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Original article

Psychological factors are associated with local and generalized pressure pain hypersensitivity, pain intensity, and function in people with chronic shoulder pain: A cross-sectional study

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ABSTRACT

Objectives: To explore the association between psychological factors and shoulder pain intensity, function, as well as local and generalized pressure pain hypersensitivity.
Design: a cross-sectional study.
Methods: 90 participants with chronic shoulder pain were included. Pressure pain thresholds determined the presence of pain hypersensitivity. Pain intensity, function, pain self-efficacy, emotional distress, and pain catastrophizing were also assessed. Analyses were adjusted for gender and age.
Results: The diagnosis of depression (yes/no answer) was associated with both greater local (standardized $\beta = -0.19$[95%CI -0.37 to −0.00]) and generalized (standardized $\beta = -0.20$[95%CI -0.39 to −0.01]) pressure pain hypersensitivity. Greater pain self-efficacy was associated with lower local pressure pain hypersensitivity (standardized $\beta = 0.19$[95%CI 0.04 to 0.38]). The standardized beta coefficient for the diagnosis of depression indicated that this variable showed the strongest association with pressure pain hypersensitivity. Additionally, greater pain self-efficacy was associated with lower pain intensity (standardized $\beta = -0.34$[95%CI -0.51 to −0.17]) and better function (standardized $\beta = -0.47$[95%CI -0.63 to −0.30]). Greater pain catastrophizing was associated with more pain intensity (standardized $\beta = 0.35$[95%CI 0.18 to 0.52]) and worse function (standardized $\beta = 0.26$[95%CI 0.10 to 0.43]). The standardized beta coefficients for pain catastrophizing and pain self-efficacy indicated that both variables showed the strongest association with shoulder pain intensity and function, respectively
Conclusion: Psychological factors were associated with local and generalized pressure pain hypersensitivity, pain intensity, and function in people with chronic shoulder pain.

1. Introduction

Shoulder pain is a highly prevalent and costly condition (Kuijpers et al., 2006; Kuye et al., 2012; Luime et al., 2004; McBeth and Jones, 2007; Picavet and Schouten, 2003; Virta et al., 2012), which often leads to functional disability (MacDermid et al., 2004; Östör et al., 2005) and health loss (MacDermid et al., 2004). Shoulder complaints are associated with sleep disturbances (Khazzam et al., 2018), depression (Khazzam et al., 2018), work absenteeism (von Knoch et al., 2016), and healthcare utilization (e.g. opioid consumption) (Lentz et al., 2018). People with shoulder pain often consult primary care (Jordan et al., 2010; Linsell L, Dawson J, Zondervan K, Rose P, Randall T, Fitzpatrick R,

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2006). Unfortunately, a great percentage of shoulder pain (60%) presented in general practice do not completely recover 12 months after the onset (van der Windt DA, Koes BW, Boeke AJ, Devillé W, De Jong BA, 1996).

Pain hypersensitivity is a key factor to explain the persistence of symptoms in some individuals with shoulder pain (Noten et al., 2017; Sanchis et al., 2015). Local pain hypersensitivity is initially an adaptive process which alerts us to potentially harmful situations (Gangadharan and Kuner, 2013). When tissues heal, local pain hypersensitivity returns to normal baseline values (Latremoliere and Woolf, 2009). However, pain hypersensitivity can persist after the tissues heal, propagating to other body areas unrelated to tissue input (Meeus and Nijs, 2007). This phenomenon is known as generalized pain hypersensitivity (Woolf, 2011). Clinically, generalized pain hypersensitivity is measured by experimental, mechanical, thermal, and chemical methods (Nielsen et al., 2009). It is characterized by hypersensitivity to particular stimuli such as heat or cold (Nielsen et al., 2009), but also by fatigue, stress-intolerance, etc. (Nijs et al., 2010).

Generalized pain hypersensitivity is common in individuals with chronic shoulder pain (Borstad and Woeste, 2015; Noten et al., 2017; Sanchis et al., 2015). For example, Noten et al. (Noten et al., 2017) reported in their review that people with chronic shoulder pain disorders such as subacromial impingement syndrome or rotator cuff pathology often report greater generalized mechanical pain hypersensitivity when compared with healthy controls (Noten et al., 2017). Unfortunately, the complexity of these pain processing mechanisms is enormous (Harte et al., 2018; Phillips and Clauw, 2011). Many factors such as sociodemographic (Pribicevic, 2012), genetic (George et al., 2016), psychological (Martinez-Calderon et al., 2018a,b), occupational (Linaker and Walker-Bone, 2015), and biomechanical (Karas et al., 2011) factors, among others, may be all involved in the development and maintenance of shoulder pain and disability. Of all these factors, psychological factors are probably the most influential in determining how individuals with chronic pain perceive, process, interpret, and cope with their pain (Edwards et al., 2016; Vlaeyen and Linton, 2000). A large body of evidence found that negative psychological factors are associated with more pain intensity (Luque-Suarez et al., 2019; Martinez-Calderon et al., 2019) and pain hypersensitivity (Huysmans et al., 2018; Nijs et al., 2017). For example, pain catastrophizing has been associated with greater presence of generalized pain hypersensitivity in chronic low back pain (Huysmans et al., 2018). Kinesiophobia is associated with and predict more clinical pain intensity and disability in chronic musculoskeletal pain (Luque-Suarez et al., 2019). Inversely, pain self-efficacy is considered a psychological protective factor, which predict a better prognosis (lower levels of pain intensity and disability) in people with chronic musculoskeletal pain (Martinez-Calderon et al., 2018a,b). Thus, psychological factors become targeted outcomes in clinical practice (O'Sullivan PB, Caneiro JP, O'Keeffe M, Smith A, Dankaerts W, Fersum K, 2018; Schütze et al., 2018).

Specifically in chronic shoulder pain, a recent systematic review concluded that psychological factors such as emotional distress or positive expectations of recovery are associated with respectively poorer or better shoulder pain outcomes (Martinez-Calderon et al., 2018a,b). However, this systematic review concluded that the overall quality of the evidence was very low in terms of risk of bias, inconsistency, indirectness, and imprecision of the findings, and thus, further research in this field was recommended (Martinez-Calderon et al., 2018a,b). Additionally, while the association between psychological factors and pain hypersensitivity has been explored in subacute shoulder pain (Kindler LL, Valencia C, Fillingim RB, George SZ, 2011), to our knowledge, this association has not yet been studied in people with chronic shoulder pain. The understanding of the underlying causes, specifically the role that psychological factors play in shoulder pain intensity, function, as well as local and generalized pressure pain hypersensitivity, may be crucial in designing targeted interventions that may lead to better outcomes for this population.

The aims of the present cross-sectional study were: (i) to explore the potential association between psychological factors (pain self-efficacy, pain catastrophizing, emotional distress) and the presence of local and generalized pain hypersensitivity (measured with pressure algometry) in people with chronic shoulder pain; (ii) to further investigate the potential association between the psychological factors previously mentioned with shoulder pain intensity and function, measured through self-reported questionnaires.

2. Material and methods

2.1. Design

A cross-sectional design was conducted. This study was performed and reported according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) criteria (http://www.strobe-statement.org) (von Elm et al., 2007). The study was also conducted according to the Declaration of Helsinki. Ethical approval was obtained from the "X" Ethics Committee (28042016).

2.2. Participants

A convenience sample of 90 individuals with chronic shoulder pain were recruited through physiotherapists from four primary care centres. Physiotherapists, who were previously trained by the research team, screened participants for eligibility. Participants meeting the eligibility criteria were invited to participate in this study. The inclusion criteria were:

(i) at least 18-year-old participants.
(ii) Chronic shoulder pain (with a pain duration for more than three months) according to the multidimensional diagnostic criteria for chronic pain (Dworkin et al., 2016).
(iii) Shoulder pain defined as non-specific shoulder pain, subacromial pain syndrome, rotator cuff tendinopathy, adhesive capsulitis, instability without trauma, superior labral anterior posterior lesion, acromioclavicular pathology and/or shoulder osteoarthritis. Diagnosis were carried out by physiotherapists through clinical tests based on the recommendations of McClure and Michener (2015).

The exclusion criteria were:

(i) Shoulder pain due to systemic diseases such as rheumatoid arthritis.
(ii) Shoulder pain due to neurological diseases or injuries such as stroke.
(iii) Shoulder pain originated from the cervical region.
(iv) Individuals with shoulder pain receiving/planned for shoulder surgery.
(v) Inability to provide informed consent and/or complete written questionnaires.

2.3. Procedures

Physiotherapists screened participants for eligibility and explained the study protocol. Participants who satisfied our inclusion criteria were invited to participate and sign an informed consent form. Afterwards, they completed a set of self-reported questionnaires assessing shoulder pain intensity, function, pain catastrophizing, pain self-efficacy, emotional distress, and sociodemographic data. Furthermore, pressure algometry was evaluated to determine the presence of local and/or generalized pressure pain hypersensitivity.

2.4. Measurements

Demographic data: Age, gender, height, duration of symptoms, shoulder diagnostic label, and the presence of depression (with yes/no answer) were collected through a self-reported questionnaire.

Shoulder pain intensity and function: The Shoulder Pain and Disability Index (SPADI) assessed the presence of shoulder pain intensity and function. This tool is a standardized 13-item questionnaire, being each item score from 0 to 10. This tool has two subdomains: a pain scale (5 items) and a function scale (8 items). The composite SPADI score ranges from 0 to 130, with greater scores reflecting worse pain and function. The minimal clinically important difference for SPADI has been reported as 8 points (Paul et al., 2004). This tool presents an internal consistency of 0.90 Cronbach's α (Hill et al., 2011; Roy et al., 2009). The Spanish version of SPADI, which has been validated for use in Spanish language, was used (Torres-Lacomba et al., 2015).

Pain sensitivity procedures: local and generalized pressure pain hypersensitivity were assessed by pressure pain detection threshold (PPDT) assessments. PPDT assessments were conducted using a hand-held pressure algometer with a 1-cm-diameter probe (Commander™ Algometer de JTECH Medical). PPDTs were assessed unilaterally (only on the affected shoulder) at both upper trapezius and infraspinatus to evaluate the presence of local pain hypersensitivity and at the anterior tibialis to evaluate the presence of generalized pain hypersensitivity. A standardized protocol for evaluating PPDTs was used (Rolke, 2006; Rolke et al., 2006a,b. A rate of 1 kg/s was applied. Participants were instructed to report the precise moment when the sensation changed from pressure to slightly unpleasant pain. The amount of pressure in kilograms (kg) on that precise moment was recorded. This process was repeated twice unilaterally at each site, with a 1-min rest interval. The average of these two measurements was used as PPDT in the data analysis. Lower PPDT scores indicate the presence of greater pain hypersensitivity. The PPDT measurement shows good test-retest reliability in chronic musculoskeletal pain (intraclass correlation coefficient = 0.93 to 0.97; standard error of measurement = 0.70 to 0.66 kg/cm^2) (Mutlu and Ozdincler, 2015) and particularly in shoulder pain (intraclass correlation coefficient = 0.78 to 0.85; standard error of measurement = 0.39–0.70 kg/cm^2) (De Groef et al., 2017).

Pain Self-efficacy: The Pain Self-Efficacy Questionnaire (PSEQ) evaluated the presence of self-efficacy for pain. This tool contains 10 items measuring the one confidence to perform certain activities despite pain (Nicholas, 2007). Each item is scored using a 7-point Likert scale, where 0 = not confident at all and 6 = completely confident. The total score ranges from 0 to 60, with greater scores indicating greater self-efficacy for pain. PSEQ presents an internal consistency of 0.92 Cronbach's α (Asghari and Nicholas, 2001).

Pain catastrophizing: The Pain Catastrophizing Scale (PCS) evaluated catastrophic thinking about pain. It consists of 13 items describing different thoughts and feelings that individuals may have when experiencing pain. Each item is scored on a 5-point scale, where 0 = not at all and 4 = all the time. A general score and scores on three subscales (helplessness, magnification, and rumination) are obtained. The total score ranges from 0 to 52, with greater scores indicating greater pain catastrophizing. This tool has shown an internal consistency of 0.87 Cronbach's α (Osman et al., 1997). The Spanish version of PCS was used (García Campayo et al., 2008).

Emotional distress: The Hospital Anxiety and Depression Scale (HADS) evaluated the presence of emotional distress. This tool contains 14 items, seven concerning anxiety (HADS-A) and seven for depression (HADS-D). Each item is scored using a 4-point Likert scale, where 0 = absence of symptoms and 3 = maximum symptoms. Scores for each subscale ranges from 0 to 42 (Pallant JF, 2005), considering the total scores the emotional distress construct. Greater scores indicate greater emotional distress. This tool has shown an internal consistency of 0.83 Cronbach's α for HADS-A and 0.82 Cronbach's α for HADS-D (Bjelland et al., 2002). The Spanish version of HADS was used (Herrero et al., 2003; Tejero A, Guimera E, Farré JM, 1986).

2.5. Sample size estimation

A sample size of 90 individuals with chronic shoulder pain was estimated at the beginning of the study. This was based on the assumption of fifteen individuals per predictor (age, gender, presence of a diagnosis of depression, pain catastrophizing, pain self-efficacy, and emotional distress) were needed in the linear regression model (Austin and Steyerberg, 2015).

3. Data analysis

Descriptive and exploratory statistics and the Kolmogorov-Smirnov test were conducted to analyse the distribution and normality of the variables. Pearson's correlation analyses determined the presence of a significant association between psychological measures (pain catastrophizing, pain self-efficacy, and emotional distress) and shoulder pain intensity, function, as well as local and generalized pressure pain hypersensitivity. The strength of the correlation was interpreted according to the criteria of Dancey & Reidy as perfect (0.9–1); strong (0.7–0.9); moderate (0.4–0.6); weak (0.1–0.3), and zero (0) (Dancey and Reidy, 2007).

Five linear multivariate regression analyses were built to observe the direct association between psychological factors and shoulder pain intensity, function, as well as local and generalized pressure pain hypersensitivity. Independent variables (age, gender, presence of a diagnosis of depression, pain catastrophizing, pain self-efficacy, and emotional distress) which reported a statistical significance in the bivariate analysis, were put into the models through regression by forward steps, using local and generalized PPDTs measures, SPADI-pain, and SPADI-function as dependent variables. However, emotional distress did not explain significant variance in any dependent variable, and thus, emotional distress was removed from the analysis. In chronic pain, gender and age were found to be associated with poorer outcomes (Bartley and Fillingim, 2013; Boerner et al., 2018; Sorge and Strath, 2018; Sorge and Totsch, 2017) (Cassou B, Derriennic F, Monfort C, Norton J, 2002), thus, all the analyses were adjusted for these two variables.

Changes in R^2 were estimated, as well as collinearity, autocorrelation, homoscedasticity, and linearity through correlation matrix, Durbin-Watson's coefficient, tolerance, variance inflation factor, and analysis of residuals. A p-value less than 0.05 was used to determine significance. All the analyses were carried out with SPSS 25 statistical package (IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY: IBM Corp.).

4. Results

4.1. Sample characteristics

More than half of participants were females (73%). The mean age of the whole sample was 54.9 years [SD 10.0; range 54]. Most of participants reported shoulder pain symptoms for more than 12 months. The most common shoulder pain diagnosis was subacromial pain syndrome (Table 1).

Relation between shoulder function, shoulder pain intensity, local and generalized pressure pain hypersensitivity and psychological factors

Correlations between shoulder function, shoulder pain intensity, local and generalized pain hypersensitivity and psychological factors are reported in Table 2.

Local and generalized pressure pain hypersensitivity, pain catastrophizing, pain self-efficacy, and the presence of depression

A total of three regression analyses were separately conducted for PPDT at the infraspinatus, PPDT at the upper trapezius, and PPDT at the anterior tibialis as dependent outcomes. Greater PSEQ was associated with greater PPDT at the upper trapezius. The presence of depression

Table 1
Sample characteristics.

	Mean (SD; range)
Age (yrs)	54.9 (10.0; 54)
Height (m)	1.6 (0.1; 0.5)
PPDT upper trapezius (kg/cm^2)	4.9 (3.7; 20.0)
PPDT infraspinatus (kg/cm^2)	4.9 (3.4; 17.1)
PPDT anterior tibialis (kg/cm^2)	7.6 (4.8; 24.1)
Pain and function (SPADI total score 0-130)	75.6 (28.4; 122)
Number and percentage of individuals regarding duration of symptoms	3–6 months = 11 (12.2%) 6–12 months = 18 (20.0%) >12 months = 61 (67.8%)
Number and percentage of individuals with the following shoulder diagnosis	Subacromial impingement syndrome = 67 (74.4%) Calcific tendinitis of shoulder = 10 (11.1%) Supraspinatus tears = 4 (4.4%) Adhesive capsulitis = 9 (10%)
Pain self-efficacy (PSEQ final score 0-60)	37.5 (15.3; 60)
Pain catastrophizing (PCS final score 0-52)	22.4 (14.0; 51)
Emotional distress (HADS final score 0-42)	19.9 (6.1; 35)
Number and percentage of individuals regarding presence of a diagnostic of depression	No = 79 (87.8%) Yes = 11 (12.2%)

PPDT = pressure pain detection threshold; SPADI = the shoulder pain and disability index; PSEQ = the pain self-efficacy questionnaire; PCS = the pain catastrophizing scale; HADS = the Hospital anxiety and depression scale; SD = standard deviation; p = p-value; Differences statistically significant: *p < .05; **p < .001.

Table 2
Correlations [95%CI] between shoulder function, shoulder pain intensity, local and generalized pain hypersensitivity and psychological factors.

	PPDT at the infraspinatus	PPDT at the upper trapezius	PPDT at the anterior tibialis	SPADI-pain	SPADI-function
PSEQ total score	0.24* [0.03 to 0.43]	0.28* [0.06 to 0.46]	0.12 [-0.08 to 0.32]	−0.43** [-0.59 to −0.25]	−0.55** [-0.68 to −0.38]
PCS total score	−0.11 [-0.31 to 0.10]	−0.19 [-0.38 to 0.01]	−0.06 [-0.27 to 0.14]	0.41** [0.22 to 0.57]	0.34** [0.15 to 0.51]
HADS total score	0.01 [-0.19 to 0.22]	−0.04 [-0.25 to 0.17]	0.05 [-0.16 to 0.25]	0.10 [-0.10 to 0.30]	0.13 [-0.07 to 0.33]

PPDT = pressure pain detection threshold; SPADI = the shoulder pain and disability index; PSEQ = the pain self-efficacy questionnaire; PCS = the pain catastrophizing scale; HADS = the Hospital anxiety and depression scale; Differences statistically significant: *p < .05; **p < .001.

was associated with lower PPDT at the infraspinatus and lower PPDT at the anterior tibialis. The standardized beta coefficient for the diagnosis of depression indicated that this variable showed the strongest association with pressure pain hypersensitivity (see Table 3). The predictive value of the regression model for both local PPDTs was moderate (PPDT at the infraspinatus $R^2 = 0.28$; PPDT at the upper trapezius $R^2 = 0.30$) with a good adjustment for both (PPDT at the infraspinatus variance inflation factor<1.1 and tolerance over = 0.9; PPDT at the upper trapezius variance inflation factor<1.1 and tolerance over>0.93). The predictive value of the regression model for generalized PPDT was weak (PPDT at the anterior tibialis $R^2 = 0.16$) but the adjustment was good (variance inflation factor<1.2 and tolerance over>0.81) (Table 3).

Table 3
Linear regression analysis with PPDT at the infraspinatus, PPDT at the upper trapezius, and PPDT at the anterior tibialis as outcome measures.

	Standardized β	p	95% confidence interval for Standardized β Lower limit	Upper limit
PPDT at the infraspinatus				
Diagnosis of depression	−0.19	.046*	−0.37	−0.00
PSEQ total score	0.14	.146	−0.04	0.32
PPDT at the upper trapezius				
PSEQ total score	0.19	.046*	0.04	0.38
PCS total score	−0.15	.116	−0.36	0.03
PPDT at the anterior tibialis				
Diagnosis of depression	−0.20	.043*	−0.39	−0.01

Differences statistically significant: *p < .05. All the regression models were adjusted for gender and age.

Pain intensity (measured with SPADI-pain), function (measured with SPADI-function), pain self-efficacy, and pain catastrophizing

A regression analysis was built using pain intensity (SPADI-pain) and function (SPADI-function) as outcome measures. Greater pain self-efficacy was associated with lower pain intensity and better function. Greater pain catastrophizing was associated with more pain intensity and worse function. The standardized beta coefficients for pain catastrophizing and pain self-efficacy indicated that both variables showed the strongest association with shoulder pain intensity and function, respectively (see Table 4). The predictive value of the regression model was moderate for both SPADI-pain ($R^2 = 0.42$) and SPADI-function ($R^2 = 0.36$) with a good adjustment (variance inflation factor<1.03 and tolerance over>0.94) (Table 4).

5. Discussion

The purpose of this cross-sectional study was twofold: (i) to explore the potential association between psychological factors (pain self-efficacy, pain catastrophizing, and emotional distress) and the presence of local and generalized pain hypersensitivity in people with chronic shoulder pain and; (ii) to further investigate the potential association between the psychological factors previously mentioned with shoulder pain intensity and function. Considering the first aim, this study found that: (i) the diagnosis of depression was associated with greater local (lower PPDT at the infraspinatus) and generalized (lower PPDT at the anterior tibialis) pressure pain hypersensitivity and; (ii) greater pain self-efficacy was associated with lower local (greater PPDT at the upper trapezius) pressure pain hypersensitivity. The standardized beta coefficient for the diagnosis of depression indicated that this variable showed the strongest association with pressure pain hypersensitivity. Previous evidence, which supports our findings, has highlighted the association between self-efficacy and pain hypersensitivity in low back pain (Smart et al., 2012). Smart et al. (2012) reported that generalized pain hypersensitivity was cross-sectionally associated with lower levels of self-efficacy. Depression has been also associated with pain hypersensitivity (Adams and Turk, 2015). Adams and Turk (2015) reported that depression is a consistent factor in people with chronic pain, specifically in central sensitivity syndromes as fibromyalgia. On the other hand, pain catastrophizing and emotional distress were not associated with local and generalized pressure pain hypersensitivity. Our sample reported low levels of pain catastrophizing and emotional distress. These results may explain why both psychological factors were

Table 4
Linear regression analysis with shoulder pain intensity and function measured with SPADI as the outcome measure.

	Standardized β	p	95% confidence interval for Standardized β Lower limit	Upper limit
Pain intensity-SPADI-pain				
PSEQ total score	−0.34	<.001*	−0.51	−0.17
PCS total score	0.35	<.001*	0.18	0.52
SPADI-function				
PSEQ total score	−0.47	<.001*	−0.63	−0.30
PCS total score	0.26	.002*	0.10	0.43

Differences statistically significant: *p < .05. The regression model was adjusted for gender and age.

not associated with pressure pain hypersensitivity.

Regarding the second aim, our study found that: (i) greater pain self-efficacy was associated with lower pain intensity and better function; (ii) greater pain catastrophizing was associated with more pain intensity and worse function and; (iii) emotional distress was not associated with both pain intensity and disability. The standardized beta coefficients for pain catastrophizing and pain self-efficacy indicated that both variables showed the strongest association with shoulder pain intensity and function, respectively.

Pain self-efficacy refers to the belief that one can execute a determined action while in pain (Nicholas, 2007). Supporting our results, Jackson et al. (2014) reported that self-efficacy was associated with lower pain and better function, through the analysis of 86 studies including people with heterogeneous chronic pain conditions. Martinez-Calderon et al., 2018a,b) concluded that baseline self-efficacy predicts lower pain intensity and better function over time, through the analysis of 27 longitudinal studies including people with chronic musculoskeletal pain.

A large amount of evidence also reinforces the role that pain catastrophizing (Edwards et al., 2011; Leung, 2012; Martinez-Calderon et al., 2019; Quartana et al., 2009) and depression (Edwards et al., 2011) play in chronic pain outcomes. The fear-avoidance model of pain has received great attention in the context of chronic musculoskeletal pain (Norton and Asmundson, 2003; Vlaeyen and Linton, 2000, 2012). This model hypothesises that pain-related cognitions and emotions such as pain catastrophizing and pain-related anxiety facilitate hypervigilance and avoidance behaviours (Asmundson GJG, Norton PJ, 2004; Vlaeyen and Linton, 2000). With these behaviours, sedentarism and immobilization become common in individuals with chronic pain. In the long term, this situation favours the development and maintenance of chronic pain and disability that has been associated with more pain and depression states (Vlaeyen and Linton, 2000).

Our results partially agree with this theoretical framework due to greater pain catastrophizing was associated with more shoulder pain intensity and disability. However, there was not an association between emotional distress and both shoulder pain intensity and disability. In this sense, another theoretical framework, the biopsychosocial model of chronic pain (Gatchel et al., 2007), highlights that psychological factors cannot exclusively explain the maintenance of chronic pain. Multiple pathways can determine the development and persistence of chronic pain and pain-related disability. The number and duration of episodes, fluctuations of symptoms, the biopsychosocial profile of every individual, and how they use health care, can vary considerably in individuals with chronic pain (Bartley and Fillingim, 2013; Gatchel et al., 2007; Maly and Vallerand, 2018; Pearce, 2002). These factors may mediate and moderate the association between emotional distress with pain intensity and disability in individuals with chronic shoulder pain.

The findings of this study highlight the importance of pain self-efficacy and pain catastrophizing in how individuals with chronic shoulder pain perceive their pain. A large number of studies found that pain-related cognitions such as pain catastrophizing and pain self-efficacy are moderators of treatment response after physical and cognitive-behavioural interventions in chronic pain (Burns et al., 2012; Racine et al., 2016; Sherman et al., 2013; Smeets et al., 2006; Spinhoven et al., 2004; Turner et al., 2007; Vowles et al., 2007).

Furthermore, both factors has been shown to be modifiable through different interventions (Picha and Howell, 2018; Schütze et al., 2018), and therefore, potential targets for preventive approaches. Thus, clinicians should pay attention to these factors when both assessing and treating people with chronic shoulder pain.

This study presents several limitations which must be recognized. The impact that psychological factors play in shoulder pain intensity, function, local and generalized pressure pain hypersensitivity cannot be determined due to the cross-sectional nature of this study. Further prospective cohort studies are needed to clarify how these factors are interrelated over time. These studies will be valuable for the development of targeted interventions in chronic shoulder pain populations. A number of analyses were performed which may increase the likelihood of type I error. The sample size was small ($n = 90$), hence, analyses for testing mediation and moderation effects on shoulder pain outcomes cannot be conducted. Further research including large chronic shoulder pain samples are required. Sixty-seven individuals included in this study presented subacromial pain syndrome. Individuals with adhesive capsulitis (10%) were included in the same group that individuals with subacromial pain syndrome. Given the different clinical course and characteristics of adhesive capsulitis population, our results should be taken with caution. We encourage researchers to further investigate other shoulder pain conditions (e.g. shoulder instability) to corroborate these findings. Pain catastrophizing, pain self-efficacy, and emotional distress were evaluated. However, psychological factors such as optimism or fear were not analysed. Evidence has showed the importance of these factors in chronic shoulder pain (Martinez-Calderon et al., 2018a, b). Further research exploring the role that these factors play as prognostic factors of the progression of shoulder pain outcomes are warranted. Finally, sociodemographic, genetic, psychological, occupational, and biomechanical factors, among others, may be all involved in the development and maintenance of shoulder pain and disability. However, cultural, contextual, occupational, social, and genetic factors were not evaluated. These factors may behave as potential confounding variables in the association between psychological factors and shoulder pain intensity, function, local and generalized pressure pain hypersensitivity.

6. Conclusions

This cross-sectional study provided preliminary evidence about the association between psychological factors and pain intensity, function, as well as local and generalized pressure pain hypersensitivity in people with chronic shoulder pain. The diagnosis of depression was associated with more local (lower PPDT at the infraspinatus) and generalized (lower PPDT at the anterior tibialis) pressure pain hypersensitivity. Greater pain self-efficacy was associated with lower local (greater PPDT at the upper trapezius) pressure pain hypersensitivity. The standardized beta coefficient for the diagnosis of depression indicated that this variable showed the strongest association with pressure pain hypersensitivity. Additionally, greater pain self-efficacy was associated with lower pain intensity and better function. Greater pain catastrophizing was associated with more pain intensity and worse function. Emotional distress was not associated with any dependent variable. The standardized beta coefficients for pain catastrophizing and pain self-efficacy indicated that both variables showed the strongest association with shoulder pain intensity and function, respectively. However, the causality between these factors cannot be established because the cross-sectional nature of this study.

Disclosures

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Ethical approval

Ethical approval was obtained from the Costa del Sol Ethics Committee (28042016).

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