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Understanding Pain and Depression in Back Pain: the Role of Catastrophizing, Help-/Hopelessness, and Thought Suppression as Potential Mediators

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Abstract
Purpose The cognitive mediation hypothesis describes the influence of psychological factors on the relationship between pain and depression such as cognitions of catastrophizing and help-/hopelessness. More recent research also emphasizes the role of suppression of negative thoughts and experiences such as pain. However, there is little research investigating direct and indirect effects of these contrasting cognitions.
Method A total of 164 acute and sub-acute non-specific back pain patients participated in this study. Pain intensity, depression, and pain-related cognitions were measured using questionnaires, such as the Beck Depression Inventory and the Kiel Pain Inventory. Data were analyzed using structural equation modeling.
Results The results of the path analysis support the hypothesis that cognitive coping strategies have a mediating effect on pain and depression. Consistent with previous research, we found that pain had no direct relation with depression. Help-/hopelessness had a direct path to depression, whereas catastrophizing had an indirect effect via increased help-/hopelessness. The current results also indicate that thought suppression mediated the relationship between pain and depression via both direct and indirect effects.
Conclusion Cognitive mediators, such as help-/hopelessness, catastrophizing, and thought suppression, have a significant impact on depression in patients with acute and sub-acute back pain. The current results may aid in the optimization of treatments for these patients by focusing attention toward the modification of dysfunctional cognitive pain-coping strategies.

Keywords Depression · Sub-acute back pain · Catastrophizing · Help-/hopelessness · Thought suppression

Introduction

Back pain is a leading health problem in Germany and in other industrialized nations [1–4]. This health issue is intensified by the co-occurrence of depression in patients with back pain [5–7]. Currie and Wang [8] reported a point prevalence of 19.8 % for depression in chronic back pain patients and found that having back pain was the strongest predictor of depression. Depressive symptoms in acute or sub-acute pain are among the risk factors for pain chronification in the long run and are part of the yellow flag guidelines [9]. Furthermore, it is remarkable that not only clinically relevant depression but also mild depressive symptomatology seem to be sufficient for pain chronification. Higher depression scores can be assessed in chronic low back pain, while lower depression scores can be assessed in early phases of pain [10–13]. To prevent the development of depressive symptoms in pain patients and consequently reduce the risk for chronification, there is a need for precise clinical strategies and treatment recommendations for therapists and patients. Therefore, the role of precise mechanisms that contribute to the development of depression in the context of pain needs to be clarified.

Previous literature has discussed different mechanisms that might potentially influence the relationship between pain and depression [14–17]. A specific focus has been directed toward

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the cognitive mediation hypothesis, which assumes that dysfunctional cognitions mediate this relationship. According to the cognitive mediation hypothesis, psychological factors influence the development of depression in chronic pain patients [18]. However, the exact mechanisms still remain unclear. On the other hand, it is confirmed that pain exacerbation activates different reactions, such as catastrophizing and negative emotional conditions. The person suffering from pain has to regulate these reactions to prevent the development of depression [17].

A pilot study using path analysis in patients suffering from chronic back pain investigated the pain responses of catastrophizing, help-/hopelessness, and thought suppression as mediators of the relationship between pain and depression [19]. The results showed that pain did not influence depression directly. The pain responses had direct effects on depression, while catastrophizing and thought suppression showed indirect effects via help-/hopelessness on depression.

Several cognitive behavioral models of chronic pain [10, 20–23] describe these variables as responses to pain. Catastrophizing is described as a maladaptive response to painful experiences, which results in fear of movement, avoidance behaviors, disability, depression, and an increase in pain [22, 24]. Studies have confirmed that catastrophizing is a predictor of disability, pain, and depression [25–27], and they have revealed a direct link to depression [27–29].

Most studies used catastrophizing as a construct of magnification, rumination, and helplessness [30]. Helplessness is consistent with the concept that no effective coping strategy can be initiated. Abramson [31] reported feelings of helplessness as part of catastrophizing, which has an indirect influence on depression. Help-/hopelessness has also been discussed as a cause of depression [31–33]. A separate measurement of both these constructs can be used with the Kiel Pain Inventory (KPI) [34], which was developed on the theoretical basis of the Avoidance-Endurance Model (AEM) [35], as well as on the cognitively oriented coping model by Lazarus and Folkman [36]. Catastrophizing refers to the putative stressfulness of the pain stimulus, whereas help-/hopelessness measures the cognitions associated with low levels of behavioral control.

The third mediating pain response investigated in the pilot study [19] was the tendency to suppress negative thoughts or experiences such as pain. More recent research has shown that thought suppression may influence the characteristics of dysfunctional patients [20, 24, 37–39], leading to persistent pain and disability in association with behavioral endurance [40]. Wegner and colleagues [41] defined thought suppression as a non-focused search for distraction from pain, which often fails. These failures cause emotional distress and depressive moods. Furthermore, a rebound effect causes an increase in pain-related thoughts and, consequently, leads to help-/hopelessness and a depressive mood [41–44]. Both catastrophizing and thought suppression were followed by feelings of helplessness and hopelessness. In addition to experimental studies [42, 45–48] on the pathogenesis of depression in the context of pain, there is a lack of clinical examinations, such as randomized controlled trials (RCTs) of pain-related thought suppression. Most of the research that has been conducted in this area is focused on the cognitive mediation hypothesis, which has been directly tested in chronic pain samples. However, to prevent the development of depressive symptomatology, there is a need for examinations in acute and sub-acute pain populations. In the current study, we are interested in the role of these mechanisms in relation to the early phase of pain to compare it with the results from the chronic pain sample [19]. Based on previous research and on the pilot study, we suggest that the mediating effect of pain responses on depression can be measured in acute and sub-acute patients as well as in the chronic pain sample. We examined the influence of catastrophizing, help-/hopelessness, and thought suppression as putative mechanisms in the relationship between pain and depression in a sample of patients with sub-acute low back pain. We hypothesized that pain and depression would show no significant relationship after the mediating pain responses were added. We also hypothesize that catastrophizing, help-/hopelessness, and thought suppression will directly influence depression. Moreover, we suggest that catastrophizing and thought suppression will additionally influence depression indirectly via help-/hopelessness.

Materials and Methods

Participants

A total of 164 patients, 49.4 % male and 50.6 % female, with acute (26.8 %) or sub-acute (73.2 %) non-specific back pain were recruited consecutively from general and orthopedic practitioners in the Bochum area for participation in this study. Inclusion criteria were the following: patients were between the ages of 18 and 70 years and had experienced an acute or sub-acute (for less than 90 days) thoracic or lumbar pain episode for the first time. Exclusion criteria were the following: the existence of circumscribed spinal diseases, for example, fractures, neoplasm, or herniated disks, and comorbid psychiatric diagnoses such as active psychosis or mania, acute suicidal risk, and substance misuse.

Procedures

Study criteria were checked by the physicians using a standardized evaluation sheet. The selected patients were invited to participate and informed about the study. They signed a declaration of consent. The Ruhr-University Bochum Ethics Committee approved this study. Patients completed questionnaires

and self-report measures regarding demographic data, such as gender, age, and education level. Additional measures regarding clinically relevant descriptions of pain- and depression-related data were collected.

Measures

Pain intensity was measured using an 11-point numerical self-rating scale (from 0=no pain to 10=worst pain imaginable) regarding patients' experiences during the last 7 days.

Depression was measured with the Beck Depression Inventory (BDI) [49], which is a widely used 21-item self-report measure regarding the severity of the depressive symptoms experienced. Participants reported their affective, cognitive, motivational, behavioral, and biological symptoms of depression. Every item has four possible responses, with scores from 0 to 3 to describe the severity of the depression. Total scores ranging from 0 to 9 indicate no or minimal depressive symptoms, scores ranging from 10 to 18 indicate mild depression, scores ranging from 19 to 29 indicate moderate depression, and scores ranging from 30 to 63 indicate severe depression. Total scores of 21 or greater are described as representing clinically relevant depression [50]. The psychometric properties of the BDI have been reviewed by Beck et al. [50]. Using the BDI and not the structured clinical interview for DSM-IV (SCID) [51], the gold standard assessment for mental disorders, is often discussed in the literature. In addition to refusing BDI assessment in pain patients because of misleading outcomes [52, 53], there are also results that support the BDI in assessing depressive symptoms in pain samples [54–56]. It is important to consider that the BDI is only a symptom screening and not a diagnostic instrument [49]. In this study, it was necessary to measure only the depressive symptoms that may develop into depressive disorder. BDI was chosen to reduce time and financial resources in the current study. Furthermore, the results of this study are comparable with our previous studies and results using the BDI.

Pain-related cognitions were measured with the subscales for help-/hopelessness, catastrophizing, and thought suppression from the Kiel Pain Inventory [34], which includes 34 items assessing pain-related cognitive reactions. Using a 7-point numerical self-rating scale (from 0=never to 6=each time), patients rated how often they experienced the relevant cognitions while suffering from pain in the past 14 days. Higher scores indicate a higher tendency toward these cognitions. The thought suppression subscale consists of four items ("Pull yourself together!", "It is important for me now to hold on.", "Don't make such a fuss!", "It is important not to let myself go now.") and describes the tendency to suppress pain-related thoughts and emotions. The KPI is the older version of the Avoidance-Endurance Questionnaire (AEQ) [57]. The four thought suppression items in the KPI remained the same in the AEQ, which is available in several languages, including English.

The help-/hopelessness subscale of the KPI consists of nine items and describes a disposition to focus on lack of hope (e.g., "It seems the pain will never ease up."). The catastrophizing subscale consists of five items (e.g., "It isn't a serious illness, is it?") and describes the threatening aspects of pain. Cronbach's α was 0.91 for help-/hopelessness, 0.84 for catastrophizing, and 0.78 for the thought suppression subscale [34]. The widely used Pain Catastrophizing Scale (PCS) [30] was not yet available in the German language during the onset of the study. The Coping Strategies Questionnaires (CSQ) [58, 59] and the catastrophizing subscale measure helplessness as a part of catastrophizing [60, 61]. The KPI uses a separate measurement of these both constructs. Therefore, it is possible to investigate whether help-/hopelessness itself influences the relationship between pain and depression, while catastrophizing and thought suppression would indirectly affect depression via help-/hopelessness. The KPI scales were validated by previous cross-sectional and prospective investigations [34, 62, 63].

Disability was assessed by the Pain Disability Index [64, 65]. This 7-item self-reported measure was developed to assess pain-related disability. The PDI consists of seven domains: family/home responsibilities, recreation, social activities, occupation, sexual behavior, self-care, and life support activities. Each item is rated from 0 (no disability) to 10 (worst disability). There is evidence of good reliability of the PDI [66, 67]. In the current study, the German version of the PDI was used [68].

Statistical Analysis

Path analysis was chosen as special case of structural equation modeling for this cross-sectional study. This method permits the examination of a priori formulated hypothesis that have multiple intercorrelations. Different indicators may describe the adaptation of the empirical data to the theoretical model structure. Descriptive data are represented as the number of subjects and percentages or as means and standard deviations. In general, the sample size in structural equation modeling plays an important role. Small samples are related to the alpha and beta-errors as well as technical errors [69]. All these factors might in sum reduce the power of a study [70]. Kline [69] explained that complex models with many parameters need lager samples and simpler models need smaller samples. The current study consists of a modest model with only few parameters. In accord with the *N:q rule*, which considers the sample size and the number of parameters in relation, the current sample size seems to be adequate [71]. Statistical calculations were conducted with the Statistical Package for Social Sciences (SPSS 22) [72] and with the related package Analysis of Moment Structures (AMOS) [73] using the

Maximum Likelihood method. AMOS is frequently used for performing path analysis. Byrne [74] recommends to use the following fit indices to assess the model fit:

Chi-Square Test (x^2-test) The chi-square index and the chi-square adaptation test provide information about the validity of a model. Good model adaptation is evident when the chi-square value is non-significant. However, the chi-square value should be interpreted conservatively, as it is a measure of the adaptation goodness of the whole model. Therefore, it accepts high values when complicated models deviate slightly from the empirical variance covariance matrix [74].

Root-Mean-Square-Error of Approximation (RMSEA) The RMSEA is an inferential statistical measure that determines whether a model is similar to reality. In contrast with the chi-square test, the RMSEA does not test the absolute correctness of a model; rather, the model's complexity is determined by its degrees of freedom. According to Brown and Cudeck [75], the RMSEA's values can be interpreted as follows: RMSEA≤0.05 is a good model fit, RMSEA≤0.08 is a satisfactory model fit, and RMSEA≤0.10 is an unacceptable model fit.

Comparative Fit Index (CFI) This comparative evaluation takes into account the saturation and independence of the model formulated by the user. The CFI is an incremental fit measure that compares default and independent models. When the default model differs only slightly from the independent model, this measure has a value close to zero. When the value is higher than 0.9, there is clear improvement in the default model compared to the independent model [74].

Results

Study Sample

Table 1 presents the essential sociodemographic characteristics of the study's sample.

Table 1 Sociodemographic data (N=164)

Variables	N	%
Gender		
Male	81	49.4
Female	83	50.6
Age		
18–30 years	20	12.2
31–40 years	41	25.0
41–50 years	43	26.2
51–60 years	42	25.6
61–70 years	18	11.0
Family status		
Single	29	17.7
Married	96	58.5
Cohabiting	15	9.1
Divorced	17	10.4
Widowed	5	3.0
Highest education		
Basic high school education	55	33.5
higher education	42	25.6
secondary education	61	37.1
Another graduation	4	2.4
Occupation		
Full time	81	49.4
>Part time	23	14.0
<Part time	17	10.4
Housewife/man	16	9.8
Education	9	5.5
Jobless or unemployed	7	4.3
Other	11	6.7
Days not working		
Certified sick	51	31.1
One week	28	17.1
Two weeks	13	7.9
Four weeks	3	1.8
Eight weeks	5	3.1
More than 8 weeks	2	1.2
Duration of pain		
0–42 days (acute)	144	87.8
43–84 days (sub-acute)	20	12.2

Descriptive Statistics

The average pain intensity (experienced during the last 7 days) was 4.62 (standard deviation (SD)=2.15). The average depression was 6.73 (SD=6.0). The average disability was 2.48 (SD=1.83). With regard to pain-related cognitions, the average for thought suppression was 2.77 (SD=1.27), which was the highest when compared to help-/hopelessness (M=1.86, SD=1.29) and catastrophizing (M=1.78, SD=1.21).

An evaluation of the depressive symptoms rates showed that only 3.7 % of patients experienced clinically relevant depression, whereas 70.7 % showed no symptoms of depression and 25.6 % experienced a mild form of depression.

Correlations

Table 2 presents the correlations for the variables which establish the base of the path analysis.

Table 2 Bivariate correlations between pain intensity (last 7 days), depression (BDI), help-/hopelessness, catastrophizing, and thought suppression

	(a)	(b)	(c)	(d)	(e)
(a) Pain intensity (last 7 days)	–	–	–	–	–
(b) Depression	0.28*	–	–	–	–
(c) Help-/hopelessness	0.35*	0.32*	–	–	–
(d) Catastrophizing	0.33*	0.24*	0.67*	–	–
(e) Thought suppression	0.15	0.24*	0.25*	0.15	–

*$p<0.01$ (one-tailed)

Path Model

For the a priori formulated path model (see Fig. 1, based on the pilot study [19]), the fit indices indicated an acceptable model fit: χ^2 (df=1)=2.02, p=0.15, RMSEA=0.08, and CFI=0.99.

Pain had a significant influence on catastrophizing (β= 0.34; critical ratio (CR)=4.46; p 0.001) and a significant effect on help-/hopelessness (β=0.13; CR=2.09; p 0.05). The path between catastrophizing and help-/hopelessness (β= 0.61; CR=9.96; p 0.001) was also significant. In addition, the paths between thought suppression and help-/hopelessness (β=0.14; CR=2.40; p 0.05), help-/hopelessness and depression (β=0.22; CR=2.15; p 0.05), and thought suppression and depression (β=0.16; CR=2.15; p 0.05) were significant. There were no significant connections for the paths between pain and thought suppression (β=0.14; CR=1.72), catastrophizing and depression (β=0.019; CR=0.19), or pain and depression (β=0.16; CR=1.87) (see Fig. 1). The standardized indirect effects mirror the effects above. Pain indirectly influenced help-/hopelessness with an effect of 0.23 via catastrophizing and thought suppression. The indirect effect of pain on depression was 0.11. This effect was influenced by catastrophizing and thought suppression. These variables also indirectly influenced depression via help-/hopelessness. Thought suppression had an indirect effect of 0.03 on depression via help-/hopelessness. Catastrophizing had an indirect effect of 0.13 on depression via help-/hopelessness. The direct effects explain 8 % of the variance of depression. The indirect effects additionally explain 16 %. In total, 24 % of the variance of depression is explained by the cognitive pain responses represented in this model.

Discussion

Based on the cognitive mediation hypothesis and on the previous pilot study [19], we suggested that the mediating effect of the pain responses on depression can be measured in acute and sub-acute patients. We hypothesized that pain and depression show no significant relationship after the mediating pain responses were added. Moreover, we hypothesized that catastrophizing, help-/hopelessness, and thought suppression would directly influence depression, while catastrophizing and thought suppression additionally would influence depression indirectly via help-/hopelessness.

Overall, the fit indices indicated an acceptable model fit, with the result that the a priori formulated path model represented the empirical data. Consistent with previous studies [18, 19], the present study supports the assumption that pain itself is not a sufficient condition for depression. According to the hypothesis, pain and depression showed no significant relationship after the pain responses were added to the path model. Other variables seem to mediate this relationship. This is the first study that investigated the mediating effects of catastrophizing, help-/hopelessness, and thought suppression on depression in acute and sub-acute pain patients.

In the present study, help-/hopelessness played a central role in the relationship between pain and depression. Pain intensity was positively related to help-/hopelessness, and help-/hopelessness was in turn positively related to depression. Furthermore, we confirmed the hypothesis that there was an indirect mediating effect of catastrophizing via help-/hopelessness. These results are consistent with those in the literature. Klasen and colleagues [19] reported a direct relation between help-/hopelessness and depression and an indirect

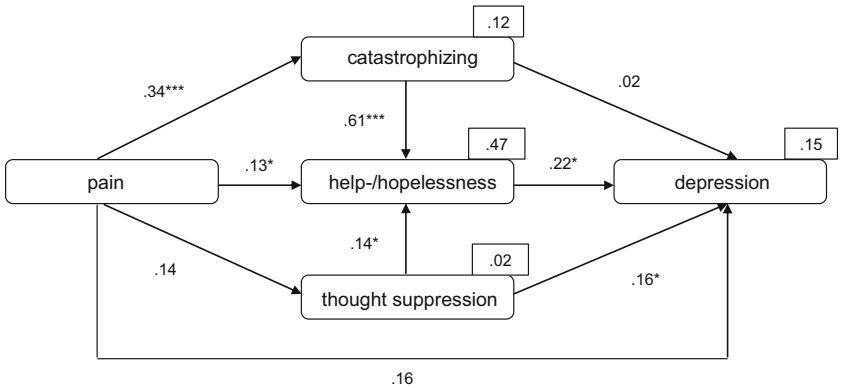

Fig 1 Path model with standardized path coefficients and multiple correlation coefficients (*squares* at the variables). *$p<0.05$; ***$p<0.001$

path between catastrophizing and depression in a sample of chronic back pain patients. More recently, Fahland and colleagues [76] showed in a large population-based longitudinal study that help-/hopelessness was a direct mediator between pain and depression.

In contrast to previous findings [27–29], catastrophizing did not reveal a direct path to depression in the current study. This inconsistency in results has been discussed in various ways in the literature. Sullivan and colleagues [30] conceptualized the construct of catastrophizing by developing the PCS, which consists of magnification, rumination, and helplessness. Furthermore, Sullivan and colleagues [61] assigned these three dimensions to the primary and secondary appraisal processes as part of a transactional model of stress [36]. The transactional model describes the interaction of two appraisal steps with regard to a special stressor. The first appraisal includes judgments about whether the stressor is stressful, and the second appraisal determines whether there are coping strategies. The authors assigned magnification and rumination to the first appraisal and helplessness to the second appraisal. The current results lead to the suggestion that in early phases of pain it is not catastrophizing itself but the thoughts of having no control about the pain experience that might influence the development of depression. Therefore, the use of the KPI [34], which allows a separate measurement of catastrophizing and help-/hopelessness, seems to be confirmed. The relation of these two constructs is reflected in the significant correlation of r=0.67 in the current study. Additionally, the correlation is higher than the inter-correlation of the subscales in the KPI and also of the subscales of the Avoidance-Endurance Questionnaire (AEQ) [57] which is the shortened version of the KPI. The correlations of these two constructs seem to be higher in acute and sub-acute samples than in chronic pain samples [19], which seem to be a noteworthy finding.

The indirect path between catastrophizing and depression, which we found in our study, is in line with the main assumptions of cognitive behavioral models [10, 20–23]. These do not expect a direct relation between catastrophizing and depression. For example, Vlaeyen and Linton [10] suggested that catastrophizing leads to pain-related fear, avoidance, and hypervigilance, which in turn, lead to disuse, depression, and disability. However, several modifications of the fear-avoidance model are discussed [77–79], indicating the need for an extension of the fear-avoidance pathway. In their topical review, Linton and Bergbom [17] suggested conceptualizing catastrophizing as a mechanism in emotion regulation and, therefore, as part of both pain and depression.

In addition to catastrophizing, thought suppression represents a strategy of emotion regulation, which is suggested to occur automatically as well as more deliberately [41]. Thought suppression, as a potential mediator between pain and depression, has rarely been included in previous pain research. As expected, thought suppression revealed a direct positive association with depression in the present sample of acute and sub-acute patients. This is consistent with Klasen and colleagues [19], who found a direct path in chronic back pain patients. Moreover, we detected an indirect path via an increase in help-/hopelessness. The indirect path between thought suppression and depression via help-/hopelessness is consistent with the assumptions of Wegener and colleagues' [41] theory of ironic processes, which declared that patients often fail to distract themselves from their pain. The unfocused search for distraction causes emotional distress and depressive mood. In contrast, focused distraction leads to a decrease in pain [80].

Further, Wegner and colleagues described a rebound effect with unwanted thoughts and sensations occurring more often, leading to an increase in pain distress and help-/hopelessness [41–44]. The relationship between thought suppression and distress has already been shown in patients with obsessive-compulsive disorder [81]. The authors showed that there was an increase in distress after focusing on unwanted thoughts and after following a thought suppression condition. However, participants of a control condition (acceptance) reported decreased distress. Wenzlaff and Luxton [82] investigated the relationship between thought suppression and dysphoria in a student sample: healthy volunteers who were classified as high suppressors showed an increase in rumination and dysphoria after a phase of high stress compared with low suppressors. In the field of low back pain, our results on thought suppression and depression are in line with the avoidance-endurance model of pain [20]. This model conceptualizes thought suppression as a part of the distress-endurance pathway leading to affective distress and depressive mood, even in the short term [40]. In the long term, thought suppression, in association with behavioral pain persistence, is assumed to cause overuse/overload of physical structures and, finally, an increase in pain and disability [83].

To understand the relationship of stressful experiences and the worsening of mood, emotional regulation should also be considered. Catastrophizing, help-/hopelessness, and thought suppression represent different aspects of emotion regulation, processes suggested to be relevant not only in psychopathology but also in pain [17]. In a recent meta-analysis on emotion regulation in different affective disorders, suppression revealed positive associations with depression [84]. In contrast, strategies such as acceptance, reappraisal, and problem solving showed negative correlations with depression and were labeled as adaptive [84]. Different sub-processes of emotion regulation are distinguished with regard to pain [37, 38, 85]. Lumley and colleagues [38] reported a more broadly defined approach of emotional processes. In addition to the modulation of emotions, the authors considered emotional awareness, expression (expression vs. suppression), and experiencing. Many patients suffering from pain show a suppression reaction

after perceiving pain, followed by an emergence of different negative consequences such as distress, depressive moods, and an increase in pain [41–44, 86]. The present results also demonstrate this in relation to depression.

Several limitations should be mentioned, as they have the potential to guide future research. Although the results from the current study show an acceptable model adaptation, they should be interpreted with caution [74]. The use of a cross-sectional study design should be especially noted. Path analyses do not provide unequivocal proof of causality in cross-sectional designs. Alternatively, a prospective study design should be used in future studies, although this type of design is more complex and cost-intensive. Furthermore, all data were measured by self-assessment questionnaires; all cognitive pain responses were assessed with the KPI. These data represent subjective ratings of the participants. Especially in the measurement of cognitive and emotional strategies, the participants need to be able to reflect and remember their own reactions [87]. Future studies should focus on objective and experimental measurements to repeat the current results.

Moreover, the previous results show quite a low mean BDI score in comparison to chronic pain samples. Lower levels of depression have often been shown in patients with acute and sub-acute pain [10–13]. In the current study, approximately 29.3 % of our sample revealed mild (25.6 %) or clinically relevant (3.7 %) depressed symptomatology. Interestingly, epidemiological data of chronic pain are comparable with these values [88]. Nevertheless, the depression scores in the present study were sufficient to measure strong mediating effects of the relationship between pain and depression. However, the results were in line with previous research. Depression scores in acute pain were lower compared with depression scores from studies in chronic pain populations [10–13].

However, a detailed comparison with the results by Klasen and colleagues [19] displays lower correlations in the present study than in the chronic pain sample. This is congruent with the results of Scholich and colleagues [89], who investigated the possible impact of assessment time on the height of correlations. Correlations between psychological variables, pain intensity, and disability have been shown to be low at the start of a treatment, especially in early phases of back pain. After 6 months, when treatment has been finished, these correlations increased significantly. Kovacs and colleagues [90–92] presented comparable results. Due to these results and the higher correlations in the chronic back pain sample [19], we assume that we would find increased correlations in a follow-up measurement of the present sample. Therefore, it is crucial that future research should employ prospective study designs. Additionally, future studies should integrate already known factors influencing the relation between pain and depression to build a multifactorial explanation model that might explain a high number of variance. In the long run, it might be possible to determine treatment strategies from such a model.

Finally, although there are limitations, it is important to note that this was the first study investigating catastrophizing, help-/hopelessness, and thought suppression as potential mediators between pain and depression in a sample of patients with sub-acute back pain. Consistent with studies by Rudy and colleagues [18] and Klasen and colleagues [19], the present study supports the hypothesis that pain is not a sufficient condition for depression. Cognitive pain responses, such as catastrophizing, help-/hopelessness, and thought suppression, seem to mediate the relationship between pain and depression not only during chronic pain [19] but also during early phases of back pain.

The results of the present study have implications for improving the treatment strategies used with back pain patients. One central aspect that should be included in back pain treatment is the modification of dysfunctional cognitive pain responses. Increased attention is already focused on this aspect with regard to treatment strategies for patients with chronic back pain. Additionally, the modification of dysfunctional cognitive pain responses should be fostered to reduce the probability of depression during the early phases of treatment for back pain. This is in line with current results [93, 94], confirming the prominent role of depressed symptomatology as a risk factor for the development of chronic pain.

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Compliance with Ethical Standards All procedures followed in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2000. Informed consent was obtained from all patients being included in the study.

Conflict of Interest The authors declare that they have no competing interests.

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