCQ		.22	.1	2.20	.030	.02, .41
b		-.99	.41	-2.41	.018	-1.80, -.18
c'		8.01	5.17	1.55	.124	-2.24, 18.26
Index of Moderated Mediation		**Index**	**SE**			**95% CI**
PCS		-.21	0.13			-.51, -.03
Moderator Level	**PCS**	**Conditional Indirect Effect**	**SE**			**95% CI**
-1SD	2.78	-7.31	3.06			-14.33, -2.34
Mean	11.14	-9.10	3.54			-17.01, -3.02
+SD1	19.50	-10.89	4.23			-20.27, -3.53

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

A similar moderated mediation analysis was conducted on non-verbal working memory (Figure 4). The index of moderated mediation was significant (index = -.30, 95% CI: [-.66, -.05]), again indicating that the indirect effect of state-level pain catastrophizing was moderated by trait-level pain catastrophizing. The results of the Johnson-Neyman analysis indicated that this conditional indirect effect was significant at all levels of the moderator, including at the mean (PCS = 11.23; *b* = -12.09, 95% CI: [-21.72, -3.03]) and one standard deviation above (PCS = 19.58; *b* = -14.62, 95% CI: [-25.62, -3.87]) and below (PCS = 2.87; *b* = -9.56, 95% CI: [-18.89, -2.12]) the mean (Table 8).

Table 8. Results of moderated mediation of non-verbal working memory.

Path		***b***	**SE**	***t***	***p***	**95% CI**
Pain Group ➔ SCQ		6.57	1.37	4.79	<.001	3.85, 9.29
PCS ➔ SCQ		0.03	0.07	0.37	0.716	-.12, .17
Pain Group x PCS ➔ SCQ		0.23	0.1	2.33	0.022	.03, .42
b		-1.32	0.5	-2.66	0.009	-2.31, -.33
c'		14.22	6.28	2.26	0.026	1.74, 26.69
Index of Moderated Mediation		**Index**	**SE**			**95% CI**
PCS		-0.3	0.16			-.66, -.05
Moderator Level	**PCS**	**Conditional Indirect Effect**	**SE**			**95% CI**
-1SD	2.87	-9.56	4.29			-18.89, -2.12
Mean	11.23	-12.09	4.77			-21.72, -3.03
+SD1	19.58	-14.62	5.53			-25.62, -3.87

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

5. DISCUSSION

The current study investigated the role of pain catastrophizing in the pain—working memory nexus. Participants in the pain group experienced greater state-level catastrophizing than the control group, which was associated with worse verbal and non-verbal working memory performance. Moreover, trait-level catastrophizing moderated this mediation effect, as participants in the pain group who reported higher (vs. lower) trait-level catastrophizing experienced greater state-level catastrophizing, which led to worse performance on the verbal and non-verbal working memory tasks.

Participants in the pain group reported significantly higher pain and state-level pain catastrophizing than the control group, which supported hypothesis 1 and also aligned with previous research (Campbell et al., 2010; Sturgeon & Zautra, 2012). Moreover, these findings were important to my latter hypotheses by functioning as a manipulation check. They supported the contention that the ischemic pain task was indeed painful enough to differentiate between groups and provoke significant levels of state-level pain catastrophizing. Surprisingly, ten participants in the control group reported pain at some point during the task. Although it is unclear why this occurred, these participants may have reported pain (in its absence) due to demand characteristics – i.e., because they thought that was expected of them by the researcher. Alternatively, it is possible that they perceived the loose BP cuff as painful due to anxiety and conditioned expectations from prior painful experiences caused by BP cuffs (e.g., in medical settings), creating a nocebo effect (Colloca & Benedetti, 2007; Jensen et al., 2012).

T-tests revealed no significant group differences in performance on either working memory task, failing to support hypothesis 2 and conflicting with much of the experimental pain

literature (Buhle & Wager, 2010). The few dissenting studies speculated that their null results were explained by pain stimuli not taxing those neurocognitive processes required to complete the tasks (Attridge, Keogh, & Eccleston, 2019). This seems unlikely to be the culprit in the current study, as there were significant indirect effects (discussed below). Instead, it may have been an issue of sample size and power. O'Rourke & MacKinnon (2015) demonstrated that even a single mediation analysis increased statistical power over simpler analyses of total effects. Thus, between-group differences via t-tests may have been undetected due to the study being underpowered (O'Rourke & MacKinnon, 2015). Nonetheless, this possible type II error does not detract from the results or implications of the mediation and moderated mediation analyses. While the assumptions of the (outdated) causal steps approach to mediation may have been violated without significant between-group differences, the more contemporary bootstrapping approach used by the PROCESS macro does not require significant group differences or a significant total effect (Baron & Kenny, 1986; Hayes, 2017).

Participants in the pain group experienced more state-level pain catastrophizing than the control group, and this greater catastrophizing resulted in worse working memory performance, supporting hypothesis 3. This suggests that verbal and non-verbal working memory deficits for people in pain are, at least partly, due to catastrophizing about that contemporaneous pain. These findings expand upon the existing literature by providing stronger evidence for state-level pain catastrophizing as a causal mechanism in the pain—working memory nexus. While other studies have found that working memory deficits are associated with pain catastrophizing, they were unable to draw causal inferences due to a reliance on correlational designs (Baker et al., 2016; Galvez-Sánchez et al., 2018; Legarreta et al., 2016). The current results thereby lend further support to a resource-depletion model of cognitive dysfunction related to pain – i.e., pain

catastrophizing consumes limited cognitive resources, leaving less available for working memory (Solberg Nes, Roach, & Segerstrom, 2009).

Interestingly, the pain group (vs. control) performed better on the working memory tasks after controlling for state-level pain catastrophizing, although this difference was statistically significant only for the non-verbal task. These results were contrary to the existing literature; for example, Buhle & Wager (2010) found that working memory performance progressively worsened as they incrementally intensified the pain-inducing stimulus. Instead, my findings may be partially explained by the relationships among pain, arousal, and attention. Seminowicz & Davis (2006) found that pain led to faster reaction time on tasks with minimal cognitive demands. Amongst several competing explanations, they reasoned that pain led to increased alertness and arousal (which are fundamental components of working memory), which then brought attention to bear on the cognitive task, reducing reaction times (Baddeley, 1992, 2010). Thus, the ischemic pain induction used herein may have brought more arousal, alertness, and attention to the working memory tasks in the pain group (vs. control), resulting in a positive direct effect after controlling for state-level pain catastrophizing.

Regardless of their origin or clinical significance, these unexpected results may help explain the non-significant total effects in my mediation models. Total effects are calculated as the sum of indirect and direct effects (Hayes, 2017). The indirect effect for each mediation was negative and stronger than the positive direct effect. Adding these effects together led to smaller, negative, non-significant total effects. Thus, the differences in sign and strength between the indirect and direct effects meant that state-level pain catastrophizing masked the effect (also known as “suppression”) of pain on working memory when it was not included in the model (i.e., in the t-tests as part of hypothesis 2) (MacKinnon, Krull, & Lockwood, 2000). Statistical

suppression may provide an alternative explanation of why some studies failed to find significant relationships between pain and working memory (Berryman et al., 2013; Mazza et al., 2018). By not assessing it as a mediator, state-level pain catastrophizing may have masked relationships that were actually significant.

Supporting hypothesis 4, participants in the pain group who reported higher ratings of trait-level pain catastrophizing tended to engage in greater state-level pain catastrophizing, which led to worse performance on both verbal and non-verbal working memory tasks. This suggests that a person's trait tendency to catastrophizing about pain increases the likelihood and intensity of catastrophizing about pain while it occurs, thereby causing greater decrements in working memory. By focusing on working memory dysfunction and employing an experimental design, the current study expands on the correlational work of Sturgeon & Zautra (2012), who used a similar moderated mediation model with state- and trait-level pain catastrophizing predicting mood-based outcomes.

My results may also resolve the inconsistent and, at times, contradictory findings of the few studies researching the role of pain catastrophizing in the pain—cognitive function nexus. For example, in their study of fibromyalgia syndrome, Galvez-Sánchez et al. (2018) assessed pain catastrophizing with the corresponding CSQ subscale (a trait-level measure), but interpreted the measure as a state-level process (i.e., pain catastrophizing interrupting cognitive function) in correlational and multiple regression analyses. Specifically, they found pain catastrophizing was significantly correlated with worse performance on components of the Trail Making Test (a measure of attention, processing speed, executive function), but not with performance on a test of verbal learning and memory (Galvez-Sánchez et al., 2018). Legarreta et al. (2016) performed correlational analyses between PCS scores (a trait-level measure) and cognitive test scores in a

heterogeneous pain sample, and found an opposite pattern of results to Galvez-Sánchez et al. (2018) – there were no significant relationship between pain catastrophizing and the Trail Making Test, but pain catastrophizing was significantly correlated with poorer learning and recall on tests of verbal learning and memory. These divergent findings may be due to imprecision in the measure and interpretation of state vs. trait pain catastrophizing, as well as the use of correlational design and analyses. Conversely, my study demonstrates that an experimental design and moderated mediation analyses of state- and trait-level pain catastrophizing reveal previously obscured relationships of pain with cognitive function, thereby highlighting key mechanisms by which they occur.

5.1. Limitations

There were several noteworthy limitations to this study. Participants were (relatively) young, healthy, and mostly Caucasian undergraduate students of a large midwestern university. While the results may be applicable to other undergraduate populations, or even other healthy, young adults outside of higher education settings, the external validity may still be limited. For example, older adults might have more overall chronic pain experience than my sample (62.7% had no personal experience with chronic pain), which could lead to differential effects of state-level and trait-level pain catastrophizing. Samples with more chronic pain experience may compare the experimental pain and its associated state-level catastrophizing with that of their prior chronic pain. Alternatively, individuals with more (e.g., doctoral degree holders) or less (e.g., those lacking high school diplomas) education may have significant differences in available cognitive reserve, which could potentially affect the degree to which pain catastrophizing impacts working memory.

Relatedly, the results may not generalize to people with current chronic pain, who were explicitly excluded from participating. The ischemic pain paradigm was intended to replicate the sensation of clinical musculoskeletal pain, but it was a temporary, acute experience that lacked the chronicity, consequences (e.g., disability), and other unique facets of chronic pain. This possibly resulted in different experiences of pain catastrophizing, and, therefore, different ramifications for working memory performance. As this study only featured acute pain, it is difficult to tell if there are differences between acute vs. chronic experiences of pain and pain catastrophizing from just my results.

Translating the current results to other research on chronic pain is further complicated by inconsistency and heterogeneity in the clinical samples used across the extant research. In two recent meta-analyses, the associations between pain and working memory were assessed in samples with either singular diagnoses (e.g., all fibromyalgia or chronic low back pain) or a mix of chronic pain conditions. The former has the advantage of clarity through controlling for variation in diagnosis and symptomatology, though this limits external validity to other pain conditions. The latter may be more generalizable across different pain conditions and etiologies but may introduce additional variance, thus confounding the results.

Other limitations were introduced by the cognitive testing procedures. Although the backwards digit span and Corsi block tapping tasks are well-established verbal and non-verbal working memory tests in other pain research, several alternatives have been employed (e.g., the Paced Auditory Serial Addition Test) (Berryman et al., 2013; Mazza et al., 2018; Tombaugh, 2006). It is difficult to draw conclusions across the literature when there is such heterogeneity in how working memory constructs are being operationalized and measured. In their meta-analysis, Berryman et al. (2013) noted that this is an ongoing issue in research on the pain—working

memory nexus, which may account for some of the aforementioned inconsistencies. Relatedly, my results may not be applicable to other cognitive functions that appear to be impacted by acute and chronic pain (Berryman et al., 2014; Buhle & Wager, 2010; Galvez-Sánchez et al., 2018; Higgins et al., 2018; Jongsma et al., 2011; Legarreta et al., 2016; Mazza et al., 2018; Moore et al., 2017; Oosterman et al., 2011; Park et al., 2001).

5.2. Future Directions

To address the aforementioned sample limitations, future research could replicate this study with other healthy samples that are more diverse in terms of education level. Education has been shown to moderate the relationships of pain catastrophizing with pain intensity and distress (Edwards et al., 2010). Similarly, education may moderate the relationship between pain and working memory, because it is associated with the size of cognitive reserve and may buffer the deleterious effects of pain conditions, as it does for neurodegenerative conditions (Le Carret et al., 2003; Martins Da Silva et al., 2015). Replicating this study with chronic pain samples is also warranted. This could be performed through multiple studies with different singular chronic pain conditions (e.g., all fibromyalgia syndrome vs. all chronic headache and migraine) or a single study of diverse chronic pain conditions.

Another priority for future research is to incorporate sleep measures. Pain and sleep have a complex, multifaceted relationship (Finan, Goodin, & Smith, 2013). Sleep disturbances are associated with subjective complaints of working memory dysfunction in chronic pain populations, though their relationship with objective tests of working memory (e.g., digit span) is less consistent (C-oté & Moldofsky, 1997; Dick, Verrier, Harker, & Rashiq, 2008; McCracken & Iverson, 2001; Suhr, 2003). These inconsistencies may be the consequence of different methods

of assessing sleep quality and disturbances (e.g., sleep efficiency vs. total hours slept) (Glass, 2009). Combined with my results, this body of research calls for future studies to incorporate sleep disturbance as an additional mediating variable in the pain—working memory nexus, possibly in a parallel mediation alongside state-level pain catastrophizing.

These replications and extensions of the current study would stimulate interventional research to ameliorate the cognitive consequences of pain. Evidence-based interventions for pain catastrophizing would be a logical starting point. In particular, cognitive behavioral therapy (CBT) and acceptance and commitment therapy (ACT) have demonstrated small-to-moderate and moderate-to-large effect sizes, respectively, in treating pain catastrophizing, and are efficacious for treating pain in general (de C Williams, Eccleston, & Morley, 2012; Hughes, Clark, Colclough, Dale, & McMillan, 2017; Schütze et al., 2018). Future research could synergize these studies with the current one to explore whether CBT and ACT yield improvements in working memory function for chronic pain populations. Furthermore, as CBT and ACT are effective in treating insomnia and other sleep problems, future research could investigate the mechanisms (i.e., state-level pain catastrophizing vs. sleep disturbances vs. a dynamic relationship between them) by which these interventions improve working memory function in chronic pain (Daly-Eichenhardt, Scott, Howard-Jones, Nicolaou, & McCracken, 2016; Taylor & Pruiksma, 2014).

In addition to expanding my results within the domain of working memory, it might also be fruitful to replicate my research for other cognitive abilities. There is reason to suspect that certain abilities, particularly executive functions, would be prime candidates for extending this line of work. Executive functions and working memory are related, yet distinct, cognitive abilities (Baddeley, Chincotta, & Adlam, 2001; Berryman et al., 2014; Hester & Garavan, 2005;

Mazza et al., 2018; Miyake, Friedman, Rettinger, Shah, & Hegarty, 2001). Since executive functions are impacted by chronic pain, pain catastrophizing's role in the pain—executive function nexus is likely in similar direction, if not degree, to that of working memory (Berryman et al., 2014).

5.3. Conclusion

Overall, findings of the current study support the role of pain catastrophizing as a causative factor in the relationship between pain and working memory dysfunction. Future research should expand upon these results to explore whether a similar causal relationship holds in chronic pain samples, which could then allow for development or adaptation of interventions to reduce pain catastrophizing and thereby ameliorate working memory dysfunction in these patients.

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APPENDIX

A.1: Demographics Questionnaire

1) Age: _____

2) Sex/Gender:

- ☐ Male
- ☐ Female
- ☐ Other (please specify): _____

3) Race:

- ☐ Asian/Pacific Islander
- ☐ Black/African American
- ☐ Native American/Eskimo/Aleut
- ☐ White/Caucasian
- ☐ Other (please specify): _____
- ☐ Multiple racial backgrounds (please describe): _____

4) Ethnicity:

- ☐ Hispanic or Latinx or Spanish origin
- ☐ Not Hispanic or Latinx or Spanish origin

5) What is your marital status?

- ☐ Single
- ☐ Married
- ☐ Widowed
- ☐ Divorced
- ☐ Separated
- ☐ Cohabitating

6) What is your annual income?

- ☐ $0 - $25,000
- ☐ $25,001 - $50,000
- ☐ $50,001 - $75,000
- ☐ $75,001 - $100,000
- ☐ $100,001 or more

7) What is your work status? (check all that apply)

- ☐ Not working
- ☐ Student
- ☐ Part-time employee
- ☐ Full-time employee

8) What is your student status?

- ☐ Freshman
- ☐ Sophomore
- ☐ Junior
- ☐ Senior
- ☐ Other (please specify): _____

9) College Major (no abbreviations, please): _____

10) Current cumulative (overall) GPA: _____

11) Current major GPA: _____

12) Rate your level of personal experience (i.e., your own prior experiences) with chronic pain (pain lasting longer than 3 months):

- ☐ No experience
- ☐ Minimal experience
- ☐ Some experience
- ☐ Much experience
- ☐ Extensive experience

A.2: Pain Catastrophizing Scale

PCS

Client No.: _____ Age: _____ Sex: M(__) F(__) Date: _____

Everyone experiences painful situations at some point in their lives. Such experiences may include headaches, tooth pain, joint or muscle pain. People are often exposed to situations that may cause pain such as illness, injury, dental procedures or surgery.

We are interested in the types of thoughts and feelings that you have when you are in pain. Listed below are thirteen statements describing different thoughts and feelings that may be associated with pain. Using the following scale, please indicate the degree to which you have these thoughts and feelings when you are experiencing pain.

0 – not at all **1** – to a slight degree **2** – to a moderate degree **3** – to a great degree **4** – all the time

When I'm in pain …

1. ☐ I worry all the time about whether the pain will end.
2. ☐ I feel I can't go on.
3. ☐ It's terrible and I think it's never going to get any better.
4. ☐ It's awful and I feel that it overwhelms me.
5. ☐ I feel I can't stand it anymore.
6. ☐ I become afraid that the pain will get worse.
7. ☐ I keep thinking of other painful events.
8. ☐ I anxiously want the pain to go away.
9. ☐ I can't seem to keep it out of my mind.
10. ☐ I keep thinking about how much it hurts.
11. ☐ I keep thinking about how badly I want the pain to stop.
12. ☐ There's nothing I can do to reduce the intensity of the pain.
13. ☐ I wonder whether something serious may happen.

…Total

A.3: Coping Strategies Questionnaire-Revised

Name: _____ Date: _____

Individuals who experience pain have developed a number of ways to cope, or deal with their pain. Below are a list of things that patients have reported doing when they feel pain. For each activity, I want you to indicate, using the scale below, how much you engage in that activity when you feel pain and how effective this activity is in controlling pain, where a 0 indicates never, a 3 indicates sometimes, and a 6 indicates always. You can use **any point along the scale.** Remember, for each item you will be making two ratings.

Frequency

0 1 2 3 4 5 6

Never Sometimes Always

Effectiveness in controlling pain

0 1 2 3 4 5 6

Never Sometimes Always

1. When I feel pain, I try to feel distant from the pain, almost as if the pain was in somebody else's body.

Frequency _____ Effectiveness in controlling pain _____

2. I try to think of something pleasant.

Frequency _____ Effectiveness in controlling pain _____

3. It's terrible and I feel it's never going to get any better.

Frequency _____ Effectiveness in controlling pain _____

4. I tell myself to be brave and carry on despite the pain.

Frequency _____ Effectiveness in controlling pain _____

5. I tell myself that I can overcome the pain.

Frequency _____ Effectiveness in controlling pain _____

6. It's awful and I feel that it overwhelms me.

Frequency _____ Effectiveness in controlling pain _____

7. I feel my life isn't worth living.

Frequency _____ Effectiveness in controlling pain _____

8. I pray to God it won't last long.

Frequency _____ Effectiveness in controlling pain _____

9. I try not to think of it as my body, but rather as something separate from me.

Frequency _____ Effectiveness in controlling pain _____

10. I don't think about the pain.

Frequency _____ Effectiveness in controlling pain _____

11. I tell myself I can't let the pain stand in the way of what I have to do.

Frequency _____ Effectiveness in controlling pain _____

12. I don't pay any attention to it.

Frequency _____ Effectiveness in controlling pain _____

13. I pretend it's not there.

Frequency _____ Effectiveness in controlling pain _____

Frequency						
0	1	2	3	4	5	6
Never			Sometimes			Always

Effectiveness in controlling pain						
0	1	2	3	4	5	6
Never			Sometimes			Always

14. I worry all the time about whether it will end.

Frequency _____ Effectiveness in controlling pain _____

15. I replay in my mind pleasant experiences in the past.

Frequency _____ Effectiveness in controlling pain _____

16. I think of people I enjoy doing things with.

Frequency _____ Effectiveness in controlling pain _____

17. I pray for the pain to stop.

Frequency _____ Effectiveness in controlling pain _____

18. I imagine that the pain is outside of my body.

Frequency _____ Effectiveness in controlling pain _____

19. I just go on as if nothing happened.

Frequency _____ Effectiveness in controlling pain _____

20. Although it hurts, I just keep on going.

Frequency _____ Effectiveness in controlling pain _____

21. I feel I can't stand it anymore.

Frequency _____ Effectiveness in controlling pain _____

22. I ignore it.

Frequency _____ Effectiveness in controlling pain _____

23. I rely on my faith in God.

Frequency _____ Effectiveness in controlling pain _____

24. I feel like I can't go on.

Frequency _____ Effectiveness in controlling pain _____

25. I think of things I enjoy doing.

Frequency _____ Effectiveness in controlling pain _____

26. I do something I enjoy, such as watching TV or listening to music.

Frequency _____ Effectiveness in controlling pain _____

27. I pretend it's not a part of me.

Frequency _____ Effectiveness in controlling pain _____

A.4: Generalized Anxiety Disorder 7-Item Scale

Over the last 2 weeks, how often have you been bothered by the following problems?	Not at all sure	Several days	Over half the days	Nearly every day
1. Feeling nervous, anxious, or on edge	0	1	2	3
2. Not being able to stop or control worrying	0	1	2	3
3. Worrying too much about different things	0	1	2	3
4. Trouble relaxing	0	1	2	3
5. Being so restless that it's hard to sit still	0	1	2	3
6. Becoming easily annoyed or irritable	0	1	2	3
7. Feeling afraid as if something awful might happen	0	1	2	3
Add the score for each column	+	+	+	
Total Score *(add your column scores)* =				

If you checked off any problems, how difficult have these made it for you to do your work, take care of things at home, or get along with other people?

Not difficult at all _____
Somewhat difficult _____
Very difficult _____
Extremely difficult _____

A.5: Patient Health Questionnaire 8

QUESTIONNAIRE

Over the last 2 weeks, how often have you been bothered by any of the following:

		Not at all	Several Days	More than half the days	Nearly every day
PHQ.1	Little interest or pleasure in doing things?	☐ 0	☐ 1	☐ 2	☐ 3
PHQ.2	Feeling down, depressed or hopeless?	☐ 0	☐ 1	☐ 2	☐ 3
PHQ.3	Trouble falling or staying asleep, or sleeping too much?	☐ 0	☐ 1	☐ 2	☐ 3
PHQ.4	Feeling tired or having little energy?	☐ 0	☐ 1	☐ 2	☐ 3
PHQ.5	Poor appetite or overeating?	☐ 0	☐ 1	☐ 2	☐ 3
PHQ.6	Feeling bad about yourself-or that you are a failure or have let yourself or your family down?	☐ 0	☐ 1	☐ 2	☐ 3
PHQ.7	Trouble concentrating on things, such as reading the newspaper or watching television?	☐ 0	☐ 1	☐ 2	☐ 3
PHQ.8	Moving or speaking so slowly that other people could have noticed? Or the opposite—being so fidgety or restless that you have been moving around a lot more than usual?	☐ 0	☐ 1	☐ 2	☐ 3

A.6: Pittsburgh Sleep Quality Index

During the past month,

1. When have you usually gone to bed? _____
2. How long (in minutes) has it taken you to fall asleep each night? _____
3. What time have you usually gotten up in the morning? _____
4. A. How many hours of actual sleep did you get at night? _____
   B. How many hours were you in bed? _____

5. During the past month, how often have you had trouble sleeping because you	Not during the past month (0)	Less than once a week (1)	Once or twice a week (2)	Three or more times a week (3)
A. Cannot get to sleep within 30 minutes				
B. Wake up in the middle of the night or early morning				
C. Have to get up to use the bathroom				
D. Cannot breathe comfortably				
E. Cough or snore loudly				
F. Feel too cold				
G. Feel too hot				
H. Have bad dreams				
I. Have pain				
J. Other reason (s), please describe, including how often you have had trouble sleeping because of this reason (s):				
6. During the past month, how often have you taken medicine (prescribed or "over the counter") to help you sleep?				
7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?				
8. During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?				
9. During the past month, how would you rate your sleep quality overall?	Very good (0)	Fairly good (1)	Fairly bad (2)	Very bad (3)

A.7: Recent Sleep Quality Questionnaire

1. Estimate the total time you slept last night: _____

2. Compare this amount to your usual time asleep:
    ____ More than usual
    ____ About the same as usual
    ____ Less than usual

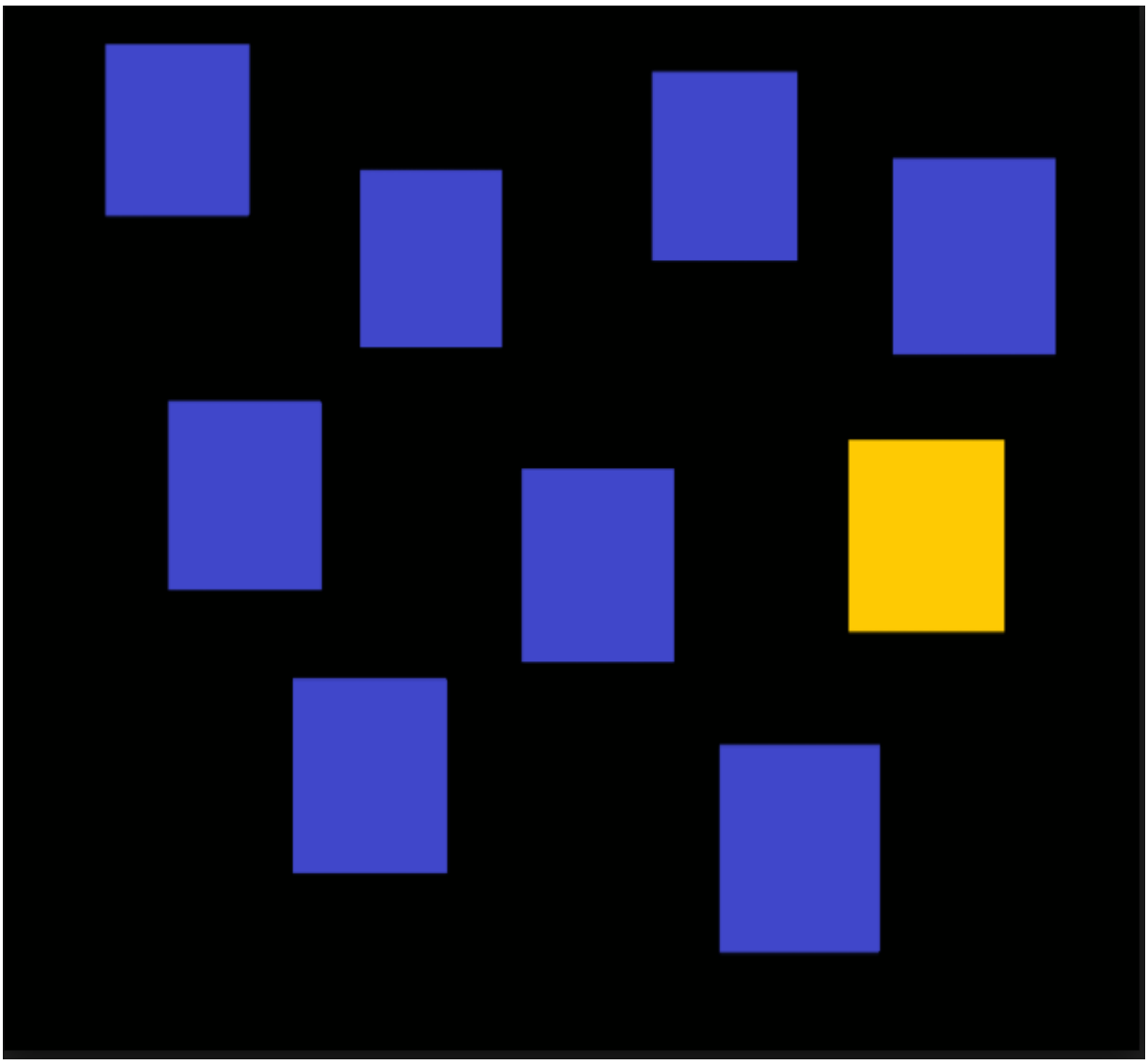
3. How would you describe your sleep last night compared with your typical night?
    ____ Better than usual
    ____ About the same as usual
    ____ Worse than usual

4. Did you get enough sleep last night?
    ____ Slept too little
    ____ Slept just the right amount
    ____ Slept too much

A.8: Backwards Digit Span

Backwards Digit Span				Item Score 0 Or 1
1	1)	6—4	*4—6*	
	2)	7—5	*5—7*	
	Pain Rating 0-100:			
2	1)	9—2—6	*6—2—9*	
	2)	5—7—4	*4—7—5*	
	Pain Rating 0-100:			
3	1)	9—7—2—8	*8—2—7—9*	
	2)	8—6—9—4	*4—9—6—8*	
	Pain Rating 0-100:			
4	1)	3—4—8—5—6	*6—5—8—4—3*	
	2)	6—8—4—5—1	*1—5—4—8—6*	
	Pain Rating 0-100:			
5	1)	8—1—4—7—3—5	*5—3—7—4—1—8*	
	2)	6—5—8—4—2—7	*7—2—4—8—5—6*	
	Pain Rating 0-100:			
6	1)	2—6—3—9—4—1—8	*8—1—4—9—3—6—2*	
	2)	8—2—6—9—3—7—4	*4—7—3—9—6—2—8*	
	Pain Rating 0-100:			
7	1)	8—1—2—6—7—3—4—9	*9—4—3—7—6—2—1—8*	
	2)	3—4—6—5—1—8—2—7	*7—2—8—1—5—6—4—3*	
	Pain Rating 0-100:			
8	1)	4—9—1—3—6—8—5—7—2	*2—7—5—8—6—3—1—9—4*	
	2)	8—6—5—2—4—9—3—1—7	*7—1—3—9—4—2—5—6—8*	
	Pain Rating 0-100:			
Participant:			Total Score:	/16

A.9: Corsi Block-Tapping Task



A.10: Situational Catastrophizing Questionnaire

Directions: For the following questions, we are interested in the types of thoughts and feelings that you had while you were participating in these pain procedures. Listed below are several statements describing different thoughts and feelings that may be associated with pain. Using the following scale, please indicate the degree to which you had these thoughts and feelings during this pain testing session.

0 - not at all	1 - to a slight degree	2 - to a moderate degree	3 - to a great degree	4 - all the time

1. _____ I worried about when it would end.

2. _____ I thought that the pain might overwhelm me.

3. _____ I felt that I couldn't stand it.

4. _____ I couldn't stop thinking about how much it hurt.

5. _____ I kept wishing that it would be over.

6. _____ I felt that the procedures were awful.

A.11: NASA Task Load Index

Name	Task	Date

Mental Demand — How mentally demanding was the task?

Very Low — Very High

Physical Demand — How physically demanding was the task?

Very Low — Very High

Temporal Demand — How hurried or rushed was the pace of the task?

Very Low — Very High

Performance — How successful were you in accomplishing what you were asked to do?

Perfect — Failure

Effort — How hard did you have to work to accomplish your level of performance?

Very Low — Very High

Frustration — How insecure, discouraged, irritated, stressed, and annoyed wereyou?

Very Low — Very High