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Psychologic Influence on Experimental Pain Sensitivity and Clinical Pain Intensity for Patients with Shoulder Pain

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Abstract

Pain-related fear and pain catastrophizing are two central psychologic factors in fear-avoidance models. Our previous studies in healthy subjects indicated that pain-related fear, but not pain catastrophizing, was associated with cold pressor pain outcomes. The current study extends previous work by investigating pain-related fear and pain catastrophizing in a group of subjects with shoulder pain, and included concurrent measures of experimental and clinical pain. Fifty nine consecutive subjects seeking operative treatment of shoulder pain were enrolled in this study (24 females, mean age = 50.4, sd = 14.9). Subjects completed validated measures of pain-related fear, pain catastrophizing, and clinical pain intensity and then underwent a cold pressor task to determine experimental pain sensitivity. Multivariate regression models used sex, age, pain-related fear, and pain catastrophizing to predict experimental pain sensitivity and clinical pain intensity. Results indicated that only pain-related fear uniquely contributed to variance in experimental pain sensitivity (beta = −.42, $p < .01$). In contrast, sex (beta = −.29, $p = .02$) and pain catastrophizing (beta = .43, $p < .01$) uniquely contributed to variance in clinical pain intensity. These data provide additional support for application of fear-avoidance models to subjects with shoulder pain. Our results also suggest that pain-related fear and pain catastrophizing may influence different components of the pain experience, providing preliminary support for recent theoretical conceptualizations of the role of pain catastrophizing.

Indexing Words

Chronic pain; biopsychosocial; pain-related fear; pain catastrophizing; quantitative sensory testing; fear-avoidance model

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Perspective
This study provided additional information on how specific psychological variables potentially influence experimental and clinical pain. In this sample of subjects with shoulder pain, we replicated findings from our previous studies involving healthy subjects, as fear of pain was uniquely associated with experimental pain sensitivity. In contrast, pain catastrophizing emerged as the sole psychological variable related to clinical pain intensity.

Introduction

The Fear-Avoidance Model of Musculoskeletal Pain (FAM) is a biopsychosocial conceptualization of chronic pain that has received considerable theoretical and empirical attention.[15] According to this model, a chronic and disabling pain condition may develop from an initial injury through a reciprocal process involving fear and avoidant behavior. Specifically, the acute injury results in heightened pain-related fear, which leads to avoidance of activities that may produce pain. Persistent avoidance of this kind may lead to a disuse syndrome notable for increased pain sensitivity, psychologic distress, and chronic disability. Pain catastrophizing, a cognitive process in which pain is interpreted as highly threatening and overwhelming,[23,27] is also highlighted as a factor of importance in the FAM. Thus, two related but separate constructs, pain-related fear and pain catastrophizing, are hypothesized to be involved in the development of chronic pain and disability.[15]

Consistent with the current version of the FAM, several clinical studies have investigated pain-related fear and catastrophizing, primarily for patients with low back pain.[3,18,33,32] The experimental pain literature includes fewer studies that have concurrently examined these constructs. As a result, the pain literature does not clearly indicate if separate assessment of pain-related fear and catastrophizing is indicated because these constructs uniquely contribute to the pain experience. Our prior experimental studies have focused on investigating the influence of pain-related fear and catastrophizing on cold pressor task (CPT) outcomes for healthy subjects. Collectively these studies have demonstrated that of the two primary psychologic factors associated with the FAM, fear of pain is the more robust predictor of pain sensitivity outcomes from a CPT.[8,12] This finding was consistent across different pain indices and when alternative measures and assessment time points (pre-task vs. “in-vivo”) of pain catastrophizing were utilized.[12]

One of the remaining issues in this line of research is to determine whether fear of pain and pain catastrophizing influence experimental pain sensitivity in persons with clinical pain conditions. This is an important question to consider since the experience of clinical pain may increase the relevance of pain catastrophizing, not only for the clinical pain experience, but also in the experimental context.[2] The extant literature is marked by studies that have primarily used healthy participants and, thus, cannot directly speak to this issue. Given the many advantages of experimental pain paradigms to investigate biopsychosocial processes (e.g., stimuli standardization, methodological control), expansion of this literature to include persons with clinical pain conditions is an important pursuit.

Therefore, the purpose of the current study was to investigate if psychologic factors central to the FAM (fear of pain and pain catastrophizing) were uniquely associated with experimental pain sensitivity and shoulder pain intensity. Our previous related studies included healthy participants participating in a CPT. The present study also employed a CPT, but enrolled participants with ongoing shoulder pain. This study advances previous work in this area by simultaneously examining associations of pain-related fear and catastrophizing to determine if each uniquely contributes to experimental pain sensitivity and clinical pain intensity for patients with a pain condition. We hypothesized that consistent with our previous studies,[8,12] fear of pain would have a stronger association with experimental and clinical pain reports, in comparison to pain catastrophizing.

Materials and Methods

Subjects

Subjects with evidence of rotator cuff pathology and seeking operative treatment of shoulder pain were considered for enrollment in this study. Subjects provided informed consent for study

participation following guidelines set forth by the University of Florida's Institutional Review Board for Human Subjects.

The following inclusion criteria were adopted for this study: a) between 18 – 85 years of age, b) complaints of pain limited to anterior, lateral, or posterior shoulder, c) documented or suspected rotator cuff tendinopathy (evidence from clinical examination or imaging studies) including small (< 1 cm), medium (1 – 3 cm), and large (3 – 5 cm) tears, d) documented or suspected adhesive capsulitis (evidence from clinical examination or imaging studies), e) documented or suspected labral lesion (evidence from clinical examination or imaging studies), and f) scheduled for arthroscopic procedure. Exclusion criteria for this study were determined by subject history and consisted of the following: a) current complaints of neck, elbow, hand, low back, hip, knee, or ankle pain lasting greater than the past 3 months, b) massive or complete rotator cuff tear (defined as tear > 5 cm), c) documented shoulder osteoarthritis or rheumatoid arthritis, d) prior shoulder surgery within the past year or current complaints of pain from prior shoulder surgery, e) current shoulder fracture, tumor, or infection, f) previously diagnosed chronic pain disorder (including, but not limited to irritable bowel syndrome, fibromyalgia, temperomandibular disorder, and chronic low back pain), g) current medical management for psychiatric disorder (defined as taking 2 or more psychiatric medications), and h) current gastrointestinal or renal illness.

Measures

Demographic information—Sex, age, pain medication status, work-status, marital status, and involved upper-extremity were collected from each subject.

Self-report questionnaires—Psychological factors were assessed with previously validated self-report questionnaires commonly used in pain research. The specific psychological factors assessed were consistent with a current fear-avoidance model and were utilized in our previous studies.

Fear of pain was assessed with the Fear of Pain Questionnaire (FPQ-III).[1,16,17] The FPQ-III is a 30-item scale that measures fear about specific situations that would normally produce pain. Individual FPQ-III items range from 1 (not at all) to 5 (extreme), and the total scale has a range of 30 to 150. Examples of items from the FPQ-III include: (1) "Receiving an injection in your arm" (2) "Falling down a flight of concrete stairs" and (3) "Getting a paper cut on your finger". Psychometric studies[1,16,17] suggest acceptable levels of reliability and validity for the FPQ-III.

Pain catastrophizing was assessed with the Pain Catastrophizing Scale (PCS).[6,31,27] The PCS is a 13-item scale that assesses the degree of catastrophic cognitions an individual experiences while in pain. Individual PCS items range from 0 (not at all) to 4 (always), and the total scale has a range of 0 to 52. Examples of items from the PCS include: (1) "I worry all the time about whether the pain will end" (2) "I keep thinking about how much it hurts" and (3) "There's nothing I can do to reduce the intensity of the pain." Higher PCS scores indicate greater frequency of cognitions related to helplessness, magnification, and/or rumination of the experienced pain. Psychometric studies[6,31,27] suggest acceptable levels of reliability and validity for the PCS, both at the item and total scale levels. FPQ-III and PCS were reported as total scores for the purposes of this study.

Clinical shoulder pain was assessed with the Brief Pain Inventory (BPI), which includes a numerical rating scale (NRS) for pain intensity [29,4,5]. The BPI assesses pain intensity over 3 conditions, and since the present pain intensity rating coincided with performance of experimental procedures it was used for the purposes of this study.

Procedure—The University of Florida Institutional Review Board approved this protocol. All study procedures were completed 3–5 days before scheduled shoulder surgery. After providing informed consent, subjects were then administered the demographics form and self-report questionnaires. Next, they completed a CPT as per our previous studies,[8,12] which involved submerging their un-involved hand (extremity opposite of one with shoulder pain) in a circulating water bath maintained at 2°C (+/− .5). Subjects were asked to keep their hand immersed for as long as they could tolerate, but were instructed that they could withdraw at any time. The CPT was limited to 3 minute duration, but subjects were not informed of that time limit. During the CPT subjects indicated a) the time at which the cold sensation first began to feel painful (pain threshold); b) NRS ratings of pain intensity at 15 second intervals starting at hand immersion; and c) time to withdrawal from CPT (pain tolerance). All CPT procedures were administered by a male research assistant.

Data Analysis

Descriptive statistics were generated for demographic and self-report data, and correlation analyses characterized the bivariate relationships among the pain and psychological measures. For ethical reasons, patients' pain medications were not discontinued prior to research procedures. Therefore, a preliminary analysis compared CPT pain outcomes and clinical pain reports based on pain medication status. If pain medication had an influence on the pain measures, it would be considered as a covariate for the primary analysis. A second preliminary analysis utilized principal component analysis (PCA) to reduce the pain measures from the CPT to a single variable representing experimental pain sensitivity. The rationale for this decision was that it provided a single dependent variable for the subsequent regression models (parsimony), and our previous studies reported consistent findings for the predictive models regardless of whether threshold, tolerance, or intensity ratings were the dependent variable.[8,12]

The primary analyses involved construction of multivariate, hierarchical regression models to predict experimental pain sensitivity and clinical pain intensity. Sex and age were first entered into the models to account for any variance attributed to these factors. Next, fear of pain and pain catastrophizing were entered into the models to account for additional variance attributed to these psychological factors. Separate regression models were computed for the experimental and clinical pain measures, with each model containing the same independent variables entered in the same order. In addition to standard regression statistics, Variance Inflation Factor (VIF) coefficients were reported to assess for multicollinearity among the independent variables. This analytic approach was consistent with that employed in our previous studies.[8,12]

All analyses were performed with SPSS for Windows, Version 15.0 (SPSS Inc, Chicago, IL) and a type I error rate of 0.05. Although the construction of multiple regression models does increase the risk for Type I error inflation, we believe this concern is mitigated by the fact that the current study was a follow-up to a previous study with very specific hypotheses. Therefore, no adjustments were made to the Type I error rate.

Results

Fifty nine subjects were enrolled in this study and the descriptive data for this sample is provided in Table 1. As expected, there was a significant, positive association between fear of pain and pain catastrophizing (Table 2). Fear of pain tended to have significant associations with experimental pain sensitivity measures, whereas pain catastrophizing was correlated with clinical pain intensity (Table 2). There were no significant differences in the experimental pain sensitivity or clinical pain measures based on pain medication status ($p > .10$ for all comparisons), so this variable was not utilized as a covariate in the primary analyses. PCA indicated that 1 experimental pain sensitivity factor (eigenvalue = 2.0, 67.7% variance) was

extracted from the measures used in this study. Factor loadings were consistently high and in the expected direction for threshold (.85), NRS rating at 15 seconds (−.81), and tolerance (.81). Regression method was used to generate a factor score that represented experimental pain sensitivity in the subsequent regression models.

The final regression models for predicting experimental pain sensitivity and clinical pain intensity are reported in Table 3. In the first step of the model for experimental pain sensitivity, sex and age explained 7.3% ($p = .14$) of variance. The addition of fear of pain and pain catastrophizing explained an additional 16.1% ($p < .01$) of variance. In the final model only fear of pain (beta = −.42, $p < .01$), was a unique predictor of experimental pain sensitivity, such that higher fear of pain was associated with lower experimental pain sensitivity. In the first step of the model for clinical pain intensity, sex and age explained 12.7% ($p = .02$) of variance. The addition of fear of pain and pain catastrophizing explained an additional 18.5% ($p < .01$) of variance. In the final model, sex (beta = −.29, $p = .02$) and pain catastrophizing (beta = .43, $p < .01$) were unique predictors of clinical pain intensity. Female sex and greater pain catastrophizing was associated with greater clinical pain intensity.

Discussion

The purpose of this study was to investigate the influence of key psychologic constructs associated with the FAM on experimental pain sensitivity and clinical pain intensity. Fear of pain and pain catastrophizing were the primary variables of interest given their central role in the FAM and they were targeted in our previous studies involving healthy subjects. The current study extended our previous work by considering these variables in a cohort of subjects with clinical shoulder pain, and by investigating experimental as well as clinical pain measures. The present results were consistent with our previous studies and demonstrated that fear of pain was a unique and robust influence on experimental pain sensitivity during a CPT. In contrast, and contrary to our hypotheses, pain catastrophizing emerged as the only psychological variable uniquely associated with clinical shoulder pain intensity.

The influence of fear of pain on experimental pain sensitivity in persons with clinical shoulder pain is a novel finding. Although the influence of psychologic variables on CPT pain indices have been widely reported for healthy subjects,[7,8,12,21,27,28] few studies have included samples of subjects actively experiencing musculoskeletal pain. In our studies the relationship between fear of pain and experimental pain sensitivity has generally been consistent with what is postulated in the FAM regardless of whether the sample consisted of healthy subjects or those with shoulder pain. That is, fear of pain has a negative association with pain tolerance and threshold times and a positive association with ratings of pain intensity during the CPT. As such, the relationship between fear of pain and experimental pain sensitivity appears to be robust, at least when pain is evoked via the CPT. We have now reported this association across three separate studies, two involving healthy subjects,[8,12] and the current study involving subjects with clinical shoulder pain. Collectively, these studies provide fairly strong evidence that fear of pain is a unique and robust predictor of experimental pain sensitivity, even in multivariate models that include sex, anxiety, and pain catastrophizing.

In the current study we were able to further investigate the relationship between selected psychological variables and pain perception by utilizing subjects with shoulder pain and recording a concurrent measure of pain intensity. When the same model was used to predict clinical pain intensity, fear of pain was not associated with shoulder pain intensity ratings. Instead, pain catastrophizing was the only psychological variable uniquely associated with clinical pain intensity. Specifically, the association was positive such that greater pain catastrophizing was associated with higher pain intensity ratings. This association is consistent with what is postulated in the FAM and has been previously reported for other musculoskeletal

conditions.[13,14,26,18,3] The novel finding from this study is that this association was observed in patients with shoulder pain.

This study allowed for a unique investigation of the influence of psychological variables on different components of pain perception. The observed differences in psychological influence on clinical shoulder pain are not altogether surprising, given the distinctive differences between experimental and clinical pain experiences. Interestingly, fear of pain was associated with experimental pain, a shorter duration and less threatening stimulus. In contrast, pain catastrophizing was associated with clinical pain, a longer duration and putatively more threatening stimulus. These findings are consistent with current views of the FAM, as well as recent theoretical and empirical developments on the role of pain catastrophizing. A diathesis-stress hypothesis of catastrophizing was recently proposed by Buenaver and colleagues.[2] This hypothesis posits that catastrophizing may function as a diathesis; that is, it is normally distributed and dormant among the general population and does not prove detrimental until a pain condition emerges. Thus, pain catastrophizing requires the presence of a significant pain experience that is sufficiently intense and/or threatening before it is activated. There is some support for this diathesis-stress formulation in the literature.[2,11] Additional support for the diathesis-stress theory can be drawn from the present findings that pain-related fear and catastrophizing are differentially associated with experimental pain sensitivity and clinical pain intensity, respectively. Interestingly, our results suggest that catastrophizing's influence may be specific to clinical reports of pain intensity and not affect all dimensions of the pain experience, even in subjects with clinical pain conditions. Additional work in this area is certainly needed, with a particular focus on experimental studies that manipulate the level of threat from pain stimuli and longitudinal clinical investigations that directly test the temporal aspects of these variables.

The primary focus of this manuscript was on the psychological influence on pain perception, but we did observe an influence of participant sex. This is also potentially novel information because previous sex-difference studies have largely included healthy subjects and experimental pain sensitivity,[20] and our study suggests a similar association with sex and experimental pain sensitivity. It is less clear, however, if sex differences are present for clinical pain intensity ratings. The influence of sex was as would be expected from the experimental literature, with women reporting higher shoulder pain intensity ratings in this study. Sex differences in the frequency of specific pain conditions have been widely reported in the literature. In contrast, comparisons of intensity ratings for similar groups are not as widely reported and reports are mixed.[22,30,9] Although only preliminary conclusions can be drawn from our data, these results suggest that sex differences may exist in experimental pain sensitivity and pain intensity ratings for subjects with shoulder pain.

Several limitations of this study should be noted. This was a cross-sectional study involving correlation analyses, so no conclusions can be made regarding the temporal aspects of these relationships. Furthermore, we did not attempt to experimentally manipulate any of the psychological constructs. Therefore, we were not able to make cause and effect statements about the relationship of fear of pain and pain catastrophizing to experimental pain sensitivity or clinical pain intensity. Another limitation is that we only utilized CPT to determine experimental pain sensitivity. This was done to replicate the methodology from our previous studies involving healthy subjects.[8,12] While the CPT is considered to be a strong experimental pain model,[19] the CPT does have some drawbacks, such as evoking an autonomic response in addition to reports of pain and frequently creating a bimodal distribution of pain responders (high and low pain sensitivity). Pain can be induced via chemical, electric, and ischemic stimuli, and these stimuli may have different psychological associations. Thus, caution should be used when generalizing the current results to other experimental pain paradigms. Furthermore,

subjects with chronic pain conditions were excluded from study participation, so these results can not be applied to individuals with chronic pain syndromes.

For ethical reasons, patients' pain medications were not discontinued and although we did not find obvious differences in pain outcomes, it is conceivable that these results could be influenced by medication status. Therefore, another limitation is that we did not precisely measure pain medication status by considering type or dosage because pre-operatively these subjects are most commonly taking a non-steroidal anti-inflammatory drug or no medication at all. We also did not measure menstrual cycle phase for the female subjects, but the influence of this factor is likely minimal based on a recent review.[25] Another limitation to consider is that moderate amounts of variance were explained in the regression models, suggesting that factors other than what were included in this study have the potential to influence experimental pain sensitivity and clinical pain intensity. Finally, the psychological questionnaires were completed by subjects following standard instructions on the form, so the assessment of fear of pain and pain catastrophizing was not specific to experimental or clinical pain.

The results of the current study provide possible direction for future study. Our studies so far have focused on the CPT, and future studies should attempt to replicate these findings by using experimental pain stimuli that do not invoke autonomic responses or create high and low pain sensitivity groups. For example, our previous study involving patients with low back pain reported associations of fear-avoidance beliefs and pain catastrophizing with thermal pain stimuli.[10] In particular, these results suggest that psychological manipulation of responses to CPT might be better served by focusing on constructs associated with fear of pain, as opposed to pain catastrophizing. Our repeated finding that fear of pain influenced experimental pain sensitivity when pain catastrophizing was controlled may partially explain why attempts at experimentally manipulating pain catastrophizing during CPT have not been successful.[24] These results may also guide future study by suggesting that pain catastrophizing is a viable component to include in assessment of clinical pain in patients with shoulder pain. Pain catastrophizing has a long-standing association with low back pain and arthritis pain, but evidence that it is also associated with shoulder pain is not widespread. Future longitudinal studies that incorporate pain catastrophizing will help to determine its utility in predicting development of chronic shoulder pain.

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Table 1

Descriptive statistics for operative shoulder pain cohort (n = 59)

Variable	Values	% or SD
Sex (# females, %)	24	40.7%
Age	50.4	14.9
Hand dominance (# right handed, %)	38	64.4%
Work-related shoulder pain (# yes, %)*	8	13.6%
Employment status (# employed, %)*	30	60.0%
Marital status (# married, %)*	32	54.2%
Education*		
Less than high school	1	2.0%
Graduated high school	12	24.0%
Completed some college courses	20	40.0%
Graduated college	9	18.0%
Completed some post-graduate courses	2	4.0%
Completed post-graduate degree	6	12.0%
Pain medication (# medication, %)	27	45.8%
Fear of pain questionnaire	70.6	21.4
Pain catastrophizing scale	13.4	9.6
Cold pressor - pain threshold (time in seconds)	11.4	10.8
Cold pressor - pain intensity at 15 seconds (0 – 100 NRS)	47.1	32.3
Cold pressor – pain tolerance (time in seconds)	75.8	81.7
Cold pressor – pain intensity at tolerance (0 – 100 NRS)	84.5	19.3
Brief Pain Inventory – present pain intensity (0 – 10 NRS)	3.9	2.5

All variables are as means and standard deviations or number and percentage, NRS – numerical rating scale

* - 9 subjects did not complete this information

Table 2

Associations of Psychologic and Pain Measures

Variable	2	3	4	5	6	7
1. Fear of pain (FPQ-III score)	.291*	−.281*	.377**	.313*	−.004	.186
2. Pain catastrophizing (PCS score)		.162	.045	−.074	−.003	.464**
3. CPT pain threshold			−.543**	0.553*	.139	.017
4. CPT pain intensity rating at 15 seconds				−.455**	.274*	.225
5. CPT pain tolerance					.097	−.243
6. CPT pain intensity rating at tolerance						.046*
7. BPI present pain intensity						

* - $p < 0.05$,

** - $p < 0.01$

FPQ-III – Fear of pain questionnaire, PCS – Pain catastrophizing scale, CPT – Cold pressor task, BPI – Brief pain inventory

Table 3

Final regression models for experimental pain sensitivity and clinical pain intensity

	R^2	B* (std error)	Beta*	p-value*	VIF*
Experimental Pain Sensitivity	0.23			<.01	
Age		.008 (.009)	.12	.38	1.13
Sex		.516 (.265)	.25	.06	1.14
Fear of pain		−.019 (.006)	−.42	<.01	1.12
Pain catastrophizing		.020 (.014)	.19	.16	1.15
Clinical Pain Intensity	0.31			<.01	
Age		.003 (.020)	.02	.88	1.13
Sex		−1.469 (.604)	−.29	.02	1.12
Fear of pain		.003 (.014)	.03	.81	1.11
Pain catastrophizing		.111 (.032)	.43	<.01	1.14

* - Reported values are from the final model, sex coded for regression model (0 = female, 1 = male)