



Clinical pain research

Chronic low back pain and the transdiagnostic process: How do cognitive and emotional dysregulations contribute to the intensity of risk factors and pain?



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HIGHLIGHTS

- Transdiagnostic approach is an innovative way to understand pain and psychological factors.
- Cognitive and emotional processes are moderator factors.
- Difficulties in emotional regulation play an important role in the increase in negative affects.

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ABSTRACT

Background and aims: Based on a transdiagnostic approach, this study assesses the impact of cognitive and emotional processes (difficulties in emotional regulation, impulsiveness, rumination and somatosensory amplification) on the psychological risk factors of chronic low-back pain.

Methods: The study was carried out with 256 patients with chronic low-back pain. All the variables were assessed through a booklet of 10 validated questionnaires. Multiple regression analysis and moderation analysis were performed.

Results: Predictors included in multiple regression models explain 3%–42% (adjusted R^2) of the variance in psychological risk factors. Moreover, analyses reveal a significant moderator effect of somatosensory amplification on the link between fear-avoidance beliefs linked to work and pain intensity ($F_{(3;250)} = 12.33$; $p = .00$), of somatosensory amplification and brooding on the link between depression and functional repercussions (FR) on everyday life ($F_{(3;252)} = 13.36$; $p = .000$; $F_{(1;252)} = 12.42$; $p = .00$), of the reflection dimension of rumination on the link between the helplessness dimension of catastrophizing and FRs on sociability ($F_{(3;252)} = 37.02$; $p = .00$). There is also a moderation analysis with a significant trend concerning the lack of emotional awareness and the difficulties in controlling impulsive behaviours.

Conclusions: Our results indicate an important role of some dimensions of difficulties in emotional regulation, somatosensory amplification and rumination in the increase in negative affects and dysfunctional beliefs, and in the links between those psychological risk factors and pain/disability.

Implications: This study identifies some cognitive and emotional dysregulations substantially involved in work-related chronic pain. This contribute to put in place psychotherapeutic protocols to tackle these deficits and dysregulations in a relevant way.

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1. Introduction

Pain, in an occupational context when it concerns musculoskeletal disorders (such as low back pain), is a particularly important

phenomenon in most countries. Low back pain, with a 60–90% incidence in France (6–8% becoming chronic) [1], is growing rapidly and resulting in more sick leave, which can sometimes be prolonged. Traditional biomedical variables cannot fully account for the development of disability: the intensity, or the severity of the pain, being responsible for only 10% of prolonged disability [2]. In fact, this issue of pain and disability is today considered a complex biopsychosocial phenomenon in which different factors interact with each

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other. The question of pain chronicization is of increasing interest to public authorities and the research community due to the significant costs it entails for society, companies and individuals. The aim of research has been to identify the modifiable risk factors, which could form the basis of targeted interventions to prevent the development of prolonged disability [3,4]. However, although studies have identified many psychosocial factors as predictors of chronicization or the development of disability, their prognostic value is low if these factors are taken individually [5]. Thus, researchers now recognize the need for a multidimensional perspective. Moreover, it is now known that psychological, and especially emotional, factors are closely linked with pain. Yet, the treatment of these comorbid factors is in its infancy due to the lack of a clear theoretical understanding of the mechanisms involved in their links [6].

Recently, an emerging approach restarts the debate on the understanding and treatment of psychopathological disorders: the “transdiagnostic” (or “processual”) approach [7,8]. This approach postulates that certain psychological processes contribute in a causal way to the development and maintenance of various symptoms observed. Moreover, it allows to account for the phenomenon of comorbidity by showing that different psychopathological disorders can be underpinned by common psychological processes. These different processes can be conceptualized in terms of “vulnerability factors”, distinguishing from “risk factors: we can define “risk factors” as a significant set of factors associated with an increased probability of occurrence of a disorder and “vulnerability factors” as a subset of individual endogenous factors that can serve as mechanisms in the development of a disorder. Vulnerability factors moderate the effects of risk factors [9]. Adopting a transdiagnostic approach has many clinical implications in the field of pain [6]: assessing the effect of factors and their interactions will help to understand the link between pain and emotions, improve knowledge about the processes involved and thus strengthen the effectiveness of treatments. Yet, this approach has not been the subject of many studies in chronic pain. It is within this perspective that the aim of this research is to study the impact of cognitive and emotional processes, called vulnerability factors on the psychological risk factors. We choose to study 3 major processes, which are transversal factors in several disorders and are thus conceptualized as transdiagnostic processes [8]: (difficulties in) emotional regulation, somatosensory amplification and rumination. These factors may act as moderators of the relationships between risk factors and pain.

We thus put forward the following hypotheses:

- the levels of vulnerability factors contribute significantly to the increase in levels of risk factors;
- the levels of vulnerability factors moderate significantly the links between risk factors, pain intensity and functional repercussions.

2. Methods

2.1. Participants

This study was carried out within the regional organization offering support and expertise called “Lombaction”, part of the occupational diseases consultation department of Angers University Hospital, which aims to reduce the impact of chronic low back pain in the work environment. This organization is aimed at people with an occupational problem (either at work or on sick leave) due to their incapacitating chronic low back pain.

In total, the study sample consisted of 256 subjects, 120 men (46.88%) and 136 women (53.12%), aged from 21 to 61 years, with a mean age of 41.74 (SD = 8.94).

Regarding their occupational status, 219 were employed (92 at work, 113 on sick leave with a recognized occupational disease, 8 working part-time for health reasons) and 37 were not employed (34 without a job, 1 disabled, 2 without a job and with a recognized occupational disease).

Concerning the duration of the low back pain, 25 subjects reported having had back pain for less than 1 year, 107 for between 1 and 5 years and 124 for more than 5 years.

2.2. Material

We compiled a booklet of various validated questionnaires in order to assess:

- Pain:

Intensity (visual analogue scale): intensity of pain was assessed by a visual analogue scale. This is a tool easy and quick to use which results in a pain intensity score between 0 (no pain) and 10 (maximum pain experienced).

Functional repercussions (Dallas Pain Questionnaire) [10]: the Dallas questionnaire is a self-administered quality of life questionnaire exploring the functional repercussions of pain in four areas of life (“everyday life activities”, “work and leisure”, “mood” and “sociability”). Participants responded to the 16 items using a visual scale, segmented to allow for better gradation. The evaluation is done by assigning a value to each segment. Elementary values are summed and multiplied by a constant factor to evaluate the repercussion on each area.

- Risk factors:

Depression (Beck 13-item Depression Inventory) [11]: The BDI-13 is a 13-items scales that measures intensity of depression. Each item (as “sadness”, “suicidal thoughts” or “self-image”) includes 4 different proposals that are graded from 0 to 3. The participant choose the one with which he feels most in agreement, depending on how he feels during the last fifteen days. The sum gives an overall score of intensity of depressive syndrome.

State-anxiety (Spielberger State-Trait Anxiety Inventory) [12]: The STAI-Y, frequently used by researchers and practitioners, examines state and trait anxiety independently. For the state-anxiety scale (Y-A form), participant should refer to what he feels “right now”. The Y-A scale comprises 20 items rated on a 4-point Likert scale (1 = almost never, 4 = almost always).

Catastrophizing (Pain Catastrophizing Scale) [13]: the Pain Catastrophizing Scale is one of the most widely used tool for assessing catastrophizing thoughts related to pain. Participants responded to the 13 items on a 5-points Likert scale (0 = not at all, 4 = all the time). This scale gives a total score of catastrophism and a score for each sub-dimension of this concept: rumination, magnification and helplessness.

Fear-avoidance beliefs (Fear-Avoidance Beliefs Questionnaire) [14,15]: this is a tool to measure the participant’s beliefs about how work and physical activity affect his or her low-back pain. It is composed of 16 items divided into 2 subscales: a scale measuring fear-avoidance beliefs related to physical activity (items 1–5), a scale measuring fear-avoidance beliefs related to work (items 6–16).

- Vulnerability factors:

Emotional regulation (Difficulties in Emotional Regulation Scale DERS-F) [16]: the DERS-F is an instrument developed to identify difficulties of participants in four domains involved in the regulation of emotions. This scale has 36 items and participants responded to each item on a 5-point Likert scale (1 = almost never, 5 = almost always). Factorial analysis revealed 6 factors: 1) nonacceptance of emotional response, 2) difficulties in adopting goal-oriented behaviours, 3) difficulties in controlling impulsive behaviours, 4) lack of emotional awareness, 5) limited access to

emotion regulation strategies and 6) lack of emotional identification or clarity. The cotation gives a score for each of these dimension.

Somatosensory amplification (Somatosensory Amplification Scale) [17]: this scale is designed to assess the propensity of individuals to focus on bodily information and to assign ordinary sensations to serious somatic symptoms. The scale is composed of 10 items (example: “I am often aware of various things that happen in my body”). Participants responded to items on a 5-point Likert scale (1 = not all true, 5 = extremely true).

Rumination (Ruminative Response Scale) [18]: Ruminative Response Scale evaluates the frequency of ruminative thoughts by 10 items corresponding to different suggestions. Participant ranks to what extent this suggestion corresponds to him or her by using a 4-point Likert Scale (1 = almost never, 4 = almost always). We then obtain a score for each dimension of the rumination: brooding (5 items) and reflection (5 items).

2.3. Procedure and data analysis

The first step of the procedure was to distribute the booklet containing the different questionnaires to the patients coming to consult the Lombaction network. It was sent by post, in advance, with the appointment for the consultation.

All the adults consulting Lombaction and presenting chronic low back pain diagnosed according to the HAS criteria were included in the study. Non-French-speaking patients and those who could not read or write were eliminated. Questionnaires that were not fully completed were later excluded.

These data were then processed using the statistics software SPSS 22.

First, the necessary assumptions for using the tests were checked (normal or non-normal distribution of variables) and the correlations between variables were verified. Next, stepwise regressions with backward elimination method were carried out, as recommended for more exploratory research. According to this method, the initial model includes all the variables and the software removes the variable making the weakest contribution to the model and repeats the procedure until all the variables retained contribute significantly to improving the explained variance. Lastly, moderation analyses were carried out, with the help of PROCESS under SPSS, in order to test the effect of a moderator variable on the link between the independent and dependent variable. According to our hypothesis, vulnerability factors are considered moderating variables, influencing the effect of risk factors on pain and disability. Indeed, from a theoretical point of view, a mediator describes a process through which the independent variable is likely to influence the dependent variable. The independent variable triggers the action of a mediator, which in turn influences the dependent variable. In other words, the effect of the predictor on the criterion must be zero when the effect of the mediator is eliminated. A moderator is a variable that affects the relationship (by direction or intensity) between the independent variable (the predictor) and the dependent variable (the criterion).

3. Results

Means and standard deviations obtained for each scale within our sample are presented in Table 1.

For average pain intensity at the time of questionnaires, subjects assessed the impact of their pain as being high in activities of daily living, work and leisure, and moderate in mood and sociability.

According to the existing cut-off scores that we can find in validation studies, patients in this sample present moderate depression score, low levels of anxiety-state, high catastrophism

score and high level of fear-avoidance beliefs related to physical activity (there is no cut-off for those related to work).

First, we hypothesized that the levels of vulnerability factors contribute significantly to the increase in the levels of risk factors. The results of descending multiple linear regression analyses for each risk factor are presented in Table 2. They reveal that the model that best fits the explanation of depression includes the variables “Difficulties in controlling impulsive behaviours” (DERS-F scale), “Limited access to emotional regulation strategies” (DERS-F scale), “Rumination-reflection” and “Rumination-brooding”. These predictors explain 42% (adjusted R^2) of the variance in depression ($F_{(1;249)} = 47.24, p = .00$). Concerning the state-anxiety, the predictor variables “Negative urgency” (impulsivity scale), “Rumination-reflection” and “Rumination-brooding” explain 30% (adjusted R^2) of the variance in the state-anxiety ($F_{(1;249)} = 37.75, p = .00$). The model that best fits the explanation of catastrophizing includes the variables “Somatosensory amplification” and “Rumination-brooding”. These predictors explain 25% (adjusted R^2) of the variance in catastrophizing ($F_{(1;249)} = 42.81, p = .00$), 25% (adjusted R^2) of the variance in the amplification dimension ($F_{(1;249)} = 40.76, p = .00$) and 21% (adjusted R^2) of the variance in the rumination dimension ($F_{(1;249)} = 34.36, p = .00$). Concerning the helplessness dimension, the regression analysis shows that the model that best fits the explanation of this dimension includes the variables “Limited access to emotional regulation strategies” (DERS-F scale), with “Somatosensory amplification” and “Rumination-brooding” explaining 18% (adjusted R^2) of the variance in the helplessness dimension ($F_{(1;249)} = 19.31, p = .00$).

Lastly, the results demonstrate that the model that takes account of the variance in fear-avoidance beliefs linked to physical activity only includes the variable “Limited access to emotional regulation strategies”. This factor explains 3% of the variance in beliefs linked to physical activity ($F_{(1;249)} = 9.77, p = .00$).

In the same way, only somatosensory amplification is a significant and positive predictor ($B = 0.17$), explaining 3% of the variance in fear-avoidance beliefs linked to work ($F_{(1;249)} = 2.26; p = .01$).

In addition, we hypothesized that the levels of vulnerability factors significantly moderate the links between risk factors, pain intensity and functional repercussions (Table 3).

The analyses reveal a significant moderator effect of somatosensory amplification (Mo) on the link between the fear-avoidance beliefs linked to work (VI) and pain intensity (VD) (Model: $R^2 = 0.01$; $MSE = 4.30$; $F_{(3;250)} = 12.33$; $p = .00$). The interaction between VI and Mo predicts a 2% increase in the level of pain intensity (R^2 change = 0.02; $F_{(1;250)} = 8.16$; $p = .01$).

The moderation analyses show, with a significant trend, two moderators involved in the link between depression and functional repercussions (FR) on everyday life activities: somatosensory amplification (Model: $R^2 = 0.14$; $MSE = 198.67$; $F_{(3;252)} = 13.36$; $p = .000$) and the brooding dimension of rumination (Model: $R^2 = 0.12$; $MSE = 203.85$; $F_{(1;252)} = 12.42$; $p = .00$). Regarding somatosensory amplification, the interaction between VI and Mo predicts a 1% increase in the level of FRs on everyday life activities (R^2 change = 0.01; $F_{(1;252)} = 3.15$; $p = .07$). Concerning the brooding dimension of rumination, the interaction also predicts an increase of 1% (R^2 change = 0.01; $F_{(1;252)} = 3.19$; $p = .08$). There is also a moderation analysis with a significant trend concerning the lack of emotional awareness (DERS-F) involved in the link between the helplessness dimension of catastrophizing and the FRs on everyday life activities (Model $R^2 = 0.19$; $MSE = 187.30$; $F_{(3;152)} = 21.63$; $p = .00$). The interaction between VI and Mo predicts a 1% increase in the level of FRs on everyday life activities (R^2 change = 0.01; $F_{(1;252)} = 3.41$; $p = .07$).

The analyses reveal a moderator effect, with a significant trend, of difficulties in controlling impulsive behaviours (Mo) on the link between the helplessness dimension of catastrophizing (VI) and

Table 1Means and standard deviations of variables within a sample of patients suffering from chronic low-back pain ($N = 256$).

			Mean	σ
Pain and disability	Pain intensity		4.95	2.16
	Functional repercussions	Everyday life activities	62.24	15.10
		Work and leisure	62.11	19.49
		Mood	37.56	24.83
		Sociability	30.27	23.51
Risk factors	Depression		7.89	6.11
	State-anxiety		42.48	13.78
	Catastrophizing	Total	26.93	11.72
		Rumination	9.29	4.23
		Magnification	4.97	3.08
Vulnerability factors (processes)		Helplessness	12.77	5.89
		Fear-avoidance beliefs	16.80	6.24
		Work related	34.79	15.04
		Nonacceptance of emotional response	13.95	4.92
		Difficulties in adopting goal-oriented behaviours	10.36	3.62
		Difficulties in controlling impulsive behaviours	12.19	4.71
		Lack of emotional awareness	13.74	4.79
		Limited access to emotion regulation strategies	17.16	6.54
		Lack of emotional identification	12.51	3.88
	Rumination	Brooding	10.17	3.19
		Reflection	8.66	2.97
	Somatosensory amplification		24.68	6.37

Table 2

Multiple linear regressions for the psychological risk factors.

	Non-standardized coefficients		Standardized coefficients	t	p
	B	SD	Beta		
<i>Depression</i>					
(Constant)	−5.925	1.204		−4.923	.000
Difficulties in controlling impulsive behaviours	−.225	.124	−.173	−1.809	.072
Limited access to emotional regulation strategies	.217	.090	.232	2.413	.017
Rumination-reflection	.215	.119	.104	1.804	.072
Rumination-brooding	1.078	.112	.562	9.632	.000
<i>State-anxiety</i>					
(Constant)	12.641	3.268		3.869	.000
Negative urgency	.710	.294	.153	2.418	.016
Rumination-reflection	1.835	.278	.424	6.595	.000
Rumination-brooding	.558	.264	.113	2.110	.036
<i>Catastrophizing</i>					
(Constant)	3.941	2.763		1.263	.208
Somatosensory amplification	3.72	.111	.202	3.357	.001
Rumination-brooding	1.401	.222	.381	6.319	.000
<i>Catastrophizing-magnification</i>					
(Constant)	−.917	.730		−1.256	.210
Somatosensory amplification	.070	.029	.146	2.402	.017
Rumination-brooding	.399	.059	.413	6.805	.000
<i>Catastrophizing-rumination</i>					
(Constant)	1.288	1.023		1.259	.209
Somatosensory amplification	.152	.041	.229	3.706	.000
Rumination-brooding	.417	.082	.314	5.079	.000
<i>Catastrophizing-helplessness</i>					
(Constant)	1.795	1.570		1.144	.254
Limited access to emotional regulation strategies	.116	.052	.129	2.207	.028
Somatosensory amplification	.141	.058	.153	2.419	.016
Rumination brooding	.541	.118	.293	4.584	.000

FRs on work and leisure (VD) (Model: $R^2 = 0.11$; $MSE = 344.25$; $F_{(3;252)} = 11.05$; $p = .00$). The interaction between VI and Mo predicts a 1% increase in the level of pain intensity (R^2 change = 0.01; $F_{(1;252)} = 3.01$; $p = .08$).

The analyses also reveal a significant moderator effect of the reflection dimension of rumination (Mo) on the link between the helplessness dimension of catastrophizing (VI) and FRs on sociability (VD) (Model: $R^2 = 0.33$; $MSE = 376.28$; $F_{(3;252)} = 37.02$; $p = .00$). The interaction between VI and Mo predicts a 3% increase in the level of FRs on sociability (R^2 change = 0.03; $F_{(2,252)} = 15.51$; $p = .00$).

Our results show no significant moderation analyses concerning the links between risk factors and functional impact on mood.

4. Discussion

4.1. A severe test of the emotional regulation system

First, our results indicate an important role of some dimensions of lack of emotional regulation in the increase in negative affects and dysfunctional beliefs linked to pain. Only a few studies on populations different from ours have observed that (i) the pain

Table 3

Model testing the moderator effect of vulnerability factors (Mo) on the link between psychological risk factors (VI) and pain and disability (VD).

	Coeff.	SE	t	p
<i>Pain intensity (VD)</i>				
Somatosensory amplification (Mo)	.007	.019	.383	.702
Fear-avoidance beliefs linked to work (VI)	.036	.019	3.98	.000
Interaction = VI × Mo	-.004	.001	-2.86	.005
<i>FR on everyday life activities (VD)</i>				
Somatosensory amplification (Mo)	.374	.153	2.44	.004
Depression (VI)	.734	.137	5.38	.000
Interaction = VI × Mo	-.036	.021	-1.78	.078
<i>FR on everyday life activities (VD)</i>				
Rumination-brooding (Mo)	.104	.420	.245	.205
Depression (VI)	.914	.204	4.47	.000
Interaction = VI × Mo	-.044	.036	-1.79	.076
<i>FR on everyday life activities (VD)</i>				
Lack of emotional awareness (Mo)	-.109	.177	-.618	.537
Catastrophizing-helplessness (VI)	1.12	.150	7.48	.000
Interaction = VI × Mo	.047	.025	1.85	.066
<i>FR on work and leisure (VD)</i>				
Difficulties in controlling impulsive behaviours (Mo)	-.178	.263	-.678	.498
Catastrophizing-helplessness (VI)	1.04	.205	5.06	.000
Interaction = VI × Mo	.061	.035	1.73	.084
<i>FR on sociability (VD)</i>				
Rumination-reflection (Mo)	1.86	.423	4.35	.000
Catastrophizing-helplessness (VI)	1.71	.207	8.28	.000
Interaction = VI × Mo	.238	.061	3.94	.000
<i>FR on sociability (VD)</i>				
Rumination-brooding (Mo)	2.25	.472	4.78	.000
Catastrophizing-helplessness (VI)	1.47	.230	6.35	.000
Interaction = VI × Mo	.118	.063	1.89	.060

response varies according to emotional regulation and the intensity of emotions [19,20], (ii) emotional regulation is an important factor in psychological well-being and social functioning [21], and (iii) emotional regulation predicts a reduction in negative affects independently of other measures of adjustment [22]. Linton and Bergbom developed a model, based on the link between depression and chronic pain, called “The Örebro Model of Behavioral Emotion Regulation of Pain” in order to understand how a limited access to emotional regulation strategies can play a central role in the chronization of pain/negative emotion comorbidity [23]. According to these authors, chronic pain and depression are both recurrent and cyclic in nature: patients in pain experience periods when they feel quite well alternating with periods when pain and/or a depressive mood reappear, which can trigger negative feelings and reactivate catastrophic worries linked to their previous experiences. In turn, these worries exacerbate negative affects as well as the perception and intensity of the pain. Ultimately, these episodes severely test the emotional regulation system. If emotional regulation is successful, the difficult emotions experienced are managed using different strategies, and the episodes of pain and negative affects are less intense and frequent. However, if this regulation system fails, the negative affects are magnified and then trigger episodes

of depression and intense pain [23]. In addition, it seems that the dimension “Lack of emotional awareness” is a moderator factor. The concept of alexithymia, defined as finding it difficult or even being unable to identify, differentiate, and describe emotions, has been the subject of several studies aimed at exploring its links with chronic pain [24,25]. However, it remains a controversial concept due to the absence of a clearly defined theoretical and clinical status. More broadly, it appears that in our sample of patients suffering from chronic low back pain, their difficulty in recognizing their emotions is associated with greater psychological distress and disability. The ability to name, understand, explore and express an emotion favours the integration of stressful events linked to chronic pain [26].

4.2. A deficit in attentional processes

As described above, difficulties in emotional regulation affect not only the increase in pain and its associated negative affects but also the attention that the patient pays to this pain. This can be put into perspective with another attentional process strongly involved in the increase in psychological risk factors: somatosensory amplification. Our results show that somatosensory amplification significantly predicts the increase in fear-avoidance beliefs and in catastrophizing and its three dimensions and seems to be an important moderator factor. Attentional processes are activated in all situations involving the subject and are thus actively involved in emotional regulation due to their participation in triggering and extinguishing emotion [27]. This concept could thus partly explain how dysfunctional cognitions and negative affects about pain can increase perception and disability in patients with chronic pain. Some studies have suggested that somatosensory amplification could be associated with the perception that the patient has of his/her painful condition, with a greater tendency of back pain patients to magnify their physical sensations [28,29]. Other studies have measured the impact of hypervigilance, a concept close to somatosensory amplification, on the perception and intensity of pain but with contradictory results to ours: a greater hypervigilance towards nociceptive stimuli was associated with a lower sensitivity to pain [30]. However, this study was carried out in a context of experimentally-induced acute pain. Crombez and colleagues noted that focusing on acute pain is a normal phenomenon that helps to preserve the organism [31]. Paying greater attention to pain enables the development of better adjustment strategies than if the pain was avoided [30,32,33]. However, if the pain becomes chronic and is by definition always present, individuals with a greater propensity to develop hypervigilance and increased nociceptive sensations will thus develop more negative beliefs and affects in response to pain. The over-activation of attention provokes the maintenance of fear-avoidance beliefs and catastrophizing about pain and raises their impact on pain and disability, in the same way as difficulties in emotional regulation. The dysfunctional attention paid to pain will increase its perceived intensity, while emotional regulation difficulties will increase its unpleasant nature.

4.3. Rumination: vulnerability in the cognitive regulation process

Another process seems to be strongly involved in the links between risk factors and pain/disability within our population. In fact, rumination seems to be an important predictive factor of the variance in risk factors as well as a moderator factor. It has recently been suggested in the literature that rumination may be considered a transdiagnostic process, i.e. central to several disorders such as eating or addictive behaviour disorders as well as anxiety disorders, which like depression, are risk factors for chronic

pain [34]. However, to our knowledge, no research has investigated the role, in a strict sense, of these ruminations in a chronic pain context. Only one qualitative study reveals that patients suffering from chronic pain spend much of their time thinking about their pain, its causes and its consequences, and that these patients tend to consider rumination a positive way to resolve problems [35]. Yet, rumination seems to be a dysfunctional strategy, and the inability to resolve the problems linked to pain in turn fuels the future negative content of rumination. These false beliefs are similar to those reported by patients suffering from depression. In addition, rumination has been conceptualized as a key cognitive phenomenon in understanding several emotional disorders, particularly depression [36,37]. This is consistent with our results insofar as rumination contributes largely to the variance in levels of depression and moderates significantly the link between depression and disability. Rumination leads to an increase in depressive symptoms in several ways: i) it increases the bidirectional relationship between negative mood and negative cognitions, ii) it interferes with the resolution of problems by leading the subjects to adopt a more pessimistic and fatalistic system of thought and iii) it reduces social support by generating situations in which the potential help of one's entourage is rejected [38].

Moreover, our results show that rumination has important links with catastrophizing. Recently, Flink, Boersma and Linton proposed a new conceptualization of catastrophizing from the angle of a repetitive negative way of thinking. For these authors, this process also includes worry and rumination. This way of thinking is characterized by abstract thoughts, an intrusive aspect and difficulty for the subject to become disengaged [39]. The authors believe that this abstract and repetitive cognitive activity is a dysfunctional avoidance strategy, which aims to regulate negative emotions. In fact, treating the emotion on an abstract and superficial level inhibits intense emotional reactions in the short term but, in the long term, this avoidance hinders the treatment and integration of emotional and somatic responses and prevents the patient from dealing with the pain in an adapted way.

5. Conclusion

The transversal nature of this study limits the scope of its results. Apart from identifying the predictive value of certain factors, we cannot determine the order of appearance of different variables or the causal relationship between them. Setting up a longitudinal study could be an interesting avenue of research. Such a study could assess the evolution of risk and vulnerability factors, and determine how the latter intervene. In addition, this would enable patient profiles most at risk of developing chronic pain and persistent disability to be identified in advance. Nevertheless, this research contributes to improving knowledge about the factors that may be involved in the development of disability, which is crucial in understanding chronic low back pain as their roles have still not been clearly established. It is also indispensable to be able to integrate these factors into clinical practice. Finally, this study identifies the deficiencies substantially involved in work-related chronic pain, enabling psychotherapeutic care to be put in place to tackle these deficits and dysregulations in a relevant way.

Ethical issues

The ethical procedure corresponds to the research approach according to the Psychologist's Code of Ethics. Informed consent of patients was required. This study follows the ethical recommendations of the Helsinki Declaration and has been validated by the Ethic Board of the hospital.

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Conflict of interest

None declared.

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