Cognitive fusion and depressive symptoms in women with chronic pain

Cognitive fusion and depressive symptoms in women with chronic pain: a longitudinal growth curve modelling study over 12-months

Abstract

This study aims 1) to explore individual differences in women with chronic pain (CP) in regard to pain intensity, functional impairment, cognitive fusion and depressive symptoms, and 2) to longitudinally test whether cognitive fusion is a significant predictor of depression symptoms, while controlling for pain intensity and functional impairment, over a 12-month period. This study follows a longitudinal design, and was conducted in a sample of 86 women with CP who responded to an online battery of questionnaires in three equally-spaced assessment moments. In order to explore the growth trajectory of variables of interest, latent growth curve models were examined. Also, correlation analyses were conducted between demographic and illness-related variables and depressive symptoms, as well as between all variables in all assessment moments. Cognitive fusion and functional impairment (but not pain intensity) were significantly associated with baseline levels of depressive symptoms. Cognitive fusion significantly predicted the growth trajectory of depressive symptoms, while pain intensity and functional impairment did not. No demographic (age, marital status, education, socio-economic) nor illness-related variables (number of CP diagnoses, duration of CP, taking medication) were associated with depressive symptoms at any point. These results suggest that the trajectory of depressive symptoms in women with CP is not predicted by the intensity of pain nor pain-related functional impairment, but rather by the tendency to get entangled with internal experiences (e.g. thoughts, emotions, physical sensations) that may or may not be related to pain-specific contents. Clinical implications are discussed.

Keyword: chronic pain; depression; cognitive fusion; pain intensity; functional impairment.

Key Practitioner Message

- Pain intensity and pain-related functional impairment at baseline are not correlated with depressive symptoms at 6-months and 12-months;
- Depressive symptoms at baseline are correlated with pain-related functional impairment at 6-months and 12-months;
- Cognitive fusion, but not pain intensity nor pain-related functional impairment, predicts changes in depressive symptoms over a 12-months period;
- Results suggest the importance of addressing the entanglement with internal experiences (i.e. cognitive fusion) not necessarily related to pain when dealing with depressive symptoms in women with chronic pain.

Introduction

Depressive symptoms are a common experience in chronic pain (CP) (e.g. Jobski, Luque-Ramos, Albrecht, & Hoffmann, 2017; McDonald, Shellman, Graham, & Harrison, 2016). Studies have found a wide-ranging prevalence of depression in CP patients (e.g. Bair, Robinson, Katon, & Kroenke, 2003; Ho, Li, Ng, Tsui, & Ng, 2011), and CP patients with lower socioeconomic status (van Hecke, Torrance, & Smith, 2013) and lower level of education (Averill, Novy, Nelson, & Berry, 1996) seem to present higher levels of depressive symptoms. The common interaction between depressive symptoms and pain, as well as their overlap in emotional and physical complaints, have long raised questions regarding the causal relationship between pain and depression (e.g. Wörz, 2003). Indeed, the relationship between depression and chronic pain is complex (e.g. Brown, 1990; Wörz, 2003), with some studies suggesting a reciprocal relationship (e.g. Kroenke, Wu, Bair, Krebs, Damush, & Tu, 2011). However, some longitudinal results suggest that neither pain intensity nor pain disability per se significantly predict depression (e.g. Lerman, Rudich, Brill, Shalev, & Shahar, 2015). However, given that the co-occurrence of depression and CP yields greater negative interference on patients´ health and functioning (Rayner, Hotopf, Petkova, Matcham, Simpson, & McCracken, 2016), it is crucial to have a better understanding of the mechanisms through which depression and CP interact over time.

The role of psychological factors in the aetiology of CP symptomatology is widely recognized (e.g. Gatchel, Peng, Peters, Fuchs, & Turk, 2007), including the presence of depressive symptoms in CP (Turk, Okifuji, & Scharff, 1995). However, the majority of studies have focused on the content of specific thoughts and beliefs (e.g. Crombez, Eccleston, Van Damme, Vlaeyen, & Karoly, 2012), rather than on the psychological processes that underlie different thoughts. Indeed, there seems to be a

growing interest in transdiagnostic psychological processes, and in process-based psychological approaches (Hayes & Hofmann, 2017). The Psychological Flexibility Model (PFM) is a transdiagnostic approach that suggests that psychological suffering is the result of excessive entanglement with internal experiences (e.g. thoughts, emotions, physical sensations), which in turn leads to actions that are guided by avoidance rather than by meaningful personal values and goals (Hayes, Strosahl, & Wilson, 1999). During the last two decades, the PFM has been a useful lens through which psychological suffering in CP is explored (Hughes, Clark, Colclough, Dale, & McMillan, 2017; McCracken, Barker, & Chilcot, 2014a; McCracken & Vowles, 2014), and several studies have found that psychological (in)flexibility reduces the impact of CP disability (e.g. McCracken & Velleman, 2010) and depressive symptoms in CP (Scott, Hann, & McCracken, 2016). However, although the impact of all core components of psychological (in)flexibility has been studied in laboratory (see Levin, Hildebrandt, Lillis, & Hayes, 2012 for an in depth discussion), some have been neglected in CP studies, perhaps due to a lack of suitable measures (McCracken & Morley 2014).

Cognitive fusion is a central component of psychological inflexibility. It is defined as the tendency to get entangled with one´s internal experiences, instead of looking at them as transient internal events (Greco, Lambert, & Baer, 2008; Hayes, Luoma, Bond, Masuda, & Lillis, 2006). In the context of CP, cognitive fusion is the underlying process of getting caught up in thoughts such as “this pain will never go away” or “this is unbearable”, and its pervasiveness comes from the fact that it cuts across different types of thought-contents that are relevant in CP (e.g. catastrophic, ruminative, hopeless, depressive) (McCracken & Morley 2014). Indeed, one study found that cognitive fusion mediates the relationship between pain catastrophizing and

disability in young people with CP (Solé et al., 2016). Nonetheless, although some studies have pointed out its relevance as a predictor of emotional distress and depression (e.g. Gillanders et al., 2014), few studies have explored cognitive fusion in CP (McCracken, DaSilva, Skillicorn, & Doherty, 2014b; Scott, McCracken, & Norton, 2016). Those that did, found that cognitive fusion correlates with pain interference and quality of life (Wicksell, Renöfält, Olsson, Bond, & Melin, 2008), and with depression in CP (McCracken et al., 2014b). Recently, one study found that cognitive fusion uniquely predicts depressive symptoms, and it mediates the association between pain intensity and depressive symptoms in women with CP (Carvalho, Pinto-Gouveia, Gillanders, & Castilho, 2018), however the interpretation was limited by its cross-sectional design. Nevertheless, although results seem to corroborate the proposition that cognitive fusion is a detrimental process associated to negative outcomes in CP (Wiksell, Lekander, Sorjonen, & Olsson, 2010; Wicksell, Renöfält, Olsson, Bond, & Melin, 2008), longitudinal studies are needed to establish temporal associations between variables. One online 4-month longitudinal study conducted in muscle disorders found that cognitive fusion prospectively predicts life satisfaction and anxiety, but not depressive symptoms (Graham, Gouick, Ferreira, & Gillanders, 2016). Similarly, another study in a sample of older adults (age > 65) found that cognitive fusion did not mediate changes in depressive symptoms following a psychological intervention (Scott, Daly, Yu, & McCracken, 2017). However, an 18-month online longitudinal study found that cognitive fusion predicted changes in depressive symptoms in a sample of participants with irritable bowel disease (Trindade, Ferreira, & Pinto-Gouveia, 2018). These findings call for the necessity of more studies with robust methodologies and statistical analyses, in order to better understand the relationship between cognitive fusion, pain and depressive symptoms in CP over time.

Thus, the current study expands on previous literature by exploring the role of cognitive fusion on changes in depressive symptoms, while controlling for pain intensity and functional impairment, in a three-wave 12-month longitudinal design, in women with chronic pain. Based on previous literature suggesting that these variables tend to be stable over time (Dunn, Campbell, & Jordan, 2013; Trindade et al., 2018), we did not expect large changes in the outcome of depressive symptoms. Still, we hypothesized that cognitive fusion (but not pain intensity nor functional impairment) would predict the small amount of change in depressive symptoms that may be observed over twelve months.

Method

The current study is part of a larger one that aims to explore the role of psychological processes in predicting changes in depressive symptoms in adults with chronic pain. The study follows a time-lagged design with three assessment points: baseline (T0), 6-months (T1) and 12-months (T2).

Participants

Eighty-six women with musculoskeletal CP filled out an online survey with socio-demographic and medical questions, and self-report measures. Participants presented a mean age of 50.73 (*SD* = 10.84), and the majority completed high-school (*n* = 26; 30.2%) or a bachelor´s degree (*n* = 37; 43%). The majority of participants were employed (*n* = 63; 73.3%) and married (*n* = 51; 59.3%) or divorced (*n* = 20; 23.3%). More than half of our sample presented a middle socioeconomic status according to their occupation (n = 42; 48.8%).

Participants had fibromyalgia (*n* = 74; 86%), low-back pain (*n* = 11; 12.8%), arthrosis (*n* = 10; 11.6%) and/or rheumatoid arthritis (*n* = 9; 10.5%). Patients could report more than one diagnosis, with 77.9% (N = 67) having one diagnosis and 22.1% (N = 19)

having two or more. Diagnoses were provided by one or more medical doctors, such as the rheumatologist (*n* = 68; 79.1%), general practitioner (*n* = 14; 16.3%), psychiatrist (*n* = 7; 8.1%), and/or by other medical specialties (*n* = 70; 81.4%). Regarding duration of CP, 51 participants had CP for more than 10 years (59.30%), 23 from 5 to 10 years (26.70%), and 12 from 1 to 5 years (14%). Finally, the majority of our sample (N = 66; 76.7%) did not have psychotherapy for the last 12 months, and 34 of which (39.5%) reported that they never had psychotherapy. In terms of depressive symptoms, participants were not subject to a clinical diagnostic assessment. According to participants´ scores on DASS-21 (see measures section), our sample was composed of women with normal (n = 65), mild (n = 9) and moderate (n = 12) levels of depressive symptoms.

Procedure

Participants were collected through five national CP associations that advertised the study among patients through their mailing list. Interested patients were directed to a secure survey which comprised the battery of questionnaires. Before completing the survey, participants gave their informed consent and were informed about the purpose and confidentiality of data. The study was accessed by 479 participants, of which 246 completed the research battery (51%) at baseline. The sample was selected through the following inclusion criteria, which were self-reported: a) having constant or sporadic pain, unrelated to oncological disease, for three months or more; b) age above 18 years; c) having access to an online device to complete the survey. No exclusion criteria were considered. Nine men and six non-Portuguese women were further excluded from the study in order to have a homogeneous sample regarding gender and nationality. Eighty-six participants completed all three assessment points between February 2017 and March

2018. This study was granted approval by the Scientific and Ethics Committee of the University where the first author is conducting his research.

Measures

Participants filled out the following self-report measures at the three assessment points:

Pain intensity

Numerical Pain Rating Scale (NPRS; Hartrick, Kovan, & Shapiro, 2003; Ferreira-Valente, Pais-Ribeiro, & Jensen, 2011). This 11-item unidimensional scale is widely used to assess of pain intensity. NPRS presents 11 numbers from 0 ("No pain") to 10 ("Worst imaginable pain"); higher scores thus indicate greater pain intensity. Using ratings from: 1) current pain; 2) highest pain in last 24h; 3) lowest pain in last 24h, a single score of average pain intensity was created. The current study found good values of Chronbach´s alpha in all three assessment points (see Table 2).

Functional impairment

Work and Social Adjustment Scale (WSAS; Mundt, Marks, Shear, & Greist, 2002). The WSAS is a 5-item measure of functional impairment in five domains: work, home management, social leisure activities, private leisure activities, and family and other relationships. Items are rated on a 9-point scale from 0 ("no impairment") to 8 ("very severe impairment"). Higher scores indicate greater functional impairment. The original study found good internal consistencies in different samples (from α = .79 to α = 94). The current study also found good internal consistencies in all three assessment points (see Table 2).

Cognitive fusion

Cognitive Fusion Questionnaire-7 (CFQ-7; Gillanders et al., 2014; Costa, Marôco, & Pinto-Gouveia, 2017). This is a 7-item measure of cognitive fusion (e.g., "I get so caught

up in my thoughts that I am unable to do the things that I most want to do"), which is rated on a scale from 1 ("Never true") to 7 ("Always true"). The CFQ-7 showed good internal consistencies in its original (αs from 0.88 to 0.93 across five samples) and Portuguese (αs from 0.89 to 0.94 across three samples) validations. See Table 2 for values of Cronbach alphas in the current study.

Depression symptomatology

Depression Anxiety Stress Scales (DASS-21; Lovibond & Lovibond, 1995; Pais-Ribeiro, Honrado, & Leal, 2004). This is a measure of depression, anxiety, and stress symptomatology over the previous week. Its 21 items are rated from 0 ("Did not apply to me at all") to 3 ("Applied to me very much, or most of the time"). Only the depression symptomatology subscale was used in this study. This subscale presented showed good internal consistencies in DASS-21's original (α = 0.88) and Portuguese (α = 0.85) validation studies. The current study found good internal consistency (see Table 2).

Statistical analyses

Descriptive and frequency analyses were conducted in order to examine demographic and medical characteristics. Associations between variables were analysed through Pearson correlation coefficients (Cohen, Cohen, West, & Aiken, 2003). These analyses were performed using SPSS (v. 24.0; IBM Corp, 2016).

Structural equation modelling (AMOS, version 22.0; Arbuckle, 2013) was used to perform latent growth curve models (LGM) (Willett & Sayer, 1994; Windle, 1997), which estimated the growth trajectory of pain intensity, functional impairment, cognitive fusion, and depression symptomatology. Latent growth curve modelling is a longitudinal analysis that calculates growth over a period of time. Each growth curve has a baseline level (the intercept factor) and a rate of change over time (the slope factor). The intercept factor is constant and consists of the initial level of the variable (intercept mean) and its

individual differences between participants (intercept variance). The slope factor refers to the average rate of change (slope mean) and individual differences in patterns of growth (slope variance). The association between the intercept and slope factors, when positive, indicates that the lower the baseline, the larger the growth, and when negative, that the greater the baseline, the lower the growth.

Analyses of the effect of hypothesised predictors on the growth of depression symptomatology (outcome variable) were also conducted. The model of depression symptomatology was thus conditioned by adding pain intensity and cognitive fusion (model 1) and functional impairment and cognitive fusion (model 2) as predictors of the intercept and the slope factors. This analysis allows to test whether these predictors account for individual differences in basal levels of depression symptomology and for the differences in the rate of growth of this outcome.

The adequacy of the models was examined through several goodness of fit indices: Comparative Fit index (CFI), Tucker and Lewis Index (TLI), and Incremental Fit Index (IFI) that indicate a good adjustment to empirical data when around 0.95 (Hu & Bentler, 1999), and the Standardized Root Mean Squared Residual (SRMR) which indicates a good adjustment when < 0.08 (Hu & Bentler, 1999).

Results

Preliminary analysis

The study variables did not seem to present a significant bias to normal distribution (Skewness ranged from -0.14 to 0.85, and Kurtosis from -1.14 to -0.11) (Kline, 1998). Results from correlations of demographic and medical variables with depression symptoms are presented in Table 1. It is interesting to note that depression symptoms at any time of assessment were not significantly linked to any demographic or medical variable.

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Table 2 presents the correlations between pain intensity, functional impairment, cognitive fusion, and depression symptomatology at all times of assessment. All variables were significantly associated with each other, except for pain intensity that globally did not correlate with cognitive fusion and depression symptoms (with the exception of the significant correlations found between pain intensity at T2 and cognitive fusion at T2, and between depression symptoms at T1 and pain intensity at T2).

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Non-conditioned models

Pain Intensity

The LGM was successfully fitted to the levels of pain intensity at the three assessment times: $\chi^2_{(1)} = 0.04$, $p = 0.873$; CFI = 1.00; TLI = 1.00; IFI = 1.00; SRMR = 0.00.

Basal levels of pain intensity were significantly different among participants (b = 2.97; S.E. = 0.73; $Z = 4.09$; $p < 0.001$) around a mean of 5.06 (S.E. = 0.21; $Z = 24.23$; $p < 0.001$). The non-significant estimate of slope's mean ($b = 0.11$; S.E. = 0.09; $Z = 1.19$; $p = 0.235$) indicated that there was no significant change over time in pain intensity. The non-significant variance around the mean growth ($b = 0.13$; S.E. = 0.28; $Z = 0.48$; p = 0.634) indicates that the growth rate of pain intensity was homogeneous among participants.

Functional impairment

The LGM was well fitted to functional impairment at the three times of assessment: $\chi^2_{(1)} = 0.276$, $p = 0.599$; CFI = 1.00; TLI = 1.00; IFI = 1.00; SRMR = 0.00.

Basal levels of functional impairment presented significant differences among participants ($b = 84.07$; S.E. = 17.31; $Z = 4.86$; $p < 0.001$) around a mean level of 22.08

(S.E. = 1.05; $Z = 21.05$; $p < 0.001$). Slope's mean was found to be significant, indicating a significant tendency for functional impairment to decrease at a mean rate of 1.42/year (S.E. = 0.35; $Z = 4.02$; $p < 0.001$). This growth rate was homogeneous among participants as indicated by the significant variance around the mean growth ($b = 5.60$; S.E. = 5.30; $Z = 1.06$; $p = 0.291$).

Cognitive fusion

The LGM was adequately fitted to cognitive fusion's levels assessed at the three moments: $\chi^2_{(1)} = 4.08$, $p = 0.044$; CFI = 0.98; TLI = 0.95; IFI = 0.98; SRMR = 0.00.

Initial levels of cognitive fusion presented individual differences among participants ($b = 116.03$; S.E. = 23.83; $Z = 4.87$; $p < 0.001$) around a mean level of 23.96 (S.E. = 1.27; $Z = 18.86$; $p < 0.001$). Slope's mean was non-significant ($b = 0.22$; S.E. = 0.43; $Z = 0.52$; $p = 0.605$), which shows that there was no significant change over time concerning participants' levels of cognitive fusion. The growth rate was homogeneous among participants ($b = 0.19$; S.E. = 7.84; $Z = 0.02$; $p = 0.981$).

Depression symptoms

The LGM was successfully fitted to depression symptoms measured at the three times of assessment: $\chi^2_{(1)} = 0.276$, $p = 0.599$; CFI = 1.00; TLI = 1.00; IFI = 1.00; SRMR = 0.00.

There were differences among participants regarding the basal levels of depression symptomatology ($b = 19.28$; S.E. = 4.58; $Z = 4.21$; $p < 0.001$) around a mean level of 5.29 (S.E. = 0.53; $Z = 9.90$; $p < 0.001$). There was no significant change over time in depression symptoms ($b = 0.38$; S.E. = 0.22; $Z = 1.69$; $p = 0.09$) and the growth rate of this variable was homogeneous among participants ($b = 0.50$; S.E. = 1.86; $Z = 0.29$; $p = 0.789$).

The correlations between intercept and slope were non-significant for all variables, showing that the basal level of each variable does not seem to be associated with its rate of change across time.

Predicting change

The effects of pain intensity and cognitive fusion on the growth of depression symptoms

A conditional model was fitted to data to explore the influence of pain intensity and cognitive fusion on the growth of depression symptoms (Figure 1). This model presented an excellent fit to the data: $\chi^2_{(3)} = 4.57$, $p = 0.206$; CFI = 0.99; TLI = 0.98; IFI = 0.99; SRMR = 0.03.

Results showed that pain intensity did not present a significant impact on baseline levels of depression symptoms ($\beta = 0.06$, $p = 0.506$). Results also demonstrated that pain intensity did not influence the growth rate of this outcome ($\beta = 0.15$, $p = 0.169$).

Cognitive fusion significantly predicted both the baseline levels ($\beta = 0.64$ $p < 0.001$), and growth rate ($\beta = 0.35$, $p = 0.014$) of depression symptoms.

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The effects of functional impairment and cognitive fusion on the growth of depression symptoms

A conditional model was fitted to data to analyse functional impairment and self-compassion influence the growth of depression symptoms (Figure 2). Model fit was excellent: $\chi^2_{(3)} = 6.21$, $p = 0.102$; CFI = 0.99; TLI = 0.96; IFI = 0.99; SRMR = 0.02.

Functional impairment presented a positive effect on baseline levels of depression symptomatology ($\beta = 0.30$, $p = 0.005$). Nonetheless, functional impairment did not influence the growth rate of the outcome ($\beta = 0.13$, $p = 0.489$). Cognitive fusion, on the other hand, significantly and positively impacted on the baseline levels of depression

symptoms with an effect of 0.51 ($p < 0.001$), as well as on the growth rate of this outcome with an effect of 0.46 ($p = 0.017$).

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Discussion

The current study explored changes in pain intensity, functional impairment, cognitive fusion and depressive symptoms over a period of 12-months in a sample of women with CP, as well as the associations between these variables throughout three assessment points (baseline, 6-months, 12-months). This study examined whether changes in depressive symptoms during this period were longitudinally predicted by pain intensity, functional impairment and cognitive fusion.

Correlation results showed that the socio-demographic characteristics of our sample did not significantly associate to depressive symptoms at any time point, which is contrary to previous research suggesting that socio-economic status and level of education are associated with depression in CP (Averill et al., 1996; van Hecke et al., 2013). This might be due to a relatively low variance in our sample in terms of socioeconomic status, given that more than half of participants presented a middle socioeconomic status according to their occupation, which may have influenced this correlational result. More interestingly, our results seem to suggest that neither duration nor number of CP diagnoses significantly correlate with depressive symptoms at any time point. This seems to be in line with the ongoing discussion regarding the complex interaction between pain and depression, perhaps suggesting that there may be other variables, such as psychological processes, operating the relationship between pain and depression (Carvalho et al., 2018; Gatchel et al., 2007; Turk et al., 1995).

Correlation analyses show that cognitive fusion at baseline is significantly associated with depressive symptoms at all time points. This seems to be in line with

previous studies that suggest that cognitive fusion is associated with depressive symptoms in chronic illness (e.g. Gillanders et al., 2014) and in CP (Carvalho et al., 2018; McCracken et al., 2014b; Scott et al., 2016; Wiksell et al., 2010; Wicksell et al., 2008). Also, results show that neither pain intensity nor pain-related functional impairment were significantly associated with depressive symptoms at baseline, at 6-months and 12-month assessments. Interestingly, and perhaps contrarily to some studies that suggest that depression results from the negative impact of having CP, and not the other way around (e.g. Brown, 1990; Wörz, 2003), results show that depressive symptoms at T0 is significantly correlated with functional impairment at T1 and T2, and depressive symptoms at T1 is significantly associated with pain intensity and functional impairment at T2. Though establishing conclusions regarding causal relationships is unwarranted due to the nature of correlation analysis.

Results from latent growth curve modelling showed that participants present significant differences on their levels of pain intensity, functional impairment, cognitive fusion and depressive symptoms at baseline, and, except for functional impairment (which decreased), none of them presented significant changes over the 12-months time period. Also, results show that the change rate of all variable was homogenous, i.e., did not significantly differ between participants.

Based on these results, as well as on previous studies that suggest that depressive symptoms in CP are greatly influenced by psychological processes (e.g. Gatchel et al., 2007; Turk et al., 1995), including cognitive fusion (e.g. Carvalho et al., 2018; McCracken et al., 2014b; Scott et al., 2016), we have tested two conditional models in order to examine the specific impact of pain intensity and cognitive fusion (Model 1) and functional impairment and cognitive fusion (Model 2) on depressive symptoms over 12-months. Results showed that cognitive fusion, but not pain intensity, significantly

predicted both the baseline levels of depressive symptoms, as well as their changes over the 12-months. Similarly, although functional impairment significantly predicted the baseline levels of depressive symptoms, it did not predict changes in depressive symptoms throughout the 12-months. These results seem to suggest that changes in depressive symptoms were not explained by the intensity of pain nor the functional impairment that arise from having CP, but rather from the entanglement with internal experiences such as thoughts, emotions and physical sensations.

These results contribute to moving forward the understanding of the role of psychological processes in the aetiology of depression in CP. It suggests that cognitive fusion is a much more relevant factor in the development and/or maintenance of depression in CP than pain-specific symptoms such as pain intensity and pain-related functional impairment. These findings are based on previous research that found that cognitive fusion is associated with depressive symptoms in CP (e.g. McCracken et al., 2014b; Scott et al., 2016; Wiksell et al., 2010; Wicksell et al., 2008), and that it fully mediates the relationship between pain intensity and depressive symptoms (Carvalho et al., 2018). Furthermore, it adds to previous findings by conducting a more robust statistical procedure in a longitudinally designed study. Indeed, few studies have explored cognitive fusion longitudinally, and those that did found inconsistent results: one study in IBD found cognitive fusion to significantly predict depressive symptoms in an 18-month period (Trindade et al., 2018), another study found cognitive fusion to not predict depression in a 4-month period in a sample of participants with muscle disorders (Graham et al., 2016), and one intervention study found cognitive fusion to significantly predict changes in depression in a sample of older adults with CP (Scott et al., 2017). Given that cognitive fusion is a central psychological process in the growingly studied psychological flexibility model of CP (e.g. McCracken & Vowles, 2014), as well as theoretically

conceptualized as an underlying process in several relevant psychological processes in CP aetiology (i.e. catastrophizing and avoidance) (e.g. McCracken & Morley 2014), it is crucial to better understand the role of cognitive fusion in CP.

These findings should be interpreted with adequate caution due to its limitations. First, it should be noted that the study was conducted in a sample of women, which makes it unwarranted to extrapolate this results to other genders. Although studies suggest that CP is more prevalent in women (Fayaz, Croft, Langford, Donaldson, & Jones, 2016), future studies should replicate these results in samples of different genders. Indeed, cognitive fusion is closely related to other psychological processes, such as rumination (e.g. McCracken et al., 2014a). It seems that rumination is less prevalent in men (Johnson & Whiseman, 2013), which may yield a different impact on depressive symptoms in men with CP. Thus, more research is needed in order to understand if and how these psychological processes impact differently depressive symptoms in different genders. Also, it should be noted that participants presented levels of depressive symptoms bellow the threshold for clinical depression, which prevents us from generalizing these results to clinically depressed CP patients. Future studies should conduct multi-group analyses to explore differences in the model between women with depressive symptoms versus clinically depressed women. Additionally, this study was conducted online through self-report measures. Future studies ought to replicate it and assess pain and depressive symptoms through clinical interviews. Furthermore, sampling through pain associations may have introduced a sampling bias towards women who are already relatively well adjusted to their chronic pain, which may not reflect typical presentations in secondary care settings. Finally, the sample size did not allow the testing of more complex models that would make it possible to examine other relevant processes, such as experiential avoidance and commitment to valued action. Indeed, one possible mechanism that might

explain the nefarious role of cognitive fusion is that it leads to an increase in avoidant behaviours, thus decreasing actions that are valued and meaningful, leading to more symptoms of depression. Hence, more comprehensive models that integrate different relevant psychological processes are much needed for us to have a more complete picture of depression in CP. Finally, the current study did not collect qualitative information, which would provide us with much needed additional data on participants´ personal narratives on their pain experience, as well as the psychological processes and symptoms involved in their pain experience.

The current study provides additional data on the role of cognitive fusion that potentially yields clinical implications for psychotherapeutic management of CP. These results suggest that when dealing with depression in CP, psychological interventions should be more focused on tackling unhelpful entanglement with thoughts and emotions, rather than being overly focused on reducing pain symptoms. Nevertheless, more studies are needed to unequivocally establish the causal relationships between these variables. Future studies on the role of cognitive fusion in CP should consider task-oriented experimental designs comparing the effect of brief cognitive defusion exercises versus getting cognitively entangled on both mood and pain perception. Future studies should also consider the benefit of complementing standard self-report measures with ecological momentary assessment, which would allow us to collect information daily and with signal- or event-contingent reporting. Results from the current study corroborate the usefulness of integrating psychological approaches that promote acceptance and a shift in perspective in CP management. Specifically, acceptance- and mindfulness-based evidence-based interventions, such as Acceptance and Commitment Therapy (ACT; Hughes et al., 2017), seem to represent helpful approaches in reducing cognitive fusion through techniques that promote perspective-taking, such as mindfulness-based practices,

as well as deliteralization techniques. By promoting cognitive defusion (a core process in psychological flexibility), the person with chronic pain learns how to distance oneself from their internal experiences (e.g. catastrophizing content, depressive rumination, and the physical sensation itself), which will give rise to the ability to notice these experiences as transient ones, with an accepting and non-reactive stance to them. This ability of just noticing internal experiences without automatic reaction can ultimately broaden their behaviour repertoire, which opens the possibility for a person with chronic pain to activate behaviours and engage in valued and meaningful experiences despite their pain, instead of being overly focused on the pain experience and the control of pain.

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