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Original Research Article

Intolerance of Uncertainty Moderates the Relationship between Catastrophizing, Anxiety, and Perceived Pain in People with Chronic Nononcological Pain

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Abstract

Objective. Substantial empirical evidence has shown that intolerance of uncertainty is a central transdiagnostic feature in psychopathology and it has been suggested to be a pain-related psychological factor contributing to the experience of chronic pain. However, research in this area is virtually nonexistent. The objective of this study was to investigate associations between pain severity, catastrophizing, and anxiety in people with chronic nononcological pain, while assuming that intolerance of uncertainty moderates these relationships. **Methods**. A convenience sample of 188 individuals with nononcological chronic pain (157 women and 32 men) participated in the study. We investigated the moderated mediation of intolerance of uncertainty between anxiety and catastrophizing and between catastrophizing and pain intensity. **Results**. The full moderated mediation model accounted for significant variance in pain intensity ($R^2 = 0.148$, $P < .001$). Intolerance of uncertainty significantly moderated the interaction between anxiety and catastrophizing (B = 0.039, SE = 0.012, 95% CI [0.015, 0.063]) and between catastrophizing and pain intensity (B = -0.034, SE = 0.010, 95% CI [−0.054, −0.014]). Anxiety and intolerance of uncertainty did not interact in predicting catastrophizing, although an interaction effect was found between intolerance of uncertainty and catastrophizing in predicting pain intensity. **Conclusion**. This study is the first to address the interrelationship of intolerance of uncertainty, catastrophizing, and anxiety in relation to perceived pain intensity. The current findings support intolerance of uncertainty as a relevant psychological variable that is distinct from other relevant constructs in the setting of pain research and treatment.

Key Words: Intolerance of Uncertainty; Catastrophizing; Anxiety; Perceived Pain; Nononcological Chronic Pain

Introduction

Intolerance of uncertainty (IU) is currently understood as a complex transdiagnostic construct defined as an individual dispositional incapacity to endure aversive responses due to the perceived absence of information and the perception of uncertainty that creates a propensity to be afraid [1]. The attempt to avoid or control uncertainty eventually leads to high levels of distress and maladaptive behaviors, such as avoidance or impulsivity [2], whereby IU has been shown to play a role in a number of psychological disorders. Thus, a recent meta-analysis has provided evidence for the transdiagnostic

nature of IU, showing that there are robust moderate associations between IU and a range of disorder symptoms, such as depression, social anxiety disorder, panic disorder, agoraphobia, generalized anxiety disorder, obsessive compulsive disorder, and eating disorders [3].

Although there is substantial empirical evidence suggesting that IU is a central feature in understanding psychopathological conditions, particularly anxiety disorders [4], almost no studies have found associations between IU and physical conditions. However, the modest available research has shown that tolerance of uncertainty could play a substantial role in adaptive coping in the face of a chronic condition [5], such as chronic pain. Thus, a recent study has suggested that IU is a pain-related psychological factor contributing to the experience of chronic pain, finding a link between greater IU and increased pain intensity [6]. Furthermore, it has been suggested that uncertainty could be a significant factor in diagnoses in which the etiology of pain is unknown [7]. In fact, high IU has been shown to be associated with higher self-reported pain severity in unpredictable acute pain experiences [8] and with self-reported pain after diagnostic procedures such as endoscopies [9].

Because IU is related to cognitive vulnerability factors such as a ruminative style, which leads to a sense of a lack of control over internal and external experiences [10], it could be assumed that IU and catastrophizing share some common variance, even though they are different psychological constructs. Hence, fear either of the unknown [11] or of pain [12] are common characteristics of these variables. It has recently been found that high IU levels predicted increased pain catastrophizing in young people with chronic pain [7]. Previous research has shown a strong association between IU and excessive pain-related worry [12]. Nonetheless, investigation in this area is still in its infancy. Furthermore, although the role of IU in the progression and maintenance of chronic pain has barely been investigated, IU is considered to be an underlying feature of several emotional disorders and it has been proposed that uncertainty serves as an alarm function that biases emotional experiences towards negative events [13], as in the case of pain.

On the other hand, a sizeable empirical literature has accumulated in support of the idea that pain catastrophizing [14] and anxiety [15] are both implicated in the onset and maintenance of chronic pain by amplifying the pain response. Moreover, Burri et al. [16] found strong genetic correlations between pain catastrophizing and other measures of negative affect, such as fear of pain and anxiety sensitivity. Based on their findings, the authors concluded that participants showing high levels of anxiety sensitivity or fear of pain would have a stronger predisposition to developing pain catastrophizing tendencies. In addition, it has been found that many anxiety and mood disorders entail difficulties in coping with uncertainty [17].

We are unaware of any studies that have investigated the relative contribution of all three variables (i.e., IU, pain catastrophizing, and anxiety) in the same statistical model. However, a better understanding of how they interact with one another could contribute to improving clinical approaches. Consequently, the purpose of the current study was to investigate associations between pain severity, catastrophizing, and anxiety among people with chronic nononcological pain, while assuming that IU moderates these relationships (see Figure 1). Given that anxiety is often related to pain severity [18], it was assumed that it may heighten perceived pain intensity. Likewise, because catastrophizing also amplifies pain intensity [14], we also assumed a direct relationship between catastrophizing and pain. Since IU is related to fear of pain, which is a common feature of anxiety and catastrophizing, it was expected to moderate the association between these variables. Thus, we hypothesized that greater levels of anxiety (as the predictor) would be associated with pain intensity (as the outcome) (H_1), that catastrophizing would mediate the association between anxiety and pain intensity (H_2), and that the indirect links between anxiety and pain severity through catastrophizing would be moderated by IU (H_3, H_4) (see Figure 1). Because it is theoretically feasible that catastrophizing could be the predictor variable and anxiety the mediating variable, we also tested this alternative mediation model.

Methods

Participants

The participants comprised a consecutive sample of 188 individuals with nononcological chronic pain (157 women and 32 men), who were referred by physicians from a pain unit of a Spanish general hospital. Participants were eligible for the study if they met the following conditions: a) having continuous or intermittent noncancer pain of at least 3 months duration, b) having a pain intensity score of at least 3, c) pain appearing at least 5 days per week, d) able to understand the Spanish language, and e) able to understand the instructions and questionnaires. Exclusion criteria were as follows: a) severe injuries that required immediate surgery, b) presence of other chronic diseases involving disability, and c) major psychiatric illness.

All participants who fulfilled the eligibility criteria were informed by their doctor of the study aims and their participation was requested. The participants who accepted were contacted by telephone to make an appointment for the assessment, which was conducted by a trained psychologist at their clinic. The research was conducted in accordance with the Declaration of Helsinki and received ethical clearance by the Institutional Ethics Review Board (ERC UMA-66–2019-H) and the Regional Hospital Ethics Committee. The recruitment process for

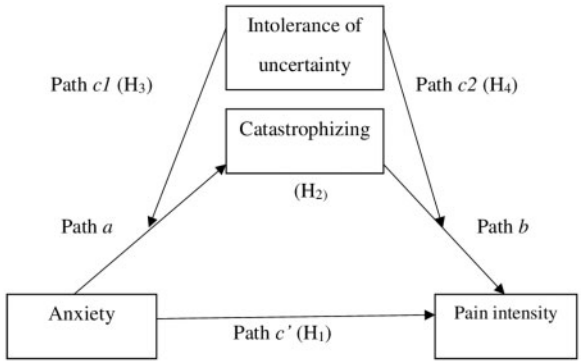

Figure 1. Theoretical moderated-mediation model of the study.

this sample was conducted from March 2018 to February 2020.

Measures

Anxiety was assessed using the anxiety subscale of the Spanish version of the Hospital Anxiety and Depression Scale (HADS) [19]. The HADS-anxiety is a seven-item self-reporting scale. Each item is rated on a 4-point scale from 1 (almost never) to 4 (often). In this study, the HADS-anxiety had a Cronbach's alpha of 0.84.

The two-item Coping Strategies Questionnaire (CSQ) was used to assess pain catastrophizing. This instrument has been shown to provide a valid and reliable measure of catastrophizing when used with chronic pain patients [20]. Respondents indicate the frequency with which they experienced two catastrophizing thoughts and feelings when in pain on a 7-point scale ranging from 0 (never) to 6 (always). In this study, the CSQ had a Cronbach's alpha of 0.86.

Intolerance of uncertainty was assessed using the Intolerance of Uncertainty Scale-short form (IUS-12) [11]. This scale is a 12-item measure of IU, which is understood as the tendency to experience negative emotional, cognitive, and behavioral reactions to uncertain situations and events. Each item is rated on a 5-point Likert-scale ranging from 1 (strongly disagree) to 5 (strongly agree). It consists of two subscales measuring prospective uncertainty intolerance and inhibitory uncertainty intolerance. In this study, we used the total scale score of the IUS-12, which had a Cronbach's alpha of 0.90.

To assess pain intensity, a composite pain intensity score was determined for each participant by calculating the average of the mildest, average, worst, and current pain. Composites of the 0–10 ratings have been shown to be highly reliable measures of pain intensity in chronic pain patients [21]. Hence, the patients were asked to rate their mildest, average, and worst pain during the past 2 weeks, as well as their current pain, on a scale ranging from 0 (not at all) to 10 (extremely painful). In this study, Cronbach's alpha was 0.85.

Data Analysis

All statistical analyses were performed using SPSS version 25.0. We analyzed the clinical and sociodemographic characteristics of the sample. Descriptive statistics and zero-order correlations between variables were then calculated for each of the continuous variables under study. Bivariate correlations were used to investigate a priori the relationships between the variables included in the model, including the association (H_1) between anxiety and pain intensity.

The mediation model and the moderated mediation model (see Figure 1) were tested using Hayes' PROCESS macro for SPSS (model 4 and model 58, respectively). Gender was included in both models as a control variable. Following the published recommendations [22], each relationship was analyzed independently with the following aims: a) to assess the mediating role of catastrophizing (H_2) in the association between anxiety and pain intensity, and b) to assess the moderated mediation of IU between anxiety and catastrophizing (H_3) and between catastrophizing and pain intensity (H_4). Sex was assigned as a covariate in the tests.

The indirect effects were tested using bias-corrected bootstrapping (n = 5,000) and 95% confidence intervals (CI) for the indices. When a 95% bootstrapped CI does not include zero, it indicates that the parameter is statistically significant. For all tests, a *P* values of less than .05 was used as a cutoff for statistical significance.

Results

Description of the Sample

Most of the participants were married or cohabiting (71%), the majority had high school education or lower (82%), and many were retired (39%). The participants had musculoskeletal pain at different locations (fibromyalgia [53%], spinal pain [28%], arthrosis [8%], and others [11%]). Their mean age was 59.90 years (SD = 10.06; age range = 27–84), and mean pain duration was 17 years (SD = 13.22). Most of the participants had been prescribed opioids (77%), and many were receiving anxiolytic medication (54%). A total of 46% of participants were prescribed opioids and anxiolytics simultaneously. Table 1 shows the main demographic and clinical characteristics of the participants.

Descriptive Statistics and Bivariate Correlations

Table 2 shows the descriptive statistics for all variables. Zero-order correlations showed significant positive associations between anxiety and both pain catastrophizing and pain intensity, between catastrophizing and pain intensity, and between IU and both anxiety and pain catastrophizing. However, no correlation was found between IU and pain intensity (see Table 2).

Table 1. Demographic and clinical characteristics of participants (N = 188)

Variable	M (SD)	N (%)
Age	59.90 (10.06)	
Sex		
Man		32 (17)
Woman		156 (83)
Marital status		
Single		10 (5)
Married/cohabiting		141 (75)
Separated/divorced		27 (14)
Widowed		10 (6)
Education		
Primary school		115 (61)
High school		55 (29)
University degree		18 (10)
Current occupation		
Active worker		33 (18)
Homemaker		52 (28)
Retired		73 (39)
Unemployed		30 (15)
Pain diagnosis		
Fibromyalgia		(38)
Spinal pain		42 (18)
Arthrosis		15 (8)
Rheumatoid arthritis		8 (4)
Others		21 (11)
Length of pain, years	17.58 (13.21)	

Mediation Model Analysis

A mediation model was computed to investigate the mediation effect of catastrophizing on the association between anxiety and pain intensity. The hypothesis that anxiety and pain intensity would be significantly related was upheld, as shown by the bivariate analysis ($r = 0.20$, $P < .01$) (H_1). The mediation model explained 9% of the variance of pain intensity ($F = 6.322$, $R^2 = 0.087$, $P < .001$). A significant positive association was found between anxiety and catastrophizing (path *a*: B = 0.158, SE = 0.025, $P < .001$, 95% CI [0.109, 0.207]) and catastrophizing was found to mediate the association between anxiety and pain intensity (H_2) (path *b*: B = 0.232, SE = 0.059, $P < .001$, 95% CI [0.115, 0.349]).

Moderated Mediation Model Analysis

A moderated mediation model was computed in order to test whether the level of IU would moderate the indirect relationships between anxiety and pain intensity via catastrophizing. Table 3 provides a summary of the model.

Overall, the full moderated mediation model accounted for significant variance in pain intensity ($R^2 = 0.148$, $P < .001$). The direct relationship (path *c*) of anxiety to pain intensity was significant (B = 0.054, SE = 0.022, 95% CI [0.011, 0.098]). Intolerance of uncertainty significantly moderated the interaction between anxiety and catastrophizing (path *a*) (B = 0.039, SE = 0.012, 95% CI [0.015, 0.063]) and between catastrophizing and pain intensity (path *b*) (B = -0.034, SE = 0.010, 95% CI [−0.054, −0.014]). However, the interaction terms indicate that anxiety did not interact with IU to predict catastrophizing (H_3) (path *c1*: B = −0.004, SE = 0.002, 95% CI [−0.008, 0.000]), thus not supporting the moderation effect in this case, whereas catastrophizing interacted with IU to predict pain intensity (H_4) (path *c2*: B = 0.010, SE = 0.005, 95% CI [0.001, 0.019]), thus supporting the moderation effect. Therefore, these results suggest that IU did not moderate the relationship between anxiety and catastrophizing, whereas IU moderated the association between catastrophizing and pain intensity.

To facilitate the interpretation of the interaction effect, we plotted predicted pain intensity by catastrophizing for both low IU and high IU (1 SD below the mean and 1 SD above the mean, respectively). The results show that under a 95% bootstrapped CI, the indirect effect reached statistical significance for mean (95% CI [0.115, 0.349]) and high (+1 SD) (95% CI [0.173, 0.529]) IU values, but not for low (−1 SD) (95% CI [−0.026, 0.251]) ones (see Figure 2). Thus, IU significantly moderated the relationship between catastrophizing and pain intensity, showing that the mediated association was stronger at higher levels of IU. In summary, the relationship between anxiety and pain intensity was mediated by catastrophizing only at high levels of IU.

Alternative Mediation Model

We tested a theoretically plausible alternative mediation model in which catastrophizing was used as the predictor variable and anxiety as the mediator. The mediation model explained 8% of the variance of pain intensity ($F = 8.383$, $R^2 = 0.083$, $P < .001$). Catastrophizing significantly and positively predicted anxiety (B = 1.146, SE = 0.178, $P < .001$, 95% CI [0.795, 1.496]), but anxiety did not mediate the association between catastrophizing and pain intensity (H2) (B = 0.029, SE = 0.021, $P = .176$, 95% CI [−0.013, 0.072]). Therefore, this alternative model did not receive empirical support. Hence, a moderated mediation model was not analyzed for these variables.

Discussion

The main aim of this study was to test a model of interactions between anxiety (as the predictor), catastrophizing (as the mediator), and pain intensity (as the outcome) in which IU would moderate the role of the mediator. As hypothesized, IU moderated the association between anxiety and catastrophizing, and between catastrophizing and pain intensity. Nevertheless, anxiety and IU did not interact in predicting catastrophizing, although an interaction effect was found between IU and catastrophizing in predicting pain intensity. Specifically, high levels of IU moderated the relationship between catastrophizing and pain intensity. Hence, catastrophizing affected the effect of anxiety on pain intensity only at high levels of IU.

Table 2. Means, standard deviations, ranges (minimum and maximum values), and correlations between study variables (N = 188)

Variable		*M*	SD	Range	1	2	3	4
1	Anxiety	19.85	5.57	7–28	–	0.45***	0.37***	0.26***
2	Catastrophizing	5.79	2.08	2–8		–	0.34***	0.26***
3.	IU	36.07	11.76	12–60			–	−0.05
4.	Pain intensity	7.11	1.54	1–10				–

***$P < .001$.

Table 3. Summary of the moderated-mediation model

R	R^2	F	df1	df2	*P*
0.385	0.148	6.322	5.000	182.00	< .001

Pathway		B	SE	*P*	LLCI	ULCI
Anxiety to catastrophizing	*a*	0.116	0.026	<.001	0.064	0.169
Catastrophizing to pain intensity	*b*	0.232	0.059	<.001	0.115	0.349
Anxiety × IU to catastrophizing	*c1*	−0.004	0.002	.028	−0.008	0.001
Catastrophizing x IU to pain intensity	*c2*	0.010	0.005	.031	0.001	0.019
Anxiety to pain intensity	*c'*	0.054	0.022	.015	0.011	0.098
Sex		−0.200	0.280	.477	−0.753	0.353

Conditional indirect effects of anxiety on pain intensity at different values of IU

IU	Effect	Boot SE	*P*	LLCI	ULCI
Low (−1 SD)	0.019	0.014	–	−0.006	0.050
Medium	0.027	0.010	–	0.010	0.048
High (+1 SD)	0.023	0.014	–	−0.003	0.052

Conditional effects of anxiety on catastrophizing at different values of IU

IU	Effect	Boot SE	*P*	LLCI	ULCI
Low (−1 SD)	0.168	0.031	<.001	0.107	0.229
Medium	0.116	0.026	<.001	0.064	0.169
High (+1 SD)	0.065	0.039	.100	−0.012	0.142

Conditional effects of catastrophizing on pain intensity at different values of IU

IU	Effect	Boot SE	*P*	LLCI	ULCI
Low (−1 SD)	0.112	0.070	.111	−0.026	0.251
Medium	0.232	0.059	<.001	0.115	0.349
High (+1 *SD*)	0.351	0.090	<.001	0.173	0.529

IU = intolerance of uncertainty; LLCI = lower level 5% confidence interval; ULCI = upper level 95% confidence interval.

Given the novelty of the study, we also analyzed an alternative mediation model, in which catastrophizing was tested as the predictor and anxiety as the mediator. However, the results did not support this model.

The findings of the current study confirm that both IU and catastrophizing share some vulnerability factors [23, 24], which is in line with the results of previous research [7, 12]. In fact, IU, like pain catastrophizing, is associated with fear of pain and frequent experiences of negative emotions [12]. Of note, it has been suggested that catastrophic cognitions, which are among the most important psychological variables related to pain intensity and disability [16], could also be transdiagnostic cognitive mechanisms, which play a role in a wide variety of disorders [25]. Interestingly, although the results show no relationship between IU and pain intensity, the conditional effects of catastrophizing on pain intensity were moderated by higher levels of IU. These findings suggest that IU may act as a type of alert which biases emotional experiences towards negative ruminations, as proposed by Gu et al. [26], or that it may act as a variable that could heighten responses to negative events. Intolerance of uncertainty is therefore a relevant variable that could contribute to explaining pain adjustment through catastrophizing.

On the other hand, some studies have demonstrated shared variance between catastrophizing and anxiety in the prediction of pain intensity [27, 28], and so these variables appear to be interrelated key psychological factors that maintain pain. Thus, it is unsurprising that the current results show an association between anxiety and both catastrophizing and pain intensity. Furthermore, it

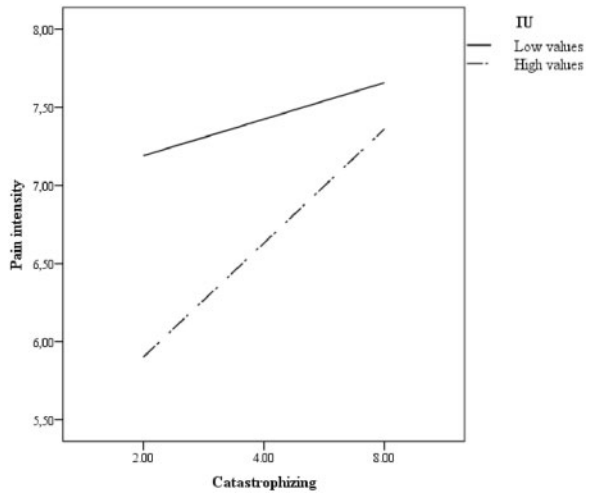

Figure 2. Interaction effect of catastrophizing and intolerance of uncertainty (IU) on pain intensity at low (-1 SD) and high (+1 SD) levels of IU. Simple slope projections of the conditional effects of catastrophizing values on pain intensity are displayed as a function of the levels of IU, which moderated the relationship between catastrophizing and pain intensity only at medium and high levels of IU.

has been proposed that similar mechanisms may be involved in the development and maintenance of chronic pain and anxiety disorders [29]. Likewise, the extensive literature on IU suggests that this variable is a key construct in anxiety, while recent results have found strong genetic correlations between pain catastrophizing, anxiety sensitivity, and fear of pain [16]. Although IU is not strictly the same as fear of pain, it is a central feature in the conceptual model of fear, which leads to anxiety and worry [1]. It thus appears that the anticipation of negative events that feature IU entail anxiety and fuel catastrophist rumination, which in turn amplifies the pain response. Nevertheless, our findings showed that anxiety did not interact with IU in predicting catastrophizing, but were independent variables. However, our findings showed that the relationship between anxiety and pain intensity was mediated by catastrophizing at high levels of IU, which would suggest that people with high levels of IU may interpret their pain experience as more threatening and, therefore, will have more catastrophizing cognitions about pain. Thus, IU appears to serve as a risk factor for a more catastrophic appraisal of pain [7]. Moreover, IU is associated with negative reactions to uncertain events, leading individuals to anticipate negative outcomes [1]. The latter aspect is also a feature of catastrophizing ruminations, which are characterized as exaggerated negative perceptions that occur during painful experiences [14]. Moreover, in the pain domain, anxiety and its nociceptive signals may act as stimuli that can trigger a cascade of catastrophic ruminations, irrespective of perceptions of uncertainty due to a perceived absence of information. In fact, catastrophizing has been considered to be the result of dispositional and situational factors [30].

The findings of the current study underlie the relevance of IU as a mechanism involved in the relationship between anxiety, catastrophizing, and pain, and confirm IU as a pain-related psychological mechanism that negatively contributes to the experience of pain [6]. Thus, given the role of IU in the onset and progression of chronic pain, a key goal in psychological pain treatment could be to reduce the level of IU. As far as we know, no studies have been conducted in which IU has been treated in people with chronic pain. However, there is empirical evidence of decreases in IU over the course of transdiagnostic treatment [31]. Such being the case, research is needed to explore the potential for adapting this psychological intervention for people with chronic pain.

Our study has several limitations. First, the cross-sectional nature of the study precludes drawing conclusions about the direction of cause-effect between variables. Future research using prospective longitudinal designs would provide more information on the causal nature of the relationships found in the current study. Second, this study was a correlational study with self-report measures. Studies that use behavioral correlates of IU would provide stronger evidence. Third, the majority of the participants were women. Sex was controlled for in the analyses conducted because it is well known that there are sex differences in pain due to biopsychosocial mechanisms (e.g., catastrophizing, anxiety) [32]. Nevertheless, the IUS-12 showed measurement invariance across sex [33] (i.e., it assesses the same construct in adult men and women). Nonetheless, the study would need to be replicated with an equal sample of men and women. Fourth, the study did not assess variables that were relevant to explaining adjustment to chronic pain, such as pain interference. Finally, although there is some evidence that there are no significant differences between trait and disorder-specific IU (e.g., generalized anxiety disorder, panic disorder, and social anxiety disorder) [34], the paucity of studies conducted in the domain of chronic pain urges further research in this area.

Conclusion

Despite the limitations of this study, it is the first to address the interrelationships between IU, catastrophizing, and anxiety in relation to perceived pain intensity. The current findings support the concept of IU as an important psychological variable distinct from other relevant constructs in the setting of pain research and treatment. Although further research is needed to better understand the interactions between these factors, the results suggest that treatment strategies could be aimed at reducing the level of IU and how best to address it clinically in the setting of multidisciplinary pain treatment. This newly emerging field within pain research warrants further study.

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