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Dynamic pain–emotion relations in chronic pain: a theoretical review of moderation studies

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Current developments in chronic pain research are changing the focus in the study of pain–emotion relations from the identification of general patterns to the study of dynamic and context-related interactions manifesting both within and between individuals. This shift towards understanding variation at both intra- and interpersonal levels has significant clinical implications for psychological adjustment to chronic pain conditions, and thus represents an important topic for both clinical and health psychology. This article reviews the existing theoretical explanations of these dynamics and their emerging empirical support, and suggests further areas of investigation. A literature search identified research on moderators of pain–emotion relations in chronic pain; existing theories were also examined from this perspective. A theoretical analysis revealed several important contributions, including the concepts of affect differentiation, generalised discrimination ability, resilience, vulnerability, coping, emotion regulation and desynchrony, which are described here together with the relevant empirical research and clinical implications. Important areas for development are the clarification of the common elements and opposing predictions and the empirical examination of mediating mechanisms. Several methodological issues are discussed. This review identifies a rich theoretical basis for research into pain–emotion moderation, and suggests that further examinations of such relationships might hold important clinical consequences.

Keywords: pain; emotion; moderation; chronic pain; pain–affect

In comparison to other health conditions, chronic pain is a special case, in that its main symptom, pain itself, is simultaneously a sensory and emotional experience. Therefore, understanding the role of emotion in pain is central to our efforts of improving psychosocial adjustment to chronic pain. Moreover, since pain is a prevalent symptom in most health complaints, the study of pain–emotion relations can be considered a topic of strategic interest to health psychologists. However, chronic pain research and pain management interventions have focused predominantly on cognition and behaviour, and by comparison our understanding of emotion is still incipient. Although numerous studies have tackled pain–emotion relations from different perspectives, a coherent image is yet to emerge. The present review attempts to bring together various theoretical contributions in an effort to

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clarify one important aspect of this relationship: its dynamic quality, as shown by the increasing number of studies reporting moderating factors for pain–emotion relations. Thus, our review also aims to contribute to the current shift from solely cognitive and behavioural models towards recognising the contribution of emotion in chronic pain management.

Emotion has been studied from various perspectives in chronic pain research. In the study of pain perception, the motivational–affective dimension has been considered an essential element, complementary with the sensory–discriminative and cognitive–evaluative dimensions (Melzack & Katz, 2001). Immediate pain unpleasantness and secondary pain-related affect have been identified as distinct stages subject to different sensory and cognitive influences (Price, Riley, & Wade, 2001). Most psychotherapeutic approaches to chronic pain management have considered emotion as part of their theoretical foundation, from early psychoanalytic accounts (Engel, 1959) to more recent contextual cognitive–behavioural therapies (e.g., Dahl, Wilson, Luciano, & Hayes, 2005). Moreover, research has targeted the role of specific emotions and emotion regulation strategies in chronic pain adjustment, such as anger expression (Bruehl, Chung, & Burns, 2006), fear of pain (Lethem, Slade, Troup, & Bentley, 1983), fear of re-injury (Leeuw, Peters, Wiers, & Vlaeyen, 2007; Vlaeyen, Kole Snijders, Rotteveel, Ruesink, & Heuts, 1995) or anxiety sensitivity (Asmundson, Norton, & Allerdings, 1997).

In the last 20 years, an increasing number of studies have identified various intra- and interindividual characteristics which influence the relationships between different aspects of the chronic pain experience. Several moderators of the pain–emotion link have been examined, and some studies have proposed theoretical explanations for the interactions identified. This growing body of research suggests that there may be no broadly applicable relations between pain and emotion in chronic pain, but rather variable relations depending on many personal and contextual factors.

Given the promising results but also the increasing number and variety of these types of studies, a theoretical analysis of this research area becomes imperative. For the researcher, it would encourage the consideration of alternative explanations of pain–emotion moderation effects and the refinement of research designs and hypotheses. For the practitioner, it would stimulate a better understanding of these dynamics, and the various influences potentially applicable to individual cases. For the wider health psychology community, it would provide an insight into the complexity of pain–emotion relations in chronic pain and the methodological requirements for studying such dynamics; this insight might both enhance understanding of pain as a symptom common to various health conditions and provide an example of studying contextual dependence, which may be applicable to other research questions in health psychology. The present review aims to perform such analysis, and to offer important clarifications regarding the proposed moderators, the mechanisms via which they might exert their influence, the most suitable research methods and the theoretical gaps in need of further empirical research. We hope that this analysis and clarification will help focus further research efforts in this area and situate the interpretation of their findings within the wider theoretical landscape of chronic pain adjustment, while also enhancing understanding of pain, emotion and context-dependence for a broader audience.

Methods

Search strategy

Publications were retrieved via a broad search in relevant databases (PubMed, PsycINFO and Web of Science accessed on 25 November 2010 and covering all available years up to November, Week 3, 2010) using a selection of keywords: pain, emotion, affect, mood, anger, depression, anxiety, fear, sadness, shame, happiness, joy, moderator, dynamic, interactions and interpersonal differences. The search syntax is presented in Appendix A. A total of 1550 articles were identified (506 PsychINFO and 761 PubMed, of which 235 articles were in both databases; 863 in Web of Science, of which 518 were shared with PsychINFO and PubMed). Other relevant works referenced in the selected publications were retrieved manually. Each publication was examined for relevance to the topic of the review and related inclusion and exclusion criteria, resulting in 68 journal articles selected (of which 14 reported null results).

Selection criteria

The main selection criterion for the literature search was the inclusion of empirical results regarding moderating influences on the dynamic relation between pain and emotion, as our focus was on the personal and contextual characteristics which influence the relations between pain perception and emotional experience in chronic pain sufferers. Therefore, we excluded studies that focused on other measures of illness severity or disability if no pain reports were included, and also excluded studies that targeted other psychological variables without a relevant emotional component. Concepts such as pain catastrophising, acceptance, anxiety, self-efficacy and depression were considered relevant for our emotion focus due to their substantial affective content.

As our focus was on examining the experience of living with chronic pain, the studies selected had to describe chronic pain populations, irrespective of age. We also excluded studies of cancer-related (malignant) pain. Although benign and malignant pain are not considered distinct physiologically, cancer pain is more closely connected with tissue pathology and treatment toxicity and has different time implications particularly in its terminal stages (Jacobson & Mariano, 2001). The potential differences in affective dynamics justify this exclusion criterion and recommend the separate investigation of malignant pain. We excluded experimental studies on normal populations, on acute pain following medical interventions, comparisons between healthy and chronic pain samples (i.e., group membership as moderator) and studies examining the effect of therapeutic interventions (i.e., treatment as moderator).

As moderation is a quantitative construct, we selected only those studies that addressed differences at intra- or interpersonal levels in a quantitative design (thus excluding qualitative studies).

Importantly, we also included studies reporting and discussing null moderation results. However, as the majority did not detail the theoretical aspects of their moderation analyses, they are mentioned in the text only when relevant for our theoretical analysis, and detailed separately in Table 2 of Appendix B. Their importance to theory testing is detailed in the 'Discussion' section.

Our search was limited to English language journal articles, excluding dissertation abstracts, non-English articles and book chapters. However, this search was supplemented with an analysis of main theories in chronic pain with regards to pain–emotion relations, as described in other sources in the literature, including books and book chapters. Theoretical literature cited in the articles reviewed was included in the review process recursively and supplemented by the authors' prior knowledge of chronic pain theory.

Literature review process

The selected articles were examined from several perspectives. Firstly, we extracted information about the specific chronic pain condition that characterised the sample, the sample size, the research design and the data analysis methods used in the moderation analyses. Secondly, the variables included in the analysis as predictors (independent variables), outcomes (dependent variables) and covariates (control variables) were identified, together with the instruments used. Thirdly, and most important for our review, the specific interactions identified and the interpretations provided by the authors were extracted.

Given our theoretical focus, we considered that additional details regarding the methodology and results (such as effect sizes and parameter estimates) would not be relevant for our aim, which was to provide a preliminary theoretical map of an emerging field within the space constraints of a topical review. Certainly, examining this information would have been essential if our purpose were to weigh the evidence regarding these theoretical accounts. The interested reader may refer to the original research articles and assess their methodological rigorousness and the practical significance of their results.

Many studies examined the moderators of the pain–emotion association as one of several research hypotheses; we summarise here only the analyses related to the topic of this review.

Results

Summary

Sixty-eight reports of empirical studies investigating moderators of pain–emotion relations have been published in the journals accessed via PubMed, PsychINFO and Web of Science database, from 1987 to November 2010. Details regarding the sample characteristics, research design, data analysis methods, variables measured, interactions identified and interpretations provided are included in Tables 1 and 2, in Appendix B.

The most frequently studied chronic pain condition was rheumatoid arthritis (RA), considered in 25 studies, followed by heterogeneous samples (17), fibromyalgia (FM, 8), osteoarthritis (OA, 6) and chronic low back pain (4). Other specific conditions (multiple sclerosis, reflex sympathetic dystrophy syndrome, spinal cord injury, temporomandibular disorder, etc.) were considered only in single studies.

Most studies have been conducted on adult populations, except six studies focusing on children and/or adolescents. Most studies were conducted on mixed gender samples, a few on women only (10) and only one study on male veterans.

A substantial number of studies have been conducted by two research centres, University of Connecticut (15) and Arizona State University (12), also in collaboration with each other or with other centres, while other centres contributed a limited number of studies to this research topic.

In terms of research design, 35 were cross-sectional, 4 experimental and 30 longitudinal, among which 23 were diary studies involving weekly or daily measurements (2 articles included 2 studies in the same report). However, among the longitudinal studies, only 15 studies (among which 8 were diary studies) actually examined time-lagged interaction effects.

In terms of statistical methods for data analysis, 40 studies used hierarchical multiple regression analysis, 18 used hierarchical linear modelling, while other methods were used in fewer studies: analysis of variance (5), Fisher's z test (3), correlations or comparison of correlations without Fisher's z test (2), multigroup structural equation modelling with equality constraints (2), pooled time-series regression analysis (1), general linear mixed modelling (1).

The empirical reports included in this review and the related theoretical literature reveal a rich and varied landscape of factors influencing pain–emotion relations, which will be discussed in the next sections (see Figure 1 for a summary). All theoretical models reviewed have tackled pain–emotion relations as part of the broader context of chronic pain adjustment, and thus the following discussion should not be interpreted as a full exposition of the theories we refer to.

The most detailed theoretical contribution to this topic has been brought by the dynamic model of affect (DMA; e.g., Zautra et al., 2001), which has developed specific predictions for chronic pain conditions and has obtained substantial

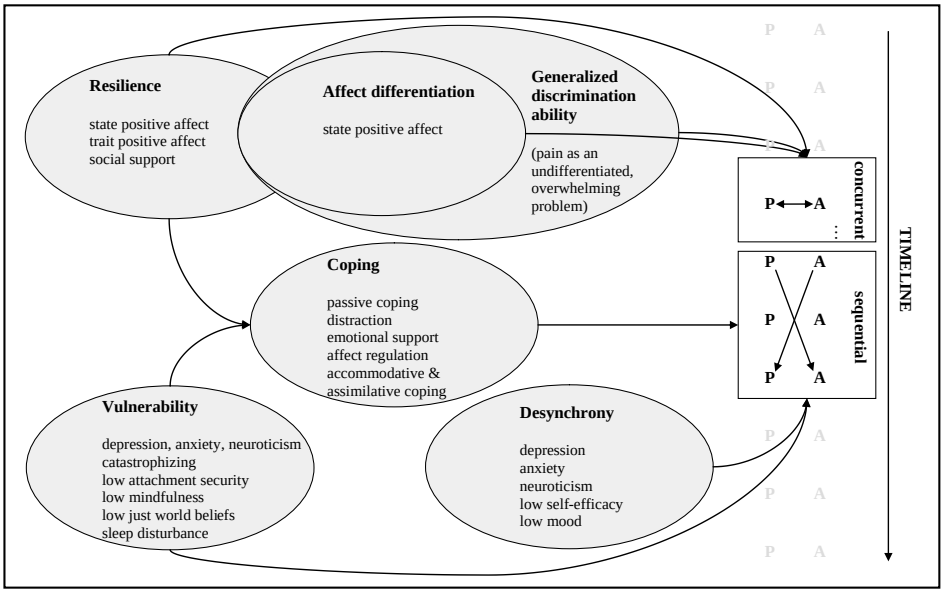

Figure 1. Graphical interpretation of pain–affect moderation literature: in the continuous interaction between pain (P) and affect (A), multiple factors are proposed to intervene. They may influence their simultaneous relations (affect differentiation, generalised discrimination ability, resilience) or their time-lagged relations (resilience, vulnerability, coping, desynchrony).

empirical support. The starting point of the DMA is affect differentiation, which we adopted as our starting point and will be the topic of the first section. However, other theoretical approaches to chronic pain, while not directly focused on pain–emotion relations, refer to a generalised difficulty of differentiating between various aspects of chronic pain adjustment, including pain and emotion. The implications of this hypothesised difficulty, described in the second section, suggest new avenues for research and complement the predictions of affect differentiation within the DMA.

A second aspect of the DMA addresses the buffering role of positive affect in the relation between pain and negative affect, discussed in the third section. Although its predictions are largely overlapping with affect differentiation, the buffering hypothesis stipulates different mechanisms related to coping and links positive affect with other factors such as social support. A complementary view is represented by the diathesis–stress model (Banks & Kerns, 1996), which stipulates an interaction between vulnerability factors (e.g., depression and neuroticism) and illness-related stressful events in increasing the psychological impact of the condition. Vulnerability hypotheses (described in Section 4) are distinguished from buffering hypotheses by their focus on detrimental, as opposed to beneficial, influences. However, they both suggest an important role for cognitive processes and coping mechanisms as pain–emotion moderators, which are described in Section 5.

A contrasting prediction is offered by the concept of desynchrony (Phillips, 1977, as cited in Lethem et al., 1983). It stipulates an increased negative impact of distress on adjustment to chronic pain under certain conditions when pain stimulation is low. Desynchrony-consistent findings are described in Section 6.

1. Affect differentiation – positive affect and negative affect merge when pain increases

Several empirical studies reviewed (Potter, Zautra, & Reich, 2000; Strand et al., 2006; Zautra et al., 1997, 2001, 2005, 2007) have tested predictions of the DMA in chronic pain (also in Davis, Zautra & Smith, 2004; Reich, Zautra & Potter, 2001; Zautra, Reich, Davis, Potter, & Nicolson, 2000). The initial focus of the DMA has been to clarify a longstanding controversy regarding the distinctiveness between positive and negative affect. While factor analytic research has supported mostly a single-dimension model, research on the impact of life events and the impact of methodological factors on affect measurement has found evidence for a two-dimensional structure. To reconcile these contrasting findings, the DMA proposed a context- and person-dependent model of affect, where stress is a central contextual influence on the variable relationship between positive and negative affect. In essence, it stipulates that under stress people tend to perceive their emotional life in a single positive–negative dimension, while in normal circumstances they tend to perceive positive and negative affect as independent dimensions.

Stress is defined as a state of increased uncertainty (understood as information processing). It represents a departure from current expectations, especially an undesirable one, and therefore demands an adaptive response, which invariably requires a reduction in uncertainty. Under non-stressful conditions, maintaining independent affective dimensions involves maximal uncertainty and is cognitively demanding, but it is also adaptive, since it offers maximum information and thus increases the organism's ability to respond flexibly to diverse environmental stimuli. Under stress, the additional ensuing uncertainty competes for resources and

increases the pressure for reduced uncertainty, which overrides the benefits of differentiation and leads to the reduction of affect independence. Thus, separate affect dimensions are merged to maintain a stable uncertainty level (Potter et al., 2000; Zautra et al., 1997, 2001).

The DMA predictions extend beyond the issue of chronic pain adjustment, but they have specific implications for pain. The DMA stipulates that pain, as a stressful stimulus, results in an increased correlation between reports of positive and negative affect (Zautra et al., 2005). Thus, painful episodes increase the inverse association between positive and negative affect reports, as the associated cognitive demands lead to adopting simpler representations of emotional experience. Moreover, the decreased predictability and controllability of chronic illness and pain influence the uncertainty (stress) levels and pressures for merging affective dimensions, leading to increased correlations in more uncontrollable health conditions (Zautra et al., 1997). Such limited information processing also affects stress-related variables, including pain, and so the DMA predicts that the associations between reports of pain and affect are also moderated by individual differences and contextual factors (Davis et al., 2004)

The DMA proposes that 'potential individual differences in the ability to sustain affective differentiation during pain and other stressors' (Davis et al., 2004, p. 1133) influence the strength of association between affective dimensions at the inter-individual level. In other words, people differ in their tendency to perceive positive and negative affect as a single dimension in times of stress. Cognitive structure (i.e., the propensity for complex processing, measured by Response to Lack of Structure subscale of the Personal Need for Structure Scale) showed a moderating role in the positive–negative affect relation in a chronic pain sample (Potter et al., 2000). Mood clarity (as a trait measure of emotion regulation) interacted with positive affect to predict negative affect levels in a sample of women with arthritis (Zautra et al., 2001). In emotion research, concepts such as emotion granularity (Barrett, 2006) reflect similar issues of distinguishing between different aspects of the experience in order to generate more adequate behaviours.

The affect differentiation processes stipulated by the DMA also affect perceptions of the social world, i.e., the differentiation between supportive versus disregarding behaviours from partners, between perceived support and negative social ties or between interpersonal distress and sense of support. The partner's affective differentiation is also hypothesised to influence the patient's ability to sustain affective complexity in face of stressful events or chronic pain (Davis et al., 2004).

The mediating mechanisms proposed refer to attention and information processing. During stress and uncertainty, attention is focused on the negative information relevant for a quick adaptive response to the current threat, at the expense of positive information; thus, the positive and negative dimensions are fused in a single bipolar continuum. For individuals suffering from chronic pain, due to the demands on cognitive resources that they already face, this process is especially powerful (Davis et al., 2004).

The process is explained in terms of 'stress-induced narrowing of the range of attention, increased difficulty in performance of complex judgements, and more unified, "single-minded" response to environmental inputs' (Zautra et al., 1997, p. 82). Physiological mechanisms related to regulation of norephinephrine, oxytocin

and endogenous opioids following stress are also considered related to affect differentiation (Zautra et al., 2000, 2001). The role of catecholamine and opioid mechanisms in pain-related positive affect regulation in FM is supported by recent evidence for a moderating role of the catechol-*O*-methyltransferase gene (COMT/val^{158}met) and the opioid receptor gene (OPRM1/asn^{40}asp) in the pain–positive affect relations (Finan et al., 2010).

It is important to highlight that affect differentiation within the DMA refers to the simultaneous relations between positive affect, negative affect and pain, which have been shown to follow the predicted pattern in samples of OA, RA and FM (Potter et al., 2000; Strand et al., 2006; Zautra et al., 1997, 2001, 2005, 2007). The authors acknowledge the difficulties in establishing causal relations and therefore in linking the model to clinical intervention based on interactions between concurrent measurements.

Affect differentiation suggests that conceptualising pain and positive and negative affect as distinct dimensions in clinical practice may be useful in clinical diagnosis, as these dimensions provide distinct information in some contexts. Moreover, visualising both increased positive affect and reduced negative affect as therapeutic outcomes may assist chronic pain sufferers and health care professionals in reaching a definition of quality of life broader than the lack of negative consequences of painful stimulation, and thus open to new therapeutic goals (Zautra et al., 2001). It also raises an important clinical question: could training in affect differentiation be useful in improving adjustment to chronic pain conditions? The research reviewed above cannot provide a satisfactory answer, as it does not study exhaustively the possible mechanisms, or the likely existence of longitudinal causal relations. These are further explored in the next sections.

2. Generalised discrimination ability – separating pain from its emotional consequences enables response flexibility

Several theoretical contributions describe generalised difficulties in chronic pain sufferers to discriminate between various aspects of their illness experience. These suggest that affective differentiation as described by the DMA might be just a special case of discrimination ability.

In his seminal work on operant–behavioural chronic pain management, Fordyce (1976) described a 'vicious cycle effect' where the frequent association between distress and pain makes discrimination between these states increasingly difficult, which he termed 'discrimination error'. This statement suggests that increased illness duration might lead to an increased difficulty to discriminate between pain and distress, at least unless other factors intervene to loosen this association (i.e., via an operant–behavioural intervention, or different environmental sources of reinforcement).

The ability to discriminate between various aspects of the pain experience can actually be considered one of the main targets of cognitive–behavioural therapy (CBT; Turk, Meichenbaum, & Genest, 1983), which starts with the assessment and reconceptualisation of the sufferer's situation. In essence, this stage targets the transformation of an undifferentiated, overwhelming problem into distinct, manageable problems. Acceptance and commitment therapy (ACT) follows on similar lines, as chronic pain acceptance involves the discrimination between the presence of pain

and the availability for value-based activities. Discriminating between pain and emotion is also reflected in the ACT concept of relational framing and in its therapeutic goal of changing not the content but the function of mental events by enhancing the flexibility of the relational framing in which the events participate (Hayes, Strosahl, & Wilson, 1999).

Other theoretical models of illness and chronic pain adjustment make similar distinctions. The self-regulatory model and its related parallel processing model of pain distress (Leventhal & Everhart, 1979) also highlight the necessity of a distinction between the sensory–cognitive aspects of pain (or any other health symptom) and its emotional aspects. The clinical application to diminishing acute pain related to medical interventions via conscious exposure to sensory information prior to medical procedures is a powerful argument for the value of this discrimination ability. Eccleston and Crombez's (1999) cognitive–affective model (CAM) of the interruptive function of pain also includes a discussion on the dissociation between pain and threat: the threat value of the pain stimulus moderates its selection over competing stimuli/demands, thus enhancing its interruptive function. Crombez, Eccleston, Baeyens, van Houdenhove, and van den Broeck (1999) found that high pain intensity interacted with high levels of pain-related fear in predicting increased attention interference in a laboratory task in a heterogeneous chronic pain sample, which suggests a facilitatory effect of fear on the negative effect of pain on attention.

While these theoretical statements are consistent with the DMA in broader terms (especially given the important role of emotional distress), their focus is rather on the distinction between pain as a sensory stimulus and its associated distress as a motivational component linked directly to behavioural responses, as opposed to an issue of the structure of emotional experience. As differentiating between the various specific aspects that compose the general problem of adjusting to chronic pain is a central issue in pain management, it is surprising that most theories mentioned above have not yet been translated into specific predictions related to the ability to differentiate between various aspects of the pain experience (including pain-related distress) and tested via moderation hypotheses.

This generalised differentiation ability may reside in attention-based and physiological mechanisms specified by the DMA. As the DMA proponents also state (Potter et al., 2000), CBT pain management might work not by reducing negative affect via decrease in maladaptive thinking but by managing to 'unlink central neurosystems responsible for cognitive processing of environmental, affective, and somatic stimuli by encouraging more differentiated appraisals and responses' (p. 196). According to the CAM (Eccleston & Crombez, 1999), operating a distinction between the pain stimulus and its affective (threat) value may enable a reinterpretation of the signal and thus a potential decrease not in its sensory properties but in its ability to motivate the interruption of ongoing activities and initiation of escape behaviours. Also, the simultaneous presence of competing environmental demands might also reduce pain's interruptive function by taking priority over pain and inducing dissociation between pain and emotion and replacing escape behaviours with approach behaviours motivated by competing goals.

However, other mechanisms might also play a role, such as the associative mechanisms mentioned by Fordyce (1976), or other cognitive or linguistic phenomena described in CBT and ACT approaches. The exploration of alternative

mechanisms for the ability to differentiate between pain perception and pain-related distress would potentially lead to identifying a broader range of strategies for chronic pain management, and importantly to an increased understanding of how current interventions work.

3. Resilience and the buffering hypotheses – positive emotions and social support protect against the negative emotional effects of pain

The DMA discusses the role of positive affect as a source of resilience as an explanation complementary with affect differentiation for the significant interaction between concurrent measures of positive emotion and pain in predicting concurrent negative affect. The buffering hypothesis of positive affect states that increases in positive affect during times of stress have a protective effect on the consequences of stress on negative affect (e.g., Zautra et al., 2001). The authors suggested that the buffering effect of positive affect may be a result of the lack of affect differentiation during times of stress, which makes the presence of positive affect more relevant to well-being due to the increased inverse correlation with negative affect. However, they acknowledged the different implications of the two alternative interpretations, and advanced that assessing coping effort and cognitive structure might differentiate between them in further studies (Zautra et al., 2005).

While the buffering effect of positive affect as described by the DMA refers to intrapersonal variations and is mediated by coping and emotion regulation, the role of interindividual differences in positive emotions as moderators of the effect of stress on well-being are predicted by two related models: the 'broaden-and-build' model (B&B; Fredrickson & Joiner, 2002) and the conservation of resources model of stress (Hobfoll, 1989). According to Zautra et al. (2005), these two models stipulate the role of increased trait (average) levels of positive affect as predicting low negative affect in times of stress, while the DMA focuses on changes in positive affect during pain increase episodes. As predicted by these theories, high average positive affect was found to interact with high weekly pain (and interpersonal stress) to reduce the simultaneous increase in negative affect (Zautra et al., 2005). Also, the moderating effect of trait acceptance on the pain–negative affect relationship was found to be mediated by average levels of positive affect (Kratz et al., 2007).

A related hypothesis refers to the protective role of social support against the adverse effects of stressful events (Cohen & Wills, 1985), of which the effect of pain on mood may be considered a special case. In an early study on RA patients (Brown, Wallston, & Nicassio, 1989), lower perceived support and increased pain have been found to interact in predicting increased depression cross-sectionally, but not longitudinally at 6-month intervals. However, a diary study on reflex sympathetic dystrophy syndrome patients (Feldman et al., 1999) has found that daily measures of perceived support interacted with daily pain to predict next day's overall negative mood and depression (but not the opposite time-lagged relation). The authors suggest on the basis of qualitative data that these effects are due to the content of supporting interactions which usually encouraged coping and acknowledged difficulties.

According to the DMA, the interaction between positive social support and pain in predicting concurrent negative affect could be explained via its link with positive affect: 'support blends with other sources of positive affect more readily and relates

inversely with negative affective conditions under stress, regardless of the form of coping that may be encouraged by the support provided' (Zautra et al., 1997, pp. 95–96). However, the time-lagged relations identified in Feldman et al. (1999) extend the DMA proposal and also point towards coping as a mediating mechanism.

Other cross-sectional studies involving social support and social functioning suggest the opposite concomitant pain–emotion relations. Giardino et al. (2003) reported that high perceived social support (pain solicitousness) and high catastrophising interacted in predicting high affective pain; also, high catastrophising predicted high sensory pain only in people living with a spouse. The authors interpreted these results as supporting the 'communal coping model' (Sullivan et al., 2001), which states that catastrophising is a form of interpersonal coping, and its relation to pain is influenced by social and interpersonal factors, such as the solicitousness of partner's responses, or the type of relationship with the partner. A low education level (as an indicator of low socioeconomic status) was also identified to interact with high catastrophising in increasing affective (but not sensory) pain, but in decreasing social disruption, suggesting that catastrophising leads to mobilisation of the social network, especially in people with low socioeconomic status (Edwards et al., 2006). To elucidate the role of social support in the cross-sectional and time-lagged pain–emotion relations, a more comprehensive study which would consider both interpersonal and intrapersonal variation for both consecutive and sequential measurements would be necessary.

From a clinical perspective, the buffering hypotheses go one step further than affect differentiation, as they propose a causal relationship between intra- and interpersonal resources such as positive affect and social support and the impact of pain on subsequent distress. Interventions focusing on increasing positive affect at the individual and social level therefore might be able to counteract the effects of prolonged painful stimulation, although more longitudinal research is needed to test these hypotheses. The contradictory results related to social support and catastrophising indicate that distinguishing between beneficial and detrimental influences requires a more careful consideration. The next section reviews research on the latter type of factors.

4. Vulnerability priming hypotheses – interindividual characteristics which predispose to increased distress under painful stimulation, or to increased pain when distress increases

In contrast to the DMA, which essentially focuses on resilience, several other moderating factors have been studied from a clinical perspective in terms of their detrimental effects on chronic pain adjustment. The most detailed theoretical contribution in this category is the scar hypothesis (or the vulnerability/diathesis–stress model) of depression in chronic pain (Banks & Kerns, 1996), which was developed on a wide cognitive–behavioural basis including Beck's cognitive distortion model, Seligman's learned helplessness model and Lewinsohn's behavioural model of depression. It stipulates that premorbid psychological predispositions (such as negative schemata about the self, the world and the future; or the tendency to make internal, stable and global attributions; or restricted premorbid levels of instrumental activities and limited skills to obtain external reinforcers) are activated by stressful events related to pain: the symptom itself, the related impairment and

disability, the secondary social and psychological losses and the interactions with the medical system. This activation leads to processing biases (such as overgeneralisation, personalisation, absolutistic thinking and catastrophising), more frequent use of depressive attributional style, limitation in rewards and increase in punishing reinforcement, which maintain dysphoric mood and negative thought patterns (Banks & Kerns, 1996). Turk (2002) follows a similar diathesis–stress approach in describing the role of psychological factors in the perception of pain and maintaining pain and disability following traumatic injury.

Other authors distinguish between the scar and kindling hypotheses in the context of moderation analyses. The former 'proposes that a depressive episode leaves lasting changes in personality and self-concept that lead the person to be more vulnerable to affective disturbance in the future' and would be supported by a main effect; the latter 'suggests that episodes of depression increase the likelihood of future episodes by conferring greater sensitization to the stress of affective disturbance' and would support an interaction effect (Zautra et al., 2007, p. 188). In RA, the mechanisms proposed are related to inflammatory processes and central sensitisation, the latter referring to a possible disturbance in the common neural substrate of pain and emotion regulation caused by prior depression episodes and leading to increased reactivity to pain (Zautra et al., 2007). This neural substrate specifically refers to the medial pain system, which includes numerous brainstem and limbic system areas also involved in emotion processing and represents the neuroanatomic basis for the proposed kindling hypothesis (Rome & Rome, 2000).

Vulnerability hypotheses have been the focus of numerous studies. Burns et al. (1997) found that low back pain sufferers with high levels of depression reported higher levels of pain if they responded with increased lower spinal muscle reactivity to laboratory stress induction via mental arithmetic task (but not anger recall interview). In a cross-sectional study, Fifield et al. (1998) found that chronic pain sufferers with lifetime history of major depression and increased current dysphoria report increased pain, compared with sufferers with low current dysphoria, irrespective of diagnosis (definite major depression, subthreshold depression or no diagnosis). They propose that major depression may leave a 'scar' which makes the person vulnerable not only to recurrence but also to health deficits in RA. The vulnerability however affects only reports of pain, not fatigue and disability, and only if 'primed' by current dysphoric mood. Tennen et al. (2006) extended these results in a diary study of women suffering from FM and found that previously depressed individuals reported higher correlations between daily pain and venting emotions as a coping strategy (and inversely with pain coping efficacy). Also, previously depressed reported less positive affect when daily depressive symptoms and daily pain increase simultaneously. In a similar study on RA patients, Conner et al. (2006) also found support for the vulnerability priming hypotheses: despite having no main effect on current levels of pain, depression history had a significant effect on the strength of contingencies between daily pain and emotion-related experiences (positive and negative mood and venting emotions as a coping strategy). Depression status, although associated with interpersonal differences in daily ratings, did not have this moderating effect; however, it interacted with depression history and daily pain in predicting control appraisals. Zautra et al. (2007) found that RA patients with prior depression reported increased bodily and joint pain when perceived stress increased following experimental induction.

Results supporting a vulnerability priming account have also been reported in relation to other emotion-related individual differences. In a cross-sectional study of MS patients, Janssens et al. (2003) found that for patients reporting high anxiety and depression, the correlations between functional limitations and quality of life (bodily pain, physical and role-physical functioning) were higher compared with patients reporting low anxiety and depression. Goubert et al. (2004) found that high neuroticism led to higher correlations between pain and catastrophising, and thus described neuroticism as a vulnerability factor, possibly by lowering 'the threshold at which pain is perceived as threatening, and at which catastrophic thoughts about pain emerge' (p. 234), consistent with theories which view anxiety as a cognitive vulnerability to environmental stress (Eysenck, 1992). Litt et al. (2004) identified an interaction between average levels of catastrophising and momentary changes in catastrophising in predicting concurrent pain. Since catastrophising may be interpreted as indicating increased levels of negative pain-related affect (McCracken & Gross, 1993), these findings concur in supporting a vulnerability priming model.

However, the role of trait negative affect-related characteristics in the pain–emotion relation is controversial. Van den Hout, Vlaeyen, Houben, Soeters & Peters (2001) did not find a significant interaction between trait (or state) negative affectivity and failure feedback on low back pain patients' pain reports after a lifting task. In a heterogeneous sample, pain catastrophising amplified the relation between focusing attention on pain and pain threshold and tolerance during an experimental cold-pressor task, but not pain reporting (Michael and Burns, 2004). Affleck, Tennen, Urrows, and Higgins (1992) showed that increased neuroticism led to lower correlations between pain and mood, while illness duration, disability, disease activity and average daily pain all led to higher pain–mood correlations. To anticipate, these opposite results are consistent with the desynchrony phenomenon discussed in Section 6.

Attachment theory is yet another angle from which the vulnerability priming account has been approached. It stipulates that individuals construct during their development relatively stable internal working models which guide their behaviour, and that their mobilisation by threat appraisals depends on attachment patterns, i.e., the affectional bonds that the child forms with the carer to meet the need for security (Bowlby, 1969). In a study by Meredith et al. (2006), low attachment security (comfort with closeness) interacted with low self-efficacy (but not high anxiety) in predicting concurrent high pain intensity. As the self-efficacy measure used in this study has a substantial positive affectivity component (e.g., 'I can enjoy things, despite the pain'), this moderation effect might be interpreted as an increased negative association pain–positive affect in people with low attachment security. Thus, attachment style can be considered a vulnerability factor for increased pain under conditions of low positive affect.

Low mindfulness was proposed as a precursor of catastrophising in a modified fear-avoidance model, based on its interaction with increased pain in predicting increased catastrophising in chronic pain sufferers (Schütze et al., 2010). Low general just world beliefs have also been identified as vulnerability for increased distress in face of increased pain in a sample of arthritis and FM patients (McParland & Knussen, 2010). Sleep disturbance has also been presented as a vulnerability factor. Nicassio and Wallston (1992) reported that sleep disturbance interacted with pain in predicting depression 2 years later, probably via motivational deficits or

physiological mechanisms. In a longitudinal study, Valrie et al. (2008) found that low sleep quality interacted with low mood to predict increased pain the following day; however, the study does not specify whether the models controlled for prior day pain; therefore, the time-lagged causal relationships reported are questionable. A different perspective is offered by Menefee et al. (2000), who report increased depression interacting with high levels of pain to predict poor sleep quality cross-sectionally.

Vulnerability research suggests that individual characteristics such as depression history, neuroticism and attachment style may be useful to diagnose in clinical practice not only for their direct impact on pain but also for the different pain–emotion dynamics that they are associated with. Moreover, they might influence the degree to which people might benefit from different clinical approaches. For example, Zautra et al. (2008) reported that RA patients with recurrent depression benefited more from a mindfulness meditation and emotion regulation intervention than from cognitive behavioural therapy on emotion-related outcomes (positive and negative affect, coping efficacy and catastrophising), but not on pain control. The authors suggest that these differences might be due to the focus on non-judgemental awareness and cultivation of positive experiences included in the mindfulness meditation and emotion regulation intervention. Such interventions however include multiple elements; therefore, in order to better understand their interaction with patient and contextual characteristics, it is necessary to examine research on specific coping and emotion regulation strategies as contextual determinants of pain–emotion relations.

5. Coping and emotion regulation – context-dependence in chronic pain management

Whether supporting resilience or counteracting vulnerability, coping and emotion regulation strategies have a central role in emotionally adjusting to chronic painful stimulation. However, this role is also extremely complex.

Brown, Nicassio, and Wallston (1989) reported that increased use of passive coping strategies (but not active coping) interacts with increased pain to predict increased depression both cross-sectionally and after 6 months, and explained these findings in relation to the concept of 'learned helplessness' (Abramson, Seligman, & Teasdale, 1978). Affleck, Urrows, Tennen, and Higgins (1992) replicated Brown, Nicassio et al.'s (1989) study using daily measurements of coping and found that at low pain intensity the increased use of distraction and emotional support was associated with improved daily mood, while the opposite relation was found at high pain intensity levels.

Emotion regulation has also emerged as a clinically relevant factor in pain–emotion relations. In a sample of women suffering from RA, differences in emotional processing such as mood repair and affect intensity have been identified as moderators of the time-lagged relations between pain and subsequent positive and negative affect (Hamilton et al., 2005). A cross-sectional study of women with FM found further support for an interaction between affect intensity and emotion regulation (emotional processing and difficulty describing feelings) in relation to pain and fatigue (Middendorp et al., 2008). The effectiveness of emotion regulation (measured as recovery from high negative affect or low positive affect) in reducing pain levels was higher for RA sufferers of younger age, lower education and higher disease severity in a study by Connelly et al. (2007).

Johansen and Cano (2007) further explored emotion regulation in couple interactions and found, for example, that the patient's expression of increased sadness in marital interaction is related to lower pain severity reports only in couples both suffering from pain, but to high pain severity when the spouse was not a chronic pain sufferer. These findings highlight the role of emphatic communication in emotion regulation in chronic pain.

Coping and emotion regulation have also been considered in the context of gender differences in pain–emotion relations. Burns et al. (1996) found that the worst pain severity is reported by women also reporting high hostility and high anger expression, while men with high hostility but low anger expression reported more severe pain. The authors, without proposing a detailed theoretical explanation, related these findings to the psychoanalytic literature on anger suppression and research on the social impact of anger expression. Adding to previous findings on gender differences in chronic pain prevalence (i.e., higher prevalence in women due to social learning, hormonal and pain sensitivity factors), Affleck et al. (1999) showed that the impact of today's pain on tomorrow's negative mood was higher in men than in women, probably due to women's ability to limit the emotional consequences of pain better than men (i.e., use of more coping strategies). These findings were extended by Keefe et al. (2004), who showed that evening increases in pain are related to higher negative mood and lower positive mood the next morning in men only, among other gender differences related to pain coping. In contrast, Adams et al. (2008) found that higher levels of depression are associated with higher activity-related pain reports only in women. Riley et al. (2001) identified gender differences only in the relation between affect and pain unpleasantness, not pain intensity, and presented these results as supporting the sequential stage model of pain processing. The model stipulates that the individual's response to pain consists of an initial pain intensity perception, followed by a more context-influenced perception of pain unpleasantness and then by more complex pain processing which determines the implications of pain for the individual's life and generates complex emotions and suffering; a fourth stage consists of overt behavioural responses (Price et al., 2001). The selective gender influences on pain unpleasantness were considered to reflect the influence of gender-specific psychosocial factors such as pain coping, catastrophising and control (Riley et al., 2001).

The issue of control in chronic pain management has been controversial, as some authors view control as related to adaptive coping, while others associate it with reports of increased distress in the context of an essentially uncontrollable illness. Affleck, Tennen, Pfeiffer, and Fifield (1987) found that in people with increased symptom activity, perceptions of increased personal control over symptoms were associated with lower mood disturbance, while reports of increased personal control over illness course were linked with increased mood disturbance. They explained these contrasting findings by Rothbaum, Weisz, and Snyder's (1982) two-process model of perceived control, which distinguished between primary control (assimilative, directed towards changing the environment) and secondary control (accommodative, directed towards the self). As the illness course is highly unpredictable in RA, unsuccessful control attempts may lead to increased distress. By contrast, the symptoms themselves are more controllable and therefore assimilative control is in this case adaptive.

Schiaffino, Revenson, and Gibofsky (1991) investigated self-efficacy beliefs of recently diagnosed RA patients, and found that increased perceived self-efficacy is related to higher depression after a year if high pain intensity is also reported at baseline. Their results also point towards the potentially maladaptive role of control in the context of increased illness severity. In a related study, Tennen et al. (1992) distinguished between perceived control (primary control) and perceived benefits (secondary, cognitive control) and found that at high levels of pain the former is related to low mood and the latter with low activity limitations, further supporting the two-process model.

The two-process model of perceived control, among other research on control and self-regulation, has been a building block for a more comprehensive model of coping: the dual-process model (DPM) of goal pursuit and goal adjustment (Brandtstädter & Rothermund, 2002). The DPM focuses on the 'modulating influence of action orientations on information processing' (p. 120). It distinguishes between two modes of coping: assimilative (assimilating the actual situation to goals, problem-solving, pursuing goals and removing obstacles, 'stability and personal continuity') and accommodative (accommodating goals to situational constraints, problem-dissolving, deconstructing commitment, reappraising the situation, finding new goals, 'adaptive flexibility').

The authors state that 'both processes are activated by perceived or anticipated goal discrepancies, or by divergences of the factual course of personal development from the intended one' (p. 121), but the activation of one or the other is moderated by several contextual factors: the appraised characteristics of the goal, such as personal importance, centrality, substitutability (depending on the abstract or concrete 'phrasing level'), the structure of personal goals system (self-complexity) and the perceived goal attainability (depending on contextual contingencies such as action resources, on attainability beliefs and self-percepts of control, also influenced by the cultural and historical context). Another equally important factor is the 'availability and accessibility of palliative cognitions' (p. 125), i.e., cognitions which decrease the interest to pursue the current goal and help reinterpret irreversible contextual factors in a positive light. Such cognitions depend on personal knowledge and experience, temperamental dispositions, basic existential attitudes, accessibility of downward comparisons and self-attributions of personal responsibility. The model also stipulates individual differences in the propensity to use such coping modes, described as tenacious goal pursuit, the tendency towards assimilative persistence, and flexible goal adjustment, the disposition towards accommodative flexibility (pp. 135–136).

These two coping modes are complementary cognitive sets that tend to inhibit each other, although they could also work in collaboration (p. 123). The information processing in assimilative mode is characterised by high accessibility of representations of goal and action paths, and of situational contingencies and information that supports persistence and continuity (positively biased control beliefs, durability bias), as well as by increased attentional focus and a convergent processing style. This focus is complemented by a shielding and inhibition of distractive influences, conflicting information and competing action tendencies. Obstacles induce an increase in focus and shielding and goal attractiveness, to compensate for the increase in implementation costs.

Repeated unsuccessful attempts or the passing of critical timelines lead to reduced attainability beliefs and reduced 'competence to compensate for incompetence' (p. 134) beliefs. Thus, the activation of the accommodative mode leads to eliminating implementation intentions from working memory, withdrawing attention from the unsolvable problem and disregarding problem-related cues, and an increased availability of palliative cognitions due to a defocused, holistic processing style and broadened field of attention.

The authors suggest that these phenomena are possibly mediated by the activity of the dopaminergic system, by a shift of processing from left to right hemisphere and by individual differences in belief flexibility. They also indicate a possible role of endogenous opioids in accommodative responses following exposure to uncontrollable painful stimuli (Brandtstädter & Rothermund, 2002).

As control is often unattainable in chronic pain, the DPM would predict that assimilative coping would relate to increased distress, while accommodative coping would be associated with better emotional functioning, especially in situations of increased painful stimulation. Schmitz, Saile, and Nilges (1996) reported that low flexible goal adjustment interacted with high pain intensity (and disability) to predict high levels of depression. Also, Kranz et al. (2010) reported that high flexible goal adjustment was associated with increased pain willingness and activity engagement (two complementary aspects of chronic pain acceptance), particularly at high average pain intensity levels.

Other studies reported DPM-consistent results. For example, Zautra et al. (2007) found that positive emotion reports increased together with stress reports following stress induction, which they referred to as 'mounting an affective counterweight to stress', but did not detail further. The DPM's clarification of the activation of the accommodative mode following stress thus complements the DMA.

Also, Strand et al. (2007) showed that high pain readiness to change (i.e., action/maintenance) interacted with low weekly positive emotion in predicting high concurrent weekly average pain reports. The authors explained these apparently surprising results in terms of active pain coping efforts reflecting personal responsibility and therefore lowering positive affect when pain increases, or proving maladaptive and therefore increasing pain concurrently with lowering positive affect or being effective only when associated with high positive affect. Given the concurrent measurements used for the data analysis, these alternative explanations could not be distinguished. A fourth explanation could be that pain readiness to change represents a switch to the assimilative mode, which works by decreasing positive affect in conditions of stress or pain increase.

The role of accommodative coping in increasing positive affect while being related to a defocusing processing style could also provide an alternative explanation for the interaction reported by Abeare et al. (2010). Using a cross-sectional design, this study found that increased pain and increased positive affect interacted in predicting lower performance in executive functioning tests. The authors discuss this effect as the result of positive emotion requiring additional resources, of dopaminergic mechanisms, or of positive emotion being related to underreporting of pain; the DPM suggests a switch to a different coping mode. The multitude of alternative explanations highlights the need for developing more specific predictions which would differentiate them empirically.

The coping research further clarifies the role of resilience and vulnerability factors and links them with possible intervention strategies. For example, it suggests that a stepped care model of treatment might be appropriate both within and between individuals, with various factors leading to matching treatment. At low levels of pain intensity, a judicious use of emotional control strategies, rationalisation and cognitive therapy strategies for reducing catastrophising could be successful, combined with behaviour activation and re-engagement in normal activity. At higher levels of pain, greater use of mindfulness and acceptance strategies could be more suitable. This combined approach could lead to developing a flexible set of strategies for living with chronic pain.

6. Desynchrony – in some circumstances increased distress may lead to increased pain and disability independent of pain stimulation

While most studies reviewed so far suggest stronger pain–emotion associations as indicative of chronic pain adjustment, other theoretical contributions identified in our literature review support what might look like the opposite relations. For example, while the self-regulatory model underlines the clinical benefits of distinguishing between sensory and affective pain (Leventhal & Everhart, 1979), it stipulates that a lack of coherence between the various emotional and cognitive illness interpretations within the individual's belief system, and also in relation to the broader psychological and social context, may impede adequate illness management (Leventhal, Diefenbach, & Leventhal, 1992). Early CBT accounts of chronic pain refer to a desynchrony of subjective, physiological and behavioural aspects of pain as being detrimental for psychological adjustment to tension-type headache and influenced by personality, attitudes and expectations (Phillips, 1977, as cited in Lethem et al., 1983). This idea was further developed in the fear-avoidance model of exaggerated pain perception (Lethem et al., 1983), which stipulated that stressful life events, personal pain history, coping strategies and behaviour patterns increase the probability of avoidance responses and thus lead to a dysfunctional desynchrony, when affective responses are more intense than sensory responses. Desynchrony was also described between affective and sensory components of pain (Phillips and Hunter, 1981, as cited in Lethem et al., 1983); avoidance behaviours were associated only with the affective component, not the sensory component of pain, pointing to the specific properties of the affective components in stimulating escape, as also detailed in the CAM (Eccleston & Crombez, 1999).

Some empirical studies reviewed reported results which may be considered as supporting desynchrony. In Affleck, Tennen, et al.'s (1992) study, high neuroticism individuals showed lower correlations between reports of pain and mood, indicating that the distress reported by individuals high in neuroticism is partly independent of pain. Lombardo et al. (2005) expected low self-efficacy to be related to high maladaptive anger management, but found that this relation holds only at low pain levels, while at high pain intensity there were no differences in anger management between low and high self-efficacy. No theoretical explanation of this moderation effect was proposed, but the lack of association between self-efficacy and maladaptive anger management at high pain levels is supportive of the desynchrony concept. Cohen et al. (2010) have also found lower associations between pain and

measures of functioning (except social functioning) at high levels of anxiety, in adolescents suffering from chronic pain.

In an experimental study, Hadjistavropoulos et al. (2000) reported that for health anxious chronic pain sufferers somatic monitoring helped reduce reports of pain and anxiety, which the authors interpret as a temporary effectiveness. This effect however supports the self-regulatory model proposal that increasing coherence between sensory and affective domains may prove effective in chronic pain management.

Desynchrony-consistent results have been reported for different conditions. Newth and Delongis (2004) found that in RA sufferers low morning pain and low morning mood led to high evening pain, while no relationship between morning mood and evening pain emerged at high morning pain levels. The study only refers to research on neurophysiological pathways in the pain–mood relation, but the findings are also consistent with desynchrony. It suggests that, at lower levels of pain, high negative affect might lead to subsequent increases in pain independent of the pain stimulation, thus leading to a desynchrony between pain severity and its consequences. Hoff et al. (2006) found that in children with sickle cell disease reporting lower pain levels, increased depression is associated with reports of increased pain after 6- and 12-month intervals (in children with juvenile idiopathic arthritis, similar results were found at lower pain levels as reported by the caregivers). In a study of Raynaud's phenomenon, characterised by symptom aggravation in colder temperatures, Brown et al. (2001) have found an increased role of anxiety in attack-related pain in warmer temperatures, suggesting that when the role of sensory stimulation is reduced, affective factors become increasingly relevant.

The contrast between desynchrony-consistent results and the majority of the studies reviewed previously recommends a careful consideration of contextual influences in particular research and clinical settings. It suggests that aiming for pain–emotion differentiation might not be clinically adequate in any situation, and further research is necessary to identify the conditions in which coherence should be targeted.

Discussion

Summary of review

The studies reviewed above reveal a complex picture for emotional adjustment to chronic pain. To summarise, affect differentiation within the DMA describes a merging of the affective space in face of pain and stress, which also diminishes the individual's ability to perceive pain and distress as separate phenomena. Other approaches point towards a generalised discrimination ability in chronic pain sufferers, which complements the specific focus of affect differentiation. The role of positive affect in buffering the effect of pain on negative affect, although it can be considered as result of affect differentiation, may also be extended to time-lagged relations and understood in connection to the role of social support and coping. From a clinical perspective, several vulnerability factors (depression, neuroticism, low attachment security) may act in opposition with resilience resources to predispose to increased pain and distress. Both resilience and vulnerability factors are likely to operate via coping and emotion regulation strategies, whose effectiveness is largely context dependent. In some circumstances, however, synchrony between

pain and affect might be actually beneficial, and a lack of coherence might result in increased suffering.

Clinical implications

These studies have important clinical consequences. Affect differentiation recommends including both positive and negative affect in diagnosis and treatment planning. The various theoretical contributions referring to a generalised discrimination ability suggest that other broadly used therapeutic methods might work via altering pain–emotion dynamics. Resilience research points towards a causal role of positive affect and social support on buffering the effect of pain on the sufferer's life, while vulnerability research highlights the importance of diagnosing depression, neuroticism and other detrimental influences. Enhancing resilience and counteracting the sufferer's vulnerability in clinical practice is likely to be most successful when it takes into consideration the context-dependent efficacy of various coping and emotion regulation strategies. However, in some conditions pain management might need to target an apparently contradictory outcome: increasing the association between pain perception and emotion. In practice, this might translate into helping people to make a more consistent assessment of pain and emotion, particularly for those who have high trait negative affect, and under conditions of low sensory pain. Under these circumstances, a therapeutic goal could be to bring greater awareness of pain and emotion links by enhancing participant's noticing of the intensity of their pain-related affect.

Various mechanisms and intervention possibilities have been addressed in the studies reviewed, both in terms of manipulating momentary contextual influences and developing useful stable characteristics such as skills and personality traits, but it is not our goal here to insist upon their details. Rather, we hope that this review would offer the interested reader a starting point in exploring the broad range of treatment methods, but most importantly the possibility that their efficiency in altering pain–emotion relations might depend on context and person characteristics.

Theoretical implications

Therapeutic practice would benefit from an integrated theoretical model of pain–affect relations, which could be attempted on the basis of the DMA. Although not specifically developed for chronic pain, the model is consistent with most empirical results, even if they have been articulated from different perspectives. The studies reviewed suggest that the model could also be extended in several ways. As Zautra et al. (2005) states, the DMA describes the role of intraindividual changes in positive affect on simultaneous pain–negative affect relations, while the role of average levels of positive affect in predicting negative affect in times of stress is detailed in the 'broaden-and-build' model and the conservation of resources model of stress. Also, the DMA predictions for time-lagged relations are underdeveloped, while the DPM and the research on the role of coping and emotion regulation specifically state predictions regarding the relations between consequent measurements.

The most difficult to reconcile with DMA are the desynchrony-consistent results, which suggest lower pain–affect associations as indicative of low pain adjustment, while the DMA describes lower associations between reports of pain and affect as

representative of better adaptation to pain. A closer examination might indicate complementarity. Desynchrony might refer to time-lagged relationships, while affect differentiation describes relationships between simultaneous measurements. Also, the DMA places affect differentiation and resilience in the context of interindividual differences in cognitive structure and mood clarity, while desynchrony might refer to a different set of interindividual affect-related differences.

Recommendations for future research

Such integration awaits further theoretical and empirical efforts. The studies reviewed highlighted various factors acting on intrapersonal or interpersonal levels, affecting simultaneous or time-lagged pain–emotion relations, and potentially exerting a more distal (e.g., prior depression) or proximal influence (e.g., coping). Yet most moderators were studied in cross-sectional designs which cannot differentiate between the alternative theoretical explanations available. Also, various mechanisms have been proposed, from attention focus and various physiological changes to coping, yet no studies have examined self-report simultaneously with physiological or environmental moderators to test their mediating role. Importantly, the potential effects of pain–emotion relations on other aspects of chronic pain adjustment such as disability have hardly been addressed. The picture so far is incomplete, and substantial efforts are required to develop a better understanding of the complex causal chain underlying emotional adjustment to chronic pain.

An essential requirement for bringing further clarity is the consideration of several methodological aspects (see Table 1 for summary). First, an interaction model is statistically symmetrical, and the decision regarding which of the variables is considered the moderator or the predictor is not based on statistical grounds. At least two equivalent interpretations may be developed for a single moderation analysis. For example, neuroticism is presented as the moderator due to being a stable trait in some studies (Affleck, Tennen, et al., 1992), while other interpretations view stress as moderating the relation between neuroticism as a stable trait and negative affect as outcome, as 'stress creates a context within which linkages among all affect-laden features are strengthened, including the association between personality dispositions, such as neuroticism and negative affective states' (Zautra et al., 1997, p. 91).

Considering alternative hypotheses is more frequent in studies where both predictors are measured at the intrapersonal level, but the preferred theoretical interpretation is usually highlighted (e.g., Cohen et al., 2010; Zautra et al., 2001). Some studies (e.g., Burns et al., 1996) use post-hoc testing to clarify the relations between the predictor and the outcome at different levels of the chosen moderator. Although this analysis is certainly valuable to the interpretation, it does not represent a test of the theoretical decision regarding which variable represents the moderator. This choice is a theoretical assumption that precedes such moderation analyses (Cohen, Cohen, West, & Aiken, 2003, p. 269). Thus, we would argue that presenting the data from both perspectives (in the case of a two-way interaction) is essential for the theoretical clarification of the possible interpretations available.

Second, apart from time-lagged models, the outcome is also arbitrarily selected from a statistical point of view, as many authors have acknowledged (e.g., Conner et al., 2006; Kratz et al., 2007; Tennen et al., 2006; Zautra et al., 2007). Different variables, such as pain (Fifield et al., 1998), negative affect (Zautra et al., 2001) and

Table 1. Methodological considerations

Methodological issue	Recommendation
Interaction models are statistically symmetrical	Considering alternative theoretical interpretations
Outcome variables are an arbitrary statistical choice in cross-sectional designs	Considering alternative models with different variables as outcomes
Predictions at the intra- and interpersonal levels differ in the models reviewed	Using multilevel designs with multiple measurements per participant
Predictions regarding simultaneous and sequential relations differ in the models reviewed	Investigating both cross-sectional and longitudinal relations, at different time intervals
All models reviewed focus on self-reported pain and affect	Integrating questionnaire responding processes in theory development and testing
Affect differentiation is also likely to influence the structure of the psychological moderators examined by the models reviewed	Considering variability of affect structure as a source of bias in self-report measures of psychological moderators
Null results are equally informative in the empirical testing of the models reviewed	Examining negative and positive findings comparatively in relation to design differences to further delimit the generalisability of the theories

depression (Schmitz et al., 1996), have been considered outcomes in investigations of simultaneous pain–emotion relations, leading to different theoretical interpretations. The diversity of theoretical accounts identified in this review highlights the necessity of considering all possible interactions in cross-sectional designs. Moreover, our literature search revealed several other studies which included pain and emotion-related data but were not selected for the present review because the analyses performed considered pain or emotion as a covariate for a different interaction effect (e.g., Boersma & Linton, 2005; Sullivan, Sullivan, & Adams, 2002), or did not report pain–emotion moderation analyses. Examining existing data from different theoretical perspectives would help accelerate progress in this area.

Thirdly, the models differ in their predictions regarding the intra- or interpersonal level of the relationships they explain. As discussed by Zautra et al. (2005), the intrapersonal level answers 'when'-questions, while interpersonal differences address 'who'-questions. Only data sets that include multiple measurements for each participant are able to distinguish between these types of research questions. Data with one measurement level are uninformative regarding intraindividual differences, even if often the interpretation of interindividual differences is framed in intraindividual terms (e.g., Brown, Nicassio, et al., 1989). Therefore, three or more measurements per participant (Singer & Willett, 2003, pp. 9–10) should be collected in future studies where possible, and multilevel modelling should be used for data analysis. Such models may be further extended to include additional levels (e.g., community), as previously advocated by Zautra, Hall, Murray, & the Resilience

Solutions Group (2008) in the context of providing recommendations for resilience research. These extensions would allow the testing of more refined hypotheses.

Fourth, the predictions addressing simultaneous and sequential relations often differ in the theoretical accounts reviewed. As simultaneous measurements are essentially descriptive and only sequential measurements may reveal causal relationships, an investigation of both cross-sectional and longitudinal relations is central to the issue of causality. Moreover, examining different time intervals (within-day, daily, weekly, monthly, at several months' intervals etc.) would be instrumental in delimiting the degree of temporal generalisation of the relationships identified.

Fifth, as all theoretical contributions and empirical studies reviewed rely on self-report data, the interpretation needs to consider the actual processes related to questionnaire responding (Tourangeau, Rips, & Rasinski, 2000): comprehension of the particular question, retrieval of relevant information from memory, judgement (integration of information) and response (mapping the judgement on the response format and editing it according to additional criteria). This additional layer of interpretation might help clarify the mechanisms responsible for the relationships described. For example, the merging of the affective dimensions might actually reflect the inability of the respondent in stressful situations to access different positive and negative experiences, and categorise them as such. Attention and categorisation processes that participate in retrieval and integration of information in questionnaire responding need to be considered as part of the theory. Certainly, the issue of self-report in chronic pain patients should not be the main focus of research, as it is relevant to clinical practice only to the degree that it exemplifies cognitive processes of sampling and labelling the experience that affect pain management decisions (e.g., regarding activity levels, medication adherence, goal-directed actions, social interactions), which translate into overall adjustment to illness. Self-report, as an instance of experience sampling and labelling, might represent a relevant measure of such processes to the extent that it resembles how such processes work in the respondent's daily life (and not in relation to an artificial context).

Sixth, affect differentiation also warns against an important methodological pitfall in health research. It implies that for questionnaires that assess emotion-laden concepts or use emotion-related response formats, such as measures of stress, coping, health status and well-being, responses depend on the ability of the individual to keep the positive and negative dimensions distinct, which is dependent on the level of stress. This implies that the very structure of the phenomenon under study changes over time and between persons, and this needs to be accounted for as a possible source of bias (Potter et al., 2000; Zautra et al., 1997). This is especially relevant for pain–emotion moderation. If factors moderating pain–emotion relationships are measured on a single continuum from high to low adjustment and include both positively and negatively worded items, the structure of the measure itself might fluctuate depending on stress and pain levels, and these fluctuations need to be accounted for in model testing and interpretation.

Last but not least, null results should be considered equally informative in mapping out the influences of stable and contextual factors on pain–emotion relations, if the studies are of methodologically good quality. Together with an analysis of the differences in study design, these results are helpful in delimiting the area of influence of the factors considered, given the type of illness condition, the

time intervals etc. examined in the different studies. In some studies, the null results could be attributed to methodological issues such as small sample size (e.g., Ferguson & Cotton, 1996; Roberts et al., 1996) or lack of multilevel and longitudinal data (e.g., Middendorp et al., 2008; Riemsma et al., 2000). In others, data analysis choices such as the decision of testing interaction effects only for predictors with significant main effects (e.g., Plach et al., 2003) might have led to the omission of possible significant interaction effects.

On the other hand, null results are essential in clarifying and delimiting the predictions of the theoretical accounts discussed. For example, studies reporting null results regarding the moderating role of gender on pain–emotion relations but significant moderation effects in relation to other health-related outcomes such as disability (Hirsh et al., 2006; Hommel, Wagner, Chaney, & Mullins, 1998; Jones & Elklit, 2007; Kaczynski et al., 2009; Keogh et al., 2006) might help clarify the role of gender in chronic pain adjustment and need to be taken into consideration when examining such specific issues.

Considering these methodological issues in future research on pain–emotion moderation would accelerate progress in this area by refining hypotheses and facilitating the collection of critical data for testing competing explanations. Moreover, they are potentially applicable in other areas of health psychology where emotion influences health behaviours and outcomes, where pain is a relevant symptom, or where dynamic relations are likely to manifest at both intra- and interpersonal levels. Indeed, contextual and interindividual differences have gained more attention recently in health psychology. For example, more recent dual-system models of health behaviour (reviewed in Hofmann, Friese, & Wiers, 2008) propose that both self-control and impulsive influences have an impact on health-related behaviours depending on 'situational and dispositional boundary conditions' (p. 117), including emotional and sensory phenomena. Research on the moderating role of these conditions would also be enhanced by the methodological recommendations described above.

Limitations

This review was limited to moderation of pain–emotion relations as reflected in self-report. Other interactive effects on various aspects of chronic pain adjustment have been studied, such as pain duration and self-evaluation tendency in relation to depression (Jensen & Karoly, 1992), marital interaction, global marital satisfaction and their effects on depression and pain (Kerns, Haythornthwaite, Southwick, & Giller, 1990), coping and pain in relation to activity levels (Jensen & Karoly, 1991), attribution style and perceived illness control in relation to depression (Chaney et al., 1996), physiological reactivity and depression in relation to pain severity (Burns et al., 1997) and the role of gender, age, work status and litigation in depression (Averill, Novy, Nelson, & Berry, 1996). These studies suggest that the variability of pain–emotion relations is only one aspect of the highly complex and dynamic landscape of chronic pain adjustment.

Conclusion

Chronic pain adjustment crucially depends on how individuals perceive pain and respond to it emotionally. Thus, which factors influence the relation between pain and emotion is an important clinical and research question. The present review has attempted to bring together separate investigations into this issue and provide a description of the current theoretical developments. Starting from the Dynamic Model of Affect, which was identified as the most detailed and empirically supported approach to pain–emotion relations in chronic pain, several concepts were reviewed, such as affect differentiation, generalised discrimination ability, resilience, vulnerability, coping, emotion regulation and desynchrony; the empirical support was reviewed and clinical implications for pain management interventions were outlined.

The growing empirical literature exploring these relationships will benefit from further clarifications of the theoretical claims, empirical predictions and mediating mechanisms proposed. Theory testing will be enhanced by considering alternative interpretations, simultaneous and sequential relations, intra- and interpersonal moderators, and self-report processes and biases, and by interpreting both null and positive results comparatively. This theoretical and methodological analysis is intended as an invitation to the research community to further investigate pain–emotion moderation with a view to developing more effective pain management interventions, while offering a detailed picture of the state of the art in pain–emotion moderation to the broader health psychology community.

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Appendix A

Detailed Search Syntax

PubMed: www.nlm.nih.gov/pubs/factsheets/pubmed.html

pain [TIAB] AND (emotion [TIAB] OR "positive affect" [TIAB] OR "negative affect" [TIAB] OR affective [TIAB] OR mood [TIAB] OR anger [TIAB] OR depression [TIAB] OR anxiety

[TIAB] OR fear [TIAB] OR sadness [TIAB] OR shame [TIAB] OR happiness [TIAB] OR joy [TIAB]) AND (moderation [TIAB] OR moderator [TIAB] OR moderates [TIAB] OR moderated [TIAB] OR moderating [TIAB] OR dynamic[TIAB] OR interaction[TIAB])

PsycINFO: www.apa.org/psycinfo/

(pain and (emotion or "positive affect" or "negative affect" or affective or mood or anger or depression or anxiety or fear or sadness or shame or happiness or joy) and (moderation or moderator or moderates or moderated or moderating or dynamic or interaction)).ab.

Web of Science:

TS = pain AND TS = (emotion OR "positive affect" OR "negative affect" OR affective OR mood OR anger OR depression OR anxiety OR fear OR sadness OR shame OR happiness OR joy) AND TS = (moderation OR moderator OR moderates OR moderated OR moderating OR dynamic OR interaction) AND Document Type = (Article)
Databases = SCI-EXPANDED, SSCI, A&HCI Timespan = All Years

Appendix B

Table 1. Empirical studies of interaction effects related to pain–emotion relationships.

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Affleck et al. (1987)	92, RA	CS – interviews, questionnaires, medical data	HMRA	IVs: –control appraisals (personal control over disease course/symptoms/treatment and healthcare provider control over disease course/symptoms): five items –illness predictability: items from the ILS –illness-status variables: multiple measures subject to principal component analysis resulting in three components: symptom activity (includes current pain ratings), functional problems, disease severity DV: –mood: PMS-B – modified CVs: –age, education, family income, occupational status, illness duration	↑ Personal control of symptoms × ↑ symptom activity=↑ mood (also ↑ personal control of illness course × ↑ disease severity=↓ mood)	Beliefs of personal control may be maladaptive if inflexible in face of evidence of the contrary (overall illness severity), but adaptive in flare-up situations (if referring to symptom control) – the dual-process model
Brown, Nicassio, et al. (1989)	287, RA	L – postal questionnaires, two waves, 6-month interval	HMRA	IVs: –passive and active coping strategies: PMI subscales –pain: AIMS subscale DV: –depression: CES-D CVs: –functional disability (mobility, household activities, dexterity, physical activities, activities of daily living): AIMS subscales –demographics and medical status: age, education, illness duration, medication –depression (wave 1) – for longitudinal analysis	Cross-sectional (wave 1): ↑ pain × ↑ passive coping=↑ depression longitudinal: ↑ pain (wave 1) × ↑ passive coping (wave 1)=↑ depression (wave 2)	Frequent use of passive coping when experiencing high pain contributes to increased depression over time – passive coping intensifies the relation between pain and depression (interpretation associated with the concept of 'learned helplessness')

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Brown, Wallston, et al. (1989)	233, RA	L – postal questionnaires, three waves, 6-month interval	HMRA	IVs: –perceived social support: satisfaction with emotional support derived from STMSS, number of close friends and relatives, adapted from SHS –pain: AIMS subscale DV: –depression: CES-D CVs: –functional disability: AIMS subscales –demographics and medical status: age, education, illness duration	Cross-sectional (waves 1 and 2, not 3): ↑ pain × ↓ perceived emotional support = ↑ depression (interaction pain × support network and all longitudinal moderation models not significant)	Perceived emotional support might buffer the noxious effect of pain on depression (possibly by mobilising coping resources, on short term) – the buffering hypothesis of social support (cf. Cohen & Wills, 1985, as cited in Brown, Wallston, et al., 1989)
Moosbrugger & Schermelleh-Engel (1991)	103, H	CS – postal questionnaires	(M)ANOVA	IVs: –perceived competence/self-efficacy: competence subscale, PRQ (dichotomised) –trait anxiety: STAI (dichotomised) –trait depression: BDI (dichotomised) DVs: –pain intensity: PRQ subscale –pain depression: PRQ subscale –pain anxiety: PRQ subscale CV: –medical diagnosis (back vs. joint pain)	↓ Perceived competence × ↑ trait anxiety = ↑ pain anxiety, ↑ pain depression (not pain intensity) at ↑ perceived competence, no differences depending on trait anxiety levels	The authors mention other moderation effects of perceived competence in other research areas, and suggest classifying patients based on this measure; no detailed interpretation presented

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Schiaffino et al. (1991)	65 (wave 1: 101); RA, maximum 2 years after diagnosis	L – two waves, interviews (wave 1) + questionnaire (wave 2), 14-month interval	HMRA	IVs: –pain: AIMS subscale –self-efficacy: three self-report items on symptom management DV: –depression: CES-D CVs: –education, age and disease duration	↑ Pain wave 1 × ↑ self-efficacy wave 1 = ↑ depression wave 2 (but not depression wave 1) (at ↓ pain wave 1, no differences depending on self-efficacy levels)	'Believing in one's ability to handle the situation in the presence of greater pain appears to contribute to greater depression [. . .] seeking control in an uncontrollable situation may be maladaptive' (p. 156)
Affleck, Urrows, et al. (1992)	78, RA	D – 75 consecutive daily reports + initial questionnaire	HMRA	IVs: –pain coping strategies: DCI – total number of reports, number of different reports and seven categories (direct action, relaxation, distraction, redefinition, emotional expression, spiritual comfort, emotional support), transformed into relative frequencies to control for the overall coping effort –pain intensity: daily joint pain, from the RADAR, sum of 20 ratings for different joints or joint groups DV: –daily mood: condensed PMS-B, positive and negative items combined for general score CVs: –neuroticism: NEO-PI –pain control appraisals: two questions from the CSQ –age	↓ Pain intensity × ↑ coping strategies (only distraction and emotional support) = ↑ mean daily mood (opposite relationship at ↑ pain intensity)	Exploratory study of daily pain coping, moderation hypotheses based on previous studies (e.g., Brown, Nicassio, et al., 1989), without a detailed theoretical interpretation

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Affleck, Tennen, et al. (1992)	54, RA	D – 75 consecutive daily reports + initial questionnaire	COR	IV: –neuroticism: NEO-PI DV: –within-individual regression coefficients between daily pain (VAS with average daily pain and joint-specific pain in 20 areas – RADAR) and daily mood (condensed PMS-B, positive and negative items combined for general score), controlling for recording day (adjusting for autocorrelation) CVs: –illness duration –disability: AIMS –disease activity: clinical assessment –average daily pain	↑ Between-persons neuroticism = ↓ within-person association between daily pain and mood (also, ↑ illness duration, disability, disease activity and average daily pain = ↑ pain–mood association)	In persons with high neuroticism, distress is less tied to pain (and stressful circumstances generally)
Nicassio & Wallston (1992)	242, RA	L – postal questionnaires, two waves, 2-year interval	HMRA	IVs: –pain: AIMS subscale –sleep problems: three items DV: –depression: CES-D, excluding the sleep item CVs: –age, education, illness duration –functional impairment: AIMS subscales –depression (time 1)	Longitudinal: ↓ pain × ↑ sleep problems (wave 1) = ↓ depression (wave 2) (at ↓ levels of sleep problems, opposite relationship) (cross-sectional: no interaction)	'The combination might produce anergia, motivational deficits or a higher level of passive coping' (p. 520), or act physiologically on disease or mood-related processes (no detailed mechanisms proposed)

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Tennen, Affleck, Urrows, Higgins, & Mendola (1992)	54, RA	D – 75 consecutive daily reports + initial questionnaire	HMRA	IVs: –perceived control and benefits: 10 items from an inventory of psychological control appraisals –daily pain: PMS-B DVs: –daily mood: RADAR –pain-related activity limitations: one item CVs: –dispositional optimism: LOT –disease activity: clinical assessment	↑ Perceived control × ↑ (and moderate) daily pain = ↓ average daily mood (but not activity limitations) (at ↓ levels of daily pain, opposite relationship) (↑ perceived benefits × ↑ pain = ↓ activity limitations only)	The role of perceived control in adaptation to pain is moderated by pain intensity – the dual process model Two explanations: the mismatch between initial estimations of control and the subsequent pain experience produced increased distress in the patients with severe and moderate pain, or the efforts to control increased pain led to neglecting other aspects and activities that could increase mood (although such efforts were not successful)
Burns, Johnson, Mahoney, Devine, & Pawl (1996)	135, H	CS – questionnaires (pre-intervention evaluation)	HMRA	IVs: –hostility: CMHS –anger expression and suppression: AEI, anger-out (AO) and anger-in (AI) subscales –spouse punishing and solicitous responses: WHYMPI subscales –gender DVs: –adjustment (pain severity, interference with daily functioning, ability to engage in daily activities): WHYMPI subscales CVs: –depression: BDI –age	Women × ↑ AO × ↓ hostility = ↓ pain severity (and ↑ activity) Men × ↓ AO × ↓ hostility = ↓ pain severity (and ↓ interference, but non-significant if controlling for spouse punishing responses) Worse pain severity for ↑ AO ↑ hostility women, and ↓ AO ↑ hostility men (different patterns for interference and activity) (no interaction effects for AI)	No detailed explanations, the authors refer generally to theories about the role of anger in chronic pain via conversion mechanisms (Engel, 1959) or social consequences of anger expression (Lane & Hofboll, 1992, as cited in Burns et al., 1996)

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, × –"interacts with", = –"predict"	Interpretations
Schmitz et al. (1996)	120, H	CS – questionnaires	HMRA	IVs: –flexible goal-adjustment (FGA) and tenacious goal-pursuit (TGP): TGP&FGAS –pain intensity: four aggregated numeric rating scales – most, least, typical, current pain –disability: PDI –pain-related coping (cognitive restructuring, action planning, self-efficacy, diverting attention, distracting activities, relaxation): DCCP DV: –depression: CES-D CVs: –sociodemographics, pain history	↓ FGA × ↑ pain intensity (and ↑ disability) = ↑ depression No moderation effect for TGP and most coping strategies (except ↓ cognitive restructuring × ↑ disability = ↑ depression)	FGA has a protective function: it dampens the negative effects of pain experience on depression (psychological distress) – based on the dual-process model of assimilative and accommodative coping (Brandstädter, 1992, as cited in Schmitz et al., 1996)
Burns, Wiegner, Derleth, Kiselica, & Pawl (1997)	107, LBP	CS – questionnaires, lab stress induction and assessment	HMRA	IVs: –depression: BDI –combined lower paraspinal change during mental arithmetic task (CLPMA) –combined lower paraspinal change during anger recall interview (CLPARI) DV: –pain: WHYMPI subscale CV: –none reported	↑ CLPMA (but not CLPARI) × ↑ depression = ↑ pain (depression – pain correlation non-significant at ↓ CLPMA levels)	Depression as vulnerability interacts with muscle reactivity in maintaining and exacerbating low back pain The authors suggest possible cognitive mechanisms: depressed patients interpreting muscle tension as pain signals

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Zautra, Potter, & Reich (1997)	RA	D – 12 consecutive weekly telephone interviews	HRMA	IVs: –high positive/negative events weeks (>3 times individual average weekly positive/ negative life events: ISLE) versus the rest of the weeks (used as subsamples) DV: –correlations between positive and negative affect (PANAS) and negative/ positive affect and self-rated arthritis pain (three analog scales: current, average weekly and worst weekly pain) CVs: –none reported	↑ Negative events weeks = ↑ pain-negative/positive affect and positive-negative affect correlations (no differences between ↑ positive events weeks and the rest of the weeks, no differences between the means of positive/negative affect between the subsamples)	The DMA – positive and negative affect and affective correlates such as pain are separate in non-stressful conditions, but under stressful conditions they 'begin to collapse to produce a mode unified response in order to conserve finite information-processing resources' (p. 87)
Fifield, Tennen, Reisine, & McQuillan (1998)	203, RA	CS – telephone interviews (part of prospective 10-year study)	ANOVA	IVs: –lifetime major depression (definite, subthreshold, none – excluding current depression), based on current/lifetime diagnosis of major depression: DIS III-A –dysphoric mood (low vs. high): CES-D DVs: –pain/fatigue in the past week: numeric rating scale –functional ability: HAQ CVs:–fatigue, functional ability, medication (for the pain analysis)	The definite lifetime major depression, ↑ dysphoria group = ↑ pain (compared with the ↓ dysphoria groups, irrespective of diagnosis) No interactions in predicting fatigue and functional disability	Mood acts as a priming condition for previous depression to influence current pain reports. Major depression is a risk factor for increased pain reports, not as a stable trait with consistent effects, but conditional on current mood – the vulnerability hypothesis

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Affleck et al. (1999)	71, OA and 76, RA	D – 30 consecutive daily reports	HLM	IVs: –gender (level 2) –daily pain (level 1): RADAR DV: –next day negative mood: short PMS-B CVs: –daily negative mood, next day positive mood (level 1): short PMS-B	Men × ↑ pain (Day 1) = ↑ negative mood (Day 2) (compared with women)	Men 'more likely than women to report an increase in negative mood the day after a more painful day' (p. 605); women, even if they report more pain, 'might be able to limit its emotional consequences better than men'
Crombez et al. (1999)	40, H	CS – questionnaires, lab attentional interference assessment		IVs: –pain: VAS –pain-related fear: TSK-D DV: –attentional interference: numerical interference test CVs: –gender, age, education, pain duration –negative affect: D-NEM	↑ Pain × ↑ pain-related fear = ↑ attention interference	According to the CAM, 'pain-related fear facilitates and intensifies the activation of these escape patterns and, therefore, fuels the interruptive function of pain' (p. 408)
Feldman, Downey, & Schaffer-Neitz (1999)	109, RSDS	D – 28 consecutive daily reports	HLM	IVs: –perceived support: number of people from whom participant received support that day –pain: item assessing the daily pain intensity relative to the average –negative mood (overall, depression, anger, anxiety): mood checklist adapted after ABS DVs: –pain/negative mood (Day 2) CVs: –pain/negative mood (Day 1)	↓ Perceived support × ↑ pain (Day 1) = ↑ overall negative mood (Day 2) (and depression, but for anger and anxiety only a trend) (opposite differences for ↑ perceived support) (perceived support × Day 1 mood does not predict Day 2 pain)	Perceived support has a buffering effect on the pain–mood relationship (probably by encouraging coping and acknowledging difficulties)

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Hadjistavropoulos, Hadjistavropoulos, & Quine (2000)	81, H	–interview, questionnaire, experimental manipulation during physiotheraphy session	MANOVA	IVs: –attention (somatic monitoring, distraction, control): experimentally manipulated DVs: –pain: SF-MPQ –anxiety: BAI CVs: –none reported	↑ Health anxiety × somatic monitoring = ↓ affective (but not sensory) pain	Attention to sensations as an effective temporary anxiety reduction strategy
Menefee et al. (2000)	167, H	CS – questionnaire	HMRA	IVs: –everyday pain: VAS –depression: CES-D DV: –sleep-quality: PSQI CV: –none reported	↑ Pain × ↑ depression = ↓ sleep-quality	
Potter, Zautra, & Reich (2000)	Study 1: 41, RA, women Study 2: 112, FM, women	Study 1: D – 12 consecutive weekly phone interviews + initial questionnaire; Study 2: CS – questionnaire	Study 1: Z + PTSRA Study 2: Z + HMRA	Study 1: IVs: –stressful weeks: >3 times individual average ISLE scores DVs: –correlations positive/negative affect (PANAS) and affect-pain (current, worst, average – mean score, numeric rating scales) CVs: –neuroticism: NEQ subscale Study 2: IVs: –information processing (degree of cognitive simplicity): Response to Lack of Structure subscale of PNS (median split) DVs: –correlations positive/negative affect (PANAS) and affect-pain (item from SF-36) CVs: –none reported	Study 1: stressful weeks = ↑ correlations PA-NA, pain-NA and pain-PA (compared ith the rest of the weeks) No differences in neuroticism, average pain, NA or PA for people experiencing stressful weeks ↑ NA × ↑ stress = ↓ PA (for the 13 subjects with at least one stressful week) Study 2: ↑ simplicity = ↑ correlations PA-NA and pain-PA (but not pain-NA) ↓ PA × ↑ simplicity = ↑ NA	The DMA – stress and cognitive simplicity as intra- and interpersonal mediators

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Brown, Middaugh, Haythornthwaite, & Bielory (2001)	313, RP	CS – questionnaire, official temperature data	HMRA	IVs: –average daily outdoor temperature per month: recorded for participant's city from national climatic data –perceived anxiety during previous month: two items from SF-36 DV: –pain associated with attacks: three items from SF-36 CVs: –age, gender	Temperature ($>60^\circ$ F and 40–49.9°F) × ↑ anxiety = ↑ pain (compared with $<40^\circ$F)	In warmer temperatures, the role of anxiety in attack-related pain is more relevant
Zautra, Smith, Affleck, & Tennen (2001)	Study 1: 175, RA, OA – women Study 2: 89, FM-women	Study 1: D – questionnaire + weekly phone interviews (between 12 and 20 weeks – to include a stressful week, and an arthritis flare) Study 2: D – reports 3 times per day, 30 consecutive days	HLM	IVs: –mood clarity (level 2): TMMS –daily pain (level 1): one 0–100 scale (study 1), 0–6 scales for 14 body areas, averaged (study 2) –daily positive affect (level 1): PANAS (study 1), modified version of MAC (study 2) DV: –negative affect (level 1): PANAS (study 1), modified version of MAC (study 2) CVs: –week number/time of day, mean positive affect, mean pain	↑ Weekly positive affect × ↑ weekly pain = less ↑ negative affect (both studies) ↓ Weekly positive affect × ↓ mood clarity = ↑ negative affect (study 1 only, no differences at ↑ positive affect levels)	Both pain and positive affect seen as mediators in alternative interpretations. Mood clarity is associated with weaker relationship between positive and negative affect Two alternative accounts: positive affect acts as a buffer against the effects of pain on negative affect, and distinction between positive and negative emotions less clear during stress

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, × –"interacts with", = –"predict"	Interpretations
Riley, Robinson, Wade, Myers, & Price (2001)	H, 967 women, 680 men	CS – questionnaire (pre-intervention evaluation)	SEM-EC	IVs: –gender –pain-related emotions (pain-related depression, anxiety, frustration, anger, fear during past week): visual analog scales DVs: –pain unpleasantness, pain intensity (lowest, usual, highest): visual analog scales CVs: –pain duration	The strength of the emotion–pain unpleasantness (but not pain intensity) relationship is ↑ for men (constraints of group equalities for parameters in simultaneous MRAs for pain-related emotions as IVs and the six pain variables as DVs)	Pain unpleasantness is more influenced by psychosocial factors than by pain intensity – based on the sequential stage model of pain processing
Janssens et al. (2003)	98, MS	CS – questionnaire and medical assessment	Comparing COR + HMRA	IV: –anxiety and depression: HADS (median split) DVs: –correlations between functional limitations (EDSS-rated medical examination) and health-related QOL (SF-36: physical functioning, role-physical functioning, bodily pain, general health, vitality, social functioning, role-emotional functioning, mental health) CV: –fatigue	↑ EDSS × ↑ HADS = ↑ bodily pain (and ↓ physical functioning, ↓ role-physical functioning) (no Z test reported) (only physical functioning significant in HMRA)	'In patients with more symptoms of anxiety or depression [...] physical limitations may have a greater impact on the quality of their physical health as assessed by the SF-36. A possible explanation is that anxiety and depression impede coping with physical limitations and therefore result in a diminished QoL on these scales' (p. 402)

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Giardino, Jensen, Turner, Ehde, & Cardenas (2003)	74, SCI	CS – telephone interview (pre-intervention evaluation)	HMRA	IVs: –catastrophising: CSQ subscale –perceived solicitous responses from others: WHYMPI subscale –type of relationship with the targets of the ratings (spouse/partner vs. other) DV: –pain (affective, sensory): SF-MPQ CVs: – depression: CES-D –age, gender	↑ Perceived pain solicitousness × ↑ catastrophising = ↑ affective pain (also, living with spouse × ↑ catastrophising = ↑ sensory pain, no association if living with someone else)	Explained both as negative talk about pain evoking solicitous responses, or as solicitousness reinforcing catastrophising verbalisations and negative pain appraisals (also, spouse relations 'carry a higher reinforcement value, represent a more established learning history, or are perceived as a safe context in which to express pain-related catastrophising', p. 23) Social and interpersonal factors influence the catastrophising – pain relationship – the 'communal coping' model (Sullivan et al., 2001)
Keefe et al. (2004)	100, OA-knee pain	D – reports two times per day, 30 consecutive days	HLM	IVs: –gender –daily joint pain: RADAR DVs: –negative/positive mood: abbreviated PMS-B CVs: –daily coping: DCI –daily catastrophising, daily coping efficacy: CSQ subscales	Men × ↑ joint pain (evening Day 1) = ↑ negative/ ↓ positive mood (morning Day 2) (no association for women)	'Women may be better able to limit the emotional consequences of their pain' (p. 576) – extension of Affleck et al. (1999)

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Newth & Delongis (2004)	71, RA	D – reports two times per day, 7 consecutive days + questionnaires	HLM	IVs: –AM pain: visual analog scale –AM mood (depressed, anxious and hostile): ABS DV: –PM Pain: visual analog scale CVs: –age, gender, general functional disability (eight daily activities), general pain frequency, last month's AM stiffness frequency and duration, years since diagnosis: items –personality: IAS-5 –coping strategies (cognitive reframing, distancing, emotional expression, active problem-solving): from WOC-R	↓ AM pain × ↓ AM mood = ↑ PM pain (no differences at ↑ AM pain levels) (also, ↑ AM cognitive reframing and ↓ emotional expression × ↑ extraversion = ↓ PM pain)	AM mood and AM pain alternatively described as moderators 'Mood/distress can play a causal role in pain experience via shared neurophysiological pathways and associated systems' (Melzack, 1999, p. 297)

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Litt, Shafer, & Napolitano (2004)	30, TMD	D – reports four times per day, 7 days + questionnaires	HLM	IVs: –catastrophising (level 2): PRSS subscale –catastrophising (level 1): two items from CSQ (results dichotomised) DV: –momentary pain (left/right): two visual analog scales CVs: –day no. (level 1), recording no. (level 1), day no. × recording no. –general appraisal (optimism/pessimism, self-efficacy, level 3): LOT, PSES –physical and emotional sensitivity (somatisation, positive/negative affectivity, level 2): SC-90-R, PANAS –coping (monitoring, blunting, coping, level 2): MBSS, PRSS subscale –coping self-efficacy (level 1): one item –control (level 1): one item –mood (high and low arousal, negative and positive, level 1): 12 adjectives	↑ Current catastrophising (worried about pain, pain is terrible; level 1) × ↑ catastrophising (level 2) = ↑ current pain (no differences at ↓ current catastrophising levels) (only one interaction tested)	'Those high in trait catastrophisation need not react maladaptively in every circumstance. It may be possible, then, to train people to react adaptively on a situational basis, even those who have a general tendency to catastrophise' (p. 361)
Goubert, Crombez, & Damme (2004)	122, LBP	CS – questionaire	HMRA	IVs: –neuroticism: NEO-D –pain: MPI-D subscale DV: –catastrophising: PCS –pain-related fear: TSK-D CVs: –none reported	↑ Neuroticism × ↑ pain = ↑ catastrophising (only a trend for pain-related fear) (no differences at ↓ neuroticism levels)	Neuroticism 'as a vulnerability factor; it lowers the threshold at which pain is perceived as threatening, and at which catastrophic thoughts about pain emerge' (p. 239) – based on the diathesis–stress model (Eysenck, 1992)

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Sandstrom & Shanberg (2004)	36, JRD-children	CS – questionaire and teachers' ratings	HMRA	IVs: –peer rejection: single items social rejection and popularity, averaged –pain: VAS DV: –depressive symptoms: CDI CVs: –social behaviour: TCSB –peer rejection–social behaviour interaction	↑ Peer rejection × ↑ pain = ↑ depressive symptoms	Peer rejection as a vulnerability factor for depression
Lombardo, Tan, Jensen, & Anderson (2005)	564, H-male veterans	CS – questionnaires (pre-intervention evaluation)	HMRA	IVs: –pain severity: WHYMPI subscale –self-efficacy (pain, function, symptoms): PSES DV: –anger management style (anger out + anger in – anger control + 16): AEI CVs: –none reported	↑ Self-efficacy × ↓ pain intensity = ↓ maladaptive anger management (no differences at ↑ pain intensity levels)	'Self-efficacy has a positive impact on anger management only when pain levels are relatively low [. . .] It is possible that individuals with high self-efficacy and high pain intensity are more apt to feel frustrated by their inability to reduce their pain levels. This frustration may be associated with anger and the potential for maladaptive anger management' (p. 768)

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Hamilton, Zautra, & Reich (2005)	81, RA-women	D – 12–20 weekly telephone interviews, + questionnaires (initial – illness history, demographics; final – individual differences)	HLM	IVs: –affective regulation (mood repair): TMMS subscale –affect intensity (tendency to experience intensely both positive and negative affect): AIM –weekly average pain: numeric rating scale DVs: –positive/negative affect: PANAS CVs: –neuroticism: NEO-PI subscale –week number –active coping: selected items from COPE	↓ Mood repair × ↑ weekly pain (Week 1) = ↑ NA (Week 2) (opposite relationship for ↑ mood repair) ↑ affect intensity × ↑ weekly pain (Week 1) = ↑ NA (Week 2) (opposite relationship for ↓ affect intensity) ↑ affect intensity × ↑ weekly pain (Week 1) = less ↓ in PA (Week 2)	Individual differences in affect regulation moderate the prospective pain–affect relationships – based on the affect regulation literature and the DMA affect intensity as a 'double-edged sword' (p. 222)

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, × –"interacts with", = –"predict"	Interpretations
Zautra, Johnson, & Davis (2005)	124, OA and/or FM-women	D – 10–12 weekly, telephone interviews + initial questionnaires	HLM	IVs: –weekly pain: numerical rating scale –positive affect (levels 1 and 2): PANAS-X –interpersonal stress (levels 1 and 2): items from ISLE and 'overall stress' item DV: –negative affect: PANAS-X CVs: –neuroticism: B5I –week number, age, diagnosis	↑ Weekly positive affect × ↑ weekly pain = less ↑ in NA ↑ weekly positive affect × ↑ weekly interpersonal stress = less ↑ in NA ↑ average positive affect × ↑ weekly pain = less ↑ in NA ↑ average interpersonal stress × ↑ weekly pain = ↑ NA ↑ average positive affect × ↑ weekly interpersonal stress = less ↑ in NA ↑ average interpersonal stress × ↑ weekly interpersonal stress = less ↑ in NA (no time-lagged interaction tests reported, only main effects)	Interpreted within the DMA and the 'broaden-and-build' models The average × weekly interpersonal stress interaction reported as counterintuitive Preferred interpretation – the protective effect of positive affect Suggest the use of mindfulness as intervention in increasing the complexity of processing affect
Meredith, Strong, & Feeney (2006)	152, H	CS – questionnaires, (pre-intervention evaluation)	HMRA	IVs: –attachment style (relationship anxiety and comfort with closeness): ASQ –pain self-efficacy: PSEQ –anxiety: DASS21 subscale DVs: –pain intensity (pain now, highest pain, lowest pain, average pain last week – averaged): visual analog scales –disability: ODI CVs: –age, pain duration, gender	↓ Comfort with closeness × ↓ self-efficacy = ↑ pain intensity ↓ comfort with closeness × ↓ self-efficacy/ ↑ anxiety = ↑ disability (no interaction effects for relationship anxiety)	'The protective nature of high comfort with closeness in the face of chronic pain' (p. 152) – based on attachment theory, exploratory approach

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Conner et al. (2006)	188, RA	D – 30 consecutive daily ratings + clinical interviews	HLM	IVs: –depression history: SCID-I –current depressive symptoms: 5 items based on DSM-IV –daily pain: numeric rating scale DVs: –daily mood (pleasant, unpleasant): six items each –pain coping strategies (direct action, relaxation, distraction, reappraisal, vent emotions, spiritual comfort, emotional support): adapted DCI –pain coping appraisals (catastrophising, control, benefit reminding): two items from CSQ, and two single item-scales CVs: –neuroticism: NEO-FFI –age	Depression history × ↑ daily pain = ↑ venting emotions, ↓ pleasant mood and ↑ unpleasant mood (depression history has no main effect on average daily levels) ↑ current depressive symptoms × ↑ daily pain = ↑ catastrophising and ↓ reappraisal depression history × ↑ current depressive symptoms × ↑ daily pain = ↓ control appraisals	'Greater contingency between pain and emotion-related experiences may reflect a hidden vulnerability for the formerly depressed' (p. 206)
Edwards et al. (2006)	190, SD	CS – questionnaires	HMRA	IVs: –catastrophising: CSQ subscale –educational level (measure of SES): single item DVs: –sensory and affective pain: SF-MPQ –social disruption: PAIS Social Environment subscale CVs: –physical disability: HAQ –depression: BDI –age, work status, type of SD, marital status, sex, ethnicity	↑ Catastrophising × ↓ education = ↑ affective pain, but not sensory pain (↑ catastrophising × ↓ education = ↓ social disruption)	Low SES is a vulnerability, increasing the deleterious effects of catastrophising, which possibly work by sensitisation of central nervous system, or amplification of affective processing (the opposite effect on social disruption possibly shows that catastrophising results in a mobilisation of the social network in people with low SES)

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Hoff, Palermo, Schluchter, Zebracki, & Drotar (2006)	119, SCD, JIA-children	L – three waves, 6-month intervals, questionnaires and physician assessment	GLMM	IVs: –depression (wave 1): RCADS –pain (wave 1, caregiver and patient ratings): FPS DV: –pain (waves 2 and 3): FPS CVs: –age, gender, family income, physician-rated disease severity, time (waves 2 and 3)	For JIA: ↑ depression (wave 1) × ↓ child report pain only (wave 1) = ↑ pain (waves 2 and 3) for SCD: ↑ depression (wave 1) × ↓ caregiver report pain only (wave 1) = ↑ pain (waves 2 and 3)	Depression might function as a risk factor for future disease-related pain (and disability) No explanation for the difference between the two clinical groups (child vs. caregiver report moderations)
Strand et al. (2006)	43, RA	D – 8 consecutive weekly telephone interviews + baseline questionnaires	HLM	IVs: –positive affect: PANAS –weekly pain (most intense): numeric rating scale DV: –negative affect: PANAS CVs: –interpersonal stress: items from ISLE and 'overall stress' items for three areas (friends, family, spouse/partner), averaged –depression: BDI –week number	↑ Weekly pain × ↓ weekly positive affect = ↑ negative affect (less ↑ negative affect at ↑ positive affect levels) (no interaction positive affect × weekly interpersonal stress, no level 2 × level 1 interactions)	Positive affect as a resilience factor. Two alternative accounts proposed: 'narrowing of affective differentiation' and 'boost in affective resources' – the DMA

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Tennen, Affleck, & Zautra (2006)	71, FM-women	D – reports three times per day, 30 days + initial interview and questionnaires	HLM	IVs: –previous depression: DIS-III-R –daily pain intensity: three times/day (14 body areas, summed), daily pain averaged DVs: –pain control: one item –catastrophising: CSQ subscale –pain coping strategies (direct action, relaxation, distraction, positive reappraisal, vent emotions, spiritual comfort, emotional support): adapted DCI –pain coping efficacy: one item –pleasant/unpleasant mood: three times/day (happy and cheerful / sad and blue, summed), daily mood averaged CVs: –neuroticism: NEO-PI subscale –current depressive symptoms: BSI subscale	Previous depression × ↑ daily pain = ↓ pain coping efficacy, ↑ venting emotions Previous depression × ↑ daily depressive symptoms × ↑ daily pain = ↓ positive affect (but not catastrophising, other coping strategies, negative mood, pain control)	Support for the lingering vulnerability (main effect of previous depression) and priming hypotheses (interactions)

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Connelly et al. (2007)	94, RA	D – 30 daily reports + initial medical examination and questionnaire	HLM	IVs: –recovery from high NA (day-to-day changes): PANAS –recovery from low PA (day-to-day changes): PANAS –education –age –active joint count: medical examination DV: –pain: VAS CV: –none mentioned	↓ Education × ↑ NA recovery = ↓ pain ↑ active joint count × ↑ NA recovery = ↓ pain ↓ age × ↑ PA recovery = ↓ pain	Interindividual differences in affect regulation (results also influenced by differences in variability of affect regulation)
Johansen & Cano (2007)	79, H	CS – questionnaire, interaction coding of conversation recordings	ANOVA	IVs: –anger/contempt, sadness, fear, humour of patient expressed in marital interaction: SPAFF –anger/contempt, sadness, fear, humour of spouse expressed in marital interaction: SPAFF –couple pain status: one or both reporting pain DV: –pain severity: items from WHYMPI CVs: –ethnicity, marriage duration, education	↑ Patient's sadness × couple pain status (both) = ↓ pain severity (opposite when only one reporting pain) ↑ Spouse's humour × couple pain status (both) = ↑ pain severity (ns when only one reporting pain)	Empathic communication in couples as an emotion regulation mechanism in chronic pain

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Kratz, Davis, & Zautra (2007)	36 OA, 86 FM-women	D – 2–12 weekly telephone interviews + initial questionnaires	HLM	IV: –acceptance (level 2): 10 items from original CPAQ –weekly worst pain (levels 1 and 2): numeric rating scale –positive affect (levels 1 and 2): PANAS DV: –negative affect: PANAS CVs: –pain catastrophising (level 2): CSQ subscale (four of the six items) –age, diagnostic (level 2)	↓ Acceptance × ↑ pain severity = more ↑ negative affect (but not positive affect) introducing the ↓ positive affect × ↑ pain severity interaction makes the above interaction non-significant	Greater acceptance is possibly a factor of resilience in managing chronic pain, but its effects are probably mediated by levels of positive affect – based on acceptance literature and the 'broaden-and-build' model
Strand et al. (2007)	40, RA	D – 8 consecutive weekly telephone interviews + questionnaire	HLM	IVs: –pain stages of change (precontemplation, contemplation, action/maintenance; level 2): PSOCQ –weekly positive and negative affect (level 1): PANAS DV: –average weekly pain: numeric rating scale CV: –first week reporting (level 2)	↑ Pain readiness to change (action/maintenance) × ↓ weekly positive affect = ↑ weekly pain (no differences at ↓ action/maintenance levels) (no interaction or main effects of precontemplation and contemplation)	High action/maintenance means more active pain coping which potentially increases pain, and therefore lower PA reports action/maintenance impacts on pain only if integrated with positive affect to promote action Action/maintenance reflects personal responsibility for the pain management, and therefore relates to a stronger impact of pain on the positive emotion (increasing it when pain is managed, lowering it when pain increases)

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Zautra et al. (2007)	74, RA	CS – questionnaires, clinical interview (phone), lab stress induction and assessment	HLM	IVs: –prior depression (two or more depressive episodes, versus one or no episodes): SCID-I – perceived stress change after stress induction (speech task, discussion of an interpersonal conflict): numeric rating scales (scores before induction extracted from scores after induction) –positive affect: PANAS (scores before induction extracted from scores after induction) DV: –bodily pain: numerical ratings – 15 body areas (body diagram) –joint pain: RADAR CVs: –current depressive symptoms: HDI –initial pain scores: WOMAC subscales, SF-36 –physician-assessment of tenderness, swelling and disease severity	prior depression × ↑ perceived stress change = ↑ bodily and joint pain prior depression × ↑ perceived stress change × ↑ positive affect change = less ↑ bodily and joint pain (positive emotion reports increased together with stress reports)	Previous depression represents increased vulnerability to stress. Positive affect is 'protective against stress-related increases in pain for those with a history of multiple episodes of major depression' (p. 195) – the DMA and the vulnerability priming hypothesis

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, × –"interacts with", = –"predict"	Interpretations
Valrie, Gil, Redding-Lallinger, & Daeschner (2008)	670, SCD – children	D – daily reports, up to 2 months + initial interviews	HLM	IVs: –mood (one dimension, positive-negative): FAS –subjective sleep quality: visual analog scale DV: –average daily pain: visual analog scale CVs: –age, gender, level of maternal education, SCD type	↓ Mood × ↓ sleep quality = ↑ next day pain (relationship sleep-quality-next day pain decreases at ↑ mood levels) (pain × mood did not predict next day sleep quality)	Mood as a moderator of the pain–sleep relation: 'the impact of poor sleep on high pain the following day was weakened at increasing levels of positive mood' (p. 320)
Adams et al. (2008)	83, MSP	CS – medical assessment and questionnaire	ANOVA, HMRA	IVs: –gender –depression (high vs. low, scorers between 10 and 15 excluded): BDI-II DV: –activity-related pain (average pain rating during lifting task): numeric rating scale CVs: –pain severity: MPQ, the PRI index –depression scores: BDI-II	Women only × ↑ level of depression = ↑ activity-related pain	Authors suggest physiological mechanisms (differences in endogenous opioids activation influenced by hormonal factors) in addition to social role explanations, and differences in emotion regulation (e.g., greater tendency to ruminate in women)

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Middendorp et al. (2008)	403, FM-women	CS – questionnaires	HMRA	IVs: –positive and negative affect: PANAS-X –emotional processing, general emotional expression: EACS subscales –cognitive reappraisal: ERQ subscale –anger expression: SECS (based on STAI) subscale –difficulty identifying feelings, difficulty describing feelings: TAS-20 –emotional suppression: ERQ subscale –anger suppression: SECS –affect intensity (impulse strength): BEQ subscale –mental distress: average FIQ anxiety and depression items and MPI disturbed mood scale DVs: –pain: averaged FIQ pain and stiffness items and MPI pain intensity scale –fatigue: averaged FIQ fatigued and rested items CVs: –none reported	↓ Emotional processing × ↑ affect intensity = ↑ pain (and ↑ fatigue) ↑ difficulty describing feelings × ↑ affect intensity = ↑ pain (no interaction PA × NA in predicting pain or fatigue)	'The intense experiencing of emotions is not necessarily maladaptive as long as these emotions are adequately processed' (p. 165) Intervening to stimulate emotion regulation depending on the patient's emotional style could help differentiate negative affect from pain and thus increase disease control

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Abeare et al. (2010)	157, RA	CS – questionnaire and lab assessment	HMRA	IV: –pain: VAS –NA, PA: PANAS-X DV: –executive functioning: mean of standardised scores of Wechsler Letter-Number Sequencing and Stroop tests CV: –fatigue: FSS –depressed mood: AIMS2 subscale	↑ Pain × ↑ PA (but not NA) = ↓ executive functioning (but no Pain × NA interaction in predicting PA)	Maintaining PA when pain increases might require additional executive resources; 'the other side of the resiliency coin' (p. 687). Alternatively, dopaminergic mechanisms might apply, or high PA related to lower pain ratings under higher stimulus intensity, requiring more executive resources
Cohen, Vowles, & Eccleston (2010)	222, H-adolescents	CS – questionnaires	HMRA	IVs: –anxiety: SCAS –typical pain over last week: visual analog scale DV: –physical and social functioning: BAPQ –physical and social functioning parent-proxy: BAPQ-P –school attendance: one item –physician visits (patient and parent reports): one item each CVs: –clinic site, age, gender, pain type, pain duration	↑ Pain × ↓ anxiety = ↓ physical functioning (self and parent report), ↓ school attendance, and ↑ physician visits (self and parent report) (but not for social functioning) (no differences at ↑ levels of anxiety)	'When anxiety is high, anxiety rather than pain might drive avoidant behavior. On the other hand, in the absence of anxiety, it might be pain itself that leads to avoidance of physical and social events' (p. 2)

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
Finan et al. (2010)	46, FM – women	D – 30 consecutive daily reports, +genotyping	HLM	IVs: –the catechol-*O*-methyltransferase gene (COMT/val^{158}met) – the opioid receptor gene (OPRM1/asn^{40}asp) –daily pain: numerical ratings – 15 body areas (body diagram) DVs: –daily PA, NA: PANAS CV: –baseline medication use	*met/met* genotype × ↑ pain = ↓ PA (than *val/met* or *val/val* genotypes) at least one asp^{40} allele × ↑ pain = ↓ PA (than those homozygous for the asn^{40} allele) (but also ↑ NA)	The role of catecholamine and opioid systems in pain-related positive affect regulation in FM
Kranz, Bollinger, & Nilges (2010)	150, H	CS – questionnaires	HMRA	IVs: –accommodative flexibility: FGAS –pain: average for past 4 weeks, 11-point rating scale DV: –chronic pain acceptance (pain willingness, activities engagement): G-CPAQ –CVs: PA, NA (PANAS German version)	↓ Accommodative flexibility × ↑ pain = ↓ pain willingness and ↓ activity engagement	Accommodative flexibility as a 'coping competence at the dispositional level that enhances concrete coping mechanisms involving disengagement and reorientation (such as pain acceptance) at the situational level' (p. 1024)

Table 1 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Interactions ↑–high/increase, ↓–low/decrease, ×–"interacts with", =–"predict"	Interpretations
McParland & Knussen (2010)	95, OA or FM	CS – questionnaire	HMRA	IVs: –pain (current, average, worst): CPG –just world beliefs, personal and general: PBJWS & GBJWS DV: –psychological distress (somatic symptoms, anxiety and insomnia, social dysfunction, and severe depression): GHQ CVs: –age –social desirability: MCSDS –religiosity: single item	↓ General (but not personal) just world beliefs × ↑ pain = ↑ psychological distress	According to the Just World Theory (Lerner, 1980, as cited in McParland & Knussen, 2010), 'when presented with injustice (in this case pain) individuals with a strong general belief in a just world will be motivated to adopt strategies to maintain this belief by restoring a sense of justice in some way' (p. 74)
Schütze, Rees, Preece, & Schütze (2010)	104, H	CS – questionnaire	HMRA	IVs: –mindfulness: MAAS –pain: subscale BPI DV: –catastrophising: PCS CV: –none reported	↓ Mindfulness × ↑ pain = ↑ catastrophising	Low mindfulness as a vulnerability factor, precursor to pain catastrophising in a modified fear-avoidance model

Abbreviations: sample characteristics (RA, rheumatoid arthritis; OA, osteoarthritis; MS, multiple sclerosis; FM, fibromyalgia; H, heterogeneous; RSDS, reflex sympathetic dystrophy syndrome; SCI, spinal cord injury; TMD, temporomandibular disorder; LBP, low back pain; MSP, musculoskeletal pain; SD, scleroderma; SCD, sickle cell disease; JIA, juvenile idiopathic arthritis; JRD, juvenile rheumatic disease; RP, Raynaud's phenomenon), research design (CS, cross-sectional; L, longitudinal; D, diary study; EXP, experimental), data analysis (HMRA, hierarchical multiple regression analysis; HLM, hierarchical linear modelling; GLMM, general linear mixed modelling; (M)ANOVA, (multivariate) analysis of variance; COR, correlation; *Z*, Fisher's *z* test; SEM-EC, multigroup structural equation modelling with equality constraints; PTSRA, pooled time-series regression analysis).
Note: Questionnaire abbreviations – see Appendix C.

Table 2. Empirical studies of interaction effects related to pain–emotion relationships – null results.

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Pain–affect interactions tested ×–"interacts with", =–"predict"
Jensen & Karoly (1991)	118, H	CS – questionnaire	HMRA	IV: –coping: CSQ –pain severity: common factor of five numerical rating scales (current, average, most, least and average frequency) DV: –psychological functioning: common factor based on CES-D and SWLS CV: –none reported	Coping × pain = depression
Jensen & Karoly (1992)	118, H	CS – questionnaire	HMRA	IV: –comparative self-evaluation: common factor of single items (selective focus, hypothetical worse worlds, downward comparison, comparison to normative standard) –pain: numerical rating scales (current, average) DV: –depression: CES-D CV: –education, gender, age, pain duration, pain site, perceived pain control (single items)	Self-evaluation × pain = depression
Ferguson & Cotton (1996)	81, RA-women	L – questionnaire	HMRA	IV: –pain: AIMS subscale –sleep: GHQ item –social activity: AIMS subscale DV: –depression: AIMS subscale CV: –age, duration of illness, disability (and initial depression for longitudinal models)	Pain × sleep = concurrent depression Pain × sleep = depression 12 months later Pain × social activity = concurrent depression Pain × social activity = depression 12 months later (ns when sleep × social activity entered first in the equation)

Table 2 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Pain–affect interactions tested × –"interacts with", = –"predict"
Roberts, Matecjyck, & Anthony (1996)	59, OA	CS – questionnaire	HMRA	IV: –pain: AIMS subscale –social support: MISSB (emotional, informational, tangible, integrative) DV: –depression: AIMS subscale CV: –none reported	Pain × social support (squared, to test non-linear relation) = depression
Arango & Cano (1998)	31, RA	L – questionnaire, two waves, 3-month interval	HMRA	IV: –daily stress: HS –pain: present pain intensity from MPQ DV: –anxiety and depression: AIMS subscales CV: –none reported	Pain × daily stress (wave 1) = depression and anxiety (wave 2)
Riemsma, Taal, Wiegman, Rasker, Bruyn, & van Passen (2000)	197, RA	CS – questionnaire	HMRA	IV: –pain: D-AIMS2 subscale –positive support: SSLI2-I –problematic support: Dutch version of SSS DV: –depression: D-AIMS2 mood scale of CV: –sex, age, education –physical functioning: D-AIMS2 subscale	Pain × positive support = depression Pain × problematic support = depression

Table 2 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Pain–affect interactions tested × –"interacts with", = –"predict"
van den Hout et al. (2001)	76, LBP	EXP – questionnaires, interview, experimental manipulation	HMRA	IV: –trait negative affectivity: D-NEM –state negative affectivity: D-POMS –failure feedback (success vs. failure): experimentally manipulated social empathy test feedback DV: –pain: VAS (during lifting task) CV: –gender –pain: VAS (baseline)	Trait/state negative affectivity × failure feedback = pain (during lifting)
Plach, Heidrich, & Waite (2003)	156, RA – women	CS – questionnaire	HMRA	IV: –pain: AIMS2 subscale –role discrepancy: SDS DV: –anxiety: JPRI subscale –depression: CES-D CV: –income –functional status: AIMS2 subscales	Pain × role discrepancy = anxiety/depression
Roelofs, Peters, Patijn, Vlaeyen, & Schouten (2004)	40, LBP	D – reports eight times per day, 1 week + baseline questionnaire	HLM	IV: –Trait pain-related fear: TSK-D –attention to pain: items from PVAQ DV: –pain intensity: single item CV: –none reported	Trait pain-related fear × attention to pain = pain intensity (concurrent and subsequent)

Table 2 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Pain–affect interactions tested ×–"interacts with", =–"predict"
Michael & Burns (2004)	82, H	EXP – questionnaires, experimental manipulation during pain induction	HMRA	IV: –pain catastrophising: PCS –attentional focus (sensory, affective, control): experimentally manipulated during cold-pressor pain induction DV: –pain reporting after cold-pressor task: single item CV: –baseline pain reporting, medication use: single items	Pain catastrophising × attentional focus = pain reporting
Keogh, McCracken, & Eccleston (2006)	260, H	CS – questionnaire	HMRA	IV: –depression: BDI –anxiety: PASS DV: –pain (present): numerical scale CV: –none (demographics not significantly related)	Gender × depression/anxiety = pain
Quartana, Burns, & Lofland (2007)	68, LBP	EXP – questionnaires, experimental manipulation during pain induction	HMRA	IV: –pain catastrophising: PCS –attentional strategy (sensory focus, distraction, suppression): experimentally manipulated during cold-pressor pain induction DV: –pain reporting after cold-pressor task: single item (residualised change from baseline) CV: –none reported	Pain catastrophising × attentional strategy = pain reporting

Table 2 (*Continued*)

Reference	Sample (N, condition)	Research design	Data analysis methods	Variables (IV – independent/predictors, DV – dependent/outcomes, CV – control/covariates)	Pain–affect interactions tested × –"interacts with", = –"predict"
Kaczynski, Claar, & Logan (2009)	266, RHD, RAP – children and adolescents	CS – clinical interview, questionnaire, parents' ratings	SEM-EC	IV: –gender –pain intensity: interview ratings (current, lowest, highest) DV: –internalising symptoms: depression (CDI) and anxiety (RCMAS) CV: –SEM model included age, passive coping (PRI), protective parenting (ARCS), and functional disability (FDI)	Gender × pain intensity = internalising symptoms
Libby & Glenwick (2010)	57, JPFS – children and adolescents	CS – questionnaire, parents' ratings	HMRA	IVs: –stress: CHS –catastrophising: CSQ-C subscale –self-efficacy: CASE DV: –current pain and worst pain: VAS CVs: –perceived support: PSS-Fa and PSS-Fr	Hassles × catastrophising/self-efficacy = current/worst pain

Abbreviations: sample characteristics (RA, rheumatoid arthritis; OA, osteoarthritis; H, heterogeneous; LBP, low back pain; RHD, recurrent headache; RAP, recurrent abdominal pain; JPFS, juvenile primary fibromyalgia syndrome), research design (CS, cross-sectional; L, longitudinal; D, diary study; EXP, experimental), data analysis (HMRA, hierarchical multiple regression analysis; HLM, hierarchical linear modelling; SEM-EC, multigroup structural equation modelling with equality constraints). Note: Questionnaire abbreviations – see Appendix C.

Appendix C: Questionnaires Abbreviations: Titles & References (Alphabetical Order)

ABS – Affects Balance Scale (Derogatis, 1975, as cited in Feldman, Downey, & Schaffer-Neitz, 1999)
AEI – Anger Expression Inventory (Spielberger et al., 1985, as cited in Burns, Johnson, Mahoney, Devine, & Pawl, 1996)
AIM – Affect Intensity Measure (Larsen, 1984, as cited in Hamilton, Zautra, & Reich, 2005)
AIMS – Arthritis Impact Measurement Scales (Meenan, Gertman, & Mason, 1980, as cited in Brown, Nicassio, et al., 1989)
AIMS2 – Arthritis Impact Measurement Scales 2 (Meenan et al., 1992, as cited in Plach, Heidrich, & Waite, 2003)
ARCS – Adult Responses to Children's Symptoms (Van Slyke & Walker, 2006, as cited in Kaczynski, Claar, & Logan, 2009)
ASQ – Attachment Style Questionnaire (Feeney et al., 1994, as cited in Meredith, Strong, & Feeney, 2006)
B5I – "Big Five" Inventory (John, Donahue, & Kentle, 1991, as cited in Zautra, Johnson, & Davis, 2005)
BAPQ – Bath Adolescent Pain Questionnaire (Eccleston, Jordan, McCracken, Sleed, Connell, & Clinch, 2005, as cited in Cohen, Vowles, & Eccleston, 2010)
BDI – Beck's Depression Inventory (Beck & Beck, 1972, as cited in Moosbrugger & Schermelleh-Engel, 1991)
BDI-II – Beck Depression Inventory II (Beck, Steer, & Brown, 1996, as cited in Adams et al., 2008)
BEQ – Berkeley Expressivity Questionnaire (Gross, 2000, as cited in Middendorp et al., 2008)
BPI – Brief Pain Inventory (Cleeland & Ryan, 1994, as cited in Schütze, Rees, Preece, & Schütze, 2010)
BSI – Brief Symptom Inventory (Derogatis & Melisaratos, 1983, as cited in Tennen, Affleck, & Zautra, 2006)
CASE – Children's Arthritis Self-Efficacy Scale (Barlow, Shaw, & Eright, 2001, as cited in Libby & Glenwick, 2010)
CDI – Children's Depression Inventory (Kovacs, 1992, as cited in Sandstrom & Shanberg, 2004)
CES-D – Center for Epidemilogic Studies – Depression Scale (Radloff, 1977, as cited in Brown, Nicassio, et al., 1989)
CHS – Children's Hassles scale (Kanner, Harrison, & Wertlief, 1985, as cited in Libby & Glenwick, 2010)
CMHS – Cook-Medley Hostility Scale (Cook & Medley, 1954, as cited in Burns, Johnson, Mahoney, Devine, & Pawl, 1996)
COPE – COPE Inventory (Carver, Scheier, & Weintraub, 1989, as cited in Hamilton, Zautra, & Reich, 2005)
CPAQ – Chronic Pain Acceptance Questionnaire (Geisser, 1992, as cited in Kratz, Davis, & Zautra, 2007)
CPG-Chronic pain Grade (Von Korff et al., 1992, as cited in McParland & Knussen, 2010)
CSQ-Coping Strategies Questionnaire (Rosenstiel & Keefe, 1983, as cited in Affleck, Urrows, et al., 1992)
CSQ-C – Coping Strategies Questionnaire – Child version (Schanberg et al. 1996, as cited in Libby & Glenwick, 2010)
D-AIMS2 – Dutch version of Arthritis Impact Measurement Scales (Riemsma et al., 1996, as cited in Riemsma et al, 2000)
D-NEM – Dutch version Negative Emotionality Scale (Stegen et al., 1998, as cited in Crombez et al., 1999)
D-POMS – Dutch version of Profile of Mood States (Wald & Mellenberg, 1990, as cited in van den Hout et al., 2001)
DASS21 – Depression Anxiety Stress Scales 21 (Lovibond & Lovibond, 1993, 1995, as cited in Meredith et al., 2006)
DCCP – dimensions of coping with chronic pain (Geissner & Wurtele, 1992, as cited in Schmitz et al., 1996)

DCI – Daily Coping Inventory (Stone & Neale, 1984, as cited in Affleck, Urrows et al, 1992)
DIS III-A – Diagnostic Interview Survey III-A, based on DSM-III-R (Robins & Helzer, 1985, as cited in Fifield et al., 1998)
DIS-III-R – Diagnostic Interview Schedule-Version III-Revised (Robins, Helzer, Cottler, & Goldring, 1989, as cited in Tennen et al., 2006)
EACS – Emotional Approach Coping Scales (Stanton, Kirk, Cameron, & Danoff-Burg, 2000, as cited in Middendorp et al., 2008)
EDSS – Expanded Disability Status Scale (Kurtzke, 1983, as cited in Janssens et al., 2003)
ERQ – Emotion Regulation Questionnaire (Gross, 2003, as cited in Middendorp et al., 2008)
FAS – Facial Affective Scale (McGrath, de Veber, & Hearn, 1985; McGrath et al., 1996, as cited in Valrie, Gil, Redding-Lallinger, & Daeschner, 2008)
FDI – Functional Disability Inventory (Claar & Walker, 2006; Walker & Greene, 1991, as cited in Kaczynski et al., 2009)
FIQ – Fibromyalgia Impact Questionnaire (Zijlstra, Taal, Van de Laar, & Rasker, 2007, as cited in Middendorp et al., 2008)
FPS – Faces Pain Scale (Bieri, Reeve, Champion, Addicoat, & Ziegler, 1990, as cited in Hoff et al., 2006)
FSS – Fatigue Severity Scale (Krupp et al., 1989, as cited in Abeare et al, 2010)
G-CPAQ – German version of Chronic Pain Acceptance Questionnaire (Nilges, Köster, & Schmidt 2007, as cited in Kranz, Bollinger, & Nilges, 2010)
GHQ – General Health Questionnaire (Goldberg & Hillier, 1979, as cited in McParland & Knussen, 2010)
HADS – Hospital Anxiety and Depression Scale (Zigmond and Snaith, 1983, as cited in Janssens et al., 2003)
HAQ – Stanford Health Assessment Questionnaire (Fries, Spitz, Kraines, & Holman, 1980, as cited in Fifield, Tennen, Reisine, & McQuillan, 1998)
HDI – Hamilton Depression Inventory (Reynolds & Kobak, 1995, as cited in Zautra et al., 2007)
HS – Hassles Scale (Kanner et al., 1981, as cited in Arango & Cano, 1998)
IAS-5 – Interpersonal Adjective Scales – Big 5 (Trapnell & Wiggins, 1990, as cited in Newth & Delongis, 2004)
IES – Illness Attitudes Scale (Kellner et al., 1987, as cited in Hadjistavropoulos, Hadjistavropoulos, & Quine, 2000)
ILS – Illness Uncertainty Scale (Mishel, 1981, as cited in Affleck, Tennen, Pfeiffer, & Fifield, 1987)
ISLE – Inventory of Small Life Events (Zautra, Guarnaccia, & Dohrenwend, 1986, as cited in Zautra et al., 1997)
JPRI – Jackson Personality Research Inventory (Jackson, 1977, 1979, as cited in Plach et al., 2003)
LOT – Life Orientation Test (Scheier & Carver, 1985, as cited in Tennen, Affleck, Urrows, Higgins, & Mendola, 1992)
MAAS – Mindful Attention Awareness Scale (Brown & Ryan, 2003, as cited in Schütze, Rees, Preece, & Schütze, 2010)
MAC – Mood Adjective Checklist (Larsen & Diener, 1992, as cited in Zautra et al., 2001)
MBSS – Miller Behavioral Style Scale (Miller, 1987, as cited in Litt, Shafer & Napolitano, 2004)
MCSDS – Marlowe-Crowne Social Desirability Scale (Strahan & Gerbasi, 1972, as cited in McParland & Knussen, 2010)
MISSB – Modified Inventory of Socially Supportive Behaviours (Krause & Markides, 1990, as cited by Roberts, Matecjyck, & Antony, 1996)
MPI-D – Multidimensional Pain Inventory – Dutch version (Lousberg et al., 1999, as cited in Goubert et al., 2004)
MPQ – McGill Pain Questionnaire (Melzack, 1975, as cited in Adams et al., 2008)
NEO-D – Dutch version of the Big Five Personality Questionnaire (de Fruyt & Mervielde, 1998, as cited in Goubert et al., 2004)
NEO-FFI – NEO Five Factor Inventory (Costa & McCrae, 1992, as cited in Conner et al., 2006)

NEO-PI – NEO Personality Inventory (Costa & McCrae, 1985, as cited in Affleck, Urrows, et al., 1992)
NEQ – a short Neuroticism and Extraversion questionnaire (Eysenck, 1958, as cited in Potter, Zautra, & Reich, 2000)
ODI-Oswestry Disability Index (Fairbank et al., 1980, as cited in Meredith et al., 2006)
PAIS – Psychosocial Adjustment to Illness Scale (Derogatis, 1983, as cited in Edwards et al., 2006)
PANAS – Positive and Negative Affect Scale (Watson et al., 1988, as cited in Zautra, Potter, & Reich, 1997)
PANAS-X – Positive and Negative Affect Scale – Expanded Form (Watson & Clark, 1999, as cited in Zautra, Johnson, & Davis, 2005)
PANAS German version – Positive and Negative Affect Scale (Krohne et al., 1996, as cited in Kranz, Bollinger, & Nilges, 2010)
PASS – Pain Anxiety Symptom Scale (McCracken et al., 1992, as cited in Keogh et al., 2006)
PBJWS & GBJWS – Personal Belief in a Just World Scale & General belief in a Just World Scale (Dalbert, 1999, and Dalbert, Montada, & Schmitt, 1987, as cited in McParland & Knussen, 2010)
PCS – Pain Catastrophizing Scale (Sullivan et al, 1995, as cited in Goubert, Crombez, & Damme, 2004)
PDI – Pain Disability Index (Tait et al., 1987, 1990, as cited in Schmitz et al., 1996)
PMI – Pain Management Inventory (Brown & Nicassio, 1987, as cited in Brown, Nicassio, et al., 1989)
PMS-B – Profile of Mood States-B (Lorr & McNair, 1982, as cited in Affleck et al., 1987)
PNS – Personal Need for Structure (Neuberg & Newsom, 1993, as cited in Potter et al., 2000)
PRI – Pain Response Inventory (Walker et al., 1997, as cited in Kaczynski et al., 2009)
PRQ – Pain Regulation Questionnaire (Schermelleh-Engel, 1990, as cited in Moosbrugger & Schermelleh-Engel, 1991)
PRSS – Pain-Related Self-Statements Scale (Flor et al., 1993, as cited in Litt et al., 2004)
PSEQ – Pain Self-efficacy Questionnaire (Nicholas, 1994, as cited in Meredith et al., 2006)
PSES – Pain Self-Efficacy Scale (Anderson, Edwards, Dowds, Peeters-Asdourian, & Pelletz, 1995, as cited in Litt et al., 2004)
PSOCQ – Pain Stages of Change Questionnaire (Kerns et al., 1997, as cited in Strand et al., 2007)
PSQI – Pittsburgh Sleep Quality Index (Buysse et al., 1989, as cited in Menefee et al., 2000)
PSS-Fa and PSS-Fr – Perceived Support From Family and Friends Scale (Procidano & Heller, 1983, as cited in Libby & Glenwick, 2010)
PVAQ – Pain Vigilance and Awareness Questionnaire (McCracken, 1997, as cited in Roelofs et al., 2004).
RADAR – Rapid Assessment of Disease Activity in Rheumatology (Mason et al., 1992, as cited in Affleck, Urrows, et al., 1992)
RCADS – Revised Child Anxiety and Depression Scale (Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000, as cited in Hoff, Palermo, Schluchter, Zebracki, & Drotar, 2006)
RCMAS – Revised Children's Manifest Anxiety Scale (Reynolds & Richmond, 1978, 1997, as cited in Kaczynski et al., 2009)
SC-90-R – Symptom Checklist-90-Revised (Derogatis, 1977, as cited in Litt et al., 2004)
SCAS – Spence Children's Anxiety Scale (Spence, 1997, as cited in Cohen, Vowles & Eccleston, 2010)
SCID-I – Structured Clinical Interview for DSM-IV (First et al., 2002, as cited in Conner et al., 2006)
SDS-Self – Discrepancy Scale (Heidrich, 1998, Heidrich et al., 1994, as cited in Plach et al., 2003)
SECS – Self-expression and Control Scale (Van Elderen, Maes, Van der Kamp, Van der Ploeg, & Spielberger, 1999, as cited in Middendorp et al., 2008)
SF-36 – Medical Outcome Survey Short Form (McHorney, Ware, Lu, & Sherbourne, 1994, as cited in Potter et al., 2000)
SF-MPQ – Short-Form McGill Pain Questionnaire (Melzack, 1987, as cited in Hadjistavropoulos, Hadjistavropoulos, & Quine, 2000)

SHS – Social Health Scale (Donald, Ware, Brook, & Davies-Avery, 1978, as cited in Brown, Wallston et al., 1989)
SPAFF – Specific Affect Coding System (Gottman et al., 1996, as cited in Johansen & Cano, 2007)
SSL12-I-Social Support List – Interactions (van Eijk, Kempen, & van Sonderen, 1994, as cited in Riemsma et al., 2000)
SSS – social support scale measuring positive and problematic support (Revenson et al., 1991, as cited in Riemsma et al, 2000)
STAI – State Trait Anxiety Inventory (Laux, Glanzmann, Schaffner, & Spielberger, 1981, as cited in Moosbrugger & Schermelleh-Engel, 1991)
STMSS – Strong Ties Measure of Social Support (Lin & Ensel, 1981, as cited in Brown, Wallston, et al., 1989)
SWLS – Satisfaction With Life Scale (Diener, Emmons, Larsen, & Griffin, 1985, as cited in Jensen & Karoly, 1992)
TAS-20 – Toronto Alexithymia Scale (Bagby, Parker, & Taylor, 1994, as cited in Middendorp et al., 2008)
TCSB – Teacher Checklist of Social Behavior (Coie et al., 1999, as cited in Sandstrom & Shanberg, 2004)
TGP & FGAS – Tenacious Goal Pursuit & Flexible Goal Adjustment Scale (Brandstädter & Renner, 1990, as cited in Schmitz, Saile, & Nilges, 1996)
TMMS – Trait Meta-Mood Scale (Salovey et al, 1995, as cited in Zautra, Smith, Affleck, & Tennen, 2001)
TSK-D – Dutch version Tampa Scale of Kinesiophobia (Goubert et al., 2003, as cited in Crombez et al., 1999)
WHYMPI – West Haven-Yale Multidimensional Pain Inventory (Kerns et al., 1985, as cited in Burns et al., 1996)
WOC-R – Revised Ways of Coping (Folkman et al., 1986, as cited in Newth & Delongis, 2004)
WOMAC – Western Ontario and McMaster Universities Osteoarthritis Index (Bellamy et al., 1988, as cited in Zautra et al., 2007)