School of Psychology

Thinking About Thinking About Pain: Development of a Measure and Model of Pain-Related Metacognition

Robert Michael Schütze

This thesis is presented for the Degree of
Doctor of Philosophy
of
Curtin University

October 2017

Declaration

To the best of my knowledge and belief this thesis contains no material previously published by any other person except where due acknowledgment has been made.

This thesis contains no material which has been accepted for the award of any other degree or diploma in any university.

Human Ethics: The research presented and reported in this thesis was conducted in accordance with the National Health and Medical Research Council National Statement on Ethical Conduct in Human Research (2007) – updated March 2014. The research study received ethics approval from the Curtin University Human Research Ethics Committee (EC00262), Approval Number #HR23/2015 and the South Metropolitan Health Service Human Research Ethics Committee (EC00265), Approval Number 14/79.

Signature: 

Date: 20 October 2017

Statement of Author Contributions

The candidate, Robert Schütze, was responsible for all aspects of the research presented in this thesis, including study design, data collection, data analysis, interpretation, and reporting of results. The following supervision team also contributed to the research design and some aspects of analysis, interpretation and writing/editing:

- Professor Clare Rees (Principal Supervisor) [i]
- Associate Professor Helen Slater (Co-Supervisor) [ii]
- Associate Professor Anne Smith (Associate Supervisor) [ii]
- Professor Peter O'Sullivan (Associate Supervisor) [ii]

In addition, Dr Jared Campbell [iii] contributed to analysis of data for Study 2 by providing an independent risk of bias rating of included studies.

Dr Mark Catley [iv] contributed to data analysis, interpretation and writing for Study 3 by performing the Rasch analysis of item functioning.

Further details of author contributions to each study are reported in the methods sections of the individual studies.

…… Date: 20 October 2017

Robert Schütze (Candidate)

…… Date: 20 October 2017

Professor Clare Rees (Principal Supervisor)

[i] School of Psychology and Speech Pathology, Curtin University, Perth, Australia.
[ii] School of Physiotherapy and Exercise Science, Curtin University, Perth, Australia.
[iii] Faculty of Science and Engineering, Macquarie University, Sydney, Australia.
[iv] School of Health Sciences, University of South Australia, Adelaide, Australia.

Abstract

Background: Pain is a multidimensional subjective experience involving complex interactions between biological, psychological, genetic and social factors, as described by the dominant biopsychosocial model of pain. Pain catastrophising (PC), defined as an exaggerated negative cognitive-affective orientation toward pain, is one of the strongest psychological predictors of negative pain outcomes. However, the exact nature of PC and therefore how best to reduce it remains unclear, with various treatments showing efficacy. While the dominant cognitive-behavioural approaches focus on changing unhelpful catastrophic beliefs, the ruminative aspect of PC has received less attention. Self-regulatory models of rumination and worry, such as the metacognitive model, have been successfully applied to various emotional disorders but remain relatively nascent in the field of pain-related distress.

Objective: To explore the ruminative aspect of PC and develop a metacognitive model of pain-related perseverative thinking. There were four specific aims related to this:

1. Systematically review the PC intervention literature and compare pooled effect sizes of different interventions.
2. Explore the lived experience of worry and rumination among people with chronic non-cancer pain and elevated PC, including their metacognitive beliefs about pain-related thinking.
3. Develop and validate a self-report measure of pain-related metacognition suitable for research and clinical settings.
4. Develop and test a theoretical model of pain-related metacognition.

Methods: Using both qualitative and quantitative methods, four linked studies addressed these aims.

1. *Study 1:* Using a registered protocol, a systematic review of all randomised controlled trials measuring treatment-related changes in PC among adults with chronic non-cancer pain was conducted. Cochrane methodology was used to search electronic databases, assess risk of bias, and rate evidence quality according to Grading of Recommendations, Assessment, Development and

Evaluations (GRADE) criteria. Pooled effects of different interventions were compared using random-effects meta-analyses and the impact of moderators was assessed using meta-regression and sub-group analyses.

2. *Study 2:* In a qualitative study exploring metacognitive beliefs about pain-related thinking, semi-structured interviews were conducted in a tertiary care setting with 15 adults experiencing chronic (≥6 months) low back pain and very high PC (≥30 on the Pain Catastrophising Scale). Transcripts were analysed using interpretative phenomenological analysis.
3. *Study 3:* A self-report measure of metacognitive beliefs underlying perseverative thinking about pain was developed and validated. Items were generated from the results of Study 2 and metacognitive theory. A large internet sample (N=864) of people experiencing pain completed the draft scale and a battery of validation measures. Rasch analysis was used to assess item functioning and refine the scale. A second internet sample (N=510) completed the revised scale to confirm psychometric properties. Correlation and multiple regression was used to establish construct validity, while receiver operator characteristic curve (ROC) analysis was used to identify possible cut-offs.
4. *Study 4:* A preliminary model of how metacognition relates to other important pain variables was tested using the second validation sample from Study 3. Regression-based moderated-mediation analysis tested the conditional indirect effect of pain intensity on PC via rumination at varying levels of unhelpful pain metacognition.

Results:

1. *Study 1:* Seventy-nine studies (9914 participants) were included and meta-analyses showed at least nine interventions demonstrated efficacy, although evidence quality was often low. The best evidence (moderate-high quality) was found for Cognitive Behaviour Therapy (CBT), multimodal treatment (combining exercise and CBT), and Acceptance and Commitment Therapy (ACT). Effects were often inconsistent, were generally of medium strength, and had questionable clinical significance. When only studies targeting people with high PC were included, effects were larger and more consistent.
2. *Study 2:* People with elevated PC described both positive (e.g. ‘thinking helps me to cope’) and negative (e.g. ‘worry is uncontrollable’) attitudes towards pain rumination. These were often held simultaneously, creating internal

conflict. Both negative and positive metacognitions could fuel perseverative thinking. More nuanced negative metacognitions (e.g. 'worry is pointless') could help to end episodes of worry/rumination by motivating the use of concrete problem solving or active coping behaviours.

3. *Study 3:* A psychometrically sound 21-item scale – the Pain Metacognitions Questionnaire (PMQ) – was created that had two dimensions (positive and negative metacognition) assessing how useful and how uncontrollable or damaging people believed pain rumination to be. Both subscales had good retest reliability (r = .76, r = .72) and internal consistency (.86, .87). They correlated negatively with a measure of mindfulness and positively with measures of pain intensity, disability, anxiety, depression, PC, rumination, and metacognition, providing good evidence of construct validity. The PMQ also predicted unique variance in PC when other variables were controlled, and predicted clinical levels of PC.
4. *Study 4:* Rumination partially mediated the effect of pain intensity on PC, accounting for 20% of the total effect. This indirect effect was conditional on both positive and negative metacognition. Higher levels of both forms of unhelpful metacognition strengthened the indirect effect. This suggests that both strongly believing that thinking about pain helps one to solve problems or cope with pain (positive metacognition), or that rumination is harmful and uncontrollable (negative metacognition) increases the amount one ruminates as pain increases, which is associated with increased PC.

Conclusions:

There is no clear gold standard for treating PC, although CBT, ACT and multimodal treatments have the best evidence. Improving interventions for PC is likely to involve better patient-treatment matching, including identifying different clinical profiles of catastrophising. For people with a strong tendency towards repetitive negative thinking about pain, identifying and modifying underlying unhelpful metacognitions may be useful. This research shows that metacognition is relevant to worry and rumination about pain and that it can be reliably measured with a new self-report scale. Future research investigating metacognitively-informed treatments for people with high PC is warranted.

Acknowledgements

Firstly, a big thank you to my wonderful supervisors – Professor Clare Rees, Associate Professor Helen Slater, Associate Professor Anne Smith and Professor Peter O'Sullivan – an all-star cast who steered me expertly through treacherous seas. Our meetings were always full of laughter and creativity. Thank you also to Dr Mark Catley, who generously shared his Rasch wizardry for the scale development study, and Dr Jared Campbell for helping with the systematic review. Many thanks also to Dr Mark Boyes for providing valuable feedback on the draft thesis. I am grateful to Diana Blackwood for advising me on the systematic review search strategy and Professor Michael Sullivan for suggestions on interpreting clinically significant changes in pain catastrophising.

None of the ensuing pages could have been written without the generous gifts of time and energy from the many participants living with pain, whose heartfelt stories and Likert ratings provided me with the raw materials from which to craft this thesis. A big thank you. I am also grateful to the numerous people and organisations who helped connect me with these participants: Fremantle Hospital Orthopaedic Clinic and Pre-Admission Clinic (Professor Piers Yates, Joanne Morrish, Jennifer Harvey, Associate Professor Stephanie Davies, and Michelle Fulwood); Sir Charles Gairdner Hospital Department of Pain Management (Associate Professor Roger Goucke, Dr Mark Schütze, and Jane Royle); Neurospine and Brain (Dr Andrew Miles and Kate Moore); Chronic Pain Australia; Australian Pain Management Association; Pain Australia; the Australian Pain Society; Psoriasis and Psoriatic Arthritis Alliance; John H. Krantz; Lorimer Moseley; and Tim Minchin.

I was fortunate to not be homeless and hungry over the last three years thanks to the generous support of Spinnaker Health Research Foundation (formerly Fremantle Hospital Research Foundation) in the form of a Bellberry Medical Research Scholarship. Thank you.

Finally, I was lucky enough to enjoy the unending patience and support of my family during this somewhat self-indulgent intellectual journey. Thank you to my parents, Joan and Udo, for the cheerleading and childcare, and my brother Mark for helping kindle my interest in pain. My sons, Jamie and Jesse, have kept me

nourished with their endless affection and joyful spirits, for which I feel eternally blessed. Finally, I am hugely indebted to my wonderful wife, Jean, an endless source inspiration, altruism, love, and very practical support. She is truly the constitutive ground of my being. Thank you for the sacrifices that have made this thesis possible.

Table of Contents

List of Figures

List of Tables

List of Abbreviations

ACT	Acceptance and Commitment Therapy
ATT	Attention Training Technique
BPI	Brief Pain Inventory
CAS	Cognitive Attentional Syndrome
CBT	Cognitive Behaviour Therapy
CCM	Communal Coping Model
CEQ	Cognitive Errors Questionnaire
CLBP	Chronic Low Back Pain
COREQ	Consolidated Criteria for Reporting Qualitative Research
CSM	Common Sense Model
CSQ	Coping Strategies Questionnaire
CTT	Classical Test Theory
ECIP	Experience of Cognitive Intrusion in Pain
EFT	Emotional Freedom Techniques
FA	Fear-avoidance
GRADE	Grading of Recommendations Assessment, Development and Evaluation
HADS	Hospital Anxiety Depression Scale
IRT	Item Response Theory
MAAS	Mindful Attention Awareness Scale
MBCT	Mindfulness-Based Cognitive Therapy
MBSR	Mindfulness-Based Stress Reduction
MCM	Metacognitive Model of chronic pain
MCQ	Metacognitions Questionnaire
PC	Pain Catastrophising
PCCL	Pain Coping and Cognition List
PCL	Pain Cognition List
PCS	Pain Catastrophising Scale
PMQ	Pain Metacognitions Questionnaire
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
PRSS	Pain-Related Self-Statements Scale
PTQ	Perseverative Thinking Questionnaire
RNT	Repetitive Negative Thinking
SMD	Standardised Mean Difference
S-REF	Self-Regulatory Executive Function
TCQ	Thought Control Questionnaire
TSK	Tampa Scale of Kinesiophobia
VPCQ	Vaginal Penetration Cognition Questionnaire

Chapter 1 Introduction – Re-thinking over-thinking pain: What can metacognition add to our understanding of pain catastrophising?

Publication History

A shortened version of this chapter was published in the journal, *Clinical Psychologist* and is reproduced in Appendix K:

Schütze, R. (2016). Re-thinking over-thinking pain: What can metacognition add to our understanding of pain catastrophising? *Clinical Psychologist*, *20*, 147–153. http://doi.org/10.1111/cp.12105

1.1 The Problem of Pain

Pain is a multidimensional subjective experience involving complex interactions between biological, psychological, genetic and social factors, as described by the dominant biopsychosocial model of pain. [1] Pain is usually defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage," [2(p210)] although definitions incorporating motivational and contextual factors have also been proposed. [3] Pain is the most common reason for seeking medical help [4] and chronic pain – or daily pain that persists beyond the usual 3-6 month course of healing [2] – affects one in five people in Australia. [5] Chronic pain costs the economy $34-55 billion each year in treatment expenses, lost productivity, and overall burden of disease, making it the country's third most costly health problem. [6,7] In the United States, the incidence and impact of chronic pain are even higher, costing up to $635 billion annually. [8] Chronic pain is strongly associated with poor self-rated health, [9,10] lower quality of life, [11] and psychopathology, [12] leading to calls for pain to become a national health priority in Australia, [13] particularly since recent projections suggest arthritis and other musculoskeletal conditions will affect 30.2% of the population by 2032. [7]

Access to effective treatment for chronic pain is generally poor, with some studies suggesting less than 10 per cent of people receive adequate care. [14] One of the key barriers to more widespread provision of effective treatment for chronic pain is the fact that it is typically poorly understood by the community, policy makers, and health professionals themselves. [15–17] For example, chronic pain is often still viewed within a biomedical model that posits tissue damage or underlying pathology as the main driver of pain symptomatology and associated disability, rather than seeing chronic pain as a disease entity of its own. [18] Chronic pain is more usefully viewed in terms of maladaptive nervous system changes rather than merely as a symptom of unresolved injury or disease. [19–21] As a corollary, purely biomedical interventions for chronic pain, such as medication, surgery, and regional anaesthesia, generally have limited efficacy and significant side-effects, which is why multidisciplinary treatment based on a biopsychosocial approach is the gold standard for chronic pain management. [13,22,23]

1.2 Psychological Aspects of Pain

Approaching chronic pain as a disease in its own right posits psychological variables as important predisposing and maintaining factors, in addition to explaining the important role of psychological and self-management interventions (e.g. [24–27]) in the evidence-based treatment of various chronic pain disorders. [28] For example, a vast body of empirical evidence highlights how psychological factors such as neuroticism, premorbid depression, anxiety, avoidant coping style, substance abuse, trauma, and psychiatric history increase a person's vulnerability to developing chronic pain. [29–31] Similarly, the punishing experience of unresolved pain often gives rise to significant mental health problems, most commonly clinical depression, which affects 30-54% of people treated in specialist pain clinics. [32,33] This is markedly higher than depression's 12-month prevalence of 4.1% in the general population in Australia [34] and 6.9% in the United States. [35] Anxiety disorders, such as generalised anxiety disorder and post-traumatic stress disorder, are also commonly comorbid with chronic pain, as are substance use disorders. [36,37]

Although people with chronic pain therefore have higher rates of psychopathology than the general population, clinically significant symptoms are not the norm. For example, a review of studies measuring the prevalence of comorbid pain and anxiety disorders found that only 7 to 28.8% of people with chronic pain also have an anxiety disorder. [38] Even the estimates for depression in tertiary pain clinics cited above show that depression is by no means ubiquitous among people with chronic pain. This suggests that, despite the focus in this thesis on a specific form of pain-related psychological distress, the majority of people with pain cope well enough to not experience psychopathology. Nevertheless, it is well documented that people with pain face a diverse range of challenges. These include: often having no clear medical diagnosis to explain and validate their symptoms; feeling as if family, friends, colleagues and health professionals do not believe them; having limited and delayed access to effective evidence-based treatment; and experiencing increased financial burdens associated with reduced productivity and increased health costs. [13] These are experienced uniquely by different individuals,

with many coping adaptively. However, for a significant minority of people these challenges can result in a maladaptive cycle of increasing distress and pain.

As will be discussed below, the psychological sequelae of chronic pain have been explained in terms of cognitive, behavioural and environmental changes linked to heightened perception of threat, functional losses and pain-related neuro-endocrine changes known to influence mood. [39] Most importantly, these psychological consequences reciprocally impact a pain experience, possibly via mechanisms involving areas associated with perception, [20] and can contribute to the maintenance of pain through neuroplastic reorganisation of the central nervous system. This can contribute to nociceptive sensitisation [40] and attenuation of endogenous nociceptive inhibitory processes. [41,42] Thus psychological factors at the level of cognition, affect and behaviour play an important and complex role in the development and maintenance of chronic pain. [43]

1.2.1 Dualism and Psychogenesis

Current manifestations of the biopsychosocial model of pain evolved through a rejection of 'specificity theory', which posited a strict mechanistic relationship between tissue damage and pain that was rooted in Descartes' mind-body dualism. [44] The cracks in specificity theory began showing with accounts of soldiers experiencing minimal pain in the presence of extensive injuries on the battlefields of World War II. [45] Meanwhile, at the other end of the biomedical spectrum, Austrian Neurologist Sigmund Freud started writing in the late 19th century about patients who reported somatic symptoms, including pain, with no organic basis. [46] The ensuing psychoanalytic theory he developed, which shaped the foundations of modern psychology, described such pain in terms of 'hysteria', whereby unresolved unconscious conflicts manifest physically through what he called 'conversion'. [47] This view influenced clinical pain science such that notions of somatisation and 'psychogenic pain' entered the diagnostic lexicon. [48] From this perspective, pain in the absence of organic pathology serves a psychological function, for example as a form of atonement for excessive guilt, or a proxy for otherwise thwarted psychological forces like aggression, grief, and sexual drive. [48]

This psychogenic view of medically unexplained pain has been clinically influential, as reflected in the inclusion of Somatoform Pain Disorder in earlier versions of the American Psychiatric Association's diagnostic manual of mental disorders. [49] However, the diagnosis was heavily criticised [50–53] and has been removed from the latest version of this manual, the DSM-5. [54] Moreover, despite its heuristic appeal for some clinicians, this model has no empirical validity. [55] For example, a systematic review of 120 pain studies using measures of somatisation found no empirical evidence to support the most common definition of somatisation [56] as a tendency to experience and communicate distressing medically unexplained symptoms while attributing them to a physical illness. [57] Similarly, there is no evidence that psychodynamic treatments associated with a psychogenic view of pain have any efficacy in treating chronic pain conditions. [39] Moreover, raising the spectre of iatrogenesis, the psychogenic view has been criticised as stigmatising an already delegitimised group, [58] with people in pain reporting that when they perceive health professionals holding this view of pain, it can impede their access to effective treatment and contribute to worse mental health outcomes. [59]

1.2.2 The Fear-Avoidance Model of Pain

Contemporary biopsychosocial models are less stigmatising in that they frame pain-related distress and disability within a 'normal psychology' of pain, which focuses on dimensional traits that are present in the general population but become exaggerated in some people living with pain. [60,61] An example of a biopsychosocial approach that accounts for psychological factors without the simplistic psychological reductionism of somatisation is the fear-avoidance (FA) model. [62] Based on the work of Lethem and colleagues, [63] this cognitive-behavioural model was developed to explain the transition from acute to chronic low back pain [64] and has many parallels with avoidance models of phobia and anxiety disorders. [65] The FA model has been extensively researched, predominantly in musculoskeletal pain such as low back pain, whiplash and fibromyalgia, [64,66,67] and has significant heuristic value in the prediction and treatment of people with chronic pain through exposure-based cognitive behavioural interventions. [68–70]

The FA model is presented in Figure 1.1, with the clockwise path on the left showing the onset and maintenance of chronic pain as characterised by a vicious cycle of increasing pain and disability as pain-related fear develops. According to the model, pain experiences in some people lead to negative cognitions that constitute pain catastrophising. This cognitive process gives rise to pain-related fear, which in turn elicits hypervigilance to pain stimuli and avoidance of daily activities that are expected to produce pain. These avoidance behaviours are further reinforced through a reduction in fear, and tend to persist because the lack of exposure to the consequences of feared activity means there is little opportunity to correct maladaptive beliefs about pain. According to the model, avoidance of daily activities leads to increasing functional disability and mood disturbances such as depression, which results when positive environmental contingencies associated with meaningful activity are removed. [71]

The key link in this vicious cycle is the resultant maintenance of pain due to deconditioning, disablement and depression. [71] These secondary effects of pain-related fear may even exacerbate the perceived pain by lowering a person's pain threshold. [68] By contrast, when acute pain is not perceived as threatening, the maintenance of normal activities (i.e., confrontation) often promotes recovery, as depicted by the anti-clockwise path in Figure 1.1. Therefore, according to the FA-model, the way pain is approached cognitively plays a crucial role in determining whether it will develop into a cycle of chronicity or gradually recede alongside normal healing.

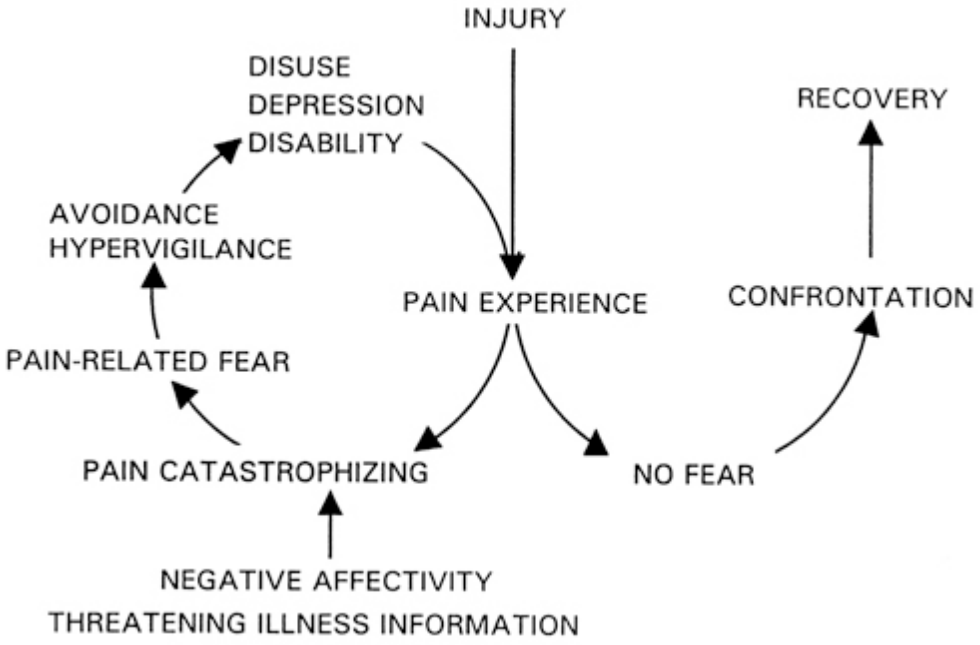


Figure 1.1. Fear-avoidance model of chronic pain. [62]

The FA model continues to be challenged and refined through empirical research. [70] For example, variations of the original model have emerged to account for factors such as: the mediating role of pain-related anxiety in eliciting avoidance behaviour [72]; the moderating role of mindfulness on catastrophising [73]; the effect of physiological activity, such as autonomic nervous system dysregulation, on pain expectancy [74]; and the effect of multisensory processing on associative learning in the conditioning of fear and pain. [75] More recent reviews of the model have called for future research to address motivational aspects of FA behaviour, particularly in better articulating how functional recovery occurs. [69] Responding to this, recent research has highlighted that there are multiple pathways out of pain-related fear, which involve making sense of pain and gaining control of the pain experience somatically, psychologically or both. [76,77]

1.3 Pain Catastrophising

Despite these refinements to the FA model, the role of cognition for pathways into and out of pain-related fear and disability remains central. The key cognitive variable in this model – pain catastrophising (PC) – is one of the most widely studied psychological variables in pain science, largely because of its strong

relationship with negative pain and health outcomes. [78] Broadly defined, PC can be considered a negative cognitive response to pain and has various dimensions, including a tendency to ruminate about the pain experience, exaggerate its threat value and underestimate one's ability to cope with it. [79] It has been defined as "an exaggerated negative mental set brought to bear during actual or anticipated painful experience". [79(p52)]

The broader concept of catastrophising has its roots in the cognitive psychology tradition of Albert Ellis [80] and Aaron Beck, [81] who separately developed psychological interventions for identifying and challenging unhelpful beliefs through evidence-testing. The application of this concept to pain began around the 1980s with three groups of researchers exploring how people cope with pain. [82] Their findings were integrated in the development of the now dominant measure of PC, the Pain Catastrophising Scale (PCS) [83] and are reflected in its three facets: rumination, [84] magnification, [85] and helplessness. [86] However, the incorporation of catastrophising into the FA model emphasised the magnification aspect of PC, describing it as "negative appraisals about pain and its consequences" [62(p319)], or "catastrophic misinterpretations of pain" [87(p1)]. This formulation focuses on the *contents* of cognition, which is associated with so-called 'second wave' psychological treatments like Cognitive Therapy, [88] rather than the *process* aspects of cognition, such as its perseverative nature in rumination and worry, which is the focus of 'third wave' psychological therapies. [89] This is discussed below in more detail.

1.3.1 Catastrophising and pain outcomes

A large body of research into the FA model and other aspects of pain now links PC to a range of negative pain outcomes. Elevated PC is associated with greater pain intensity, [79] disability, [90,91] depression, [92] anxiety, [93] work absenteeism, [94] opioid misuse, [95] and health-care utilisation. [96] As hypothesised in the FA model, empirical evidence also shows PC is associated with the transition to chronicity and its maintenance [97]. For example, a Dutch population-based cohort study of 1,571 people found that high levels of PC predicted back pain 6 months later both

for people with and those without back pain at baseline and the effect was stronger for severe back pain and pain with disability. [98]

In perioperative settings, PC has become an important predictor of pain and function following surgery, particularly joint replacement. [99] For example, in people undergoing total knee replacement, PC is the most significant psychological predictor of persistent post-surgical pain, [100–103] more so than depression, fear of movement, anxiety, or low self-efficacy. [104] One study attempting to quantify the influence of PC on pain following knee replacement estimated a mean effect size of Cohen's *d*=0.75 for the prediction of acute post-operative pain, and *d*=0.73 in predicting the development of persistent post-surgical pain, [105] both of which are medium to large effects. [106] These findings strongly point towards the need to target PC as a modifiable risk factor, particularly since the number of TKR surgeries for knee arthritis are rapidly increasing. However, up to 50 percent of patients are dissatisfied with their surgery [107] and up to 53 percent develop persistent post-surgical pain [108] despite good procedural outcomes. [109] Furthermore, TKR is a risk factor for chronic opioid use, leading to calls for psychobehavioural interventions, including those that target PC, to augment traditional analgesia in improving postsurgical outcomes. [110]

In treatment settings, PC is also an important process variable that mediates improvements during interventions such as Cognitive Behaviour Therapy, [111,112] Acceptance and Commitment Therapy, [113] exercise-based rehabilitation, [114] and multidisciplinary treatment. [115–117] Some studies using cross-lagged designs show that improvements in PC early in treatment predict later improvements in pain and disability. [115,117] A recent systematic review found that PC mediates treatment efficacy in a range of interventions for non-specific chronic low back pain (CLBP). [118] As a result, elevated PC has become a key treatment target, particularly in psychological and multidisciplinary interventions for people with chronic non-cancer pain. Research has tended to focus on musculoskeletal pain, including chronic low back pain (CLBP), [119] neck pain, [120] and osteoarthritis,[121] as well as fibromyalgia, [122] perioperative pain, [123] and more recently neuropathic pain. [117]

1.3.2 Reducing Pain Catastrophising

Several high quality systematic reviews attest to the efficacy of psychological treatments for improving various outcomes for people with chronic pain, including pain intensity, functional disability and emotional distress. [120,124,125] However, the effect sizes found in these reviews have weakened over time, with early reviews showing medium effects for CBT on pain, disability and emotional functioning, [125] while the latest review suggests only small effects. [120] It has been proposed that this decline reflects an improvement in trial design, while the quality of treatment has remained relatively unchanged. [120] Dominant CBT protocols are often a 'pragmatic mix' of components – from cognitive restructuring, to behavioural activation, activity-rest cycling, relaxation training, communication training and the use of positive coping statements – which rest on a very broad theoretical framework. [120] There is now a strong quorum of researchers advocating for more theory-driven research that explores treatment mechanisms and tests new interventions against active controls rather than waitlist, [28,126–128] echoing developments in the psychotherapy literature more broadly. [129,130] Moreover, improving the efficacy and efficiency of pain psychotherapy requires better patient-treatment matching models so that specific treatment components can be matched to individual strengths and needs. [131,132]

The same limitations found in the general pain psychotherapy literature exist in PC intervention research. Firstly, it is far from clear how best to reduce elevated PC, since a range of different interventions produce benefit. On one hand, there seems to be most evidence for CBT, with the only meta-analytic data on PC interventions coming from the latest Cochrane review of psychological therapies for chronic pain. [120] This showed that CBT reduces PC with a medium standardised mean difference (SMD) effect of -0.53 compared to waitlist at post-test. [120] However, in a high quality head-to-head trial comparing CBT, exercise (general aerobic and strength training), and multidisciplinary treatment combining CBT and exercise, all three interventions showed similar effects of moderate strength. [114] This is surprising given that exercise does not explicitly target unhelpful thinking processes. More recently, emerging so-called third wave psychological therapies such as ACT and mindfulness meditation have also shown efficacy for reducing PC, [133,134] with some suggesting large effect sizes. [135] A recent head-to-head

comparison of CBT and mindfulness meditation in people with CLBP showed both were efficacious, with mindfulness slightly superior in reducing PC in the short term. [136] While these data suggest that there are a range of different ways to reduce PC, there is no clearly superior intervention and the mechanisms that underpin this change remain unclear.

One explanation for this uncertainty might be that the construct of PC itself requires further clarification. Indeed, some have argued that PC is nothing more than psychological distress that is better captured with measures of anxiety and depression, although empirical evidence suggests otherwise. [79,137] Similarly, although catastrophising among people with generalised anxiety has been measured in structured clinical interviews in psychopathology research, [138] PC is only measured with self-report questionnaires and there are at least six of these, all capturing slightly different aspects of the construct. These include: the Pain Catastrophising Scale [83]; the Coping Strategies Questionnaire [86]; the Pain-Related Self-Statements scale [139]; the Cognitive Errors Questionnaire [140]; the Vaginal Penetration Cognition Questionnaire [141]; and the Pain Cognition List. [142] Moreover, there are several theoretical models of PC emphasising different aspects of the construct. Therefore, heeding calls for a greater focus on theory and treatment mechanisms in refining psychological interventions for chronic pain, there is a strong case for further exploring the construct of PC in order to better target it in treatment.

1.3.3 Theoretical Models of Pain Catastrophising

Early theoretical models of PC were proposed by the research group of Michael Sullivan, who developed the Pain Catastrophising Scale. [83] As noted above, selection of items for this scale drew on three experimental studies emphasising different aspects of the construct. [84–86] In developing and validating the PCS, Sullivan and colleagues [83] analysed the reported thought content of people scoring highly on this measure, finding that people high on PC tended to anticipate and exaggerate possible negative pain outcomes, dwell on the aversive features of their pain experience, and focus on their inability to cope with this experience. This echoes research into catastrophising among chronic worriers in the

psychopathology literature. [138] This operationalised PC as a cognitive process involving three distinct facets – magnification, rumination and helplessness – with rumination explaining the most variance. [83,143] Numerous studies validating the PCS have confirmed these three stable factors and found the PCS to have good reliability and construct validity, [144–147] including being non-redundant when controlling for anxiety and depression. [93] This tripartite account of PC paved the way for more theoretical discussion about its nature and role in a pain experience. [79]

1.3.3.1 Appraisal model

Perhaps the most widely accepted way to operationalise PC, the appraisal model is based on Lazarus and Folkman's [148] transactional model of stress. This model explains responses to pain in terms of primary appraisals concerning the relevance and positivity/negativity of potential stressors, along with secondary appraisals of one's coping abilities. [78] Severeijns and colleagues [149] suggest that the magnification subscale of the PCS relates to primary appraisals of threat in PC, while the helplessness subscale reflects secondary appraisals. This aspect of secondary appraisals is supported by a significant amount of empirical evidence showing an inverse relationship between PC and self-efficacy [93] and echoes findings that catastrophising amongst worriers involves exaggerated incompetency beliefs, particularly around coping ability. [150]

The appraisal model is most closely linked to the cognitive theory origins of catastrophising in the psychotherapy literature. [80,81] This model focuses on beliefs, or the contents of cognition, suggesting the beliefs associated with PC are "catastrophic misinterpretations". [87] The goal of interventions based on this model are therefore to identify and replace these negative appraisals with more realistic and helpful ones. A detailed protocol has been developed for applying traditional Cognitive Therapy techniques aimed at cognitive restructuring to elevated PC in people with chronic pain. [151] Cognitive restructuring is also a cornerstone of 'From Catastrophising to Recovery', [152] one of the few interventions that specifically focuses on PC rather than including it amongst a broad range of outcomes as often occurs in psychological and multidisciplinary pain interventions.

As noted earlier, an appraisal model also underlies how PC is traditionally formulated within the FA model. As well as promoting habituation to feared stimuli, the exposure interventions stemming from the FA model are intended to aid cognitive restructuring by providing disconfirmatory evidence of catastrophic predictions. There is evidence that such cognitive restructuring occurs for people undergoing pain-related exposure therapy, [153] much as behavioural experiments are effective cognitive interventions for a range of psychological problems. [154] However, key FA model proponents have more recently cautioned against viewing PC as merely irrational thinking, given that many of the erroneous pain beliefs underlying fear-avoidance behaviour in people with chronic pain are common in the general population and even healthcare providers. [69] This suggests there is more to PC than faulty appraisals.

1.3.3.2 Attentional bias model

The information processing approach to understanding PC that is evident in the appraisal model is also evident in the attentional bias model of PC. This posits that people with exaggerated PC have an attentional bias towards sensory and affective aspects of pain, which facilitates pain perception and causes cognitive interference. [155] Whilst intuitively appealing, particularly given the mounting evidence for attentional bias as a key maintaining factor of anxiety disorders, [156] the current evidence for this model is mixed. The most recent high quality review and meta-analysis found that the effect size of attentional bias towards pain-related words and images found in chronic pain patients is small and similar to that found in healthy controls. [157] More importantly, individual differences in anxiety, depression, fear, or pain intensity are not associated with more attentional bias. Nonetheless, there has been some notable evidence that elevated PC is associated with delayed disengagement from threat cues, [158] supporting the idea that PC produces attentional bias or hypervigilance, as predicted in the FA model. [71] However, the mixed findings around attentional bias leave this explanatory model unconvincing as a comprehensive theory of PC, although it may well be a feature associated with the negative cognitive and affective responses to pain that putatively constitute PC. This field of inquiry is evolving, however, and may well

shed further light on PC once the problems with poor reliability in experimental models that investigate attentional bias [159] are overcome.

1.3.3.3 Communal coping model

In another attempt to explain why some people show heightened negative psychological responses to pain, Sullivan and colleagues [160] proposed the communal coping model (CCM), which frames PC as an interpersonal coping strategy. The CCM suggests that exaggerated pain expressions associated with PC serve a social-communicative function. For those who adopt social and relational orientations in the face of stress, PC may serve as a way to elicit empathy and support from the social environment. [161] This hypothesis is supported by evidence that people with elevated PC exhibit more observable pain behaviours. [162] Furthermore, some studies show that the partners of these people behave in supportive ways in the face of such pain behaviours. [163,164] However, there is also evidence that this coping style is associated with more punishing or critical responses from partners, [165,166] suggesting it often backfires.

Perhaps the strongest evidence for the CCM comes from a cross-lagged partner study of 105 couples completing daily diaries of various pain outcomes. [167] Results confirmed the main tenets of the CCM, showing that patient PC is noticeable to spouses and in turn triggers a response, rather than PC emanating from spouse behaviour. Interestingly, both positive and negative spouse responses were observed at the onset of patient PC. [167] However, these responses became increasingly positive over the course of the day, suggesting that the putative goal of eliciting social support through PC is eventually achieved. [167] Similarly, a recent study of 510 female university students found PC was associated with increased solicitous partner responses and vulvovaginal pain, both cross-sectionally and prospectively. [168]

These studies highlight a potential mechanism through which PC is reinforced (i.e. operant conditioning), suggesting interventions for PC should focus on environmental factors in addition to intrapsychic ones. Nevertheless, the CCM has been criticised by some commentators as redundant and confusing, conflating what is essentially a cognitive process with its interpersonal correlates. [149]

However, this argument constructs a false dichotomy between intrapsychic and interpersonal factors, failing to acknowledge that even if the emergence of catastrophic beliefs is cognitively accounted for by an appraisal model, behavioural theory [169] suggests that behavioural and social sequelae will shape their future recurrence.

1.3.3.4 Neurobiological accounts

Another attempt to explain the aetiology and maintenance of PC has taken a neurobiological approach, investigating the various physiological, neurological and genetic correlates of PC. For example, PC has been shown to be associated with dysregulation of the hypothalamic-pituitary axis, including increased levels of cortisol, which is in turn linked to increased pro-inflammatory cytokine activity related to central nervous system sensitisation. [170,171] Similarly, descending inhibitory control of nociception through endogenous opioid pathways is attenuated during induced experimental pain states involving elevated PC. [172] There is also evidence that the brain is differently activated when people are catastrophising, such that areas involved in processing the affective components of nociception are more active, including the dorsolateral prefrontal cortex, anterior cingulate cortex and amygdala. [173] Pain rumination, which is a key aspect of PC, is also associated with nociception-facilitating changes in functional connectivity within the brain's default mode network.[174]

Finally, the question of whether trait differences in PC are due to genetic factors has been explored. A recent twin study investigating experimental pain estimated that around 37% of the variance in PC is heritable, while the remaining 63% is attributable to environmental factors. [175] While there is no clear genetic marker for PC, there is evidence of an epigenetic effect whereby the interaction of PC with specific haplotypes of the catechol-O-methyltransferase (COMT) gene may play a key role in varying levels of pain sensitivity. [176]

These findings are valuable in suggesting possible neurophysiological mechanisms by which PC is associated with increased pain and disability. However, to date there is no coherent theoretical framework to integrate these findings. [177] Just as the CCM was criticised for merely describing the interpersonal correlates of PC, a

neurobiological account seems to describe possible biological predisposing factors which in themselves are not sufficient causal factors for PC, as well as biological correlates of catastrophic thinking. However, it is likely that these correlates may be involved in the dynamic modulation of PC. For example, neuroplastic increases in functional connectivity in brain areas associated with rumination and nociception are likely to reinforce or facilitate the process over time. Similarly, recent evidence suggests a bidirectional relationship between pain intensity and PC, such that an increase in PC can cause pain to increase, while at the same time increases in pain intensity can cause increases in catastrophising. [117] There may therefore be multiple drivers of PC, including neurobiological ones, differentially operating at various clinical stages of pain disorders.

1.3.3.5 Repetitive negative thinking models

In the same way that the CCM posits a functional account of PC embedded in the social environment, more recent formulations postulate an implicit self-regulatory function for PC. For example, Flink and colleagues [178] suggest that PC is best viewed as a form of repetitive negative thinking whose function is to regulate negative affect. In particular, they suggest it is a form of 'covert avoidance' because the abstract nature of cognition involved in rumination and worry inhibits the activation of aversive images, sensations and emotions in the short term. [178] This is based on Borkovec's avoidance theory of worry, which has been influential in the anxiety literature. [179,180] Support for this theory comes from evidence that reversing avoidance through imaginal exposure tasks allows for the symptom-reducing processing of distressing images, memories and emotions, both in people with generalised anxiety and trauma-related anxiety. [181,182] The avoidance theory of worry and by extension PC, suggests that it is negatively reinforced through the relief of negative affect in the short term, although in the long term it maintains chronic distress because of delayed emotional processing. [178,180] Despite the theoretical plausibility of this model, to date there is no direct empirical evidence supporting its application to PC.

A related functional account of pain-related worry suggests it is ego-syntonic and therefore positively reinforced because people see it as a constructive form of problem solving. [178] Certainly there is evidence that some forms of worry are

normal and adaptive, leading to the resolution of certain types of problems. [183] In the anxiety literature, such normal worry is distinguished from 'pathological worry', which is perseverative, intrusive and maladaptive in that it does not result in useful solutions. [138,150,184] Furthermore, worry is just one form of repetitive negative thinking (RNT), alongside other forms like depressive rumination, which has been characterised as perseverative, passive thinking about the causes and consequences of one's symptoms. [185,186] The abstract, iterative, and difficult to control nature of both worry and rumination mean they share structural similarities despite differences in their thematic content. [187] Indeed there is significant evidence suggesting RNT is a transdiagnostic process that underlies many emotional problems, including both anxiety and mood disorders. [188,189] A recent systematic review found that RNT prospectively predicts depression, anxiety and emotional distress, particularly in people with long-term mental health conditions. [190]

The role of PC within the FA model emphasises the future-based 'if-then' style of RNT (i.e. worry) that breeds anxiety or fear. However, there is good reason to view PC as RNT more generally, involving both anxiogenic worry and depressogenic rumination, given empirical evidence of its strong association with both anxiety and depression. [78] Furthermore, self-report items from common measures of PC capture both future-based worry (e.g. PCS: "I become afraid that the pain will get worse" [83]) and brooding on the negative meaning of symptoms that characterises rumination in depression (e.g. CSQ: "I feel my life isn't worth living" [86]). Perhaps even more importantly, some studies suggest rumination is the most important facet of the PCS. For example, initial validation of the PCS showed rumination explained 41% of its variance, compared to 10% for magnification and 8% for helplessness. [83] Similarly, in the recent development of a brief, daily version of the PCS, rumination items had the highest factor loadings onto the final unidimensional factor of the 3-item daily PCS. [191] Furthermore, in a study of 86 people with pain following motor vehicle accidents, the rumination subscale was the strongest predictor of pain and disability. [137] However there are mixed findings in this area and some data suggest helplessness may be a stronger predictor of outcomes. [192,193] Despite this, the ruminative aspect of PC warrants deeper investigation.

Cognitive-behavioural models frame rumination/worry as a strategy to address unattained goals and it might therefore be viewed as a form of problem-solving behaviour, although it is often unsuccessful [184,187,194]. This problem-solving account of worry has been applied to the pain experience in Eccleston and Crombez's 'misdirected problem solving model', as depicted in Figure 1.2. [195] This model suggests worry functions to orient people in pain toward threat and motivate them to find a solution to their problems. However, its authors argue that worry persists in a perseverance loop because the problem is poorly framed in terms of reducing pain from a biomedical perspective, rather than as a problem of how to live a valued life despite pain. [195,196]

At least one empirical study supports the main tenets of this model, although it found that worry mediated between biomedical problem framing and biomedical treatment seeking, rather than biomedical problem framing mediating between worry and biomedical treatment seeking, as predicted in the original model. [197] There is also evidence that worry related to pain is experienced as more distressing than other types of worry, and does not arise from a general disposition to worry. [198] This suggests that, despite associations with trait anxiety, [199] PC as a form of perseverative worry is contextually determined by a range of factors, rather than being purely dispositional. Consistent with the goal-discrepancy hypothesis found in cognitive models of RNT, [184,187,194] the misdirected problem-solving model of pain-related worry suggests that people who worry a lot about their pain would benefit from reframing their goals to make them more attainable if they are to attenuate the worry process. [195]

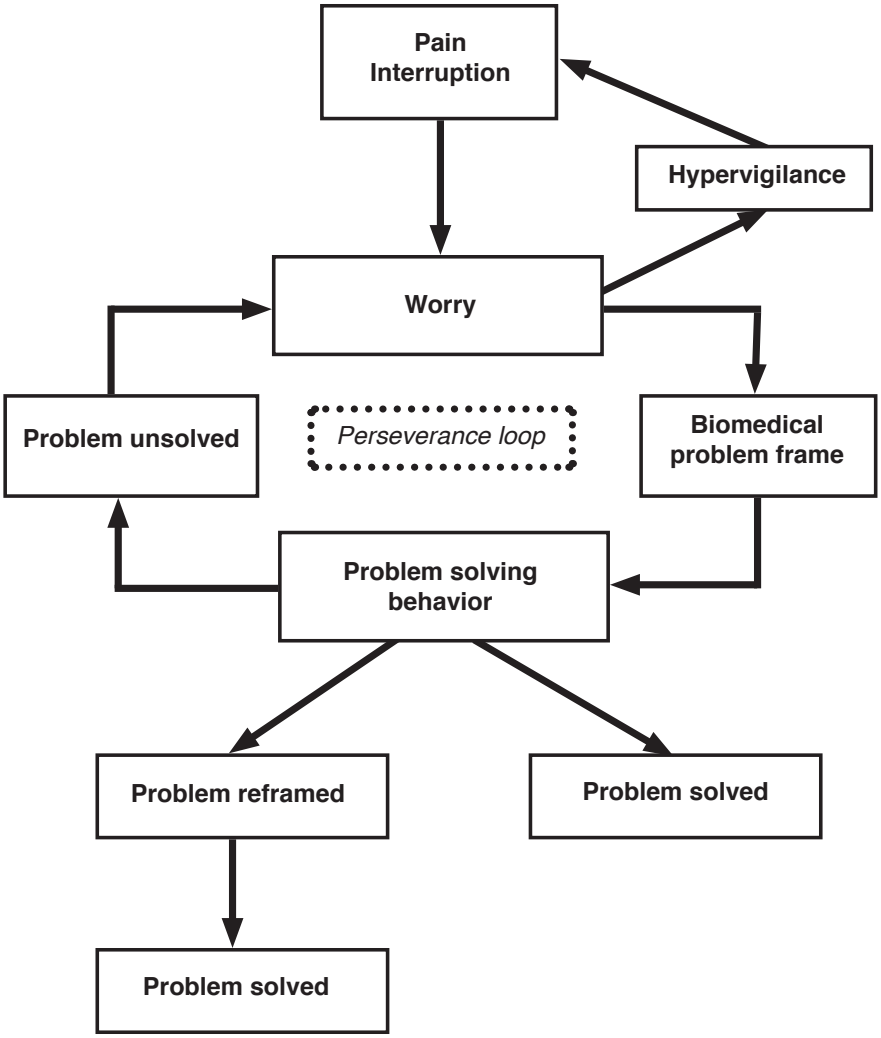


Figure 1.2. Misdirected problem solving model of pain-related worry. [195]

The functional model of PC described above focuses on the perseverative nature of catastrophic thinking about pain and suggests it has two main purposes: (1) to alleviate emotional distress by using abstract cognition to reduce contact with unpleasant images, sensations and emotions; and (2) to address threats to the self by orienting one towards threat and motivating problem solving behaviour aimed at removing these threats or achieving unresolved goals. This is reminiscent of other self-regulatory models of health behaviour, including the Common Sense Model (CSM). [200] This model describes how people with chronic illnesses try to make sense of their illness/symptoms, develop coping strategies to deal with the illness and its emotional effects, and then re-start this interpretation-action process based on how well the coping strategies work. [201] The CSM has recently been applied to fear-avoidance experiences of people with chronic back pain. [76] The

CSM articulates various pathways out of fear, [202] which was inadequately accounted for in the original FA model, as noted above. [69]

1.3.3.6 A Role for metacognition?

Another self-regulatory model that has particular pertinence to rumination and worry is the Self-Regulatory Executive Function (S-REF) model. [203,204] This information processing model suggests that the prolonged emotional distress characteristic of psychopathology stems from a maladaptive strategy for dealing with unwanted thoughts, emotions and sensations and that this strategy involves largely 'top-down' conscious processes that can be modified. [205] This unhelpful coping strategy, according to the S-REF model, is termed the 'cognitive attentional syndrome' (CAS), which is characterised by: (1) perseverative thinking in the form of worry, rumination and over-analysis; (2) attentional bias towards signals of threat, particularly internal cues such as unwanted thoughts, feelings or sensations; (3) coping behaviours that backfire and intensify distress rather than alleviate it. [205]

A key feature of this model is that once a threat is perceived, the CAS is activated and maintained by higher-order beliefs called metacognitions, which can be both positive and negative. For example, positive metacognitions ascribe a useful role to worry and rumination (e.g. "worrying helps me to solve problems"), and therefore function to increase the time and energy given to this style of cognition. [204,205] Once the ruminative process begins, negative metacognitions are activated. These are beliefs about the harmfulness (e.g. "worrying is dangerous for me") or uncontrollability (e.g. "when I start worrying, I cannot stop") of perseverative thinking, which in turn precipitate further RNT. This is because the negative outcomes of RNT themselves become threats that trigger further analysis, sometimes in the form of worry about worry ('meta-worry'). At the same time, uncontrollability metacognitions lead to people abandoning their attempts to control or disengage from RNT. Figure 1.3 depicts a metacognitive formulation of depression. A similar structure is found across other anxiety and mood problems, although disorder-specific versions have also been proposed. [206]

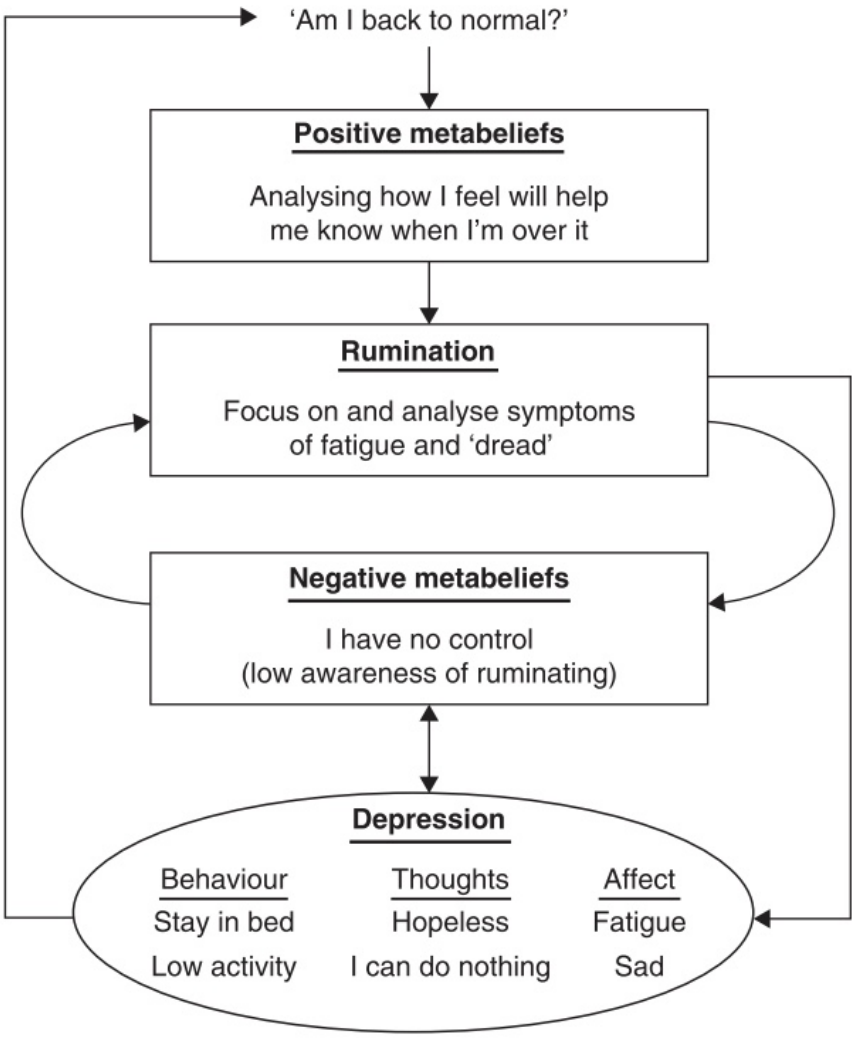


Figure 1.3. Metacognitive model of depression. [207]

The S-REF model forms the basis for Metacognitive Therapy (MCT), which focuses on dismantling the CAS using techniques such as: Socratic dialogue and behavioural experiments to challenge unhelpful metacognitions; attention training exercises to reduce hypervigilance and self-focused attention; and tasks that foster a state of 'detached mindfulness' in which RNT is less likely to take hold. [206] Although MCT was originally developed to treat pathological worry in generalised anxiety disorder, [208] there is empirical evidence for its application to depression, [207,209,210] obsessive compulsive disorder, [211,212] post-traumatic stress disorder, [213,214] health anxiety, [215,216] cancer-related distress, [217–219] smoking cessation, [220,221] and chronic fatigue. [222,223] Recent systematic reviews show MCT has efficacy for numerous anxiety and mood disorders, in some cases surpassing the efficacy of CBT. [224,225]

Interestingly, little attention has been paid to metacognition in the pain literature. This is surprising, given: (1) its centrality to models of RNT [184,207]; (2) the important role ascribed to perseverative thinking in the misdirected problem solving model of pain-related worry [195] and RNT model of PC [178] described above; and (3) the importance of the rumination subscale of the PCS in predicting other pain outcomes. [83,137] To date there are only two studies exploring the role of metacognition in PC. The first used a muscular dystrophy sample, [226] which has limited generalisability to other chronic pain cohorts. Furthermore, it used the Thought Control Questionnaire (TCQ) , [227] which does not measure metacognitive beliefs *about* worry/rumination but rather assesses the frequency with which particular maladaptive cognitive strategies are used to deal with unpleasant thoughts. Therefore, although the study claims to show an association between metacognition and PC, the way the TCQ was used conflates metacognition and cognition, rendering the results difficult to interpret.

A recent study by Spada and colleagues [228] offers more solid insights. In a community sample of 308 people, they found that positive metacognitive beliefs about worry (e.g. "worrying helps me to get things sorted out in my mind") mediated the relationship between neuroticism and PC. Meanwhile negative metacognitions (e.g. "my worrying is dangerous for me") mediated the relationship between PC and self-reported maladaptive pain behaviours. [228] There are design limitations to this study because it was cross sectional and therefore unable to show causation. From a theoretical and cognitive process perspective, it would also make more sense for the relationship between neuroticism and PC to be moderated rather than mediated by positive metacognitions, which was not tested. However, notwithstanding these limitations and the non-clinical sample, this study provides the most convincing direct evidence to date that metacognitive theory is relevant to our understanding of PC. In particular, it shows that metacognition accounts for a significant amount of variance in the relationship between someone's disposition towards negative affect and their tendency to catastrophise about pain.

In addition to this direct evidence, indirect evidence of the relationship between metacognition and PC can be found in the now extensive research on so-called third wave psychological therapies for chronic pain. Various reviews and meta-

analyses show that interventions such as Acceptance and Commitment Therapy (ACT) and Mindfulness-Based Stress Reduction (MBSR) are effective in lowering pain, disability, and emotional distress (including PC) in people with chronic pain. [229–235] While these interventions do not explicitly challenge metacognitions through reattribution exercises in the way MCT does, [206] they aim to increase mindfulness, which is often defined as a metacognitive variable because it involves a change in the way people relate to their own thoughts. [236] For example, the former cognitive therapists who blended MBSR's meditative practices with insights from Beck's cognitive theory of depression [237] to develop Mindfulness-Based Cognitive Therapy (MBCT), describe how the goal of mindfulness practice is to relate to thoughts as passing mental events rather than facts requiring further elaborative cognition. [238]

The psychological flexibility model underlying ACT similarly focuses on cultivating mindful contact with present-moment experience. This involves 'cognitive defusion' in which unwanted experiences, including negative thoughts, are experienced directly rather than elaborated upon. [239,240] While proponents of MCT emphatically differentiate their 'detached mindfulness' from meditative mindfulness, [207] a common feature shared by ACT, MBSR, MBCT and MCT is a focus on changing not the *contents* of negative thoughts, but how people view and respond to them. This could be framed as a focus on cognitive *processes* (e.g. rumination) rather that cognitive contents (e.g. beliefs).[241]

Therefore, given that mindfulness involves self-regulated, non-elaborative awareness of thoughts, feelings and sensations in the present moment, [236] it is at least theoretically antithetical to the automatic, elaborative style of cognition found in worry, rumination and therefore PC. Empirical evidence of this is found in studies that show significant negative relationships between measures of mindfulness and both worry and PC. [242] Similarly, in a cross sectional study exploring the role of mindfulness within the FA model, mindfulness was most strongly related (negatively) to PC, moderating the relationship between pain intensity and catastrophising. [73] Therefore mindfulness and PC are related constructs, [243] and appear to be associated with two ends of the perseverative thinking spectrum. Furthermore, according to the S-REF model, developing detached mindfulness results in less pronounced unhelpful metacognitions and

therefore less rumination/worry. Evidence that mindfulness interventions reduce PC (i.e. pain rumination/worry) therefore suggests that these interventions change what people think about their own thinking. Mindfulness interventions may well result in metacognitive changes which have not yet been documented. Therefore, there are considerable theoretical and empirical grounds for expecting the metacognitive model of emotional disorder to be relevant to pain-related worry and rumination, potentially shedding new light on this facet of the PC construct.

1.4 Summary and Research Aims

The dominant biopsychosocial model of pain highlights the important contribution of psychological factors in the development and maintenance of chronic pain, which is now seen as a disease in its own right, [18] rather than merely a symptom of underlying pathology. Of these psychological factors, PC has emerged as one of the strongest predictors of increased pain, disability and a range of poor health related outcomes. The evidence for this effect is strongest in musculoskeletal pain-related disorders.

There is good evidence that psychological interventions, particularly CBT, are effective components of gold standard multidisciplinary treatments for people with chronic pain, and may partly improve pain outcomes by modifying process variables such as PC. However, the small effects associated with these interventions has prompted calls to improve the content of these interventions, which often lack a coherent theoretical basis. In responding to these calls, it makes sense to target process variables such as PC, using an intervention linked to the theory of this construct. However, the nature of PC requires further clarification, particularly since a range of theoretically distinct interventions have efficacy in reducing PC.

While appraisal and interpersonal models have received most attention, recent functional accounts suggesting PC is largely a form of RNT open promising avenues for clarifying nascent aspects of this important construct. One of these is the role of metacognition in modulating pain-related rumination and worry, given that metacognition is strongly related to RNT in anxiety and mood disorders. Accumulating evidence that acceptance and mindfulness-based interventions,

which implicitly target metacognition, are effective in reducing PC, suggests metacognition is a pertinent though unexplored feature of PC.

The present research therefore attempts to address this gap in the PC literature by developing a preliminary metacognitive model of PC. Shedding further light on the construct of PC itself is a necessary first step toward improving interventions in order to offer more effective, efficient and tailored treatments to people with chronic pain and elevated PC. This may involve identifying new treatment targets (e.g. positive and negative pain metacognitions) and techniques for modifying them. This research therefore has four related aims.

> **Aim 1:** Systematically review the PC intervention literature and compare pooled effect sizes of different interventions in order to document the characteristics of interventions with efficacy in reducing PC.

Rationale: This is an important first step towards improving PC interventions since to date there have been no systematic reviews exploring how a broad range of interventions modify PC. The only meta-analysis of PC outcomes was the latest Cochrane review of psychological interventions for chronic pain, which did not include exercise, mindfulness- and acceptance-based interventions and a range of other complementary treatments. [120] Including the full array of treatments that have been used to treat PC will produce a more complete picture of the characteristics of efficacious PC interventions. Aim 1 is addressed through the systematic review described in Chapter 2 (Study 1).

> **Aim 2:** Explore the lived experience of worry and rumination among people with elevated PC, including their metacognitive beliefs about pain-related thinking.

Rationale: Although PC has been extensively researched with quantitative methods that measure PC using self-report instruments, there is a surprising lack of qualitative research exploring the lived experience of PC. Certainly, there are numerous qualitative studies in the chronic pain literature more broadly, documenting themes such as biographical disruption,[244] the social unpleasantness of being in pain, [245] challenges in seeking treatment, and confusion about uncertain pain diagnoses. [59] However, to date no research has specifically targeted a pain population with elevated PC in order to document the lived experience of catastrophising. A qualitative analysis of experiences in this cohort would be valuable in shedding light on how people make sense of their pain-related thinking, including the specific attitudes they have towards pain rumination (i.e. metacognitions). Gathering rich idiographic data about pain metacognition in this way will strengthen any further quantitative research. Aim 2 is addressed through the qualitative study described in Chapter 3 (Study 2).

Aim 3: Develop and validate a self-report measure of pain-related metacognition that can be used in research and clinical settings.

Rationale: While at least one study has documented how metacognitive beliefs about worry predict PC and pain behaviours, [228] there is no measure of metacognition tailored to a pain population. The most widely used instrument – the Metacognitions Questionnaire [246] – was developed for a generalised anxiety disorder population and mainly addresses worry rather than other forms of perseverative thinking, such as depressogenic rumination, [247] that are likely to be relevant to people in pain. There is therefore a need to develop a specific tool to measure positive and negative metacognitive beliefs about pain-related perseverative thinking, which includes both worry and rumination. Just as specific metacognitive measures have been developed for use with addiction and chronic fatigue populations [221,222], a tool tailored to people with chronic pain is likely to have better validity than a generic measure, particularly given the unique attentional demands of chronic pain. Aim 3 is addressed through the scale development study described in Chapter 4 (Study 3).

Aim 4: Develop and test a model of pain-related metacognition in a sample of people with chronic pain.

Rationale: If metacognition is to be considered as a possible treatment target, then it is important to investigate how it relates to other pain and cognitive variables. Given the review of PC and RNT literature above, it is expected that the relationship between pain intensity and PC is largely a function of how much a person worries or ruminates about their pain. In other words, it is expected that RNT will mediate the relationship between pain intensity and PC. However, according to metacognitive theory, how much one ruminates depends on positive and negative metacognitions. This suggests the relationship between the trigger (pain) and a ruminative response will be conditional on the strength of unhelpful positive and negative metacognitions. Testing this conditional indirect effect is therefore an important step in developing a model of how metacognition might fit into existing theories of PC and whether there is potential value in targeting metacognition in treatment. Aim 4 is addressed through the conditional process analysis described in Chapter 5 (Study 4).

These four aims are each addressed in four discreet empirical studies described in the following chapters.[v]

[v] This thesis is presented in the 'hybrid' format, which blends a traditional typescript thesis with a thesis-by-publication format. Each chapter represents a separate study intended to be published as an independent manuscript, although not every study has yet been published. There is therefore some overlap in the introductions to each study. Short bridging statements are also provided before each study to explain how they link to the previous study.

Chapter 2 Study 1 – How can we best reduce pain catastrophising in adults with chronic non-cancer pain? A systematic review and meta-analysis

Publication History

This paper has been accepted for publication and is currently in press with the *Journal of Pain* (see Appendix L for acceptance letter):

Schütze, R., Rees, C. S., Smith, A. J., Slater, H., Campbell, J. M., & O'Sullivan, P. (in press). How can we best reduce pain catastrophizing in adults with chronic non-cancer pain? A systematic review and meta-analysis. *Journal of Pain*.

2.1 Abstract

Pain catastrophising (PC), defined as an exaggerated negative cognitive-affective orientation toward pain, is one of the strongest psychological predictors of pain outcomes. While regularly included as a process variable in clinical trials, there have been no comprehensive reviews of how it can be modified. Using a registered protocol (PROSPERO 2016 CRD42016042761), a search of MEDLINE, PsychINFO, EMBASE, CINAHL, and CENTRAL databases was performed up to November 2016 for all randomised controlled trials measuring PC in adults with chronic non-cancer pain. Two authors independently screened studies and assessed bias risk using the Cochrane tool. Quality of evidence was rated according to GRADE criteria. There were 79 studies (N=9914) included, which mostly recruited participants with musculoskeletal pain and had low risk of bias. Meta-analyses (standardised mean difference) showed 9 interventions had efficacy compared to waitlist/usual care or active control, although evidence quality was often low. The best evidence (moderate-high quality) was found for Cognitive Behaviour Therapy (CBT), multimodal treatment, and Acceptance and Commitment Therapy (ACT). Effects were generally of medium strength and had questionable clinical significance. When only the 8 studies targeting people with high PC were included, effects were larger and more consistent. Multimodal treatment showed the strongest effects when all studies were considered, while CBT had the best evidence among targeted studies.

Perspective: Pain catastrophising is a modifiable characteristic but most interventions produce only modest benefit unless targeted to people with high PC. More research into theory-driven interventions matched to specific patient profiles is required to improve treatment efficacy and efficiency.

2.2 Introduction

Pain catastrophising (PC) is a negative cognitive-affective response to pain [248] and a large body of research shows it is a significant risk marker for adverse pain and health outcomes. [78] Elevated PC is associated with greater disability, [98] pain intensity, [79] depression, [92] anxiety, [93] work absenteeism, [94] opioid misuse, [95] and health-care utilisation. [96] A tendency to catastrophise can also predict the transition to chronicity and its maintenance, [97] with the influential fear-avoidance model of pain [62,68,70] providing an account of how PC facilitates pain, disability and distress, particularly in musculoskeletal pain. [64,66,67]

Experimental and clinical data show that PC is associated with a range of biological processes that could modulate nociception. These include: dysregulation of the hypothalamic-pituitary axis that is linked to central nervous system sensitisation [170,171]; reduced descending inhibitory control through endogenous opioid pathways [172]; increased activation of brain areas associated with affective aspects of pain [173]; and pain-facilitating changes in functional connectivity of the brain's default mode network. [174]

In treatment settings, PC is an important process variable that mediates improvements through interventions such as Cognitive Behaviour Therapy (CBT), [111,112] Acceptance and Commitment Therapy (ACT), [113] exercise-based rehabilitation, [114] and multidisciplinary treatment. [115–117] Some studies using cross-lagged designs show that improvements in PC early in treatment predict later improvements in pain and disability. [115,117] As a result, PC has become a key treatment target, particularly in psychological and multidisciplinary interventions for people with chronic non-cancer pain. Research has tended to focus on musculoskeletal pain such as chronic low back pain (CLBP), [119] neck pain [120] and osteoarthritis, [121] as well as fibromyalgia, [122] perioperative pain in the context of joint replacement, [123] and more recently neuropathic pain. [117]

However, it is still unclear how best to help people with pain to catastrophise less, since a range of different interventions produce benefit. On one hand, there seems to be most evidence for CBT, with the only meta-analytic data on PC interventions coming from the latest Cochrane review of psychological therapies for chronic pain. [120] This showed that CBT reduces PC with a medium standardised mean

difference (SMD) effect of -0.53 compared to waitlist at post-test. [120] However, in a high quality head-to-head trial comparing CBT, exercise (general aerobic and strength training), and multidisciplinary treatment combining CBT and exercise, all three interventions showed similar effects of moderate strength. [114] This is surprising given that exercise does not explicitly target unhelpful thinking processes. More recently, emerging so-called third wave psychological therapies such as ACT and mindfulness meditation have also shown efficacy for reducing PC, [133,134] with some suggesting large effect sizes. [135] A recent head-to-head comparison of CBT and mindfulness meditation in people with CLBP showed both were efficacious, with mindfulness slightly superior in reducing PC in the short term. [136]

While these data suggest that there are a range of different ways to reduce PC, there is no clearly superior intervention and the mechanisms that underpin this change remain unclear. To date, a meta-analytic approach that investigates all interventions measuring treatment-related changes in PC has not been conducted. The present study therefore attempts to fill this gap in the literature. Specifically, it aims to: (1) systematically review and describe randomised controlled trials that measure catastrophising changes in chronic non-cancer pain; (2) document and compare the pooled effects of different interventions; and (3) identify factors that moderate the efficacy of these interventions. Given evidence cited above that it is not only interventions designed to target PC that show efficacy in reducing it, [114] this review aims to examine all treatment-related changes in PC regardless of whether catastrophising was specifically targeted as a primary outcome. Although it is therefore likely that many of the included studies do not primarily target PC, it allows for an examination of a wider array of possibly efficacious treatments, rather than just those intentionally designed to reduce catastrophising in high risk cohorts.

2.3 Methods

PRISMA guidelines [249] for conducting and reporting systematic reviews were used to design this study and a review protocol was prospectively registered with PROSPERO. [250]

2.3.1 Data sources and search strategy

The primary search was conducted in the following databases: MEDLINE, EMBASE, PsycINFO, CINAHL, and CENTRAL (the Cochrane Central Register of Controlled Trials) up until November 2016. A search strategy was developed using free text words, questionnaire names, and MeSH headings according to published guidelines. [251,252] Validated search filters were used for randomised controlled trials, including the sensitivity-maximising version of the Cochrane Highly Sensitive Search Strategy for MEDLINE, [251] the CADTH filter for PsycINFO, [253] and the SIGN filter for CINAHL [254] (see Appendix A for example of MEDLINE search strategy). Reference lists of retrieved studies and relevant review articles were also hand searched.

2.3.2 Study inclusion

Studies meeting the following criteria were eligible for inclusion:

1. Participants reported any kind of chronic non-cancer pain, defined as pain lasting ≥3 months;
2. Participants were adults (≥18 years old);
3. Used at least one experimental intervention intended to reduce clinical, rather than experimental pain or pain-related outcomes;
4. Compared experimental interventions to waitlist/usual care control or an active control using a randomised controlled trial (RCT) design;
5. Analysed ≥20 participants in each treatment arm at post-test. This condition attempted to reduce the risk of bias associated with small samples and increase the likelihood that included studies would be adequately powered. This condition is consistent with the most recent Cochrane Review of psychological interventions for chronic pain, which also reports on pain catastrophising. [120]
6. Reported on changes in PC using a validated self-report measure;
7. Study was available as English language article published in a peer-reviewed journal.

The inclusion of only published studies was intended to maximise the quality of included data by ensuring it had passed peer-review, thereby reducing risk of bias. As noted earlier, this review aimed to examine all treatment-related changes in PC rather than only changes associated with studies specifically designed to reduce catastrophising. Therefore, studies treating PC as either a primary outcome or a secondary/process outcome were included, although how PC was treated within studies was recorded for the sake of moderator analysis described below.

Using these criteria, two independent assessors (R.S. and C.R. – see Statement of Author Contribution for full author names) screened the titles and abstracts of all studies identified by the search. Clearly irrelevant studies were excluded and the full text of the remaining articles was retrieved. Any discrepancies between assessors at screening were discussed and resolved by consensus. The same process was used to independently assess the full text of potentially eligible studies. If consensus was not achieved, a third assessor (H.S.) was consulted.

2.3.3 Data extraction and management

Data from included studies was extracted by one assessor (R.S.) and checked by a second (A.S.). A customised piloted data extraction form based on Cochrane guidelines [251] was used to retrieve the following information: study characteristics (design, funding, country); sample characteristics (inclusion and exclusion criteria, age, gender, pain duration, pain condition, number randomised); intervention characteristics (content, duration, format, type of therapist, total therapist contact); and outcome characteristics (PC instrument, number of participants analysed at each time point, catastrophising scores at each time point). For PC outcomes, means and standard deviations/standard errors/confidence intervals at baseline, post-test and follow-up were extracted, or alternatively change scores from baseline plus standard errors, were extracted. Only data from relevant PC subscales were extracted from studies using broader multidimensional measures (e.g. Coping Strategies Questionnaire [86]). Where insufficient data were reported for meta-analysis, it was requested by contacting study authors. Data screening was managed using Covidence systematic review software. [255] Outcomes for meta-analysis were entered into Comprehensive Meta-Analysis software [256] by one

author (R.S.) and checked by another (A.S.).

2.3.4 Risk of bias rating

Risk of bias was assessed using the Cochrane Collaboration's risk of bias tool. [257] Two authors (R.S. and J.C.) independently assessed each study in Covidence and resolved any differences through discussion to arrive at consensus. Each study was assessed against six domains in the standard tool: random sequence generation (selection bias), allocation concealment (selection bias), blinding of participants (performance bias), blinding of outcome assessment (detection bias), incomplete outcome data (attrition bias), and selective reporting (reporting bias). Since many studies were expected to involve behavioural interventions where participants cannot be blinded to intervention content, participant blinding in trials with active control groups was assessed in terms of efforts to control for expectancy effects by concealing study hypotheses. Studies only using waiting list/usual care control groups are not able to control for expectancy effects in this way and were judged at high risk of performance bias. Only participant blinding, rather than therapist blinding, was assessed since therapists delivering behavioural interventions cannot be blinded to the content they are delivering. [258]

Since PC was always measured with self-report questionnaires, which are relatively robust to detection bias, this was rated ‘low’ if participants completed these independently (e.g. at home) and ‘high’ if measures were administered by un-blinded assessors. Where missing data due to attrition had been excluded from analysis, attrition bias was judged as ‘high’ if the loss was ≥ 20% of the allocated sample. [259] However a ‘low' rating was given if intention to treat (ITT) analysis was used with robust imputation methods such as multiple imputation, or theoretically justified modelling methods that included variables as covariates which might be predictive of withdrawal. [260] Where authors did not prospectively register their trials or publish a protocol, reporting bias was judged ‘unclear’, since it was not possible to determine whether all planned outcomes and analyses were adequately reported.

Following Cochrane recommendations, risk of bias results were used to classify

studies as either at low risk of bias, or at unclear/high risk of bias. Low risk studies were defined as those having low risk ratings on at least three of the six bias categories, and also not being judged as at high risk on any critical bias category (random sequence generation, allocation concealment, incomplete outcome data or selective reporting).

2.3.5 Data synthesis and analysis

Comprehensive Meta-Analysis [256] software was used for meta-analysis. Since PC was measured with a variety of different self-report instruments as a continuous variable, pooled effect sizes were generated as standardised mean differences (SMD). [251] A random effects model was used given the expected variation in interventions included and therefore likely heterogeneity in effect sizes. Heterogeneity was assessed for statistical significance using Cochran's Q statistic and its magnitude was assessed using the I^2 statistic, which describes the percentage of variability due to true differences in effect sizes rather than due to chance. Values of 25%, 50% and 75% for I^2 were used to classify low, moderate and high heterogeneity respectively. [261] Pooled effect estimates using SMD were interpreted according to Cohen's criteria (small ≤ 0.2; moderate = 0.5; large ≥ 0.8). [106]

Four groups of analyses were planned:

1. Waitlist/Usual care controlled trials at post-test for each intervention type;
2. Waitlist/Usual care controlled trials at follow-up for each intervention type;
3. Active control group trials at post-test for each intervention type;
4. Active control group trials at follow-up for each intervention type.

Where there were multiple comparison groups and the experimental treatment was not specified in the study, the most intensive intervention was chosen as the experimental group. Active control groups included comparison treatments intended to control for expectation and other non-specific factors. Low-contrast comparisons from the same class of intervention were not included. For example, where two variants of a CBT protocol were compared in a non-inferiority trial, this was not included in the CBT versus active control meta-analyses. However, where

CBT was compared to exercise, for example, this was included. Only studies that presented a treatment as an experimental condition were included in meta-analyses of that intervention. For example, education and exercise were commonly used as attention controls but at times were studied as experimental conditions in their own right and compared against other active controls. Therefore, only studies that used education and exercise as experimental groups were included in analyses calculating the pooled effect of education and exercise. Consistent with Williams et al., [120] follow-up comparisons were included where data was available between 6 months and 12 months post-intervention, with the longest of the follow-up periods chosen when several assessments were made within this range.

2.3.5.1 Moderator and sub-group analysis

Where possible, these meta-analyses were performed on all included studies. However, they were also performed separately on the subset of studies that targeted PC as a primary outcome and whose cohorts had clinically significant levels of mean PC at baseline (based on recommendations of a score of >24 on the PCS [262]). This was done to explore effect sizes in an emulated clinical context where interventions are commonly matched to clinical risk factors (i.e. a treatment aimed at reducing PC for those with clinically significant symptoms of catastrophising). [263] This was only included as a subgroup analysis because defining clinical PC is still problematic, with published PCS cut-offs varying from 16, [264] to 20, [265] 24, [262] and 30 [143]. Furthermore, since PC exists on a spectrum, it is likely that even those with moderate elevations could benefit from reducing these symptoms through treatment. Therefore, while effect sizes in targeted cohorts were deemed important to document, so too were effect sizes across a spectrum of baseline catastrophising. Pooled effects are therefore presented separately for: a) all included studies, and b) targeted studies (see Tables 2.1-2.4).

Moderation of pooled treatment effects was also explored through meta-regression in Comprehensive Meta-Analysis. The following moderator variables were tested: risk of bias status, baseline PC, intervention duration, facilitator contact time, pain condition, year of publication, type of facilitator, whether PC was a primary outcome, PC measure, and delivery format. To use baseline PC as a moderator,

scores on the various PC measures were transformed into a common scale of 0-100. Meta-regression significance testing was relaxed to $p<.10$ due to expected low power associated with small samples. Finally, the statistical significance of differences in effect sizes between interventions in each of the analysis domains above was measured with analysis of variance using the weighted sum of squares Q statistic.

2.3.5.2 Publication bias

As per Cochrane recommendations, [251] funnel plots of each meta-analysis with at least 10 studies were inspected and tested for publication bias. Smaller meta-analyses (N < 10) were not tested due to the high probability that they would be under-powered. [251] Statistical evidence of bias through asymmetry of plots was tested using the Egger-weighted regression, with a significant *p* value suggesting possible publication bias. [266] The impact of publication bias on pooled effects was estimated using Duval and Tweedie's trim and fill method to impute likely missing studies and an adjusted effect size once these studies were included. [267]

2.3.6 Quality of evidence

Grading of Recommendations, Assessment, Development and Evaluations (GRADE) criteria [268] were used to assess the quality of evidence for each intervention in the four analyses described above. Starting with an assumption of high quality evidence since all data came from RCTs, [268] evidence quality was downgraded one category for each of the following GRADE criteria:

5. Risk of bias: > 25% of participants are from studies judged at high/unclear risk of bias according to criteria above (see Appendix C for risk of bias summary).
6. Inconsistency: Significant heterogeneity in pooled effect ($I^2 > 50\%$)
7. Indirectness of evidence: Interventions not directly compared; results unlikely to generalise; surrogate outcomes used.
8. Imprecision: Total participants <400 (based on optimal information size for a small effect, using normative approach of $\alpha=.05$, $\beta=.20$, SMD=0.2 [269])
9. Publication bias: Significant selective publication of evidence based on criteria

described above.

Based on these criteria, evidence from each analysis was rated as either high, moderate, low or very low quality, as defined by GRADE. [269]

2.4 Results

2.4.1 Search

The search strategy returned 2411 citations, with a further 54 studies identified through hand searching. As shown in the PRISMA flow chart (Figure 2.1), the final review included 79 studies. [114,116,119,121,122,135,136,270–341] Eight authors were contacted for further information or data, and four responded. Sufficient data for meta-analysis was available for 77 studies. A summary table of the characteristics of included studies is provided in Appendix B.

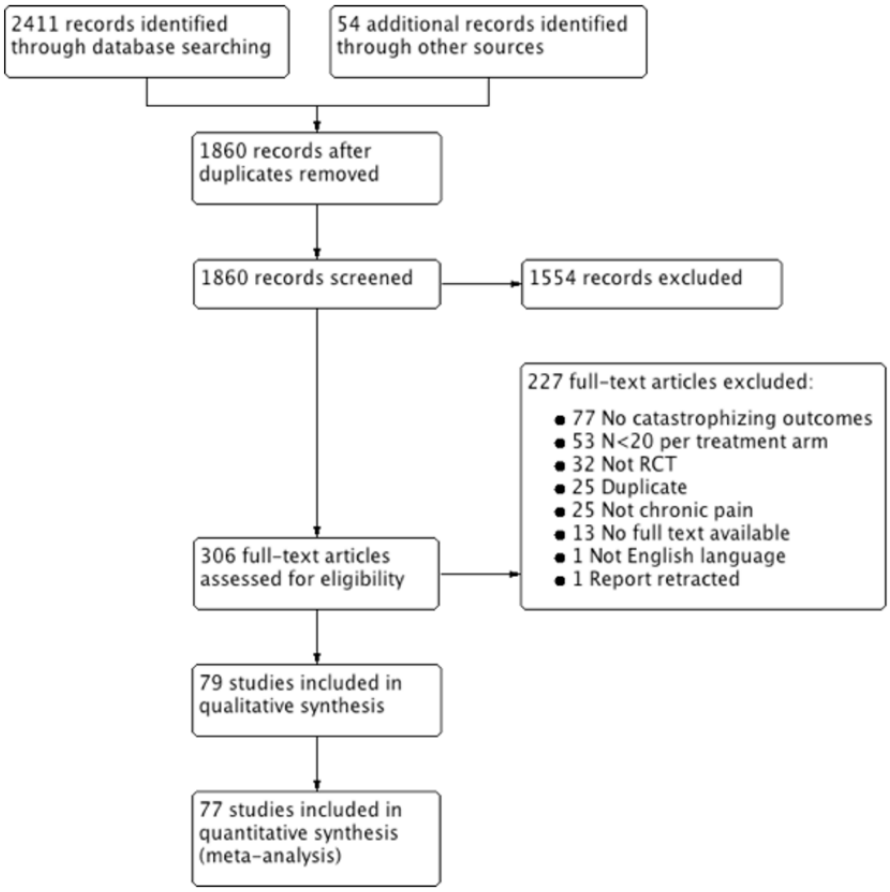


Figure 2.1. PRISMA flow chart for study selection.

2.4.2 Description of studies

Most studies originated in Europe (64.6%) and North America (22.8%), with the following countries most strongly represented: United States (21.5%), Netherlands (16.5%), Sweden (12.7%), Spain (10.1%) and Australia (10.1%). Included studies were published between 1988 and 2016, with a median publication date of 2013 (IQR = 2009-2015). Most of these RCTs used a single control group (n = 61, 77.2%), although a handful used two (n = 16, 20.3%) or three (n = 2, 2.5%) controls. The most common measures of PC were the Pain Catastrophising Scale[83] (n = 44, 55.7%) and Coping Strategies Questionnaire[86] (n = 28, 35.4%). Four studies (5.1%) used the Pain-Related Self-Statements scale,[139] and three separate studies (1.3% each) used the Cognitive Errors Questionnaire, [140] Vaginal Penetration Cognition Questionnaire,[141] and Pain Cognition List.[142] These latter three scales were not listed in the published review protocol[250] because they were only discovered during systematic database searching; however investigation of their psychometric properties justified inclusion, despite this minor protocol deviation.

Only 32 (40.5%) studies reported specifically targeting PC as a primary outcome, with most treating PC as a secondary outcome or not specifying a primary outcome. There were only 8 (10.1%) targeted studies that used PC as a primary outcome and included cohorts with high baseline catastrophising.

2.4.3 Participants

There were 9,914 participants (74% female) studied in total, with trial samples ranging from 40 to 341 people. Participants ranged in age from 27 to 82 years, averaging 48 years overall. Pain duration was only reported in 58 (73%) studies, with means ranging from 1.2 to 23 years (overall mean 8.7 years). Spinal pain – most often CLBP or neck pain – was the most common pain condition (n=24, 30.4%). Mixed pain cohorts (n=19, 24.1%) and fibromyalgia (n=17, 21.5%) were also strongly represented, although the mixed cohorts were mainly comprised of CLBP, making spinal pain by far the dominant pain condition represented overall. Baseline PC scores were available for 75 studies and when these were converted to

a 0-100 scale the mean score was 44.3 (SD = 13.6). This corresponds to a score of 23 on the Pain Catastrophising Scale (PCS), which has a possible score of 0 to 52 (higher scores mean higher PC). When scores were dichotomised based on recent evidence that a score of ≥24 on the PCS represents high PC, [262] a majority of studies (n = 43, 57.3%) were found to have low PC samples at baseline. Almost identical results were found when only the 44 studies using the PCS were included in this analysis.

2.4.4 Interventions

Seventeen different types of intervention were identified. These could be broadly grouped into: those containing mostly psychological content (n = 48, 60.8%); those involving mostly physical treatments, such as exercise, acupuncture, or manual therapy (n = 7, 8.9%); multimodal interventions involving a combination of physical and psychological content (n= 22, 27.8%); and purely pharmacological treatments (n = 2, 2.5%). Within these groupings, the most common interventions studied were CBT (n = 28, 35.4%) and multimodal interventions (n=20, 25.3%). However, all the multimodal interventions contained a CBT component, making CBT by far the most commonly used modality. The duration of interventions varied considerably, ranging from 1 day to 28 weeks (median = 8 hrs, *IQR* = 5.75-12.00). Similarly, the amount of contact participants had with treatment facilitators varied from no contact in the case of online treatments, to 126 hours (median = 14 hrs, *IQR* = 3.22-24.62). Less than half of the included studies gave as much facilitator contact to the control group as they did to the experimental group (n = 33, 41.8%).

In terms of format, most interventions were delivered face-to-face, with 34 (43%) using a group format and 24 (30.4%) delivered individually. Twenty-one (26.6%) of the included studies were predominantly self-administered using some form of media (internet, smartphone, telephone, booklet), although some web-based interventions also involved minimal therapist contact by email or telephone. The most common facilitators of interventions in the experimental arms of included studies were psychologists/psychotherapists (n = 25, 31.6%) and multidisciplinary teams (n = 18, 22.8%).

2.4.5 Risk of bias

A summary of the risk of bias assessment for the 79 studies reviewed is presented for each bias category in Figure 2.2. Most studies (n = 60, 75.9%) had 'low risk' ratings for at least half of the bias categories assessed. The median number of categories that were judged low risk for each study was four out of the six included. Using criteria described earlier for judging each study's overall risk of bias, 48 (60.8%) studies were low risk while 31 (39.2%) were unclear/high risk (see Appendix C, which provides a risk of bias assessment for each included study). Risk of bias was also related to certain study characteristics. For example, the number of bias categories judged low risk for each study was positively correlated with its sample size (Spearman's rho = .233, p = .039), publication year (Spearman's rho = .239, p = .043) and number of treatment arms (Spearman's rho = .258, p = .022). This suggests that less biased studies tended to be larger, more recent trials.

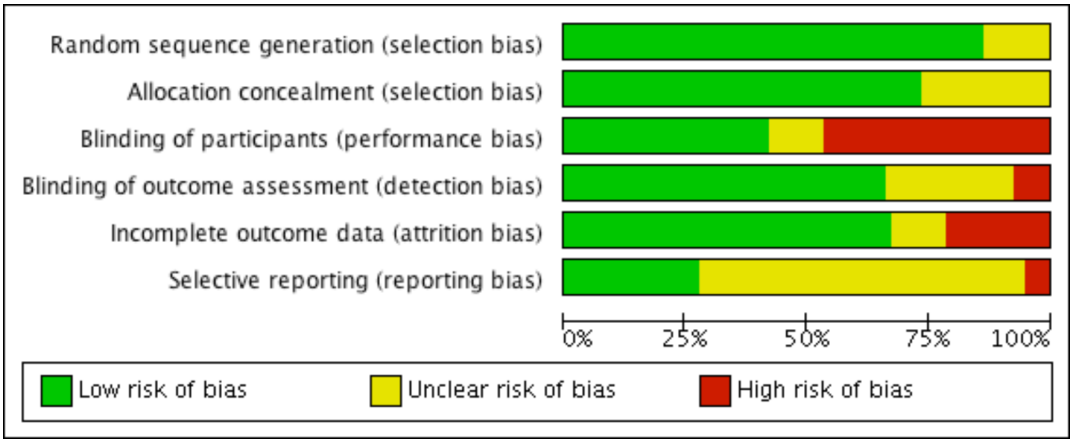


Figure 2.2. Risk of bias graph: review authors' judgements about each risk of bias item, presented as percentages across all included studies.

2.4.6 Meta-analysis of catastrophising outcomes

2.4.6.1 Intervention versus waitlist/usual care: Post-test outcomes

The effects of different interventions on PC compared to WL/UC at post-test are

summarised in Table 2.1, with 9 interventions showing efficacy. Meta-analysis was possible for five of these – ACT, CBT, exercise, mindfulness and multimodal treatment – which is depicted with forest plots in Figures 2.3-2.7. One outlier [284] with a very large effect (SMD = -6.78) was removed due to its outsized effect on the CBT meta-analysis, although inclusion did not alter the direction or significance of the pooled effect. As shown in Table 2.1, effect sizes ranged from small in the case of CBT (SMD = -0.25) to very large for graded exposure (SMD = -1.74). However, the quality of evidence was very low for the interventions with large effect sizes. The best quality evidence according to GRADE criteria was found for CBT, multimodal treatment, exercise and mindfulness (moderate quality). Multimodal treatment (medium effect) and CBT (small effect) had the largest body of evidence, but effects were inconsistent, thereby reducing confidence in their estimates. Exercise and mindfulness showed consistent medium effects but the estimates were imprecise, requiring data from more participants to justify confidence in their effect estimates. Considering only the interventions where meta-analysis was possible, the differences in pooled effect between interventions were not quite statistically significant ($Q = 9.34$, $df = 4$, $p = .053$).

Table 2.1. Effects of different interventions on pain catastrophising compared with waitlist/usual care at post-test.

Intervention †	Participants (studies)	SMD effect (95% CI)	PCS change‡	Heterogeneity Q, I^2	Moderators	Evidence quality (GRADE)
All included studies						
ACT [135,328]	263 (2)	-0.71* (-1.38, -0.04)	-6.6	6.71*, 85.10	-	Low [b d]
CBT [114,119,121,122,136,276,277,280,282,296,316,320,329,330,333]	1933 (15)	-0.25** (-0.41, -0.10)	-2.3	35.80**, 60.90	Baseline PC	Moderate [b]
EFT [275]	66 (1)	-0.87** (-1.38, -0.36)	-8.1	-	-	Very low [a b d]
Exercise [114,294]	277 (2)	-0.38*** (-0.59, -0.17)	-3.5	0.22, 0.00	-	Moderate [d]
Graded exposure [324]	70 (1)	-1.74*** (-2.29, -1.19)	-16.3	-	-	Very low [a b d]
Hypnosis [318]	59 (1)	-.32 (-0.84, 0.19)	-3.0	-	-	Very low [a b d]
Manual therapy [319]	48 (1)	-1.56*** (-2.21, -0.92)	-14.6	-	-	Low [b d]
Mindfulness [136,301]	338 (2)	-0.46*** (-0.67, -0.24)	-4.3	0.08, 0.00	-	Moderate [d]
Multimodal [114,116,285,286,322,335,338]	737 (7)	-0.63*** (-0.89, -0.38)	-5.9	18.91**, 68.28	Baseline PC; PC primary	Moderate [b]
Yoga [283]	53 (1)	-0.71* (-1.27, -0.15)	-6.6	-	-	Low [b d]
Only studies targeting elevated pain catastrophising [§]						
CBT [119,122,316,330]	288 (4)	-0.45* (-0.85, -0.06)	-4.2	8.36*, 64.11	-	Very low [a,b,,d]
Multimodal [116,285,286]	375 (3)	-0.88*** (-1.09, -0.66)	-8.2	0.42, 0.00	-	Moderate [d]

Note: * $p<.05$, ** $p<.01$, *** $p<.001$. ACT, Acceptance and Commitment Therapy; CBT, Cognitive Behaviour Therapy; EFT, Emotional Freedom Techniques; SMD, standardised mean difference. † Studies included in each pooled effect. ‡ Change in Pain Catastrophising Scale (PCS) score calculated by multiplying SMD by average standard deviation of included studies that used PCS (SD = 9.34). [a] Downgraded due to risk of bias; [b] Downgraded due to inconsistency; [c] Downgraded due to indirectness of evidence; [d] Downgraded due to imprecision, [e] Downgraded due to publication bias. [§] Targeted interventions are those that treat pain catastrophising as a primary outcome and have cohorts with clinically significant levels of catastrophising (>24 equivalent on the PCS).

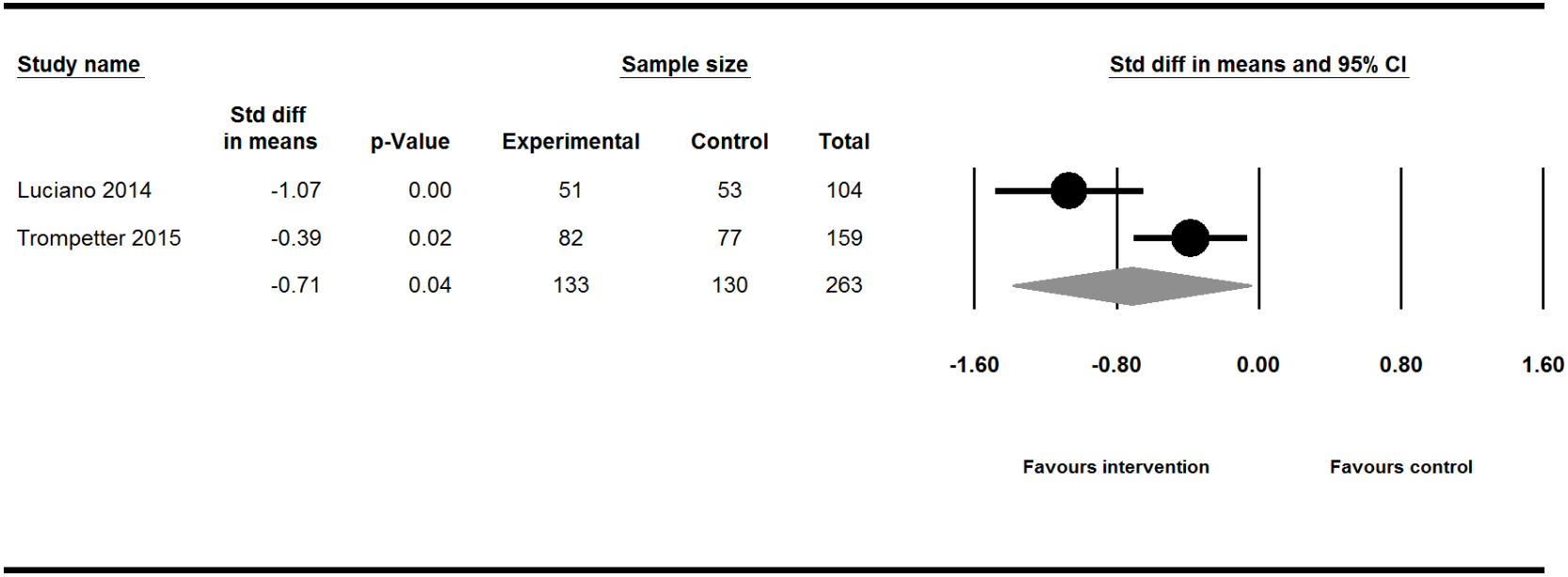


Figure 2.3. Pooled effects on pain catastrophising of Acceptance and Commitment Therapy versus waitlist/usual care at post-test.

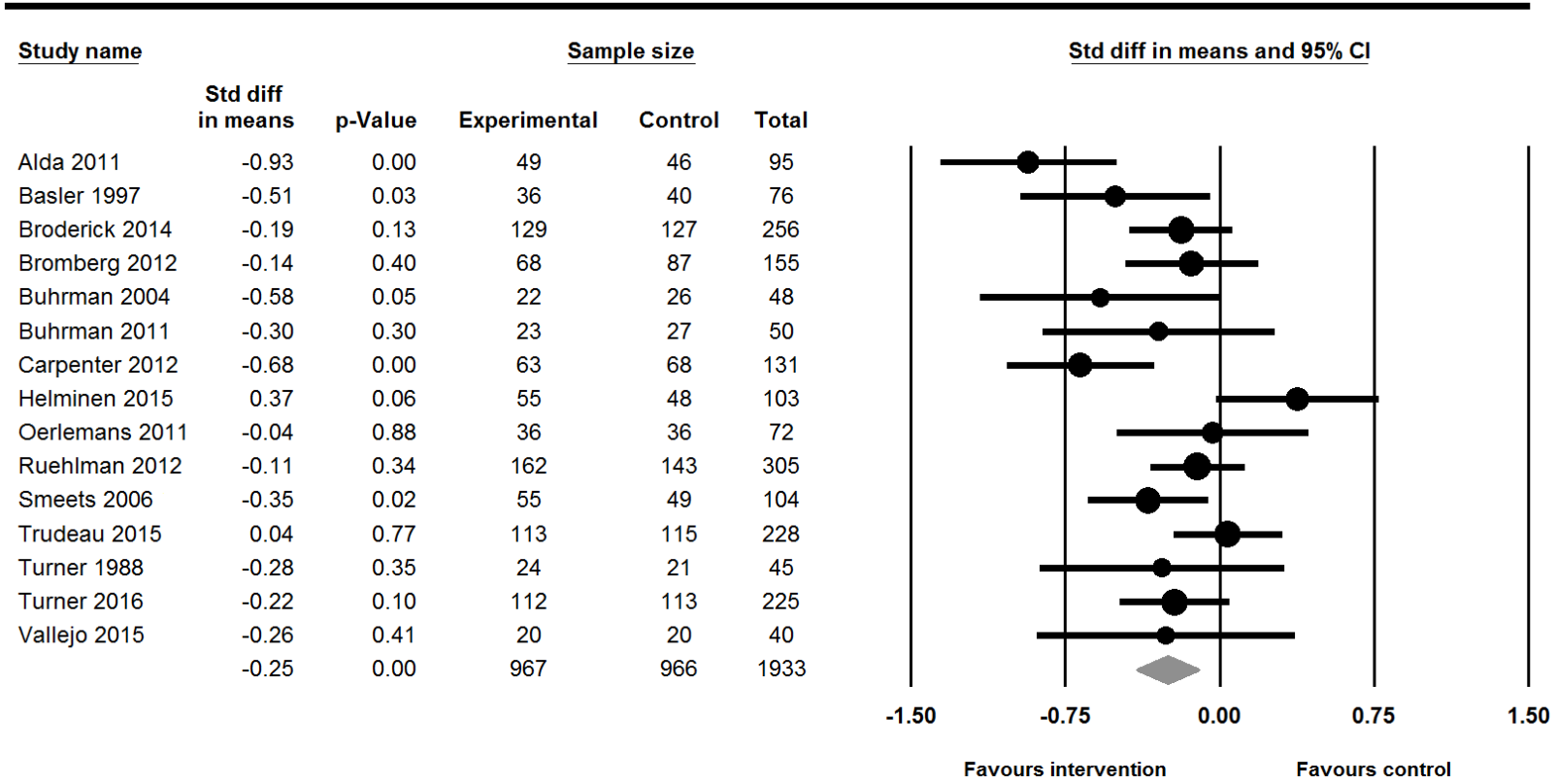


Figure 2.4. Pooled effects on pain catastrophising of Cognitive Behaviour Therapy versus waitlist/usual care at post-test.

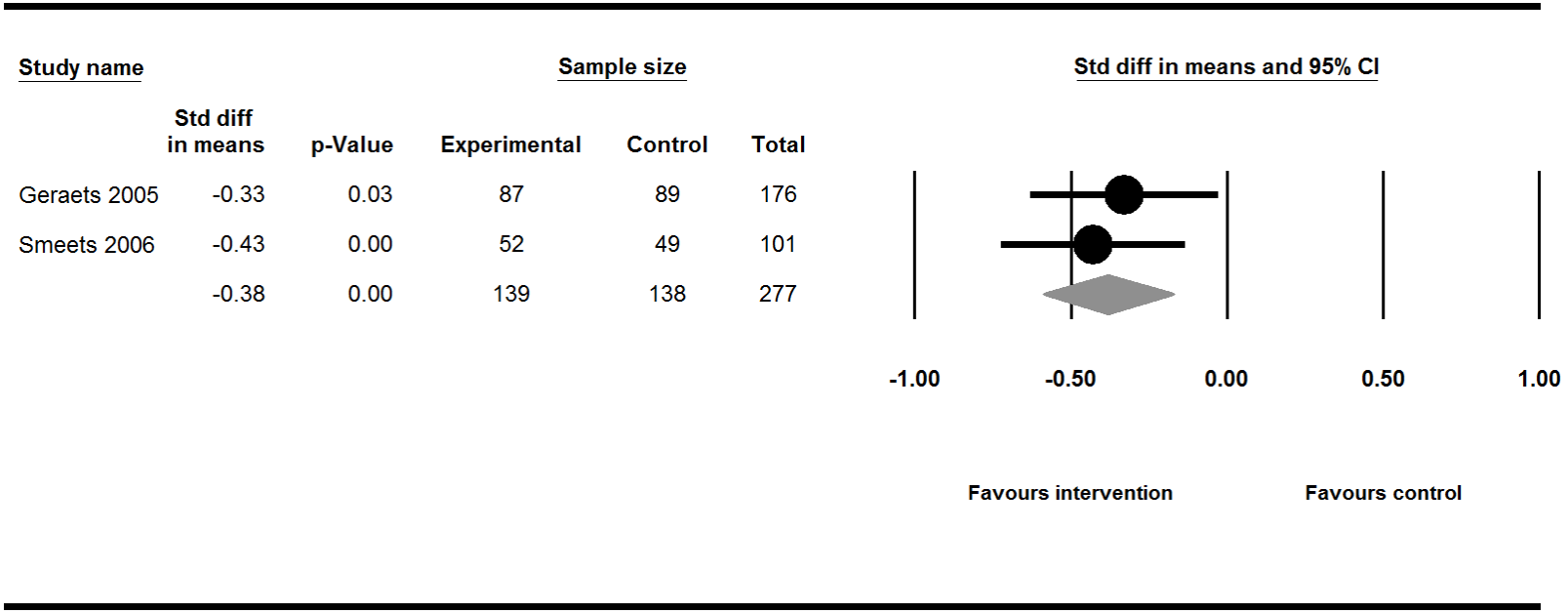


Figure 2.5. Pooled effects on pain catastrophising of exercise versus waitlist/usual care at post-test.

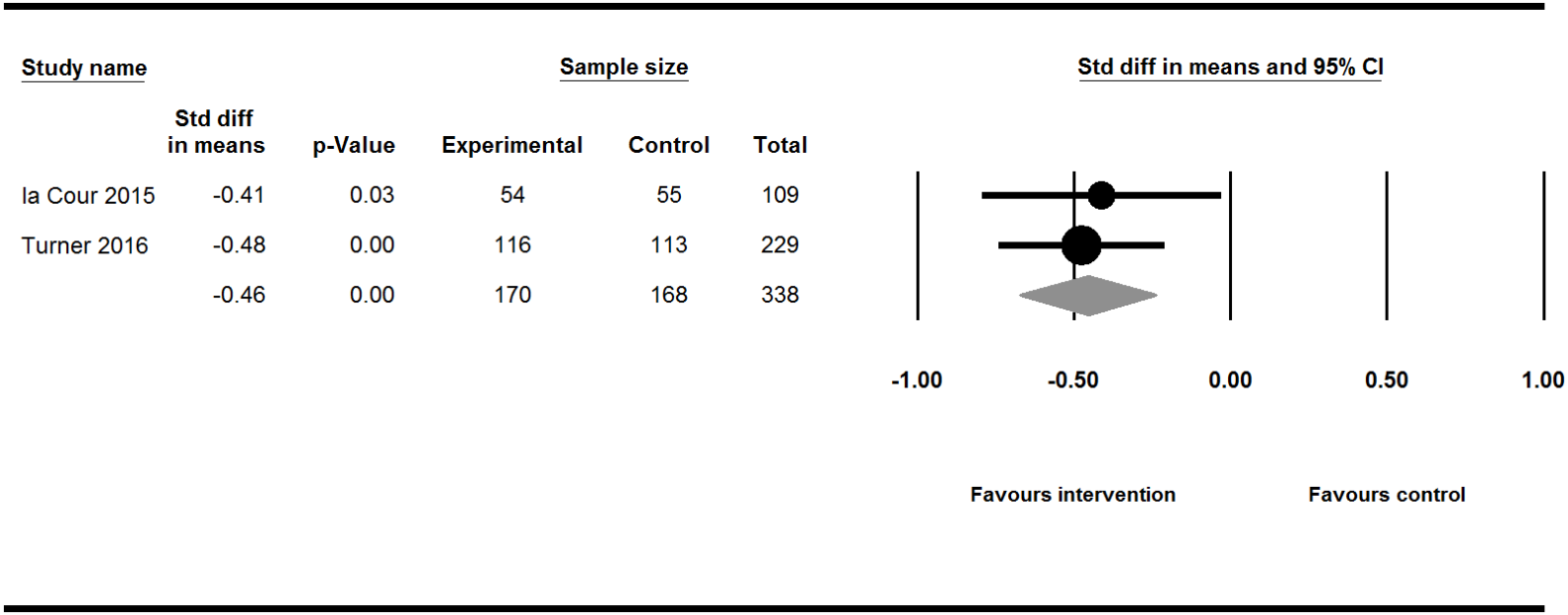


Figure 2.6. Pooled effects on pain catastrophising of mindfulness meditation versus waitlist/usual care at post-test.

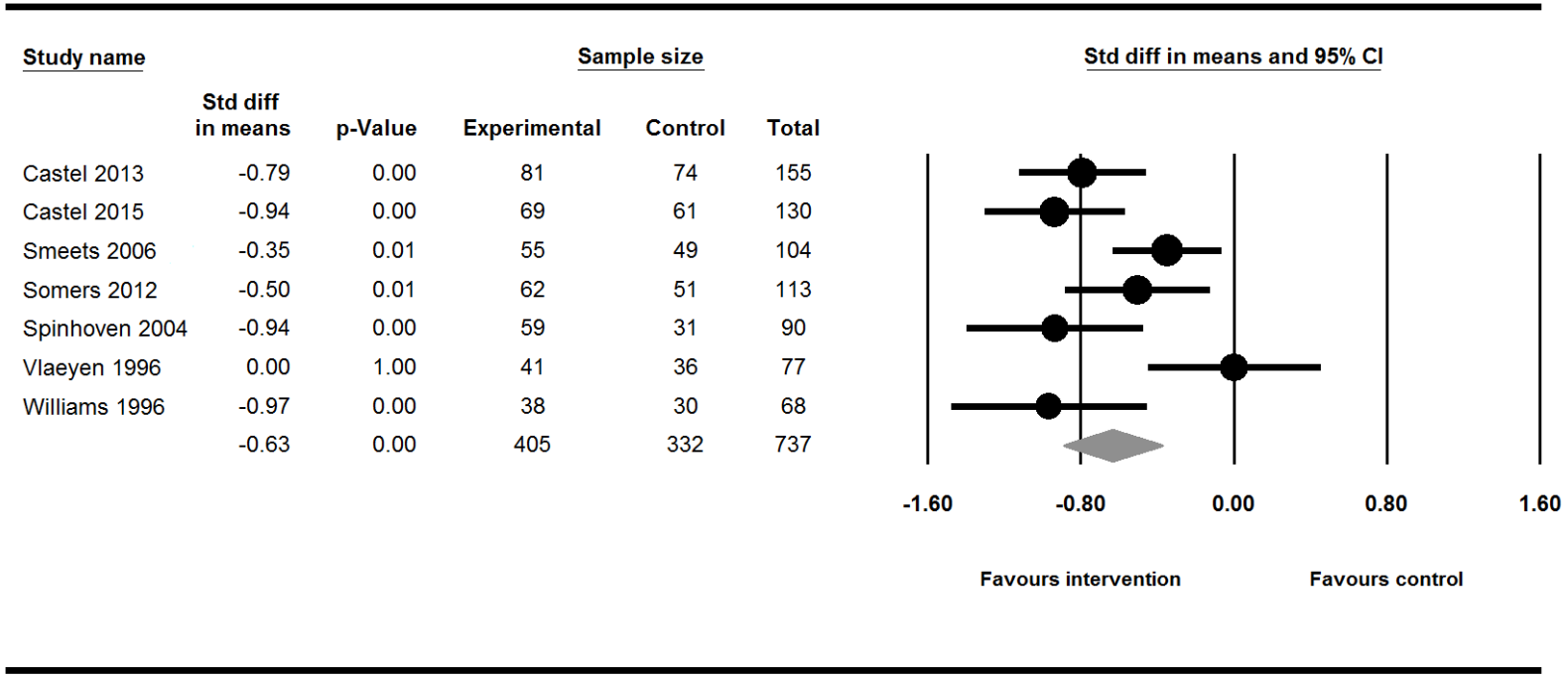


Figure 2.7. Pooled effects on pain catastrophising of multimodal treatment versus waitlist/usual care at post-test.

2.4.6.1.1 Moderator and sub-group analysis

Multimodal treatment and CBT were the only interventions with sufficient studies for meta-regression using the moderator variables described earlier. For CBT, baseline PC was a significant moderator of pooled effect (Q = 3.56, *df* =1, *p* = .06), favouring high baseline PC. The pooled effect of CBT among studies with high baseline PC was larger (SMD = -0.36, 95% CI -0.55, -0.16, *p* < .001) and less heterogeneous (Q = 18.75, *df* = 8, *p* = .02, I^2 = 57.34). Multimodal interventions were also moderated by baseline PC (Q = 4.78, *df* =1, *p* = .03) as well as whether PC was targeted as a primary outcome. As shown in Table 2.1, these moderation effects are reflected in the sub-group analyses, where including only high baseline PC studies that targeted catastrophising produced larger effect sizes for CBT (SMD = -0.45) and multimodal treatment (SMD = -0.88). A reduction in heterogeneity was also observed for multimodal treatments, but not CBT.

2.4.6.1.2 Publication bias

Only CBT had sufficient studies for an intervention-specific funnel plot. As shown in Figure 2.8, there was no evidence of asymmetry (Egger's test = -1.52, *df* = 13, *p* = .13) and no missing studies according to Duval and Tweedie's trim and fill method.

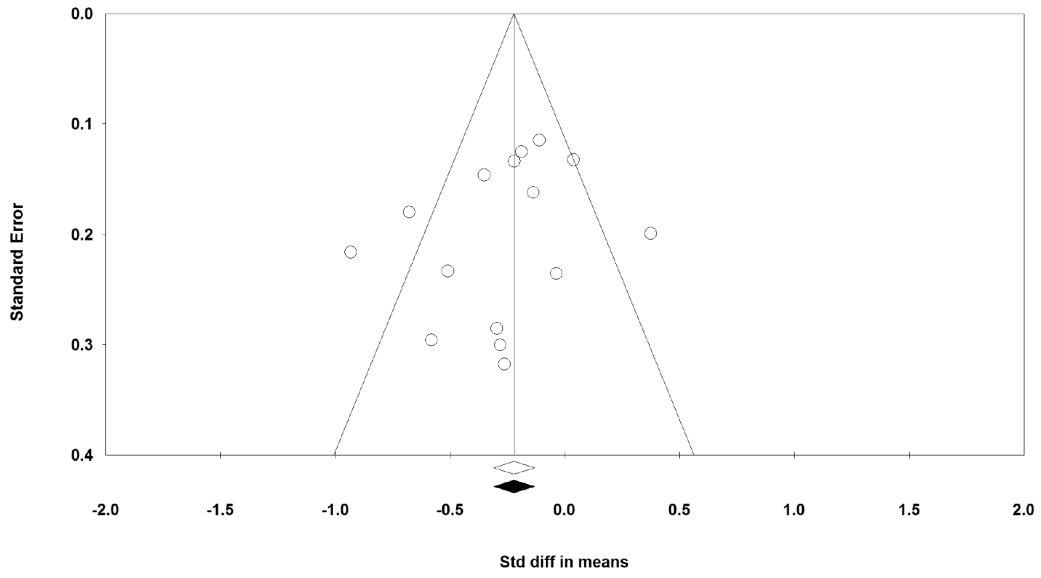


Figure 2.8. Funnel plot of observed (white) and imputed (black) studies for comparison: Cognitive Behaviour Therapy versus waitlist/usual care at post-test (N = 15 studies).

2.4.6.2 Intervention versus waitlist/usual care: Follow-up outcomes

There were fewer studies providing follow-up data than post-test data; however, the efficacy of five different interventions compared to WL/UC at 6-12 months is shown in Table 2.2. All interventions – ACT, CBT, hypnosis, mindfulness, and multimodal treatment – had significant medium effects. Meta-analysis was possible for ACT, CBT and multimodal treatment, as depicted with forest plots in Figures 2.9-2.11. The outlier [284] exerting an outsized effect on the post-test CBT meta-analysis was removed for the same reason. The quality of evidence ranged from very low (hypnosis) to moderate (CBT, Multimodal). Again, CBT had the most data (6 studies), but the considerable heterogeneity of effect sizes reduced confidence in the pooled effect estimate. As seen in Figure 2.10, this heterogeneity was mainly due to the influence of one study [122] with large effects. Multimodal treatment had more consistent effects, however the estimate was imprecise in the absence of a larger sample. The pooled effects of the meta-analysed interventions were not significantly different from each other (Q = 1.34, *df* = 2, *p* = .51).

Table 2.2. Effects of different interventions on pain catastrophising compared with waitlist/usual care at follow-up (6-12 months).

Intervention †	Participants (studies)	SMD effect (95% CI)	PCS change‡	Heterogeneity Q, I^2	Moderators	Evidence quality (GRADE)
All included studies						
ACT [135,328]	263 (2)	-0.60* (-1.06, -0.14)	-5.6	3.27, 69.45	-	Low [b d]
CBT [121,122,136,272,276,329]	1116 (6)	-0.39*** (-0.59, -0.19)	-3.6	13.04*, 61.64	Baseline PC	Moderate [b]
Hypnosis [318]	59 (1)	-.69* (-1.22, -0.17)	-6.4	-	-	Very low [a b d]
Mindfulness [136]	229 (1)	-0.46*** (-0.67, -0.24)	-4.3	-	-	Low [b d]
Multimodal [285,286]	285 (2)	-0.56** (-0.80, -0.32)	-5.2	0.44, 0.00	-	Moderate [d]
Only studies targeting elevated pain catastrophising [§]						
CBT [122]	95 (1)	-1.01*** (-1.44, -0.59)	-9.4	-	-	Moderate [d]
Multimodal [285,286]	285 (2)	-0.56*** (-0.80, -0.32)	-5.2	0.44, 0.00	-	Moderate [d]

Note: * $p<.05$, ** $p<.01$, *** $p<.001$. ACT, Acceptance and Commitment Therapy; CBT, Cognitive Behaviour Therapy; EFT, Emotional Freedom Techniques; SMD, standardised mean difference. † Studies included in each pooled effect. ‡ Change in Pain Catastrophising Scale (PCS) score calculated by multiplying SMD by average standard deviation of included studies that used PCS (SD = 9.34). [a] Downgraded due to risk of bias; [b] Downgraded due to inconsistency; [c] Downgraded due to indirectness of evidence; [d] Downgraded due to imprecision, [e] Downgraded due to publication bias. [§] Targeted interventions are those that treat pain catastrophising as a primary outcome and have cohorts with clinically significant levels of catastrophising (>24 equivalent on the PCS).

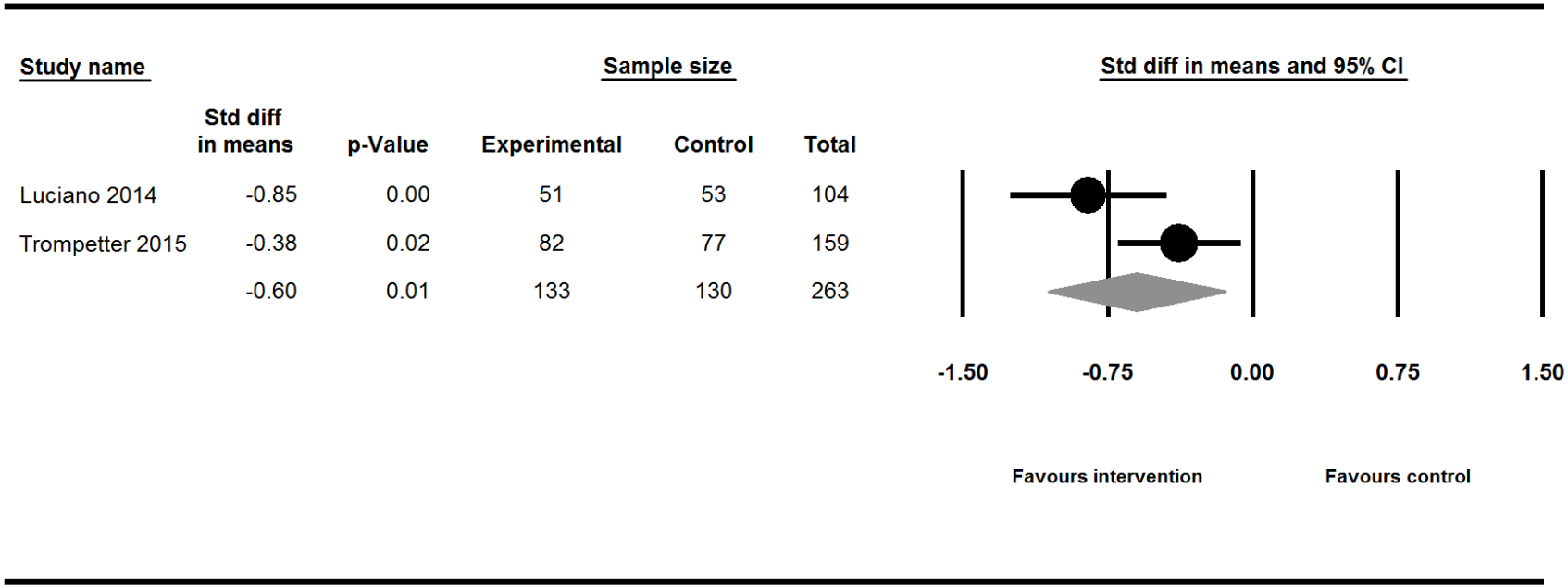


Figure 2.9. Pooled effects on pain catastrophising of Acceptance and Commitment Therapy versus waitlist/usual care at follow-up (6-12months).

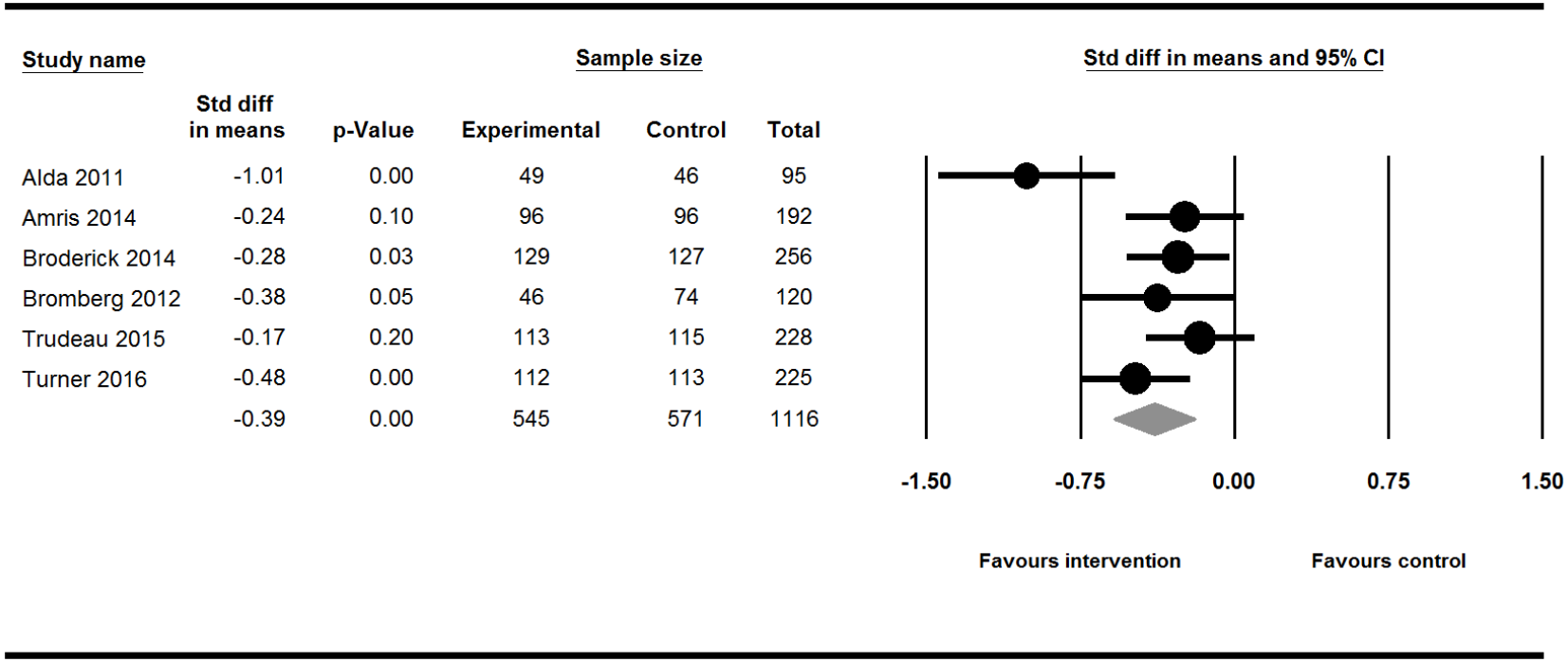


Figure 2.10. Pooled effects on pain catastrophising of Cognitive Behaviour Therapy versus waitlist/usual care at follow-up (6-12months).

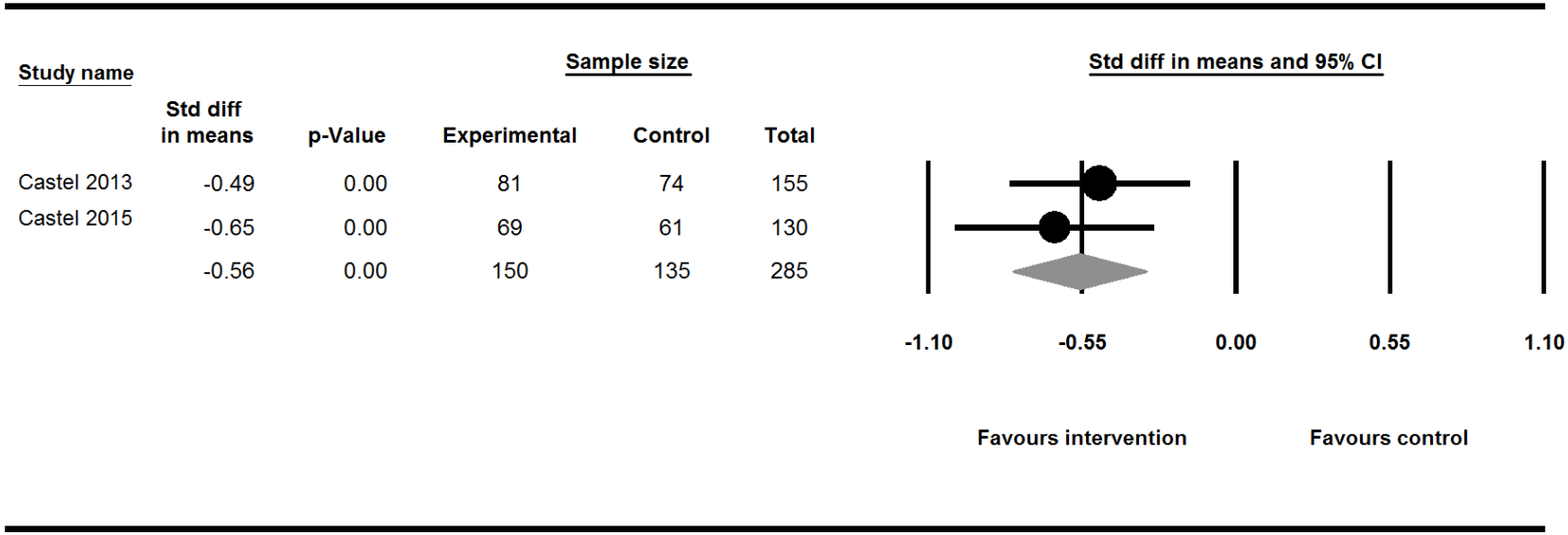


Figure 2.11. Pooled effects on pain catastrophising of multimodal treatment versus waitlist/usual care at follow-up (6-12months).

2.4.6.2.1 Moderator and sub-group analysis

The only intervention with sufficient studies for meta-regression was CBT. Baseline PC was a significant moderator of pooled effect (Q = 3.42, *df* =1, *p* = .06), favouring high baseline PC. Recalculating the pooled effect of CBT for studies with high baseline PC produced a larger effect (SMD = -0.69, 95% CI -1.31 to -0.06, $p < .05$) but increased heterogeneity (Q = 4.88, *df* = 2, *p* = .03, I^2 = 79.50). Similarly, the sub-group analysis of only targeted studies (see Table 2.2) resulted in a larger CBT effect (SMD = -1.01), although this was only based on one study.

2.4.6.2.2 Publication bias

There were not enough studies to reliably test for publication bias in any of the meta-analyses at follow-up.

2.4.6.3 Intervention versus active control: Post-test outcomes

Ten different interventions were tested against active control interventions, as represented by the 40 studies and 4191 participants in Table 2.3. Only half of these showed efficacy – ACT, CBT, exercise, hypnosis, and multimodal treatment – and effects were moderate except for multimodal treatment, which had a large effect. Meta-analysis was possible for the 7 interventions depicted with forest plots in Figures 2.12-2.18. Omitted from these analyses were studies that compared different variants of the same intervention type. [116,284,288,291,310,313,315,323,330,333,335,338] As shown in Table 2.3, there was high quality evidence for ACT (SMD = -0.44) based on four studies. Again, CBT provided the most data (12 studies); however, several studies had an unclear or high risk of bias, so the quality of this evidence (SMD = -0.47) was downgraded to moderate. Multimodal treatment had moderate quality evidence of a large effect (SMD = -1.00), which was downgraded due to high heterogeneity. All other interventions had low or very low quality evidence. Differences in pooled effects across meta-analysed interventions were not statistically significant (Q = 9.74, *df* = 6, *p* = .136).

Table 2.3. Effects of different interventions on pain catastrophising compared with active controls at post-test.

Intervention †	Participants (studies)	SMD effect (95% CI)	PCS change‡	Heterogeneity Q, I^2	Moderators	Evidence quality (GRADE)
All included studies						
ACT [135,281,300,328]	474 (4)	-0.44** (-0.69, -0.19)	-4.1	5.49, 44.32	Contact; format	High
Acupuncture [337]	126 (1)	-0.01 (-0.36, 0.34)	-0.1	-	-	Low [b d]
CBT [122,274,278,279,287,290,295,304,312,327,331,332]	1251 (12)	-0.47*** (-0.62, -0.33)	-4.4	16.18, 31.99	Facilitator; PC primary outcome	Moderate [a]
Education [292,299,305,341]	284 (4)	-0.52 (-1.14, 0.09)	-4.9	18.67***, 83.94	Baseline PC; contact	Very low [a b d]
Exercise [336]	139 (1)	-0.36* (-0.69, -0.02)	-3.4	-	-	Low [b d]
Graded exposure [302]	77 (1)	-0.34 (-0.79, 0.11)	-3.2	-	-	Low [b d]
Hypnosis [271,325]	169 (2)	-0.47** (-0.78, -0.16)	-4.4	0.04, 0.00	-	Low [a d]
Mindfulness [289,293,339]	281 (3)	-0.13 (-0.37, 0.10)	-1.2	0.26, 0.00	-	Low [a d]
Multimodal [270,273,306–309,314,317,322,334]	1258 (10)	-1.00*** (-1.54, -0.46)	-9.3	176.93***, 94.91	Baseline PC; facilitator	Moderate [b]
Pharmacotherapy [297,326]	132 (2)	-0.02 (-0.37, 0.32)	-0.2	0.52, 0.00	-	Low [a d]
Only studies targeting elevated pain catastrophising [§]						
CBT [122,312]	146 (2)	-0.84*** (-1.18, -0.50)	-7.8	0.23, 0.00	-	Low [a,d]

Education [299]	105 (1)	0.20 (-0.18, 0.58)	-1.9	-	-	Low [a,d]
Pharmacotherapy [326]	70 (1)	-0.14 (-0.61, 0.33)	-8.2	-	-	Low [a,d]

Note: * $p<.05$, ** $p<.01$, *** $p<.001$. ACT, Acceptance and Commitment Therapy; CBT, Cognitive Behaviour Therapy; EFT, Emotional Freedom Techniques; SMD, standardised mean difference. † Studies included in each pooled effect. ‡ Change in Pain Catastrophising Scale (PCS) score calculated by multiplying SMD by average standard deviation of included studies that used PCS (SD = 9.34). [a] Downgraded due to risk of bias; [b] Downgraded due to inconsistency; [c] Downgraded due to indirectness of evidence; [d] Downgraded due to imprecision, [e] Downgraded due to publication bias. [§] Targeted interventions are those that treat pain catastrophising as a primary outcome and have cohorts with clinically significant levels of catastrophising (>24 equivalent on the PCS).

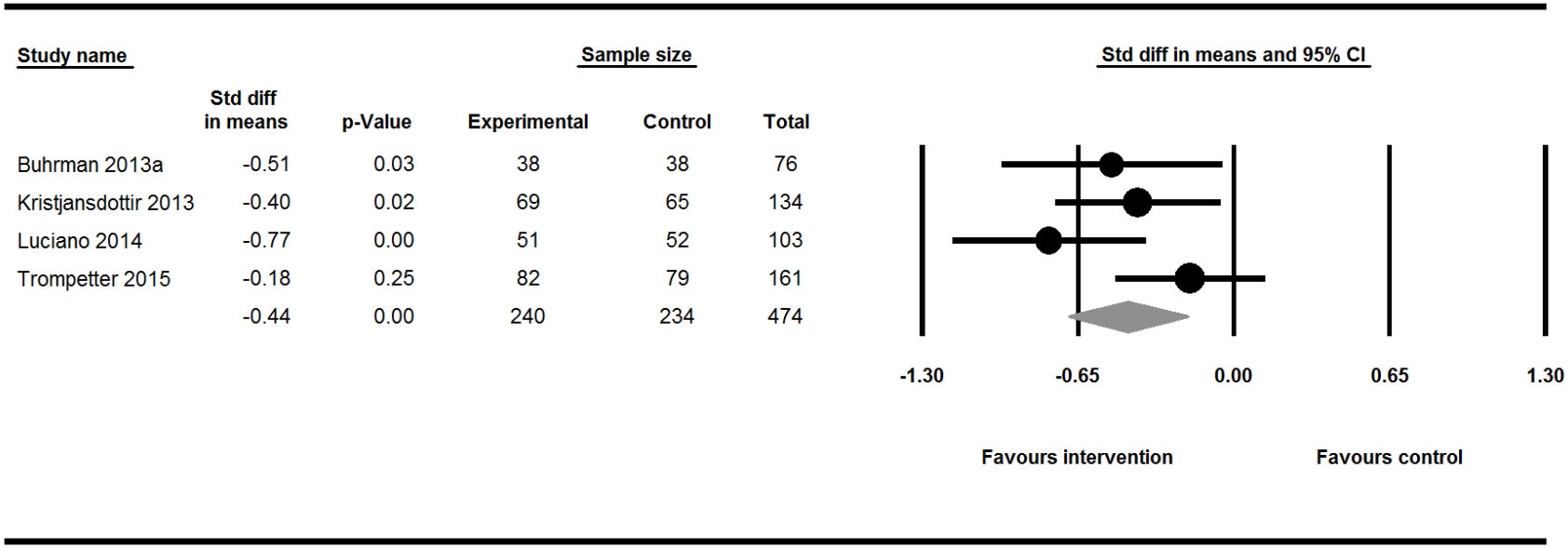


Figure 2.12. Pooled effects on pain catastrophising of Acceptance and Commitment Therapy versus active control at post-test.

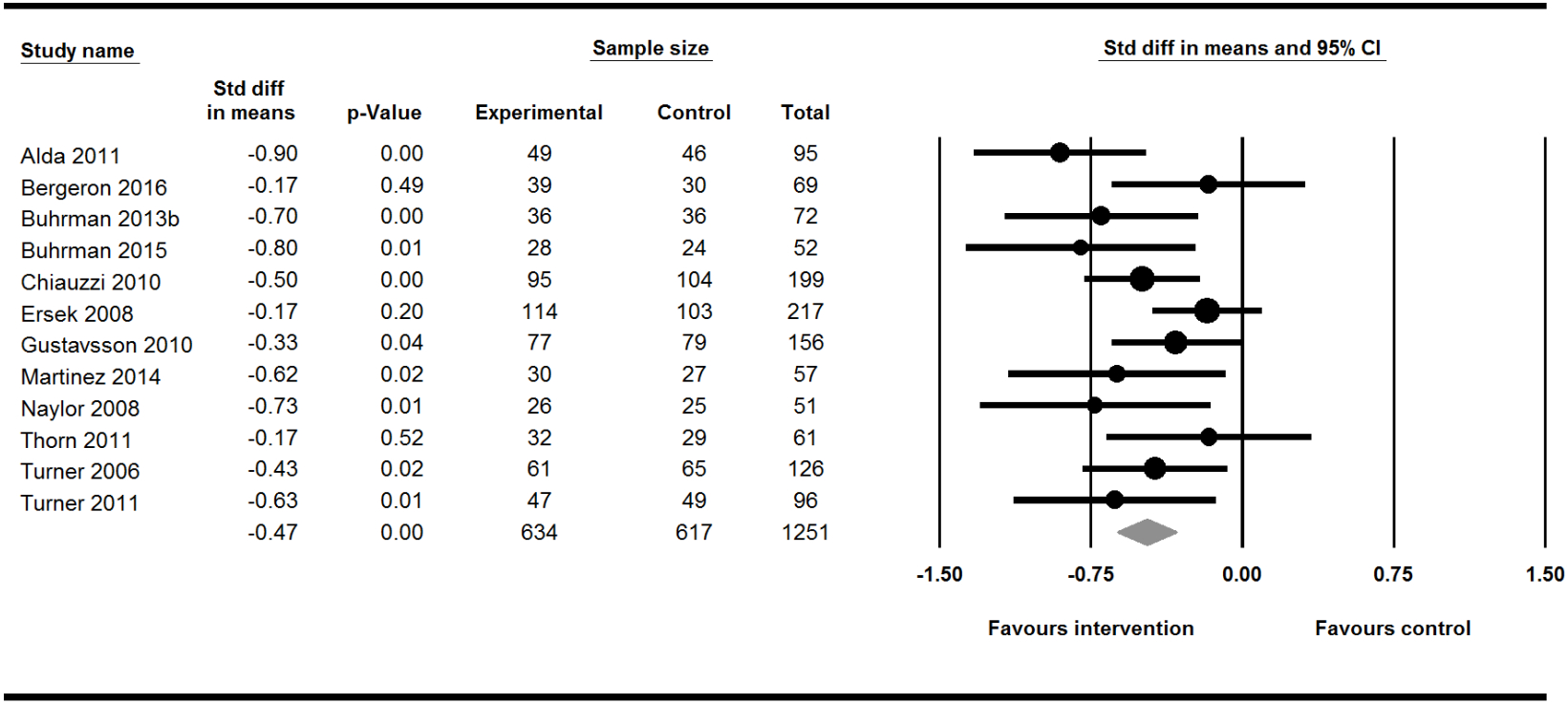


Figure 2.13. Pooled effects on pain catastrophising of Cognitive Behaviour Therapy versus active control at post-test.

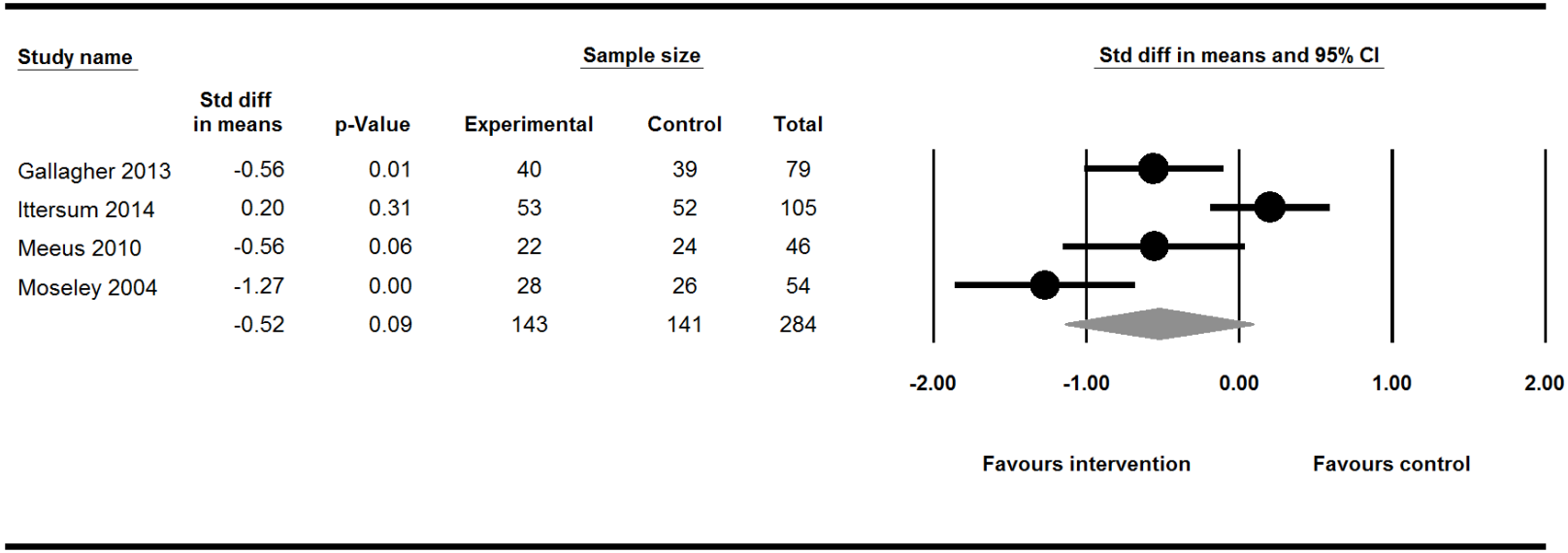


Figure 2.14. Pooled effects on pain catastrophising of education versus active control at post-test.

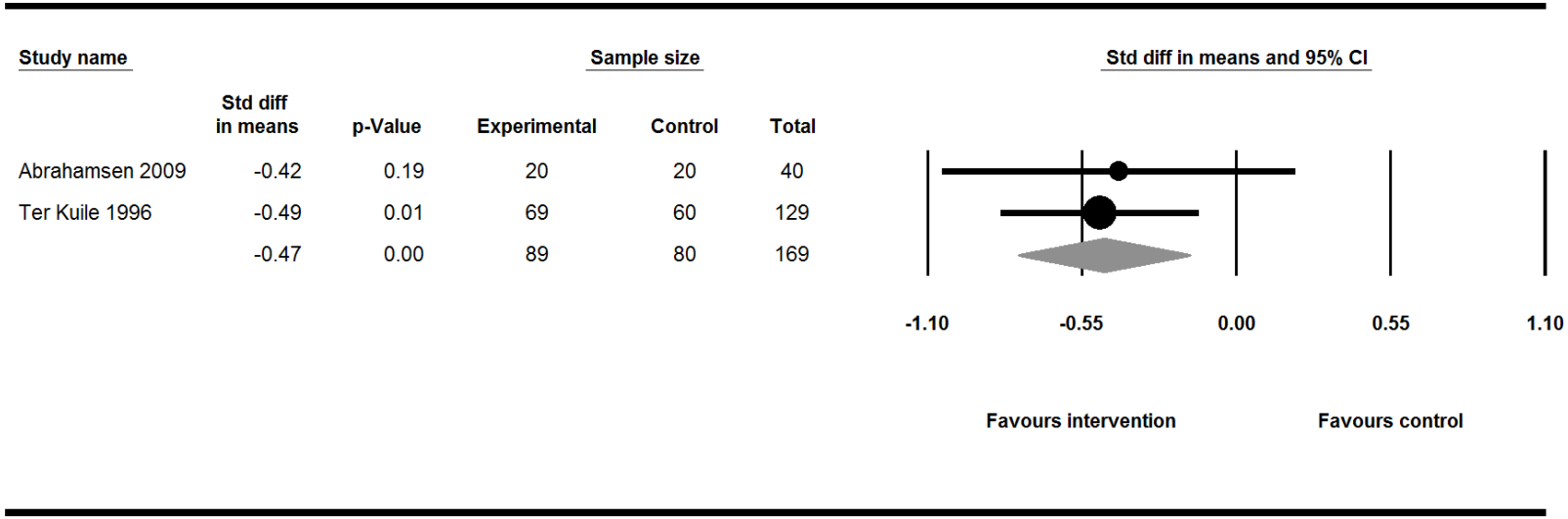


Figure 2.15. Pooled effects on pain catastrophising of hypnosis versus active control at post-test.

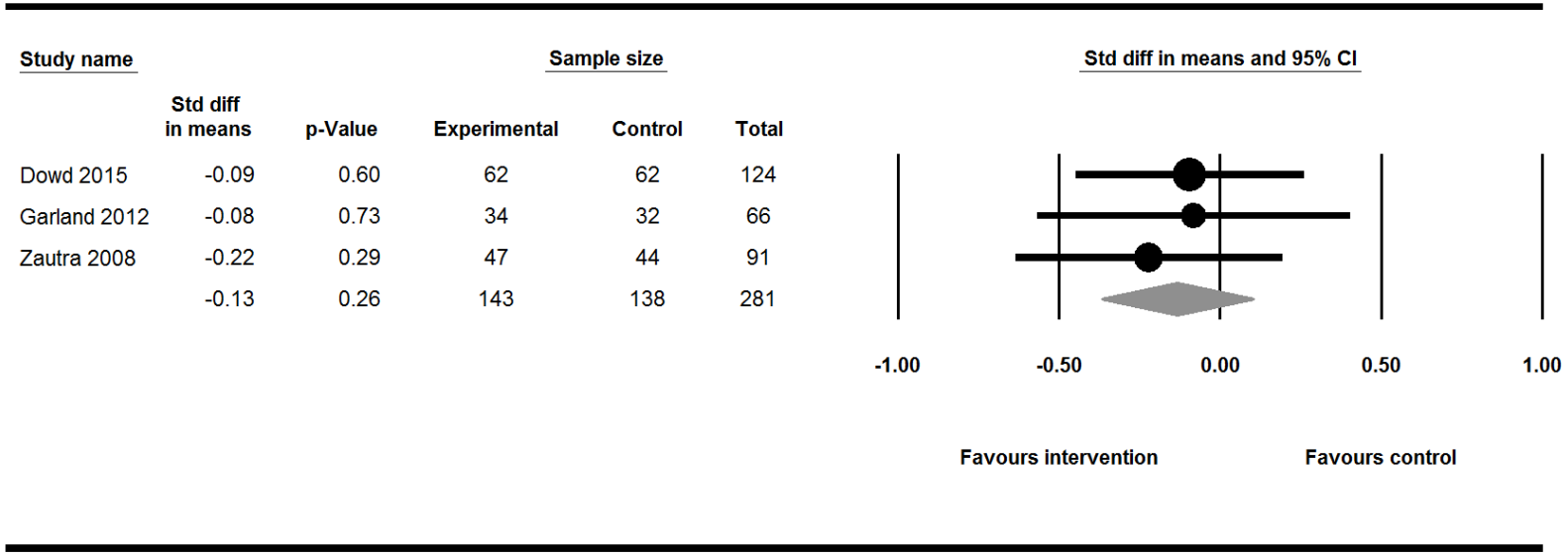


Figure 2.16. Pooled effects on pain catastrophising of mindfulness meditation versus active control at post-test.

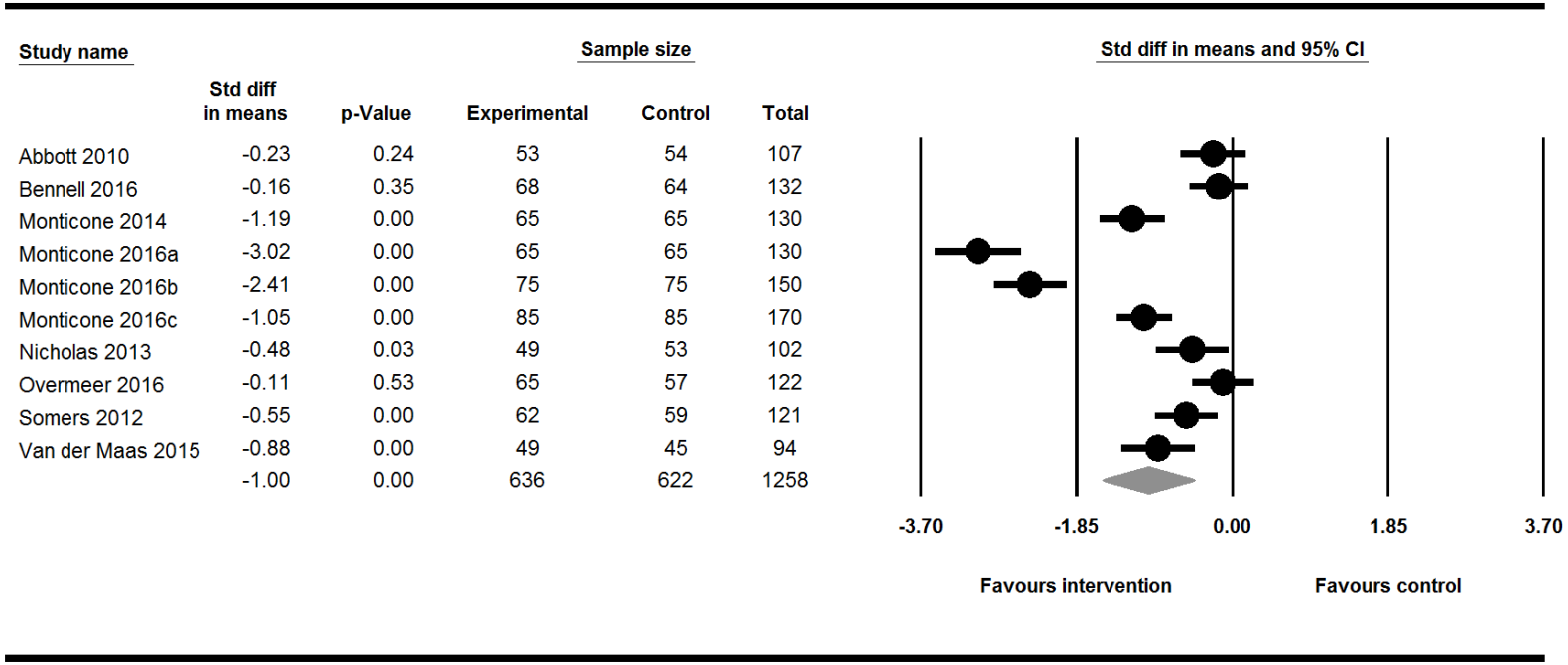


Figure 2.17. Pooled effects on pain catastrophising of multimodal treatment versus active control at post-test.

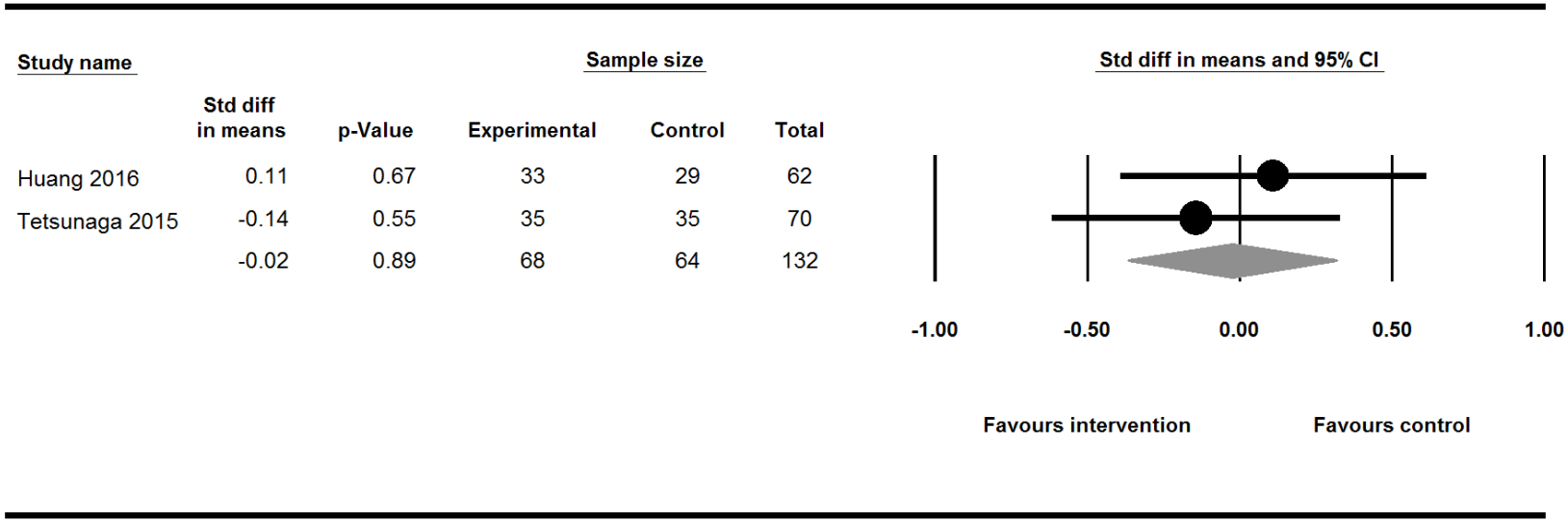


Figure 2.18. Pooled effects on pain catastrophising of pharmacotherapy versus active control at post-test.

2.4.6.3.1 Moderator and sub-group analysis

There were sufficient studies for meta-regression of ACT, CBT, education, and multimodal treatments. For ACT, facilitator contact time significantly moderated treatment effect (Q = 4.02, *df* = 1, *p* = .04), favouring more contact. Delivery format also moderated the effect (Q = 3.76, *df* = 1, *p* = .05), with face-to-face interventions superior to internet interventions. For CBT, facilitator type moderated effect (Q = 8.53, *df* = 4, *p* = .07), favouring psychologists. Whether PC was targeted as a primary outcome also influenced pooled effects (Q = 5.66, *df* = 1, *p* = .02), and limiting the CBT analysis to studies targeting PC produced larger effects (SMD = -0.65, 95% CI -0.85, -0.45. *p* < .001) with less heterogeneity (Q = 2.64, df = 3, *p* < .001, I^2 = 0.00). Baseline PC moderated the effect of education interventions (Q = 5.57, *df* = 1, *p* = .02), although unexpectedly favouring low baseline PC. Contact time influenced education effects as well (Q = 3.65, *df* = 1, *p* = .06), favouring more contact. Finally, for multimodal interventions baseline PC influenced outcome, favouring high PC as expected (Q = 4.33, *df* = 1, *p* = .04). Facilitator type also moderated multimodal outcomes, with multidisciplinary teams superior to physiotherapists (Q = 5.66, *df* = 1, *p* = .02). Limiting this meta-analysis to studies of multimodal interventions led only by multidisciplinary teams for people with high baseline PC increased the pooled effect (SMD = -1.52, 95% CI -2.45, -0.59. *p* < .001) but heterogeneity remained high (Q = 102.81, *df* = 1, *p* < .001, I^2 = 96.11).

The sub-group analyses of targeted high PC interventions showed that only CBT was effective. CBT had stronger and more consistent effects in this cohort (SMD = -0.84) but evidence quality was downgraded to low because of the small sample.

2.4.6.3.2 Publication bias

There were enough studies to test for publication bias in the CBT and multimodal intervention meta-analyses. As shown in Figure 2.19, the funnel plot for CBT displayed some asymmetry (Egger's test = -2.22, *df* = 10, *p* = .045), but trim and fill did not impute any missing studies that would alter the pooled effect size, suggesting the impact of any possible publication bias is trivial. As shown in Figure 2.20, the funnel plot for multimodal interventions also showed slight

asymmetry (Egger's test = -22.13, *df* = 8, *p* = .026), and trim and fill suggested one study in the same direction of the pooled effect was missing, which again suggests trivial effects of any publication bias.

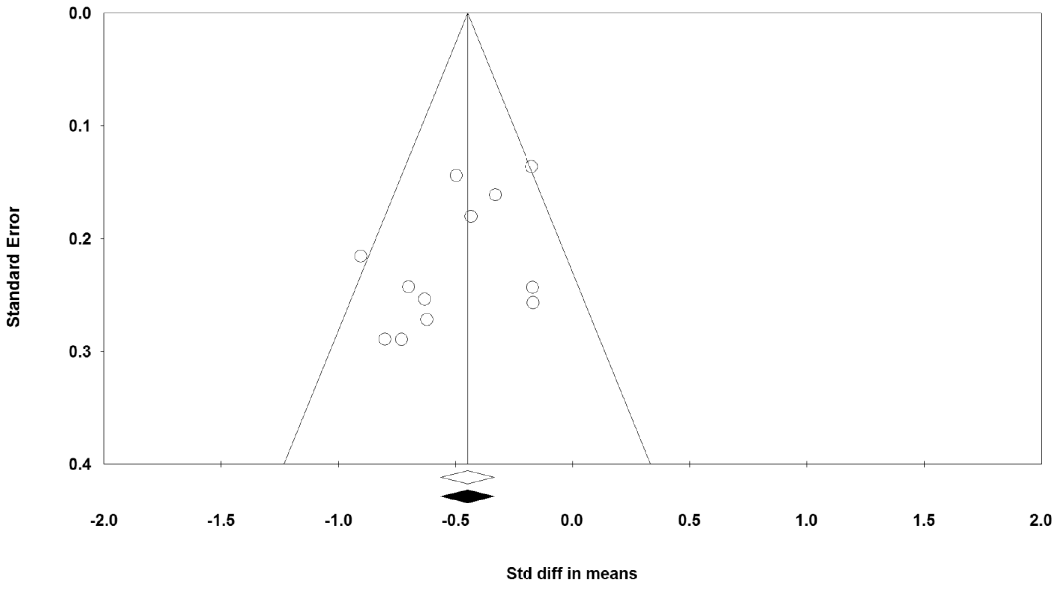


Figure 2.19. Funnel plot of observed (white) and imputed (black) studies in comparison: Cognitive Behaviour Therapy versus active control at post-test (N = 12 studies).

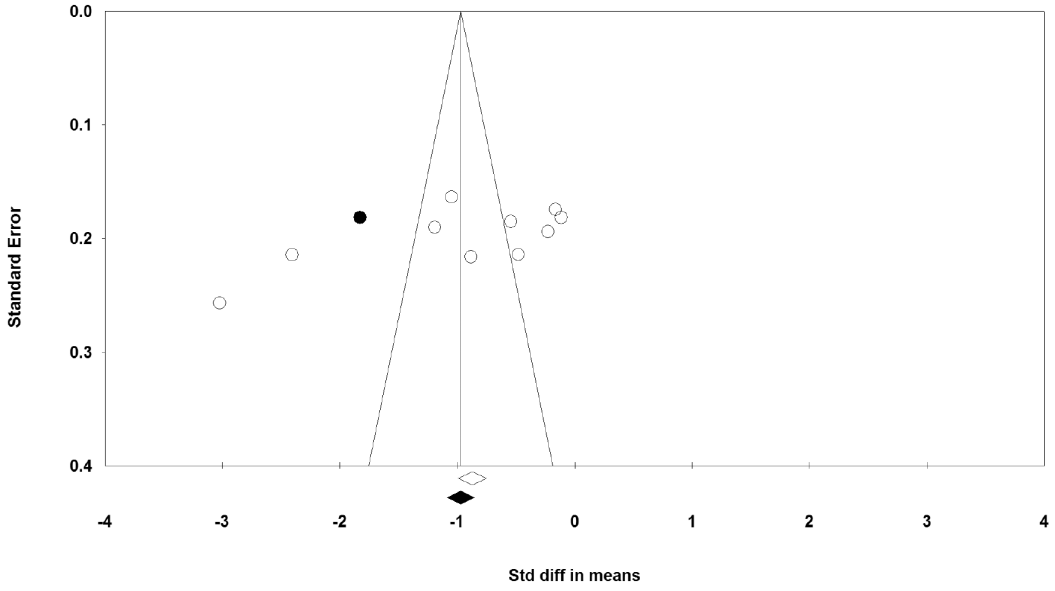


Figure 2.20. Funnel plot of observed (white) and imputed (black) studies in comparison: Multimodal treatment versus active control at post-test (N = 10 studies).

2.4.6.4 Intervention versus active control: Follow-up outcomes

Six different interventions were tested against active control groups at follow-up (22 studies, N=2653). However, as shown in Table 2.4, only 3 of these were efficacious: ACT, CBT, and multimodal treatment. These three interventions were suitable for meta-analysis, with forest plots shown in Figures 2.21-2.23. Omitted were studies that compared different variants of the same intervention type.[116,284,310,312,313,315,323,330,333,335–338,340] Like results at post-test, there was high quality evidence of a medium effect for ACT (SMD = -0.35). Moderate quality evidence was found for CBT having a medium effect (SMD = -0.30), which was downgraded due to heterogeneity. Multimodal treatment was downgraded for the same reason, yielding moderate quality evidence for a large effect (SMD = -1.39). Comparison of pooled effect sizes showed a significant difference in favour of multimodal treatment (Q = 7.51, *df* = 2, *p* = .023).

Table 2.4. Effects of different interventions on pain catastrophising compared with active controls at follow-up (6-12 months).

Intervention †	Participants (studies)	SMD effect (95% CI)	PCS change‡	Heterogeneity Q, I^2	Moderators	Evidence quality (GRADE)
All included studies						
ACT [135,281,300,328]	399 (3)	-0.35** (-0.59, -0.11)	- 3.3	2.86, 30.16	-	High
CBT [122,274,287,290,304,327,332,342]	928 (8)	-0.30** (-0.51, -0.09)	-2.8	16.08*, 56.46	Pain condition; PC primary outcome	Moderate [b]
Education [299]	105 (1)	0.15 (-0.23, 0.54)	1.4	-	-	Very low [a b d]
Graded exposure [302]	73 (1)	-0.07 (-0.53, 0.39)	-0.7	-	-	Low [b d]
Mindfulness [289]	124 (1)	0.00 (-0.35, 0.36)	0.0	-	-	Very low [a b d]
Multimodal [270,273,306–309,317,334]	1024 (8)	-1.39** (-2.27, -0.51)	-13.0	268.24***, 97.39	Baseline PC; facilitator	Moderate [b]
Only studies targeting elevated pain catastrophising [§]						
CBT [122]	95 (1)	-0.73** (-1.15, -0.32)	-6.8	-	-	Moderate [d]
Education [299]	105 (1)	0.15 (-0.23, 0.54)	-1.4	-	-	Low [a,d]

Note: * $p<.05$, ** $p<.01$, *** $p<.001$. ACT, Acceptance and Commitment Therapy; CBT, Cognitive Behaviour Therapy; EFT, Emotional Freedom Techniques; SMD, standardised mean difference. † Studies included in each pooled effect. ‡ Change in Pain Catastrophising Scale (PCS) score calculated by multiplying SMD by average standard deviation of included studies that used PCS (SD = 9.34). [a] Downgraded due to risk of bias; [b] Downgraded due to inconsistency; [c] Downgraded due to indirectness of evidence; [d] Downgraded due to imprecision, [e] Downgraded due to publication bias. [§] Targeted interventions are those that treat pain catastrophising as a primary outcome and have cohorts with clinically significant levels of catastrophising (>24 equivalent on the PCS).

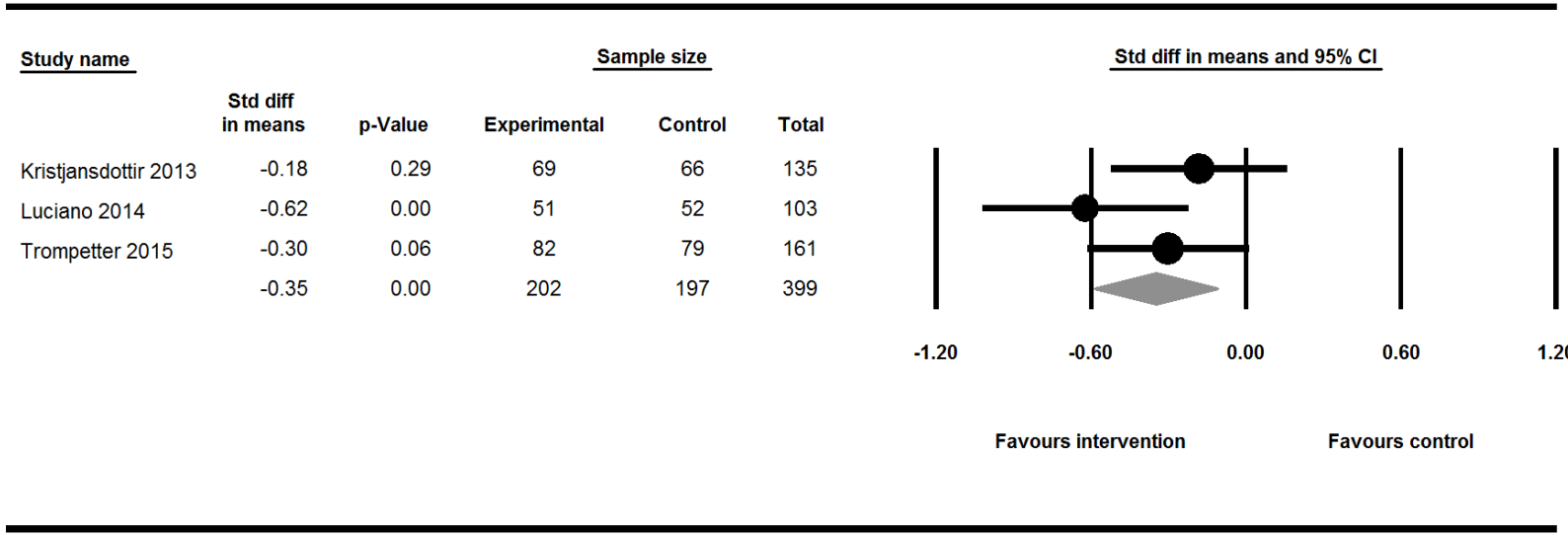


Figure 2.21. Pooled effects on pain catastrophising of Acceptance and Commitment Therapy versus active control at follow-up (6-12 months).

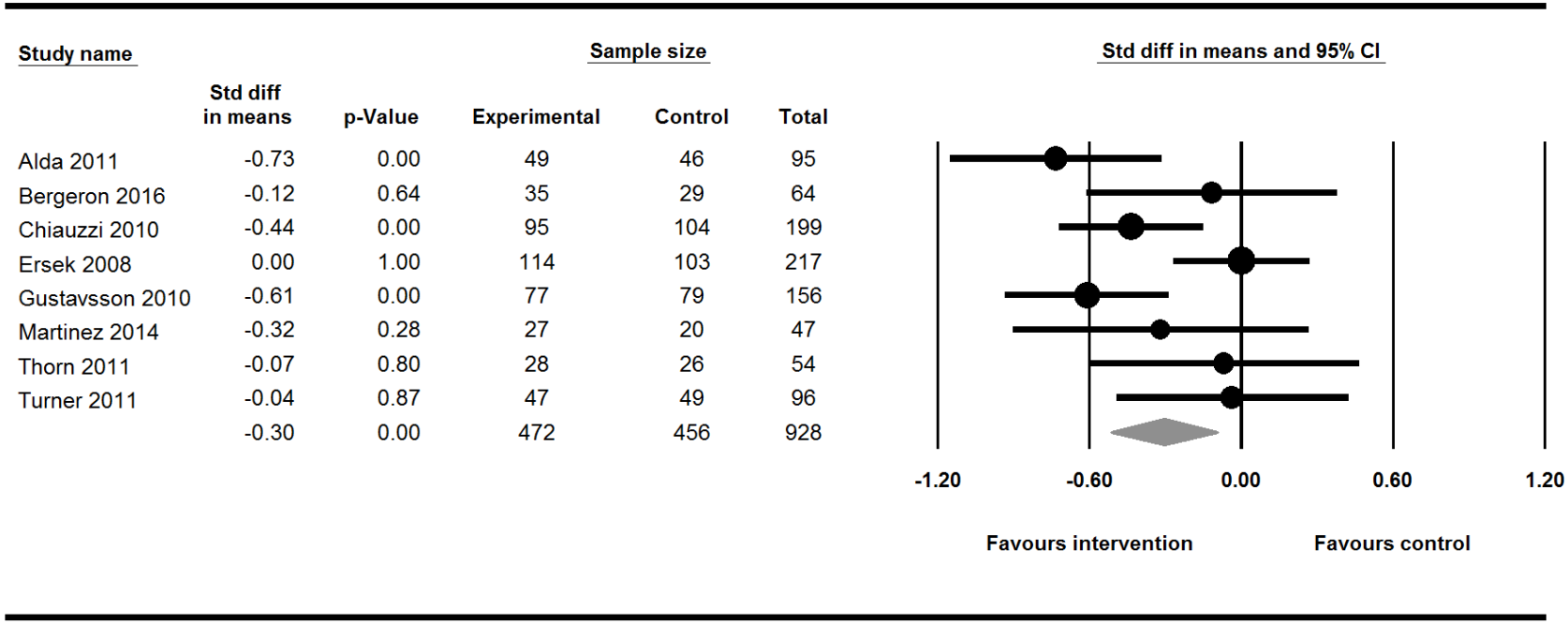


Figure 2.22. Pooled effects on pain catastrophising of Cognitive Behaviour Therapy versus active control at follow-up (6-12 months).

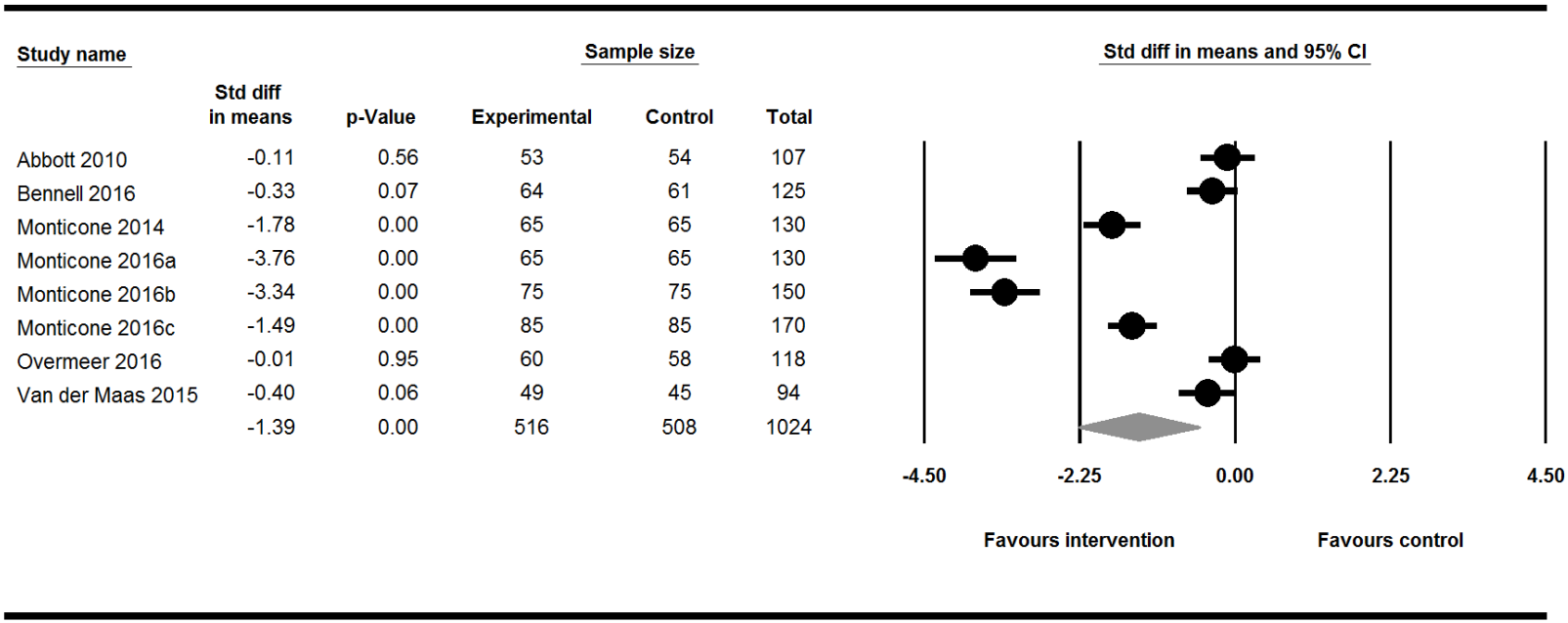


Figure 2.23. Pooled effects on pain catastrophising of multimodal treatment versus active control at follow-up (6-12 months).

2.4.6.4.1 Moderator and sub-group analysis

There were sufficient CBT and multimodal studies for regression-based moderator analysis. Pain condition moderated the effect of CBT (Q = 14.74, *df* = 5, *p* = .01), favouring spinal pain and fibromyalgia over vulvodynia and temporomandibular disorders. Targeting PC as a primary outcome also moderated CBT effects (Q = 14.74, *df* = 5, *p* = .01). Limiting this meta-analysis to studies targeting PC increased the CBT effect (SMD = -0.54, 95% CI -0.82, -0.26, *p* < .001) and decreased its heterogeneity (Q = 1.31, *df* = 1, *p* = .25, I^2 = 25.42). For multimodal interventions, baseline PC moderated effects (Q = 19.50, *df* = 1, *p* < .001), favouring high PC. Facilitator type also moderated effects (Q = 7.85, *df* = 1, *p* = .005), favouring multidisciplinary teams over physiotherapists. Limiting this meta-analysis to studies with high baseline PC and multidisciplinary facilitators increased effects (SMD = -2.95, 95% CI -4.20, -1.71, *p* < .001), but heterogeneity remained high (Q = 39.12, *df* = 2, *p* < .001, I^2 = 94.89). Sub-group analyses of only targeted interventions echoed post-test findings, with only CBT showing efficacy, albeit with greater strength (SMD = -0.73).

2.4.6.4.2 Publication bias

There were not enough studies to reliably test for publication bias in any of the meta-analyses at follow-up.

2.5 Discussion

This is the first systematic review to focus on reductions in PC using any type of intervention. There were three related aims: (1) to systematically review and describe randomised controlled trials that measure changes in catastrophising changes in chronic non-cancer pain; (2) to document and compare the pooled effects of different interventions; and (3) to identify factors that moderate the efficacy of these interventions. Considering the first of these, there is clearly a large body of literature examining treatment-related changes in PC, providing

strong evidence that PC is a modifiable characteristic. This review, using strict methodological inclusion criteria, yielded a considerable 79 studies representing 9914 people with mostly musculoskeletal pain, although it is possible that this is an incomplete retrieval of available data. However, only a minority (40.5%) of these studies targeted PC as a primary outcome. This probably reflects the fact that PC is usually seen as a process variable [94,113,343] and many of the included studies were secondary mediation analyses of broader trials. Only 8 studies targeted PC and also included cohorts with high PC, suggesting there are relatively few high-quality trials matching treatments to this particular risk factor.

While the methodological quality of the included research was generally good, with most studies (61%) receiving a low risk of bias rating, there was scope for improving performance bias, attrition bias and reporting bias. For example, more of the studies using active controls could have made explicit attempts to control for expectancy effects by concealing study hypotheses. There were also still too many studies either not accounting for attrition with ITT analysis or using ITT with unreliable imputation methods such as last observation carried forward. [260] Finally, it was disappointing that so few recent studies had a low risk of reporting bias given that publication guidelines have required prospective registration of clinical trials since at least 2005. [344,345]

The second aim of this review has perhaps the most clinical utility – documenting and comparing effect sizes across interventions. The general finding when considering all included studies was that several interventions work modestly well in reducing PC and multimodal treatments combining CBT and exercise may work best, although further high quality research is needed to confirm this. Nine different interventions showed efficacy at post-test when compared to waitlist/usual care, although considering only treatments with at least moderate quality, there was evidence for: ACT, CBT, exercise, mindfulness, and multimodal treatment. When compared to active controls, three treatments stood out: CBT, multimodal treatment and ACT. An encouraging finding was that treatment benefits were largely maintained at follow-up, which may suggest that these interventions involve skill acquisition that translates to at least medium-term behaviour change.

When all 79 studies were considered, most of the pooled effect sizes observed

were of medium strength (SMD = 0.3-0.7). Converting this to scores on the most common scale of PC suggests reductions of roughly 3-7 points on the 52-point Pain Catastrophising Scale. [83] The obvious question is whether this is clinically meaningful, which is difficult to answer with only group level data reported in the included studies. Although several methods for assessing clinical significance exist, one common approach looks for reliable change unlikely to be caused by measurement error, along with movement from a clinical range to a non-clinical range. [346] There is evidence that minimum reliable change on the PCS is around 20%, [347] while a conservative clinical cut-off for the PCS is a score of 20. [265,348] Based on this, minimum clinically significant change would be a reduction of 5 points on the PCS (baseline 24, post-test 19), which equates to an effect size of SMD = -0.54, given that the average standard deviation of the PCS in this review was 9.34. Considering only those interventions with at least moderate quality evidence, this condition was only satisfied by multimodal treatment when all interventions were considered, and by both CBT and multimodal treatment when only targeted studies were considered.

There are several plausible reasons why multimodal treatments might have shown larger effect sizes. [79] Firstly, since multimodal treatment usually combined CBT and exercise, it is possible that these components had additive effects. Considering the fear-avoidance model, if a person's catastrophising involved magnified threat cognitions relating to injury or functional limitations, [71,349] exercising without catastrophic outcomes could function as a form of behavioural experiment aiding cognitive restructuring through the provision of disconfirmatory evidence.[154] Similarly, exercise may help to shift attention away from rumination due to its attentional demands and mood effects, [350] while the use of exercise as a self-management tool could increase self-efficacy and thereby reduce helplessness. Finally, given evidence of a bidirectional relationship between pain intensity and PC, [117] the modulation of descending inhibitory control mechanisms associated with paced exercise [351] may indirectly reduce PC via pain reduction. These effects could occur in addition to the positive effects of traditional CBT components. However, another possibility based on the patient-treatment matching model discussed below, [131] is that these broader spectrum multimodal treatments have a greater chance of matching at least one treatment component to a patient strength or deficit.

Unfortunately, the evidence for multimodal treatments in this review is marred by a high level of heterogeneity in effect sizes. This was primarily due to the influence of much larger effect sizes for studies coming from a single research group. [306–309] It is difficult to account for these differences in terms of treatment content or other factors and, while there is no methodological reason to exclude these studies, this unexplained heterogeneity lowers confidence in the pooled effect estimate. Indeed, heterogeneity was a significant problem for other interventions and was the most common reason for evidence quality to be downgraded. This likely relates to the lack of consistency among interventions, although the absence of detailed manualisation of many treatments included in this review makes it difficult to compare intervention content.

It was possible to reduce heterogeneity by including moderator variables. This highlights the third aim of this study, which was to document moderators of PC treatment effect. Where meta-regression was possible, the most consistent moderators were baseline PC and whether PC was a primary outcome. Indeed, in the sub-group analyses of only studies targeting high PC, effect sizes were significantly higher and heterogeneity lower. For example, CBT versus active control at post-test increased from SMD = -0.47 to SMD = -0.84 and CBT was the only effective treatment in the active control group analyses. However, this probably reflects the lack of targeted studies using other interventions, pointing to the need for more research using other interventions with targeted samples.

In general, it is likely that effect estimates from meta-analyses that included all 79 eligible studies were diluted by the lower baseline PC scores in many samples, again suggesting more targeted studies using high risk cohorts are needed. Further research is also needed to explore whether people with moderate levels of PC still benefit from a reduction in PC, or whether others benefit from resilience-oriented early interventions that prevent future elevated levels of catastrophising.

Improving the efficacy and efficiency of our PC treatments may require re-examining the construct and, to echo several commentators, [127,352] clarifying psychological treatment mechanisms as well as developing algorithms for how to match treatment components to patient profiles. The Limit, Activate, Enhance model of psychosocial pain management moderation provides one useful framework for operationalising patient-treatment matching. [131] This model

suggests treatments need to be tailored to: a) limit a person's maladaptive coping responses, b) activate or increase their healthy behaviours, and c) enhance outcomes by optimising existing individual strengths.

Catastrophising is usually seen as a factor to limit within this model [131]; however, it is possible that sub-groups of people with elevated PC may also exist, requiring differentially targeted interventions. For example, cognitive restructuring in CBT may act as a 'limit-oriented' therapy to help someone high on the magnification subscale of the PCS who also lacks a clear understanding of their chronic pain. In addition, pain neurophysiology education, for example using the Explain Pain syllabus, [43] may serve as an 'activate-oriented' therapy to generate coping statements (e.g. "motion is lotion") that in turn may facilitate other adaptive behaviours like paced exercise. Conversely, someone whose catastrophising is characterised mainly by rumination may respond well to a third-wave intervention like ACT or mindfulness meditation which focuses on interrupting preservative thinking (i.e. limit-oriented therapy), particularly if they exhibited a characteristic that could be enhanced, such as previous meditation experience.

While future research is needed to explore this, one implication for clinicians is that there is currently no single gold standard for treating people in pain with elevated levels of catastrophising. This review shows that a range of approaches work to some extent and it seems likely that matching treatment components to specific phenotypes of patient strengths and deficits may be one way to optimise outcomes. Different strength/deficit profiles may also constitute specific catastrophising phenotypes that might be documented through further research. As others have noted,[192] research is needed to explore ways of increasing the efficacy of treatments by matching their content to particular clinically-relevant dimensions of PC rather than the construct as a whole.

2.5.1 Conclusions

A large body of evidence shows PC is a modifiable characteristic. Several interventions show efficacy; however, ignoring the poorer quality evidence, three treatments stand out: CBT, multimodal treatment and ACT. Effect sizes were generally modest and in many cases, may not be clinically meaningful. Treatments

are most likely to produce clinically significant benefits when they are targeted to people with high levels of catastrophising and CBT has the best evidence in these cohorts. Future research should focus on testing theory-driven interventions for PC in targeted samples of people with elevated catastrophising while matching treatment components to specific patient characteristics.

Chapter 3 Study 2 – 'I call it stinkin' thinkin'': A qualitative analysis of metacognition in people with chronic low back pain and elevated catastrophising

Chapter Linking Statement

The previous study shows there is no clear gold standard for reducing PC and that effect sizes are generally modest and of questionable clinical significance. This suggests there is room to improve treatments for PC. The systematic review also suggested that improving treatments may involve clarifying the construct of PC, including identifying different PC phenotypes that can then be better matched to specific treatments. Qualitative methods are well suited to gaining a deeper understanding of phenomena such as PC, shedding light on their lived experience. Study 2 therefore fills this gap in the literature, given there are no qualitative studies to date that target a sample of people with elevated PC.

The systematic review also showed that it was not only interventions based on an appraisal model of PC (e.g. CBT) that had efficacy. The high quality evidence for ACT, a so-called third-wave psychological treatment that targets cognitive processes rather than cognitive contents, suggests there may be value in investigating other cognitive process-oriented approaches to PC. Metacognitive Therapy (MCT) shares with ACT a focus on interrupting the processes of rumination and worry. Therefore, the following study qualitatively explores metacognition in people with elevated PC as a first step towards documenting pain-related metacognitions. This will pave the way for developing a measure and model of these higher order beliefs, which might then be incorporated into treatment research.

Publication History

This chapter was published in the *British Journal of Health Psychology*. The published typescript of the same text is reproduced in Appendix M:

Schütze, R., Rees, C. S., Slater, H., Smith, A., & O'Sullivan, P. (2017). 'I call it stinkin' thinkin'': A qualitative analysis of metacognition in people with chronic low back pain and elevated catastrophising. *British Journal of Health Psychology*, 1–18. http://doi.org/10.1111/bjhp.12240

3.1 Abstract

Objectives: Pain catastrophising is widely studied in quantitative pain research because of its strong link with poor pain outcomes, although the exact nature of this construct remains unclear. Focusing on its ruminative dimension, the present qualitative study aimed to explore a nascent aspect of pain catastrophising – metacognition – by documenting people's attitudes towards rumination and examining how these metacognitions might influence the course it takes.

Design: Qualitative interview study.

Methods: Semi-structured interviews were conducted in a tertiary care setting with 15 adults experiencing chronic (≥6 months) low back pain who scored highly (≥30) on the Pain Catastrophising Scale. Transcripts were analysed using interpretative phenomenological analysis.

Results: The first aim of documenting pain metacognitions revealed both positive (e.g. 'thinking helps me to cope') and negative (e.g. 'rumination is uncontrollable') attitudes towards pain rumination. These were often held simultaneously, creating internal conflict. The second aim of exploring the influence of metacognition on rumination showed that both negative and positive metacognitions could fuel perseverative thinking. However more nuanced negative metacognitions (e.g. 'worry is pointless') could help to end episodes of rumination by motivating the use of concrete problem solving or active coping behaviours.

Conclusions: While most participants described pain rumination as uncontrollable and harmful, dwelling on pain could be helpful when focused on tangible and solvable problems, thereby translating into adaptive coping behaviours that eventually interrupt rumination. Future treatments may be more effective if they are based on individualised formulations of pain catastrophising that focus on its perseverative nature and implicit function.

3.2 Introduction

Pain is a multidimensional subjective experience involving complex interactions between biological, psychological, genetic and social factors, as described by the dominant biopsychosocial model of pain. [1] Chronic pain – defined as pain that persists beyond 3-6 months [2] – is strongly associated with poor self-rated health, lower quality of life and psychopathology. [353] There is growing evidence that psychological factors influence maladaptive behavioural and nervous system changes that in themselves may perpetuate pain. [19] One of the most widely studied of these psychological constructs is pain catastrophising (PC).

Broadly defined, PC is a negative psychological response to pain and has various dimensions, including a tendency to ruminate about the pain experience, exaggerate its threat value, and underestimate one's ability to cope with it. [78,79] Although not all people in pain catastrophise, elevated PC predicts a range of negative outcomes, including greater pain intensity, [93] functional disability, [92] emotional distress, [354] health care utilisation, [96] and time off work. [90] It is also an important process variable in treatment, mediating or moderating the effects of evidence-based interventions such as cognitive behaviour therapy (CBT), exercise, and exposure in vivo. [114,355] This makes elevated PC a key treatment target in gold standard multidisciplinary pain rehabilitation programs. [120]

However, ambivalence about how best to modify PC is evidenced by the fact that a range of interventions – from exercise to cognitive therapy – are about equally effective, although effect sizes are generally modest. [114] The most evidence exists for CBT, with medium effects of d=0.53 to d=0.63 in reducing PC[vi]. [120] However, emerging evidence suggests that mindfulness-based approaches, which address the rumination aspect of PC as a form of repetitive negative thinking rather than addressing the content of catastrophic thoughts, may have larger effect sizes of d=0.62 to d=0.94. [134,356] Indeed, a recent high quality trial comparing CBT and Mindfulness-Based Stress Reduction (MBSR) found MBSR was superior in reducing PC. [136] Given this heterogeneity of treatments for PC, their modest effect sizes, and the existence of several competing theoretical models of PC, there have

[vi] This chapter was published before the results of Study 1 were finalised, which is why it is not referenced here.

been calls to re-examine this construct so that more effective and efficient interventions might be developed. [178,357,358]

In light of this, two recent models focus particularly on the function of PC. Eccleston and Crombez [195] articulate a model of pain-related worry that suggests it is a form of misdirected problem solving that ultimately backfires. Similarly, Flink and colleagues [178] argue that PC should be renamed 'catastrophic worry' to prioritise the rumination process over the contents of cognition. They describe PC as a form of *repetitive negative thinking* whose putative function is to reduce negative affect, partly by keeping cognition abstract. Indeed, the perseverative thinking aspect of PC warrants further investigation, given the dominance of the rumination subscale in accounting for most variance in the most common measure of PC – the Pain Catastrophising Scale – [83] as well as the encouraging effect sizes emerging for treatments that specifically target the cognitive process of rumination in PC[vii]. [134,356]

Exploring rumination in PC implicates metacognition, or the beliefs people hold about their own thinking. [178,358] So-called third wave psychological interventions that aim to disrupt rumination through cognitive defusion/decentring techniques encourage metacognitive change, where unhelpful metacognitive beliefs such as, "thoughts are facts" give way to metacognitions like, "thoughts are passing mental events". [238,240] This allows thoughts to be selectively – rather than rigidly – attended to through the cultivation of mindfulness.

However, arguably the most complete account of the role of metacognition in maintaining rumination is the Self-Regulatory Executive Function (S-REF) model of emotional disorder. [204,359] The S-REF model forms the basis of Metacognitive Therapy (MCT), [206] which was originally developed to treat perseverative worry in generalised anxiety disorder but has efficacy for numerous disorders of anxiety and depression, possibly even surpassing the efficacy of CBT. [224]

[vii] Consistent with this argument, PC will be defined in this paper as repetitive/perseverative thinking about pain. Although worry and rumination are often distinguished from each other based on the contents of cognition focusing on the future versus the past respectively, [374] for parsimony both forms of perseverative thinking will be treated as instances of PC and referred to as *pain rumination*.

A distinctive feature of MCT is that people's beliefs about their own thinking are key drivers of rumination, which is regarded as a self-regulation strategy. For example, a positive metacognitive belief about worry such as "worrying helps me to solve problems" fuels perseverative thinking by ascribing it a useful function, while a negative metacognition such as "my worry is uncontrollable" may also fuel such rumination by causing one to abandon efforts to disengage from it. [205] These metacognitions become targets for treatment in MCT. Recently, a correlational study provided the first evidence that these metacognitions are important aspects of PC. In a non-clinical student sample, Spada and colleagues found that positive metacognitions mediated between neuroticism and PC, while negative metacognitions mediated between PC and self-reported pain behaviours. [228]

Given these emerging findings and the theoretical relevance of metacognition to functional models of PC, the present study aimed to further explore metacognition in PC. A qualitative phenomenological approach was taken to gather rich data about metacognition from the idiographic perspective of people with CLBP and elevated PC. This idiographic approach is important given that, despite extensive quantitative research on PC, [93] as well as qualitative research on various aspects of chronic pain more generally, [360] there are no qualitative studies targeting a purposive sample of people with elevated PC, who are likely to engage in frequent rumination. Focusing on the ruminative aspect of PC, the aims of this study were therefore to: 1) document beliefs (metacognitions) about pain-related rumination in people with a tendency towards this type of thinking; and 2) explore how these metacognitions might influence the course of pain rumination.

3.3 Method

3.3.1 Participants

Fifteen Caucasian adults with CLBP (duration ≥6 months) who were seeking treatment from a medical specialist (orthopaedic surgeon, neurosurgeon or pain medicine specialist) were recruited. Small, purposive samples are typically sought for interpretative phenomenological analysis (IPA) to maintain idiographic focus

on individuals' experiences. The current sample constituted a fairly large sample for this methodology, which does not use the contested notion of saturation [361] in guiding sample size. [362] People with elevated PC were selected because elevated PC is often a treatment target, [78] so understanding how metacognition operates in this cohort is a starting point for improving their clinical outcomes in the future. The following exclusion criteria applied: insufficient English language proficiency for interview; likely cognitive interference due to high dose opiate medication (>100mg daily oral morphine equivalent); scoring below 30 on the Pain Catastrophising Scale, above which is a suggested threshold for 'severe' or clinically significant PC. [143]

Fifty-eight people were screened, with 43 excluded for scoring below 30 on the PCS. Table 3.1 summarises key demographic information for the included participants, as well as several quantitative pain outcomes based on the following psychometrically sound self-report measures, which are described more fully in Chapter 4: Brief Pain Inventory (BPI) [363] measuring pain intensity and pain interference; Pain Catastrophising Scale (PMS) [83] measuring pain catastrophising; Meta-Cognitions Questionnaire (MCQ-30) [364] measuring metacognition; and Hospital Anxiety Depression Scale (HADS) [365] measuring anxiety and depression.

Table 3.1. Participant demographics and outcomes on self-report measures of pain and psychological functioning.

Pseudonym	Sex	Age (yrs)	Marital status	Employment	Pain duration (years)	Pain intensity	Pain interference	Pain catastrophising	Meta-cognition	Anxiety	Depression
Gail	F	32	Married	Part time	3.5	7.2	7.3	47	75	10	8
Rachel	F	64	Married	Retired	4	7.5	7.7	30	44	7	12
Ramo	M	55	Married	Sick leave	.5	6.0	7.9	42	61	13	6
Pauline	F	58	Married	Home duties	47	4.0	7.9	38	51	5	7
Rhonda	F	53	Single	None - pain	10	9.0	9.1	51	91	11	12
Edna	F	71	Widow	None - pain	10	3.8	3.3	47	70	7	4
Kate	F	35	Married	Part time	8	6.5	9.1	40	67	14	11
Sandra	F	36	Married	Full time	1.5	5.8	7.9	35	62	12	4
Daniel	M	60	Married	None - pain	5	7.0	7.9	35	30	2	3
Jack	M	76	Married	Retired	1.5	3.2	0.7	30	56	5	1
Jacinta	F	55	Married	None - pain	26	6.2	6.9	30	93	12	12
Kingsley	M	46	De facto	None - pain	12	8.0	9.7	46	91	15	5
Carly	F	33	De facto	Full time	3.5	4.2	8.3	31	67	14	12
Jason	M	65	Married	Retired	44	4.8	6.9	30	58	5	7
Marlene	F	40	Single	Part time	1	6.2	6.1	32	36	8	6
Mean (SD)	-	51.9 (14.3)	-	-	8.9 (11.7)	6.9 (1.7)	7.1 (2.3)	37.6 (7.4)	63.5 (19.1)	9.3 (4.0)	7.3 (3.7)

Note: Self-report instruments used (higher scores reflect worse symptoms): Brief Pain Inventory measuring pain intensity (possible score 0-10) and pain interference (possible score 0-10); Pain Catastrophising Scale measuring pain catastrophising (possible score 0-52); Meta-Cognitions Questionnaire 30 (possible

score 0-120) measuring metacognition; and Hospital Anxiety Depression Scale measuring anxiety (possible score 0-21) and depression (possible score 0-21). Employment status ‘None – pain’ refers to being unable to work because of pain.

3.3.2 Recruitment and data collection

This study received ethical approval from the Government of Western Australia, Department of Health (SMHS 2014-079) and Curtin University (HR23/2015) and was undertaken between March and September, 2015. Nursing staff at a private neurosurgery practice and a public hospital orthopaedic surgery clinic gave interested patients a screening questionnaire (PCS) and information sheet describing the study as aiming to explore how people think when they are in pain. These people were contacted for further screening by the first author, who had no prior relationship with any participants. Eligible consenting participants were interviewed in their homes. Other consenting participants were recruited in the waiting room of a public hospital pain clinic, where they were interviewed in a consultation room. All participants completed self-report measures and informed consent on a tablet computer using Qualtrics™ software. [366]

Interviews were audio taped and lasted 41-76 minutes, using a semi-structured interview schedule as a loose structure but guided by the principle of sensitivity to context. [367] The schedule (see Appendix D) was developed based on a broad reading of the pain literature, discussion amongst the research team based on clinical and research experience, and using guidelines for eliciting metacognitions in Metacognitive Therapy, [206] including the 'metacognitive profiling' interview. [203] Three participants had a family member present, while the rest were interviewed alone. No repeat interviews were conducted, and no participants requested changes to interview transcripts or the summary of findings that was sent to them. No participants dropped out of the study.

3.3.3 Analysis

Interviews were transcribed verbatim and transcript data was managed using NVivo software. [368] Analysis was conducted by the first author according to the IPA process described by Smith, Flowers and Larkin, [367] but adapted for electronic coding within NVivo. Consistent with IPA guidelines, [367] the analytic process was iterative, moving from the specific idiographic content of the interviews to their abstracted themes and back to the idiographic again. This 'hermeneutic circle' involved shifting between the emic/insider perspective and the etic/outsider

perspective. [367] The abstracting part of the process also explicitly invoked broader contextual information from the pain science and psychological literature, responding to an identified gap in qualitative pain research [360] by looking for convergence and divergence with existing quantitative pain research.

3.3.4 Validity

Validity of findings was ensured by following guidelines for qualitative research documented in the Consolidated Criteria for Reporting Qualitative Research (COREQ). [369]. Following IPA guidelines, [367] a second author (C.R.) also audited all interview transcripts, stages of coding, field notes and coding trees to ensure internal coherency of findings. [367] Findings were discussed amongst all authors and incorporated into the final analysis. Finally, the recognition of subjectivity that is central to qualitative research involves making explicit the interpretative stance and background of the researchers. [369] All investigators have previously published qualitative research and share a biopsychosocial view of pain along with both an academic and clinical interest in PC. The first author is a male doctoral student and clinical psychologist; the second author is a psychology research academic and clinical psychologist; the remaining authors are pain science research academics and musculoskeletal physiotherapists.

3.4 Results

All participants (represented with pseudonyms below) were able to reflect on their pain-related thinking and report on not only what they thought about in relation to pain, but also to varying degrees on their attitudes towards this process and how it related to their pain and functioning. Interpretative analysis revealed a number of themes related to the two study aims of documenting pain metacognitions and exploring their influence on pain rumination. These are described below and summarised in Table 3.2.

Table 3.2. Summary of interpretative coding.

Study Aims	Themes	Salient Codes
Documenting pain metacognitions	Positive attitudes towards rumination	Rumination as problem solving
		Rumination prepares me
		Rumination helps me to cope
	Negative attitudes towards rumination	Uncontrollability of rumination
		Psychological harm
		Rumination exacerbates pain
		Social harms of rumination
		No point ruminating
How does metacognition influence pain rumination?	Rumination facilitators	Pain and pain reminders trigger rumination
		Anticipating pain triggers rumination
		Positive metacognitions fuel rumination
		Harm metacognitions trigger meta-worry
		Uncontrollability metacognitions keep rumination unchecked
	Overcoming rumination	Pain relief ends rumination
		Distraction relieves rumination
		Reframing relieves rumination
		Switching to problem solving is helpful
		Acceptance relieves rumination
		Seeing rumination as pointless helps overcome it

3.4.1 Aim 1: Documenting pain metacognitions

All participants described holding opinions about their pain-related thinking that revealed both positive and negative attitudes towards rumination. These were often held simultaneously, creating internal tension as people navigated their psychological responses to pain. The most commonly reported positive attitude towards thinking extensively about pain was a belief that it helped participants to

solve problems. This was described both in terms of the intention behind thinking about pain, and its consequences:

> Carly: I guess I'm trying to problem solve, which is failing, but I persist in trying it anyway. I mean, to some degree it helps if I'm in pain and thinking about it makes me stand up instead of sitting down, then that quite often helps. So there is some payoff. But a lot of it is just a lot of useless worry.

Here Carly describes her pain rumination as a kind of implicit self-regulation strategy, aiming to solve the problem of being in pain. However, she demonstrates an ambivalence towards this strategy that was echoed by several participants (e.g. Gail, Rachel, Ramo, Kate, Jacinta). On the one hand Carly suggests that thinking about pain can have a "payoff", but on the other hand she describes it as "failing". This passage reveals the variable nature of rumination. For Carly, helpful rumination translates into behaviour ("thinking about it makes me stand up") which produces a tangible effect ("that quite often helps"). She contrasts this to "useless worry", which by implication has no tangible behavioural output or benefit. There is a degree of anguish in Carly's recognition of the tensions between her positive and negative metacognitions, describing her strategy as failing but persisting anyway. She goes on to describe how this dialectic often results in more perseverative thinking, a kind of meta-worry.

> Carly: I spend so much time thinking about it and then, on top of that, thinking that I shouldn't be thinking about it. Then on top of that, thinking I shouldn't be beating myself up about thinking about it. It just gets ridiculous.

Jacinta shared the view that pain rumination functions as a form of problem solving. Like Carly, she depicts this as somewhat fraught.

> Jacinta: What the problem solving is, it's just to work through the exercises to see if they'll help the pain… When I've got pain, I keep thinking I'll do it and have the operation just to get rid of the pain but then they can't guarantee I'll be out of pain so that plays on my mind.

The temporal focus of Jacinta's thinking is important in this extract. Jacinta's analysis of her exercises is concretely linked to a current behaviour ("it's just to work through the exercises") and this seems to constrain her thinking. By contrast,

contemplating a treatment decision about surgery involves future behaviour and a range of distal contextual factors, raising the spectre of uncertainty ("they can't guarantee I'll be out of pain"). This uncertainty impedes a decision, leaving Jacinta's original problem unsolved and so rumination persists ("that plays on my mind"). The other factor that distinguishes Jacinta's two descriptions of pain-related thinking is the goal itself. Her goal in the first example is more explorative and realistic, being aimed at pain modulation ("to see if they'll help the pain"), whereas her goal in the second example is complete pain remission, an absolute and ambitious target given her 26-year pain history ("just to get rid of the pain"). Jacinta's experience again highlights the variable nature of pain-related rumination in terms of intention, temporal focus and outcome.

In contrast to seeing rumination as a problem-solving strategy – most commonly framed in terms of pain relief – some participants described it as a coping strategy yielding a sense of preparedness or control.

> Gail: If you think the worst and it doesn't get there, that's a positive. But if you're constantly thinking positive thoughts then anything bad happens, then you're not ready, you're not prepared for that. So I prepare myself for the worst I guess. It sounds horrible. And then anything positive that happens from that, well that's a plus.

> Marlene: I'd rather think than don't think. I don't know how to explain it to you, but when I think, I know where I'm going. If I don't think it's like I'm lost.

Despite these expressions of the benefits of rumination, positive metacognitions were overwhelmingly eclipsed by participants' pejorative statements about perseverative thinking. During the interviews, it was much easier to elicit negative metacognitions than positive ones. In fact, when asked about the benefits of thinking about pain, many people said "nothing", although upon prompting most endorsed some of the positive metacognitions noted above. On one level this reveals that people find it relatively difficult to explain why they ruminate, despite having insight into what they think about.

Negative attitudes towards rumination were easily elicited and these fell into five main categories: uncontrollability; psychological harm; pain exacerbation; social

harm; and ineffectiveness. Kate and Jason describe how pain-related thinking is difficult to control:

> Kate: It's just second nature to me… There's not much that will stop you thinking about it.

> Jason: It's insidious. It's just covert and voom.

Jason's account suggests that rumination creeps up on you and then suddenly accelerates. The word "voom" onomatopoeically evokes imagery of an explosion that cannot be contained. The word "covert" portrays rumination as something separate from the self but also hidden and somewhat sinister in that its stealth implies an intent to attack.

This sense of the rumination being threatening was more explicit in the ubiquitously expressed belief that it causes psychological harm, most commonly in the form of depression, but also stress, anxiety, anger and cognitive interference.

> Jason: It drags you down… I call it stinkin' thinkin'. There's always another phrase that comes – 'misery-go-round'.

> Rachel: I think you can get very depressed if you do, definitely. I've had – as I said, prior to this – black moments that I've been thinking about. 'This pain is never going to go away; I'm ruining everyone's lives.' That's why I just try and not think about it… I think if you sit and think about it, you're doomed.

Another harm described by about half the sample was that rumination exacerbates pain itself:

> Rachel: If I think about my pain, it makes it worse.

> Kate: It makes me notice the pain more, yes.

Kate went on to describe how this spiral of negativity creates social harm, a theme also powerfully evoked by Rhonda:

> Rhonda: It not only grabs you in one way, it also can destroy your family in others. It's a very nasty type of thing. If it doesn't destroy your body it can also destroy your relationships, your closest friends, your family. It can tear you apart… You have to put it out of your head, but it's constantly there. It never leaves you alone, it's torment, it's actually torment to you. That's all I can say. You're tormented for the rest of your life.

Rhonda's language again represents rumination as an entity separate from the self ("grabs you"; "never leaves you alone"), echoing Jason's description of a covert force. Here its harmful nature is more exaggerated, with images of destruction ("destroy your body", "destroy your family") and malevolent intent ("very nasty").

Prolonged thinking about pain was therefore experienced in different ways by this group of people. Its negative features – uncontrollable and harmful – were obvious to all, while for some people thinking about pain was a strategy for problem solving or coping. These positive and negative metacognitions about rumination were sometimes simultaneously held, creating tension and at times prompting further rumination.

3.4.2 Aim 2: How does metacognition influence pain rumination?

This highlights a crucial feature of pain rumination in these 15 people. Rather than being static and uniform, participants described rumination as fluctuating in content, impact, intensity, and duration. For example, despite scoring highly on the PCS, a quantitative measure of pain catastrophising, some participants said thoughts about pain were fleeting:

> Daniel: I don't have those sort of thoughts much anymore… Like when you ask me questions like this, that's when it comes up or when I'm talking to other people. That's when it comes up.

However, for many participants, thinking about pain seemed to occupy a large proportion of their time, energy and attention.

> Kingsley: I'm constantly in pain and constantly thinking about the pain level that I'm in and how I can get out of it… So you're pretty much constantly, constantly thinking about it.

> Carly: Yeah, it's pretty much ticking over all the time. It's got its own special part of my brain that is devoted entirely to that, I think.

Despite this variability in time spent thinking about pain, there was striking congruence in what initially triggered thoughts about pain. These included hearing other people talk about pain, anticipating pain-provoking activities, functional limitations, and, most commonly, pain itself:

> Edna: Oh yes, I think mainly when the pain is severe you do think.

Conversely, participants who described pain as triggering rumination also said it eased in response to pain relief.

> Gail: If that pain was completely blocked and I didn't feel it, straight away that worry would be gone.

Gail's comment suggests a mechanistic relationship between pain and rumination that is consistent with her uncontrollability metacognition ("About other things that I worry about? Yes, I have control of it. This back pain? No."). Yet she goes on to describe how even in the absence of pain, her attention is oriented towards future pain ("It [worry] just lingers. Yeah, it's anticipation as well, like just waiting for the unknown."). This is reminiscent of Gail's earlier comment that rumination prepares her for future threats ("I prepare myself for the worst, I guess"). This shows how a positive metacognition (prepares me) and a negative metacognition (uncontrollable) both facilitate rumination about pain: the 'prepares me' positive metacognition implicitly motivates increased attention to and processing of pain-related stimuli, and the 'uncontrollable' negative metacognition compounds inertia against attempts to disrupt rumination. This ultimately leads to a perseverative loop where both rumination and pain are maintained or exacerbated ("If I dwell on it too much it's just going to get ten times worse"), which in turn become triggers for further rumination.

Carly described experiencing a similar vicious cycle of pain and rumination, which, like Gail could even be triggered by the absence of pain.

> Carly: If I'm having a period of little or no pain, then there will inevitably come a moment where I realise that I'm in little or no pain and scan for it and become suspicious of the fact that it's not there. I know that it's coming back. Then I start thinking oh, okay, right, I'm having a good period right now. Why am I having a good period? What have I done to make this pain good right now? Is it because I saw the physio yesterday or because I slept well? Yeah, then I'm analysing as to why it's not there.

This hypervigilance seems to be fuelled by her positive 'problem solving' metacognition ("I'm trying to problem solve"). However, earlier Carly described how the tension between her positive and negative metacognitions often resulted in more rumination, in the form of meta-worry ("That kind of gets into a bit of a spiral about feeling bad about thinking about the pain"). This seems to stem from her negative metacognition that rumination is harmful:

> Carly: I've been told by various people that chronic pain is kind of a strengthening of these neural pathways and, I guess, short-circuiting of – your brain just saying all right, you're in pain now. You need to be able to weaken those pathways. So continually thinking about it is obviously not going to succeed in breaking those pathways down. So to some degree, I feel like it's self-perpetuating and I'm not helping myself by thinking about it.

Given this negative view of pain-related thinking, Carly's recognition of her thinking becomes a trigger for worrying about her own worry. Therefore, this negative metacognition acts as a potent facilitator of rumination.

However, despite reporting these episodes of perseverative thinking, Gail and Carly also described periods of diminished rumination and pain, which reinforces the observation that pain rumination is a dynamic process, or transient state, rather than an enduring trait. Highlighting this, several participants described how they disrupted or averted cycles of rumination. For example, just as Carly contrasted helpful perseverative thinking (which has a "payoff") against "useless worry", Pauline describes how she consciously shifts her thinking towards the more helpful style.

> Pauline: Sometimes, I think okay, well instead of worrying about it, try to find a solution to it. So there is that. I try to sort of think what can I do to make it better, rather than just worrying about it. Because I don't feel that worrying about it is going to really help me. But trying to find a solution, rather than worrying about it… To me, there's more important things to be done than just continually worrying about things. That, to me, is sort of like ifs, buts and maybes. You know, you just try and make decisions and get on with things.

Pauline insightfully differentiates worry from problem-solving, even highlighting the linguistic features of worry ("ifs, buts and maybes") and implicitly linking the abstract nature of worry to its lack of utility ("I don't feel that worrying about it is going to really help me"). This is reminiscent of the Jacinta's description of contemplating future surgery, where uncertainty and abstraction fuelled rumination ("that plays on my mind"). By contrast, Pauline depicts problem solving as concrete, present-focused and oriented toward a behavioural response ("what can I do to make it better").

An important feature of Pauline's path out of rumination is her negative attitude towards worry ("there's more important things to be done than just continually worrying"). This seems to motivate her to avoid getting stuck in a perseverative loop. As she says elsewhere, "Sometimes I do worry but I try not to, I try to take my mind off it in other ways". To this end she describes using self-management behaviours such as deep breathing, meditation, walking, and distraction through Sudoku puzzles.

This shows how a more nuanced negative metacognition about rumination as merely ineffective can be protective, prompting an adaptive coping response that involves concrete thinking and helpful action, in contrast to more overtly negative 'harm metacognitions' that trigger meta-worry in Carly's case. For Pauline, seeing rumination as pointless and thereby translating it into action has a positive impact on her pain experience, which subsequently diminishes the initial impetus to think about pain.

> Pauline: If I'm concentrating on something else and get my mind off the pain, it eases off quicker... I'll just start deep breathing. I'll go back to the Sudoku and it just slowly eases off.

Rachel demonstrates a similar protective metacognition, expressing the view that rumination is both pointless and modifiable:

> Rachel: I just can't sit and think, 'I'm in pain, God, I'm in pain.' I don't want to do that. I don't want to do that. I just get up and do it… I think, 'No, I'm going to get up and do something,' even if it's I've got to go for a little walk. I go out and see the chookies [chickens] or the roos [kangaroos]. Just little things that stop me thinking.

She goes on to describe how taking action not only breaks the cycle of rumination, it also eases her pain, which she attributes to the pain modulating effect of both external attention and movement. Other participants described similar experiences, most commonly using distraction to disrupt rumination, although some people described how meditation and acceptance of their pain and thoughts allowed them to ride out flare-ups without fuelling more distress.

This group of people therefore described rumination as a process that fluctuates in response to external and internal triggers, changing levels of pain, and different coping behaviours. Their beliefs about rumination – or metacognitions – seemed to influence this process by motivating attempts to transform it into a helpful form of thinking (problem solving) or shift attention away from thoughts in the case of protective metacognitions such as 'rumination is pointless'. Conversely, metacognition could fuel an unhelpful form of rumination when people believed it was uncontrollable, dangerous, or helped them to cope.

3.5 Discussion

This qualitative study of 15 people with CLBP and elevated PC aimed to provide an insight into people's attitudes towards their pain-related thinking and how these metacognitions influence the course of rumination. The most common positive metacognition was that rumination was a form of problem solving and further analysis revealed that this was only experienced as helpful if the thinking was focused on concrete and solvable problems in the short term, and if it translated into self-management behaviours. Otherwise rumination was commonly seen as unhelpful, uncontrollable and detrimental in its impact on one's mental state,

relationships and pain levels. In exploring the influence of metacognition on pain rumination it became apparent that rumination itself is a dynamic process that varies both within and across individuals. Some people, particularly those with strong uncontrollability metacognitions, described rumination as mainly triggered and resolved by pain itself. Others, holding metacognitions that rumination is pointless but modifiable, made active attempts to attenuate unproductive thinking by transforming it into effective problem-solving, taking action or re-focusing their attention.

Although this is the first qualitative study employing a purposive sample of people with elevated catastrophising, several of the themes found here are consistent with existing qualitative pain literature. For example, the negative metacognitions documented here that focus on the danger and harms associated with rumination echo previous qualitative accounts of fear, worry, distress and depression among people living with chronic pain. [77,245,360,370–373]

These findings also qualitatively articulate many of the empirical findings of quantitative research into PC. For example, several participants said PC increased the intensity or salience of their pain, echoing the strong empirical association between PC and increased pain intensity. [93] Similarly, negative metacognitions describing the deleterious effect that rumination has on mental health mirrors the robust correlation between PC and depression and anxiety in the pain literature. [78] Furthermore, the sense of rumination's uncontrollability that participants described, appears consistent with emerging evidence of neuroplastic changes in the brains of people prone to ruminating about pain, especially increased functional connectivity within the default mode network, a network associated with mind wandering. [174]

Importantly, the present findings are consistent with theoretical models emphasising the functional aspects of PC. For example, the prevalent metacognition describing rumination as a problem solving strategy is reminiscent of Eccleston and Crombez's misdirected problem solving model of pain-related worry. [195] Their contention that a perseverance loop of worry stems from problems being poorly framed, especially in terms of pain relief, was exemplified by people such as Jacinta, who described how uncertainty around whether surgery would get rid of her pain made it play on her mind. This was contrasted to instances where

she framed the problem more narrowly in scope and temporal focus, such as thinking about whether her exercises were helping her pain on a given day. Although Eccleston and Crombez's model portrays searching for pain relief as the lynch pin of worry perseveration, participants here described being able to steer away from endless rumination as long as their search for pain relief was helpfully framed. For example, 'what can I do to help the pain right now' rather than 'how can I get rid of this pain once and for all'.

This is also consistent with recent models of rumination as a transdiagnostic process in psychopathology. For example Watkins' Rumination-Focused Cognitive Behaviour Therapy for Depression aims to help people "shift from unconstructive rumination to constructive rumination" [374(p11)], such as problem solving. In our sample, helpful rumination was limited in duration because it produced decisions that articulated naturally into behavioural outputs that shifted attention away from thoughts of the self and onto activity, thereby improving pain and distress.

Our findings also support Flink and colleagues' [178] model of PC as repetitive negative thinking – which they rename 'catastrophic worry' – characterised by abstract cognition and whose putative function is to reduce negative affect. Present findings support this idea that the essential feature of PC is its perseverative nature, as evidenced by descriptions of it as a "misery-go-round" (Jason) and "pretty much ticking over all the time" (Carly). The clear articulation of positive metacognitions in this study provides the first qualitative support for Flink et al.'s suggestion that catastrophic worry is, "positively reinforced through metacognitions about dwelling on the problem as beneficial for finding a solution to the problem". [178(p218)]

Indeed, the stories of these 15 people with chronic back pain seem to support the broader contention that metacognition, or what people believe about their own thinking, influences how much they ruminate. In Metacognitive Therapy, the basic tenet linking metacognition to perseverative thinking is the notion that positive and negative metacognitions both trigger and reinforce rumination in various ways because the ultimate function of this type of thinking is self-regulation. [205] Our participants' reflections on how their thinking related to attempts to reduce pain, make treatment decisions, and prepare for future threats all illustrate this basic

self-regulatory function. Although similar findings have been documented for people with mental health conditions and addictions, [205,220] this is the first qualitative evidence that the S-REF model and metacognition more broadly is relevant in the context of pain-related cognition.

These findings suggest that, although several factors are likely to influence pain-rumination, there may be value in targeting metacognitive beliefs explicitly in people who think excessively about pain and future research is needed to explore this. Although metacognitive changes are an implicit feature of acceptance- and mindfulness-based interventions, present findings suggest the need to create individualised formulations of the function of different metacognitions in sustaining perseverative thinking for different people. For example, targeting positive metacognitive beliefs might be ineffective for someone with predominantly uncontrollability or psychological harm type metacognitions. The present findings raise the possibility that an adapted form of Metacognitive Therapy [206] may be an effective way to treat elevated PC for some people. This may not be a simple application of incumbent MCT protocols, but could incorporate other evidence-based treatment components such as pain neuroscience education [375] and exposure-based movement retraining, [376] although further research is needed to explore this.

On a practical level, the experiences of these 15 people demonstrate that scores on quantitative measures such as the PCS should be interpreted cautiously by clinicians. Our findings highlight that pain rumination, and PC in general, is more state-like than trait-like and may require multiple assessment points for validity. Clinicians would benefit from using such psychometric instruments as interview tools to facilitate dialogue and assessment from a functional-analytic perspective to see how cognitive and behavioural coping responses vary in different contexts. This contextual approach, and evidence of the variability of rumination within and across individuals in this study, may also reduce the stigma sometimes associated with pain-related distress, where people with pain can feel marginalised in treatment settings if psychological factors are identified as contributing to their pain problem. [59]

3.5.1 Conclusions

This study provides the first qualitative account of pain-related metacognition. It shows that metacognitive beliefs influence how people with high levels of PC respond to pain, including how much they ruminate. This advances our understanding of the functional aspect of PC, lending support for recent conceptualisations of PC as primarily a form of repetitive negative thinking. These insights may inform the development of more targeted interventions for people who experience poor pain outcomes associated with episodes of unhelpful rumination about their pain.

Chapter 4 Study 3 – Assessing beliefs underlying rumination about pain: Development and validation of the Pain Metacognitions Questionnaire

Chapter Linking Statement

The qualitative results reported in the previous chapter suggest metacognition is relevant to people with chronic pain who are prone to catastrophising. All participants reported various metacognitive beliefs about their pain-related thinking and some described the way these attitudes influence how rumination and worry either perseveres or resolves. These qualitative findings suggest further investigation of metacognition in people with pain is warranted, including quantitative research using larger, more representative samples. However, in order to conduct quantitative research, a psychometrically sound measure of pain-related metacognition is needed. The following chapter therefore reports on the development and validation of such a measure. This scale development study relies on the results from the previous qualitative study by using its emergent themes to inform the generation of items for the scale. The measure validated in this chapter will then allow a theoretical model of pain-related metacognition to be empirically tested.

4.1 Abstract

Metacognitions, which are beliefs about our own thoughts, can facilitate the transdiagnostic process of repetitive negative thinking underlying many psychological disorders. The present study aimed to develop a self-report measure of unhelpful pain-related metacognitions which might serve as a clinical and research tool to better understand pain catastrophising, which is a significant risk factor for adverse pain outcomes. Two phases of validation are presented. Phase 1 reports on how the Pain Metacognitions Questionnaire (PMQ) was empirically developed through a qualitative study of 20 people with chronic back (n=15) or knee pain (n=5) in tertiary care and then validated in a large internet sample of people experiencing pain (N=864). Rasch analysis facilitated item reduction, yielding a 21-item scale with two dimensions – positive and negative metacognition – assessing how useful and damaging people believe rumination about pain to be, respectively. Further validation was performed in a new sample (N=510) which replicated initial findings. Both PMQ subscales have good retest reliability (r = .76, r = .72) and internal consistency reliability (.86, .87). They correlate negatively with mindfulness and positively with measures of pain, disability, anxiety, depression, catastrophising, rumination, and metacognition, providing evidence of construct validity. The PMQ also predicts unique variance in catastrophising when other variables were controlled, and predicts 'patient' status for catastrophising. Sensitivity analysis yielded preliminary suggestions for clinically meaningful cut-offs.

Perspective: Unhelpful pain metacognitions can be validly and reliably measured using a self-report instrument. Future studies using the PMQ might shed new light on maladaptive pain-related thinking processes to develop better interventions for people prone to rumination about pain.

4.2 Introduction

Emotional distress is a common feature of the pain experience and prolonged distress can be a result of ongoing pain, [32,33] while also contributing to further pain and disability according to biopsychosocial models. [377] For example, clinically significant symptoms of anxiety and depression are frequently comorbid with chronic pain and have been described as part of the normal psychology of pain [60] given the adverse impact pain can have on attention, [157] cognition, [198] and behaviour. [70,378] Conversely, these forms of affective dysregulation can facilitate nociception through mechanisms such as central sensitisation, contributing to further chronicity and a vicious cycle of pain and distress. [177]

Emotional distress is therefore a common treatment target in pain management, with a large body of evidence showing psychological interventions such as Cognitive Behaviour Therapy (CBT) can effectively reduce emotional distress [120] and even sensitisation. [379] However effect sizes are modest and unique mechanisms of change among different interventions are still poorly understood. [28,111] Even when supposed process variables that predict pain psychopathology are targeted, such as pain catastrophising, a similar story of modest effects prevails. [380] This has prompted calls for interventions to be more theory-driven, which requires a deeper understanding of the psychological processes involved in pain-related distress. [120,128]

The present study aims to respond to this call by developing a tool to investigate underexplored features of worry/rumination about pain, which is a central aspect of perhaps the most widely studied psychological predictor of adverse pain outcomes – pain catastrophising (PC). [78,79] According to the metacognitive model of psychological disorder, worry and rumination are activated and maintained by higher order beliefs about thinking called metacognitions. [205] The most important of these are positive metacognitions, reflecting beliefs about how rumination can be helpful, and negative metacognitions, highlighting the harmful and uncontrollable aspects of perseverative thinking. These metacognitions can be reliably measured [364] and modified to effectively treat various anxiety and mood disorders using Metacognitive Therapy. [206,224] Metacognitions are also implicitly targeted in acceptance-based and mindfulness-based interventions, which have

well-established efficacy in reducing pain, disability, distress and pain catastrophising. [229,380]

While at least one study has documented how metacognitive beliefs about worry predict PC and pain behaviours, [228] there is no measure of metacognition tailored for use in pain populations. The incumbent Metacognitions Questionnaire, [246] although widely used, was developed for a generalised anxiety disorder population. However, it only addresses worry rather than other forms of perseverative thinking, such as depressogenic rumination, [247] which is relevant given frequent comorbidity of chronic pain and depression. [358] This study therefore aims to develop a psychometrically sound self-report instrument to measure positive and negative metacognitive beliefs about pain-related worry and rumination.

4.3 Phase 1: Scale development and initial validation

4.3.1 Overview of objectives

Phase 1 aimed to empirically develop items for a measure of pain-related metacognition and then test the draft scale's psychometric properties. This included: evaluating item functioning to discard poorly functioning items and reduce scale length; establishing construct validity (convergent, discriminant); and measuring reliability (internal consistency, test-retest).

4.3.2 Method

4.3.2.1 Participants

Participants were 864 adults recruited online in two ways. The first sub-sample was a convenience internet sample of adults who responded to advertisements on websites of various pain-related organisations (e.g. Chronic Pain Australia, Australian Pain Management Association, Pain Australia, Australian Pain Society, Psoriasis and Psoriatic Arthritis Alliance), social media sites (e.g. Facebook,

Twitter), and websites advertising psychological research (e.g. http://psych.hanover.edu/research/exponnet.html). Participants were told the research aimed to explore their beliefs about their own pain-related thoughts.

To be eligible, participants needed to be ≥18 years old, reside in a country where English is an official language, and be either experiencing an acute (non-cancer) pain episode at the time of participation or have a chronic (non-cancer) pain condition (≥3 months duration). Participants with less than moderate pain, defined as a pain intensity of less than three on a 0-10 numerical rating scale, [381] were screened out. Based on the total survey word count and a maximum reading speed of 450 words per minute, [382] participants completing the survey in less than 10 minutes were also screened out due to high risk of not validly completing the measures. Of the 1519 participants who started the survey, 930 did not complete it, 81 did not meet inclusion criteria and two results were omitted for completing measures too quickly. This left 506 included participants in the general internet sample.

The second sample comprised a paid sample of Amazon Mechanical Turk (MTurk) workers. MTurk (http://www.mturk.com) is an online labour market for various low cost tasks. It has been reliably used in social science and pain research, [383] with participants shown to be more demographically diverse than the university undergraduate samples and general internet samples that are often used. [384,385] Participants were given the same information about the study as the other sample but were each paid US$2. The same inclusion and exclusion criteria were applied. Of the 490 participants who started the survey, 70 did not complete it, 22 did not meet inclusion criteria, and 40 results were omitted for completion time of less than 10 minutes. This resulted in 358 included participants in the MTurk sample.

4.3.2.2 Measures

4.3.2.2.1 Demographic data

A demographic survey was compiled based on the Electronic Persistent Pain Outcomes Collaboration patient outcomes [386]. These included: age, gender, marital

status, work status, compensation status, education level, pain onset event, pain duration, pain frequency, diagnostic status, co-morbid psychological diagnosis, acute/chronic pain status, and pain site. Demographics were used to describe characteristics of the sample, as well as to establish discriminant validity.

4.3.2.2.2 Pain Metacognition Questionnaire

The PMQ was developed to assess people's unhelpful beliefs, or metacognitions, about their own pain-related thinking, which might be expected to facilitate pain rumination. Following recommendations in scale development literature, [387,388] item generation was based on both theory and empirical data. This data included the qualitative results from Study 2. [389] As reported in detail in Chapter 3, this involved in-depth interviews about pain-related metacognitions with 15 people experiencing chronic back pain and very high catastrophising (PCS ≥ 30). Using the same inclusion criteria, a further five people with chronic osteoarthritis knee pain who were scheduled for knee replacement surgery were also interviewed to provide perspectives from another clinical cohort with musculoskeletal pain. Since the analytic methods used in Study 2 (interpretative phenomenological analysis) require small, fairly homogeneous samples, [367] data from the participants with knee pain were only used in this scale development study rather than included in the IPA study in Chapter 3.

As discussed in Study 2, [389] and consistent with metacognitive theory, [206] analysis of qualitative data suggested participants' attitudes towards perseverative thinking about pain fell into two categories: positive metacognitions (thinking about pain is helpful) and negative metacognitions (thinking about pain is unhelpful or uncontrollable). The most common positive metacognition was a belief that rumination helped participants to solve problems, while the most common negative metacognitions were that it was uncontrollable and harmful, both in terms of emotional wellbeing and pain exacerbation. These themes, based on all 20 interviews, are summarised in Appendix E with illustrative quotes.

Based on these themes and metacognitive theory, [204–206] 40-items were drafted to allow for item reduction during scale validation. Items covered four positive metacognition themes and six negative metacognition themes, each represented

with four items. These were rated on a 7-point Likert scale based on evidence that this represents an optimal number of response categories, [388] ranging from 'strongly disagree' to 'strongly agree'. The draft scale was piloted on a subsample of five participants, with qualitative interviews undertaken to elicit feedback on its face validity and ease of use. The draft scale items are shown in Table 4.1.

Table 4.1. Draft 40-item version of Pain Metacognitions Questionnaire.

Subscale	Theme	No.	Item
Positive Metacognitions	Problem solving	1*	My pain won't improve unless I analyse it.
		2*	When I'm thinking about pain I'm trying to problem solve
		3	Analysing my pain will help me to find a solution and get better
		4	Thinking about pain doesn't get you anywhere [R]
	Protects me	5*	Thinking a lot about my pain protects me from getting injured.
		6	Thinking about my pain all the time means I'm more aware of my body so I'm less likely to hurt myself.
		7	I won't get injured as easily if I stay focused on my pain.
		8*	My pain would get worse if I didn't think about it a lot.
	Prepares me	9	I'm better prepared for pain if I think about it a lot.
		10*	Analysing my pain prepares me for the worst.
		11*	Focusing on the bad things about my pain helps me to enjoy the good things more.
		12*	My pain won't sneak up on me as long as I keep thinking about it.
	Coping	13*	Thinking a lot about my pain helps me to cope with it.
		14	I should stop thinking so much about my pain because it doesn't help. [R]
		15	I feel more in control when I'm thinking about my pain.
		16*	Thinking about my pain helps me to understand myself better.

Subscale	Theme	No.	Item
Negative Metacognitions	Uncontrollable	17	I can’t help thinking about my pain all the time.
		18*	When I start thinking about my pain, it’s impossible to stop.
		19	It's easy to shift my attention away from thoughts about pain. [R]
		20*	I don't try to stop thinking about my pain because my thoughts seem to have a life of their own.
	Increases distress	21*	Thinking about my pain all the time makes me feel depressed.
		22*	I’d be happier if I stopped thinking about pain.
		23*	I feel stressed if I think a lot about my pain.
		24*	I would be less anxious if I didn't focus on my pain as much.
	Increases pain	25	I have less pain when I don’t think about it so much.
		26*	I make my pain worse by analysing it.
		27	It hurts more when I think about my pain too much.
		28	My thoughts don’t affect my pain levels. [R]
	Social harm	29	I’m no fun to be around because I’m so focused on pain.
		30	People would like me more if I focused less on my pain.
		31	My family suffers because I think about my pain so much.
		32	If I could stop thinking about my pain, I would have better relationships.
		33*	I must block out my thoughts about pain.

	Must be controlled	34	When thoughts about my pain come to mind, I try to just get on with what I'm doing. [R]
		35	When thoughts about my pain grab my attention, I try to push them out of my mind.
		36*	It's important to control my thoughts about pain.
	Meta-worry	37	When I realise I'm thinking too much about my pain, I get annoyed with myself.
		38*	I worry about the negative effects of thinking too much about my pain.
		39*	I get caught in a vicious cycle of thinking about my pain and then thinking about how I wish I could stop thinking about it.
		40*	When I find myself brooding on my pain, it starts me thinking about how I'm just making things worse.

Note. Items rated on 7-point Likert scale: 1 = Strongly Disagree, 2 = Disagree, 3 = Slightly disagree, 4 = Neither agree nor disagree, 5 = Slightly agree, 6 = Agree, 7 = Strongly agree. [R] indicates reverse scored items. * Items included in final version of PMQ based on validation results.

4.3.2.2.3 Brief Pain Inventory

The BPI [363] is a 32-item instrument assessing demographic characteristics, pain intensity, medication use and functional interference. Only the 4-item pain intensity subscale and 7-item functional disability subscale of the BPI was used in the present study. Scores on these subscales range from 0 to 10, with higher scores representing more pain or disability. In cohorts of people with chronic pain the BPI has good convergent validity and internal reliability of Cronbach's $\alpha = .85$ for the pain intensity subscale and $\alpha = .88$ for the interference subscale. [390] In the present sample, the pain intensity scale had a reliability of $\alpha = .87$ while the interference scale was $\alpha = .93$. The BPI was used to establish convergent validity.

4.3.2.2.4 Pain Catastrophising Scale

The PCS [83] comprises 13 questions assessing the extent to which people experiencing pain report a strongly negative cognitive and affective response to pain or expected pain. The PCS has three subscales: rumination, magnification and helplessness. It has been widely validated, showing good construct validity and excellent internal consistency reliability, with $\alpha = .92$. [145] In the present sample, it had a reliability of $\alpha = .93$. The PCS was used to establish convergent validity.

4.3.2.2.5 Hospital Anxiety and Depression Scale

The HADS [365] is a 14-item measure of self-reported symptoms of anxiety and depression. It was designed for use in populations with health conditions and is not confounded by items assessing physiological symptoms of anxiety and depression like other similar measures (e.g. Beck Depression Inventory). The HADS has been widely validated and has good psychometric properties in musculoskeletal pain populations. [391,392] In the present sample the anxiety scale had an $\alpha = .86$ while the depression scale had an $\alpha = .84$. The HADS was used to establish convergent validity.

4.3.2.2.6 Experience of Cognitive Intrusion of Pain scale

The ECIP [383] is a recently developed 10-item questionnaire measuring three aspects of cognitive intrusion in pain: intrusion, rumination, and degree of control over pain-related thinking. Initial validation showed a single factor structure and adequate construct validity in samples with no pain, acute pain and chronic pain. It has excellent internal reliability of $\alpha = .96$. [383] In the present study, it had a reliability of $\alpha = .97$. The ECIP was used to establish convergent validity.

4.3.2.2.7 Tampa Scale of Kinesiophobia

The TSK [393] is a 17-item instrument measuring fear of movement, pain and injury. It was originally developed for use with CLBP patients [64] and has been validated in other populations of people with musculoskeletal pain, [394] as well as heterogeneous chronic pain samples [395]. The TSK has good construct validity and internal consistency reliability ranging from adequate to good ($\alpha = .76$ to $\alpha = .84$). [396,397] It had a reliability of $\alpha = .85$ in the present study. The TSK was used to establish convergent validity.

4.3.2.2.8 Metacognitions Questionnaire

The MCQ-30, [364] a shortened version of the original MCQ, [246] is a 30-item measure of metacognitive beliefs associated with worry and rumination. Higher scores on the MCQ are positively associated with obsessive-compulsive symptoms, pathological worry, and depression amongst other symptoms. It has a five-factor structure (positive metacognitive beliefs, cognitive confidence, cognitive self-consciousness, uncontrollability/danger, need for control) that correlates well with measures of worry and anxiety, thereby demonstrating good construct validity. The MCQ-30 has excellent internal consistency reliability ($\alpha = .93$) and good test-retest stability ($\alpha = .75$). [364] Internal consistency was $\alpha = .91$ in the present study. The MCQ-30 was used to establish convergent validity.

4.3.2.2.9 Mindful Attention Awareness Scale

The MAAS [398] is a 15-item instrument measuring people's self-reported moment-to-moment awareness of their actions, thoughts, sensations, emotions, and interpersonal interactions. It has good convergent and discriminant validity, excellent test-retest reliability (r = .81), and good internal consistency, with α = .87. [398] The MAAS has been found to correlate negatively with pain catastrophising. [73,243] In the present study, internal consistency of MAAS was α = .93. The MAAS was used to establish convergent validity.

4.3.2.2.10 Patient Global Impression of Change

The PGIC is a single item scale of a patient's overall evaluation of how much their condition has improved after a treatment [399]. The version used in this study is rated on a 7-point scale from "no change" to "a great deal better, and a considerable improvement that has made all the difference" [400]. Its construct validity in pain samples has been established through strong associations with pain intensity [401] and other outcomes such that it is recommend as a core outcome measure of overall improvement with treatment in pain trials, especially in interpreting clinically significant change [402]. The PGIC was used to examine whether possible changes in PMQ score changes in the test-retest analysis were related to changes in pain status.

4.3.2.3 Procedure

This study received ethical approval from the Government of Western Australia, Department of Health (SMHS 2014-079) and Curtin University (HR23/2015). Recruitment was undertaken from April-May, 2016. Participants responding to online advertisements described above were directed to a study link within the Qualtrics™ online platform, [366] which contained participant information, informed consent questions, inclusion criteria screening questions, and the measures described above. Each measure was presented in a separate screen and participants were required to answer all questions to progress through the survey and have their responses included. Participants were also asked for an email address to enter the

reward draw and be sent a link to the PMQ retest survey one week after initial assessment. This automated email was sent to the first 200 participants, since this was deemed adequate to power the test-retest correlations. The retest survey only included the PMQ and the one-item PGIC. Participants were free to withdraw at any time.

4.3.2.4 Analysis

The analytic strategy was to firstly assess item performance and remove poorly functioning items to refine the scale. Re-analysis of the shortened scale was then planned to report item functioning, scale properties and construct validity.

4.3.2.4.1 Rasch analysis of item functioning

Psychometric properties of the draft 40-item scale were evaluated with Rasch analysis using Winsteps software (v4.0.0, winsteps.com). Rasch analysis is based on item response theory rather than classical test theory and has several advantages, including producing true interval-level scales rather than ordinal ones. [388,403] Tennant and Conaghan [404] provide a useful overview of the Rasch model. The Andrich Rating Scale model was indicated and supported since the Likert-type scale categories are shared across items and no meaningful improvements in item and person statistics were noted when the Partial Credit model was applied. [405] The following components were evaluated: dimensionality, targeting, item and person fit, category ordering, internal consistency and differential item functioning.

An exploratory analysis was conducted to assess the suitability of the sample and the unidimensionality of the draft scale items. The suitability of the data for scale evaluation was assessed by comparing how well the scale items targeted the sample. Item endorsability (i.e. how easy an item was to endorse) and person agreeability (i.e. how agreeable the sample were) was assessed by visual inspection of the person-item distribution map and comparison of summary statistics. An exploratory principal components analysis of residuals (PCA) was conducted to determine whether the 40-item scale constituted a unidimensional

measure of pain metacognitions or a bi-dimensional measure of positive and negative metacognitions. The residual correlation matrix was visually inspected to identify item clusters with substantial loadings (eigenvalue greater than 2). The outcome of the exploratory PCA determined whether the scale was further considered in its entirety or as distinct subscales of related but independent constructs.

The item and person fit statistics were compared to identify items that functioned poorly. Fit statistics are chi-square–based and are reported as mean squares (in logits) with an expected value of 1 logit. The characteristic curves of items with infit (information-weighted) and outfit (outlier-sensitive) fit statistics >1.3 (model underfit) or <.7 (model overfit) were analysed. Poor person fit due to unexpected response patterns may compromise item fit. Such response patterns may reflect responder carelessness; hence, for the purpose of scale calibration, persons with infit or outfit statistics >2 or <0.3 logits were removed prior to reanalysis. [404]

Local dependence of items infers that the response on one item determines the response on another and can inflate reliability. [404] Items with residuals that correlated strongly were reviewed to determine whether they duplicate each other, and are thus redundant, or contribute to multidimensionality. To assess the function of the Likert-type scale categories, the category ordering was assessed. Seven response categories (1 – 7) were proposed, thus each item had six step-calibrations – the thresholds at which the likelihood of endorsing one category is equal to that of endorsing the next. Disordered step-calibrations are indicative of under-utilised categories and can influence the function of the scale. If disordering was detected, the Likert-type scale categories were collapsed to explore whether fewer categories improved the fit of the items.

Internal consistency reliability was assessed using the Person Separation Index (PSI) as an indicator of how reliably the scale differentiates persons of differing agreeability. A PSI value >.8 in Winsteps implies the scale is sensitive enough to distinguish between individuals with high and low agreeability, suggesting good reliability.

An analysis of Differential Item Functioning (DIF) detects whether person attributes bias the responses to items, contributing to item misfit. DIF was

conducted to assess the influence of gender (male, female) and six further characteristics, each dichotomised according to sample median: age (younger ≤ 38 years, older >38 years), pain duration (shorter ≤ 5.25 years, longer >5.25 years), pain intensity (low BPI-P ≤ 5, high BPI-P > 5), pain interference (low BPI-I ≤ 5.3, high BPI-I_t > 5.3), pain catastrophising (low PCS ≤ 20.5, high PCS > 20.5), pain cognitive intrusion (low ECIP ≤ 26, high ECIP > 26), psychological distress (low HADS ≤ 16, high HADS > 16). Statistically significant ($p < 0.01$) contrasts >.5 logits were deemed indicative of bias.

Items that exhibited excessive fit statistics or demonstrated local dependence were reviewed and considered for removal. The remaining items were then re-analysed with the persons deemed to be misfitting excluded.

4.3.2.4.2 Temporal stability

Temporal stability of the PMQ was assessed by correlating scores on the PMQ at two times, one week apart, with correlations of $r \geq .70$ providing evidence of good test-retest reliability. [388]

4.3.2.4.3 Construct validity

Construct validity was evaluated in terms of convergent and discriminant validity. Evidence of convergent validity included significant positive correlations between the PMQ and conceptually related pain outcomes (BPI, PCS, TSK, ECIP), psychological distress (HADS), and metacognition (MCQ-30), as well as significant negative correlations with mindfulness (MAAS). Evidence of discriminant validity was sought in the form of non-significant correlations between the PMQ and demographic variables having no expected *a priori* relationship: country of residence, marital status and education level. Pearson product moment correlations were calculated in IBM SPSS for Macintosh version 24.0. [406]

4.3.3 Results

4.3.3.1 Sample characteristics

The sample (N=864) was represented by 34 different countries, however most participants lived in the United States (n=364, 42.1%), United Kingdom (n=193, 22.3%), Australia (n = 223, 25.8%) or Canada (n = 20, 2.3%). Demographic characteristics of the combined sample are shown in Appendix F. Most were female (n=585, 67.7%) and the mean age was 39.7 years (*SD* = 12.6). The mean pain duration was 8.40 years (*SD* = 8.93). A large proportion were employed (n=542, 62.7%) and most were not involved in compensation claims (n=803, 92.9%). Almost all participants identified as living with chronic pain (n=814, 94.3%) and experiencing a pain episode at the time of assessment (n=854, 98.8%). Most (n=508, 58.8%) reported having been given a diagnosis for their pain condition, while around a quarter (n=215, 24.9%) reported having a comorbid mental health diagnosis from a health professional.

Pain sites experienced by the sample are also provided in Appendix F, showing that low back pain was most common (n=347, 40.2%). Average scores of the sample on the pain and psychological outcomes described above are presented in Table 4.2. This shows that on average the sample had moderate pain, [381] high catastrophising that exceeded a conservative risk threshold of 20 on the PCS, [265,348] high fear based on a TSK cut-off of 40, [407] high anxiety based on a clinical cut-off of 8 [408], and sub-clinical depressive symptoms based on the same cut-off. Within the subsample of 172 participants completing the PGIC one week after initial assessment, the median overall symptom change was 2 (“almost the same”) with an interquartile range of 2 to 4 (“somewhat better”). Only 6 participants (3%) registered a clinically significant score of 6 (“better”) or 7 (“a great deal better”). This suggests participants’ main symptoms in the re-test sample did not change significantly between the two assessment points.

Table 4.2. Scores on pain and psychological outcome measures for initial validation sample (N = 864)

Outcome	Mean	SD	Interpretation
Positive pain metacognitions (PMQ-P)	28.26	9.79	-
Negative pain metacognitions (PMQ-N)	49.32	11.94	-
Pain intensity (BPI-P)	4.89	1.66	Moderate [381]
Pain interference (BPI-I)	5.07	2.47	-
Pain Catastrophising (PCS)	21.05	10.75	High [262]
Cognitive intrusion of pain (ECIP)	25.94	15.33	-
Fear of pain (TSK)	39.69	7.61	High [407]
Depression (HADS-D)	6.93	4.29	Sub-clinical [408]
Anxiety (HADS-A)	8.92	4.66	Clinical [408]
Metacognition (MCQ)	60.95	15.05	-
Mindfulness (MAAS)	3.83	0.93	-

Note. SD = Standard deviation.

4.3.3.2 Rasch analysis of item functioning

Rasch analysis was conducted using the data from 864 persons. The exploratory analysis suggested the sample was suitable for analysis – a mean (SD) person agreeability of – 0.25 (.37) logits was comparable to the default mean item endorsability of 0.00 (.33) logits and visual inspection of the person-item map demonstrated an even distribution. Visual analysis of the PCA residual correlation matrix, however, clearly demonstrated a distinction between positive and negative metacognitions as independent constructs (see Figure 4.1). A contrast eigenvalue of 8.3 supported the finding of multidimensionality and the positive and negative item subscales were thus considered separately.

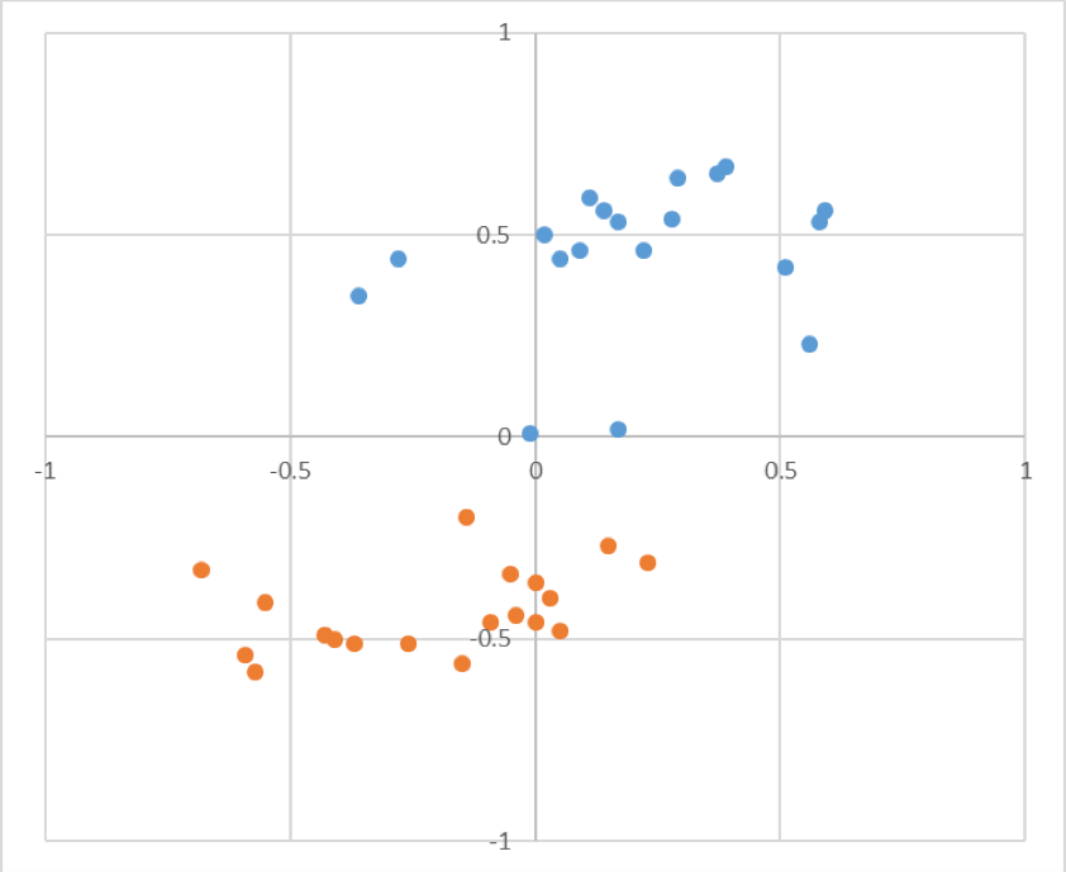


Figure 4.1. Principal Components Analysis residual correlation matrix showing the distinction between positive metacognitions (blue) and negative metacognitions (orange).

Each subscale targeted the sample reasonably well. For the positive subscale, the mean (SD) person agreeability was -.64 (.82) logits (range = -4.54 to 4.00 logits) lower than the default mean (SD) item endorsability of 0 (.38) logits (range = -.81 to.56 logits), suggesting the items targeted were relatively hard to endorse. The negative subscale items better targeted the sample with a mean person agreeability of -.12 (.51) logits (range = -3.08 to 2.04 logits) comparable with the mean item endorsability of 0 (.34) logits (range = -.62 to .80 logits). Table 4.3 shows the average item endorsability thresholds in hierarchal order where higher item thresholds indicate harder to endorse items. Seven persons (<1%) scored a minimum score on the positive subscale and no persons scored an extreme score on the negative subscale, suggesting floor and ceiling effects are negligible.

Table 4.3 also summarises the fit statistics for the two subscales. Items 2, 4 and 14 of the positive subscale were shown to underfit the model; items 2 and 14 demonstrated excessive infit and items 2, 4 and 14 demonstrated excessive outfit.

Items 25, 28 and 34 of the negative subscale were shown to underfit the model; items 25 and 28 demonstrated excessive infit and items 25, 28 and 34 demonstrated excessive outfit. Misfitting items were further explored and considered for removal.

Table 4.3. Categorical order, item endorsability thresholds and fit statistics for the draft 40-item version of the Pain Metacognitions Questionnaire (N=864).

Positive Metacognitions Subscale						
Item		Threshold	Score	SE	Infit	Outfit
12	My pain won't sneak up on me as long as I keep thinking about it.	0.56	2129	0.03	0.83	0.83
8	My pain would get worse if I didn't think about it a lot.	0.54	2144	0.03	0.88	0.91
11	Focusing on the bad things about my pain helps me to enjoy the good things more.	0.44	2247	0.03	1.15	1.29
13	Thinking a lot about my pain helps me to cope with it.	0.27	2443	0.03	0.76	0.79
9	I'm better prepared for pain if I think about it a lot.	0.25	2464	0.03	0.71	0.70
15	I feel more in control when I'm thinking about my pain.	0.14	2600	0.03	0.75	0.79
7	I won't get injured as easily if I stay focused on my pain.	0.12	2618	0.03	0.86	0.89
14	I should stop thinking so much about my pain because it doesn't help.	0.03	2732	0.03	**1.35**	**1.96**
5	Thinking a lot about my pain protects me from getting injured.	-0.04	2821	0.03	1.02	1.02
10	Analyzing my pain prepares me for the worst.	-0.08	2880	0.03	0.82	0.82
16	Thinking about my pain helps me to understand myself better.	-0.12	2922	0.03	0.86	0.90
4	Thinking about pain doesn't get you anywhere.	-0.15	2959	0.03	1.29	**1.43**
1	My pain won't improve unless I analyze it.	-0.21	3045	0.03	1.18	1.18
6	Thinking about my pain all the time means I'm more aware of my body so	-0.26	3101	0.03	1.11	1.25

	I'm less likely to hurt myself.					
3	Analyzing my pain will help me to find a solution and get better	-0.69	3649	0.03	1.19	1.19
2	When I'm thinking about pain I'm trying to problem solve	-0.81	3786	0.03	**1.35**	**1.37**
	Negative Metacognitions Subscale					
Item		Threshold	Score	SE	Infit	Outfit
34	When thoughts about my pain come to mind, I try to just get on with what I'm doing.	0.8	2170	0.03	1.24	**1.85**
31	My family suffers because I think about my pain so much.	0.41	2717	0.03	0.99	0.97
20	I don't try to stop thinking about my pain because my thoughts seem to have a life of their own.	0.35	2811	0.03	1.09	1.17
29	I'm no fun to be around because I'm so focused on pain.	0.32	2861	0.03	0.94	0.93
18	When I start thinking about my pain, it's impossible to stop.	0.28	2923	0.02	0.94	0.96
26	I make my pain worse by analyzing it.	0.21	3038	0.02	0.98	1.01
32	If I could stop thinking about my pain, I would have better relationships.	0.18	3084	0.02	0.86	0.87
30	People would like me more if I focused less on my pain.	0.15	3137	0.02	0.86	0.88
39	I get caught in a vicious cycle of thinking about my pain and then thinking about how I wish I could stop thinking about it.	0.15	3143	0.02	0.78	0.79
17	I can't help thinking about my pain all the time.	0.14	3157	0.02	1.15	1.20
27	It hurts more when I think about my pain too much.	0.11	3206	0.02	1.16	1.20
25	I have less pain when I don't think about it so much.	0.09	3231	0.02	**1.33**	**1.39**
33	I must block out my thoughts about pain.	0.04	3307	0.02	0.82	0.92
28	My thoughts don't affect my pain levels.	0	3386	0.02	**1.56**	**1.67**
40	When I find myself brooding on my pain, it starts me thinking about how	-0.02	3409	0.02	0.71	0.72

	I'm just making things worse.					
19	It's easy to shift my attention away from thoughts about pain. [R]	-0.03	3426	0.02	1.15	1.23
38	I worry about the negative effects of thinking too much about my pain.	-0.15	3616	0.03	0.92	0.92
37	When I realize I'm thinking too much about my pain, I get annoyed with myself.	-0.27	3799	0.03	0.88	0.87
24	I would be less anxious if I didn't focus on my pain as much.	-0.31	3868	0.03	0.82	0.84
21	Thinking about my pain all the time makes me feel depressed.	-0.34	3902	0.03	1.00	0.98
35	When thoughts about my pain grab my attention, I try to push them out of my mind.	-0.47	4099	0.03	1.15	1.23
22	I'd be happier if I stopped thinking about pain.	-0.5	4131	0.03	0.77	0.78
23	I feel stressed if I think a lot about my pain.	-0.52	4168	0.03	0.87	0.85
36	It's important to control my thoughts about pain.	-0.62	4293	0.03	1.05	1.03

Note. Bold type shows excessive item misfit; negative item thresholds represent items that are easier to endorse, and positive thresholds represent items that are more difficult to endorse. SE=standard error of measure; score=raw score out of 6,048 (possible score of 7 × 864 people); Threshold =item endorsability threshold (expressed in logits); infit and outfit expressed as mean square (chi-square-based fit statistic).

Seventy-nine persons demonstrated excessive underfit and 37 demonstrated excessive overfit for the positive subscale. Sixty-five persons demonstrated excessive underfit and 26 demonstrated excessive overfit on the negative subscale. Visual inspection of the PCA correlation matrices suggested items 1, 2, 3 and 4 of the positive subscale and items 25, 26, 27 and 28 of the negative subscale potentially constitute secondary dimensions. Eigenvalues of 3.2 and 4.1 respectively supported this notion. Assessment of local dependence revealed meaningful relationships between items 1, 2 and 3, items 5, 6 and 7, items 9 and 10 and items 15 and 16 of the positive subscale. Relationships between items 17 and 18, items 25, 26 and 17 and items 37 and 38 of the negative subscale were noted also. Correlated items were considered and items deemed redundant removed.

Visual inspection of the category ordering showed categories 3 and 4 of both subscales were disordered suggesting they were underutilised by the sample. The disordering was resolved by collapsing categories 2 and 3 and categories 4, 5 and 6, suggesting a 4-point Likert scale may function superiorly. The positive and negative subscales were shown to be reliable, with person separation indices of .91 and .90 respectively. However, reliability may have been inflated due to local dependence between several subscale items.

No significant differences in item functioning were observed for gender, age, pain duration or pain intensity in either subscale. Items 4 and 14 of the positive subscale were notably harder to endorse (.37 and .38 logits respectively) by people with higher levels of pain catastrophising and item 14 was notably harder to endorse (.35 logits) by people with higher levels of pain cognitive intrusion. Item 25 of the negative subscale was meaningfully biased by pain interference (.51 logits), pain cognitive intrusion (.57 logits), psychological distress (.52 logits) and pain catastrophising (.62 logits) being notably harder to endorse by persons with higher levels of each characteristic. Item 17 was also notably harder (.50 logits) to endorse by people with higher levels of pain cognitive intrusion and item 28 was harder notably harder (.52 logits) to endorse by persons with higher levels of pain catastrophising. These biases likely contributed to the misfit of these items.

Reanalysis of the positive and negative subscales was conducted on data for 814 and 809 persons respectively, after misfitting persons were excluded. Seven positive (items 3, 4, 6, 7, 9, 14 and 15) and seven negative (items 19, 25, 27, 28, 34, 35 and 37) subscale items were excluded because they functioned poorly or were deemed redundant. Four additional items related to social factors (29, 30, 31 and 32) were removed because responses to these items may reflect the impact *pain* has on people's relationships rather than the impact *thinking about* pain has on relationships. While item 2 showed misfit and local dependence with item 1, it was considered important given the prominence of the problem solving theme in the qualitative scale development study (see Study 2, Chapter 3), [389] and it was therefore retained.

Reanalysis supported the refining of the subscales. Table 4.4 shows the average item endorsability thresholds and fit statistics for the refined subscales. Targeting of the positive subscale was improved; mean person agreeability .42 logits (range

= -4.97 to 4.94 logits) compared to mean item endorsability of 0 logits; range = -1.71 to .96 logits). Targeting of the negative subscale was comparable; mean person agreeability .01 logits (range = -2.18 to 2.84 logits) compared to mean item endorsability 0 logits (range = -.65 to .61 logits). Twelve persons (1.5%) registered a minimum score on the positive subscale and 1 person (<1%) registered a maximum score. No persons scored an extreme score on the negative subscale, suggesting floor and ceiling effects are negligible for both subscales.

Table 4.4. Categorical order, item endorsability thresholds and fit statistics for the revised 21-item version of the Pain Metacognitions Questionnaire.

Positive Metacognitions Subscale (N=814)						
Item		Threshold	Score	SE	Infit	Outfit
12	My pain won't sneak up on me as long as I keep thinking about it.	0.96	2013	0.06	0.76	0.73
8	My pain would get worse if I didn't think about it a lot.	0.89	2050	0.06	0.93	0.95
11	Focusing on the bad things about my pain helps me to enjoy the good things more.	0.78	2152	0.06	1.02	1.02
13	Thinking a lot about my pain helps me to cope with it.	0.47	2308	0.06	0.75	0.75
5	Thinking a lot about my pain protects me from getting injured.	-0.21	2720	0.06	1.14	1.14
10	Analyzing my pain prepares me for the worst.	-0.29	2783	0.06	0.84	0.83
16	Thinking about my pain helps me to understand myself better.	-0.35	2816	0.06	0.89	0.89
1	My pain won't improve unless I analyze it	-0.56	2909	0.06	1.25	1.26
2	When I'm thinking about my pain I'm trying to problem solve it.	-1.71	3591	0.06	1.42	1.40
Negative Metacognitions Subscale (N=809)						
Item		Threshold	Score	SE	Infit	Outfit
20	I don't try to stop thinking about my pain because my thoughts seem to have a life of their own.	0.61	2655	0.03	1.36	1.51
18	When I start thinking about my pain,	0.53	2750	0.03	1.14	1.18

	it's impossible to stop					
26	I make my pain worse by analyzing it	0.43	2858	0.03	1.12	1.15
39	I get caught in a vicious cycle of thinking about my pain and then thinking about how I wish I could stop thinking about it.	0.35	2959	0.03	0.83	0.83
33	I must block out my thoughts about pain.	0.22	3111	0.03	0.97	1.00
40	When I find myself brooding on my pain, it starts me thinking about how I'm just making things worse.	0.15	3188	0.03	0.71	0.72
38	I worry about the negative effects of thinking too much about my pain.	-0.06	3425	0.03	0.96	0.98
24	I would be less anxious if I didn't focus on my pain as much.	-0.26	3641	0.03	0.88	0.91
21	Thinking about my pain all the time makes me feel depressed	-0.3	3689	0.03	1.13	1.11
22	I'd be happier if I stopped thinking about pain.	-0.49	3884	0.03	0.84	0.85
23	I feel stressed if I think a lot about my pain.	-0.52	3913	0.03	0.94	0.89
36	It's important to control my thoughts about pain.	-0.65	4031	0.03	1.21	1.20

Note. Bold type shows excessive item misfit; negative item thresholds represent items that are easier to endorse, and positive thresholds represent items that are more difficult to endorse. SE=standard error of measure; score=raw score out of 6,048 (possible score of 7 × 864 people); Threshold =item endorsability threshold (expressed in logits); infit and outfit expressed as mean square (chi-square-based fit statistic).

Item 2, the most readily endorsed item on the positive subscale, and item 20, the hardest to endorse item on the negative subscale demonstrated some underfit but all other items functioned well. Visual inspection of the PCA correlation matrix of the positive subscale indicated items 1 and 2 were clustered away from the other items and their residuals were marginally related (r=.30). No other meaningful patterns were detected and no meaningful patterns were detected in the negative subscale matrix, suggesting the subscales can be considered unidimensional. The positive and negative subscales remained reliable, with person separation indices of .86 and .87 respectively. Analysis of DIF revealed no meaningful biases.

4.3.3.3 Temporal stability

A sub-sample of 172 participants answered the PMQ twice, one week apart. Both the positive and negative subscales of the PMQ demonstrated good test-retest reliability, with correlations of $r = .76$ ($p<.001$) and $r = .72$ ($p<.001$) respectively. Participants' overall symptom changes on the PGIC were not correlated with one-week changes on the PMQ positive subscale ($r = .04$, $p = .58$) or PMQ negative subscale ($r = .01$, $p = .95$), suggesting PMQ variation over a week is not associated with overall changes in participants' pain.

4.3.3.4 Construct validity

Correlations between the validation measures described above and the two subscales of the PMQ are shown Table 4.5. Providing evidence of convergent validity, both subscales significantly positively correlated with measures of pain intensity, pain interference, PC, pain cognitive intrusion, fear of pain, depression, anxiety, and metacognition, as expected. These were mostly significant at a conservative alpha level of $p<.001$. As expected, the negative PMQ subscale was significantly negatively correlated with mindfulness. Contrary to predictions, the negative correlation between mindfulness and the positive PMQ subscale did not reach significance. Providing evidence of discriminant validity, the positive PMQ subscale did not correlate with *a priori* unrelated demographic variables: country ($r=.05$, $p=.14$), marital status ($r=.01$, $p=.77$), and education level ($r=-.02$, $p=.66$). The negative PMQ scale was also uncorrelated with country ($r=.04$, $p=.22$) and marital status ($r=.02$, $p=.53$), although it was very weakly associated with education ($r=.07$, $p=.04$).

Table 4.5. Correlations between the Pain Metacognition Questionnaire and validation measures in initial validation sample (N = 864)

Variable	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
Positive pain metacognitions (PMQ-P)	–									
Negative pain metacognitions (PMQ-N)	.16***	–								
Pain intensity (BPI-P)	.10**	.16***	–							
Pain interference (BPI-I)	.14***	.29***	.68***	–						
Pain catastrophising (PCS)	.28***	.48***	.45***	.59***	–					
Pain intrusion (ECIP)	.25***	.49***	.32***	.46***	.70***	–				
Pain-related fear (TSK)	.36***	.32***	.24***	.41***	.49***	.44***	–			
Depression (HADS-D)	.10**	.34***	.32***	.56***	.50***	.49***	.40***	–		
Anxiety (HADS-A)	.16***	.42***	.24***	.40***	.50***	.48***	.32***	.59***	–	
Metacognition (MCQ-30)	.32***	.39***	.08*	.19**	.42***	.43***	.32***	.41***	.61***	–
Mindfulness (MAAS)	-.06	-.26**	-.01	-.13***	-.15***	-.23***	-.12***	-.31**	-.44**	-.46**

* $p < .05$ (two-tailed). ** $p < .01$ (two-tailed). ***$p < .001$.

4.3.4 Phase 1 Discussion

Consistent with predictions, preliminary validation of the PMQ shows it has good psychometric properties. Rasch analysis shows the PMQ is two-dimensional, comprising separate subscales assessing positive and negative metacognitions. This distinction is consistent with metacognitive theory, [206] as well as the qualitative results from Study 2. [389]

Removal of poorly functioning items yielded a 21-item scale comprising 9 items assessing how helpful people believe thinking extensively about their pain is (positive metacognitions), and 12 items assessing how uncontrollable or harmful they believe such rumination to be (negative metacognitions). Both subscales had good internal consistency reliability (.86, .87), test-retest reliability (.76 .72), as well as convergent and discriminant validity. Rasch analysis suggested the PMQ should use a 4-point rather than 7-point Likert scale. These preliminary findings therefore suggest it is possible to assess metacognitions about pain rumination using a self-report measure.

4.4 Phase 2: Further scale validation

4.4.1 Overview of objectives

Phase 2 aimed to further validate the revised PMQ in a new sample to confirm that the scale functioned as expected when presented to participants as a 21-item scale on a 4-point Likert rating scale, rather than a 40-item scale on a 7-point Likert scale as initially tested.

4.4.2 Method

4.4.2.1 Participants

A sample of 510 people was recruited online through the same Amazon Mechanical Turk (MTurk) forum described in Study 2. The same inclusion and

exclusion criteria applied, except that only people with chronic pain were included this time.

4.4.2.2 Measures

Demographic questions and the following measures described in Phase 1 were used again: Brief Pain Inventory (BPI), Pain Catastrophising Scale (PCS), Experience of Cognitive Intrusion in Pain (ECIP), and Hospital Anxiety Depression Scale (HADS). In addition, the Perseverative Thinking Questionnaire (PTQ) was administered to provide a more transdiagnostic measure of repetitive negative thinking than the ECIP. The revised 21-item Pain Metacognitions Questionnaire (PMQ) was also administered, scored on a 4 point Likert scale from 0-3 (see Appendix G for final scale).

4.4.2.2.1 Perseverative Thinking Questionnaire

The Perseverative Thinking Questionnaire (PTQ) [185] is a 15-item scale assessing various aspects of rumination and worry, including three subscales: core characteristics (e.g. "The same thoughts keep going through my mind again and again"), unproductiveness (e.g. "I keep asking myself questions without finding an answer"), and capturing mental capacity (e.g. "My thoughts prevent me from focusing on other things"). Using a 4-point Likert scale, PTQ scores range from 0-60 with higher scores indicating worse rumination. The PTQ has been validated in numerous samples, with high reliability of Cronbach's $\alpha = .95$ for the full scale and subscales ranging from $\alpha = .77$ $\alpha = .94$. [185] The PTQ was used to establish convergent validity.

4.4.2.3 Procedure

The same procedures used to recruit the MTurk sample in Phase 1 were repeated, with data collected online using Qualitrics™. Since retest reliability was not sought, the measures were only completed once by each participant.

4.4.2.4 Analysis

4.4.2.4.1 Rasch analysis of item functioning

The same Rasch analysis procedures described in Phase 1 were repeated to validate the findings of the initial analysis.

4.4.2.4.2 Construct validity

The same procedures used to assess convergent and discriminant validity in Phase 1 were repeated, however with the slightly different battery of measures described above. Criterion validity was also sought by testing whether the PMQ was able to predict clinical levels of PC according to data showing a score of 24 on the PCS best identifies 'patient' status. [262] The sample was dichotomised into those above and below this cut-off. A one-way analysis of variance was then conducted in SPSS to test whether there was a difference between high and low catastrophising groups on PMQ scores for both subscales.

Further evidence of construct validity was sought using multiple hierarchical regression to test whether the PMQ would uniquely predict PC once other related variables were controlled. Predictor variables were entered into a SPSS regression equation in five blocks: (1) demographics (age, gender, marital status, compensation status, work status, education, pain duration); (2) pain intensity and interference (BPI); (3) emotional distress (HADS); (4) cognitive intrusion (ECIP) and perseverative thinking (PTQ); (5) pain metacognition (PMQ).

4.4.2.4.3 Identification of cut-off scores

Receiver Operating Characteristic (ROC) curve analysis was used to estimate preliminary cut-off thresholds for clinically meaningful scores on the two PMQ sub-scales based on their ability to predict clinical levels of PC. This was defined as scores of 24 and above on the PCS. [262] ROC curves are commonly used to analyse and visualise the ability of screening tests to accurately predict

dichotomous conditions, such as diagnostic status, by plotting a test's sensitivity against 1-sensitivity. [409,410] The area under the ROC curve (AUC) varies between .5 (depicting a test that is no better than chance at identifying the disorder), and 1.0 (depicting a perfect test that has 100% sensitivity and 100% specificity) [411]. The AUC was calculated in MedCalc for Windows, version 17.7.2 (MedCalc Software, Ostend, Belgium). Following convention, a *p*-value < .05 for the AUC indicates it is significantly different from chance (.5) and the test can therefore distinguish between cases and non-cases. [412]

Calculating cut-off scores involves balancing sensitivity and specificity. A common method that gives equal weight to sensitivity and specificity involves finding the point on the ROC curve that has the maximum vertical distance from the diagonal chance line, which is termed the Youden index (*J*). [413] Depending on the risks associated with false positives and false negatives, sensitivity or specificity can also be prioritised. Since the clinical application of the PMQ would likely involve further assessment through clinical interview, it was deemed more important to flag possible cases rather than avoid over-diagnosis. Therefore, sensitivity was prioritised over specificity, with cut-offs selected based on a sensitivity closest to 80%. Separate ROC analyses were performed for each PMQ subscale.

4.4.3 Results

4.4.3.1 Sample characteristics

Almost all the 510 participants included in this sample lived in the United States (*n* = 496, 97.3%), although another six countries were represented. Demographic characteristics of the sample are shown in Appendix H, with overall very similar characteristics to the initial validation sample. Most participants were female (*n* = 306, 60%) and the mean age was 37.5 years (*SD* = 12.4). The mean pain duration was 6.43 years (*SD* = 7.44). A large proportion were employed (n = 362, 71%) and most were not involved in compensation claims (*n* = 449, 88%). The most common site of pain was the lower back (*n* = 305, 59.8%). Mean scores of the sample on the pain and psychological outcomes described above are presented in Table 4.6, with similar symptom levels as observed in the initial validation sample.

Table 4.6. Means, standard deviations and Cronbach's coefficient alphas of outcome measures for second validation sample (N = 510)

Outcome	Mean	SD	Interpretation	α
Positive pain metacognitions (PMQ-P)	-0.73	1.98	-	.88
Negative pain metacognitions (PMQ-N)	0.29	1.51	-	.87
Pain intensity (BPI-P)	4.97	1.66	Moderate[381]	.80
Pain interference (BPI-I)	4.73	2.47	-	.92
Pain Catastrophising (PCS)	26.27	10.75	Clinical[262]	.93
Cognitive intrusion of pain (ECIP)	27.75	15.33	-	.97
Perseverative thinking (PTQ)	29.60	7.61	-	.96
Depression (HADS-D)	6.72	4.29	Sub-clinical[408]	.82
Anxiety (HADS-A)	9.09	4.66	Clinical[408]	.82

Note. PMQ scores are expressed in logits rather than raw scores.

4.4.3.2 Rasch analysis of item functioning

Rasch analysis was conducted using the data from 510 persons. Table 4.7 shows the average item endorsability thresholds and fit statistics for each subscale. Replicating the previous analysis, the positive subscale items were relatively hard to endorse, with mean (SD) person agreeability -.72 (1.56) logits (range = -5.02 to 3.29 logits) less than the mean item endorsability 0 (.74) logits (range = -1.56 to .84 logits). The negative subscale items were comparable with mean person agreeability .20 (1.28) logits (range = -4.74 to 5.15 logits) and mean item endorsability 0 (.60) logits (range = -.86 to 1.03 logits). Fifteen persons (3%) registered a minimum score on the positive subscale and six persons (<1%) registered a maximum score. Two (<1%) persons registered a minimum score on the negative subscale and seven (1%) persons registered a maximum score. These findings supported the suggestion that floor and ceiling effects are negligible.

Table 4.7. Categorical order, item endorsability thresholds and fit statistics for the revised 21-item version of the Pain Metacognitions Questionnaire (N=510)

Positive Metacognitions Subscale						
Item		Threshold	Score	SE	Infit	Outfit
12	My pain won't sneak up on me as long as I keep thinking about it.	0.84	505	0.08	1.04	1.06
8	My pain would get worse if I didn't think about it a lot.	0.75	519	0.08	0.90	0.88
11	Focusing on the bad things about my pain helps me to enjoy the good things more.	0.73	523	0.08	1.08	1.07
5	Thinking a lot about my pain protects me from getting injured.	0.25	602	0.08	0.93	0.92
13	Thinking a lot about my pain helps me to cope with it.	0.22	607	0.08	0.85	0.87
16	Thinking about my pain helps me to understand myself better.	-0.14	668	0.08	0.94	0.94
1	My pain won't improve unless I analyze it.	-0.43	715	0.08	1.03	1.04
10	Analyzing my pain prepares me for the worst.	-0.67	755	0.08	0.95	0.97
2	When I'm thinking about my pain I'm trying to problem solve it.	-1.56	900	0.08	1.25	1.24
Negative Metacognitions Subscale						
Item		Threshold	Score	SE	Infit	Outfit
20	I don't try to stop thinking about my pain because my thoughts seem to have a life of their own.	1.03	632	0.07	1.26	1.35
18	When I start thinking about my pain, it's impossible to stop	0.69	703	0.07	0.97	0.97
26	I make my pain worse by analyzing it	0.56	729	0.07	1.00	1.03
39	I get caught in a vicious cycle of thinking about my pain and then thinking about how I wish I co...	0.43	756	0.07	0.90	0.90
40	When I find myself brooding on my pain, it starts me thinking about how I'm just making things worse.	0.19	803	0.07	0.82	0.82
38	I worry about the negative effects of thinking too much about my pain.	0.04	832	0.07	0.94	0.95
21	Thinking about my pain all the time	0.03	834	0.07	1.26	1.23

	makes me feel depressed					
33	I must block out my thoughts about pain.	-0.04	847	0.07	1.07	1.08
24	I would be less anxious if I didn't focus on my pain as much	-0.5	932	0.07	0.96	0.96
23	I feel stressed if I think a lot about my pain.	-0.78	981	0.08	0.84	0.81
22	I'd be happier if I stopped thinking about pain.	-0.79	984	0.08	0.93	0.91
36	It's important to control my thoughts about pain.	-0.86	996	0.08	0.95	0.97

Note. Negative item thresholds represent items that are easier to endorse, and positive thresholds represent items that are more difficult to endorse. SE=standard error of measure; score=raw score out of 1,530 (possible score of 3 × 510 people); Threshold = item endorsability threshold (expressed in logits); infit and outfit expressed as mean square (chi-square-based fit statistic).

Item 20, the hardest to endorse item on the negative subscale, demonstrated slight underfit but all other items functioned well. Visual inspection of the PCA correlation matrices for each of the subscales revealed no meaningful patterns in the data and contrast eigenvalues of 1.8 and 1.9 for the positive and negative subscales respectively suggested unidimensionality. No evidence of local dependence was noted suggesting no redundancy in the items.

Internal consistency reliability was maintained with person separation indices of .86 and .87 replicated for the positive and negative metacognitions subscales respectively. Analysis of DIF revealed no meaningful biases. Overall, these findings support the refinement of the draft items to form two subscale measures of positive and negative metacognitions.

4.4.3.3 Construct validity

Correlations between the validation measures described above and the two subscales of the PMQ are shown in Table 4.8. Providing evidence of convergent validity, both subscales significantly positively correlated with measures of pain intensity, pain interference, PC, pain cognitive intrusion, perseverative thinking, depression and anxiety, as expected, although not so strongly as to suggest scale

redundancy (r = .1 - .56). These were mostly significant at a conservative alpha level of $p < .001$. Providing evidence of discriminant validity, the positive PMQ subscale did not correlate with *a priori* unrelated demographic variables: country (r = .02, p = .62), marital status (r = -.06, p = .18), and education level (r = .07, p = .12). The negative PMQ scale was also uncorrelated with country (r = .05, p = .30) and marital status (r = -.04, p = .39), although it was very weakly associated with education (r = -.01, p = .86).

Providing evidence of criterion validity, the PMQ predicted pain catastrophising 'patient' status. [262] For positive metacognitions, significant ANOVA results [$F(1,508) = 35.22$, $p < .001$] showed people with high catastrophising scored higher on the PMQ (M = -0.31, SD = 1.97) than people with low catastrophising (M = -1.33, SD = 1.83). For negative metacognitions, significant ANOVA results [$F(1,508) = 89.62$, $p < .001$] showed people with elevated catastrophising scored higher on the PMQ (M = 0.78, SD = 1.51) than people with low catastrophising (M = -0.41, SD = 1.21).

Further evidence of construct validity was provided by the ability of pain metacognition to predict unique variance in pain catastrophising. As shown in Table 4.9, hierarchical multiple regression showed that the PMQ predicted a further 5% of pain catastrophising when a range of demographic, pain and psychological variables were controlled (R^2 change = .05, $p < .001$).

Table 4.8. Correlations between the Pain Metacognition Questionnaire and validation measures in further validation sample (N = 510)

Variable	1.	2.	3.	4.	5.	6.	7.	8.
Positive pain metacognitions (PMQ-P)	–							
Negative pain metacognitions (PMQ-N)	.35***	–						
Pain intensity (BPI-P)	.23**	.26***	–					
Pain interference (BPI-I)	.19***	.42***	.66***	–				
Pain catastrophising (PCS)	.35***	.56***	.41***	.55***	–			
Pain intrusion (ECIP)	.24***	.51***	.38***	.62***	.67***	–		
Perseverative thinking (PTQ)	.21***	.52***	.19***	.37***	.50***	.63***	–	
Depression (HADS-D)	.10*	.34***	.35***	.62***	.41***	.50***	.48***	–
Anxiety (HADS-A)	.19***	.45***	.27***	.51***	.51***	.58***	.68***	.64***

* $p < .05$ (two-tailed). ** $p < .01$ (two-tailed). *** $p < .001$ (two-tailed)

Table 4.9. Hierarchical regression analysis showing the unique contribution of pain metacognition to predicting pain catastrophising (N = 510)

Predictor variables	R^2	ΔR^2	ΔF	Beta
Step 1	.05	.05	$F_{(7,502)} = 3.64$**	
Age				-.01
Gender				.51
Marital status				-.32
Compensation				-.28
Work status				.15
Education				-.89**
Pain duration				-.07
Step 2	.32	.28	$F_{(2,500)} = 102.00$***	
Pain intensity (BPI-P)				.80*
Pain interference (BPI-I)				.33
Step 3	.40	.08	$F_{(2,498)} = 31.34$***	
Depression (HADS-D)				-.05
Anxiety (HADS-A)				.29*
Step 4	.52	.12	$F_{(2, 496)} = 63.79$***	
Perseverative thinking (PTQ)				.00
Cognitive intrusion (ECIP)				.26***
Step 5	.57	.05	$F_{(2,494)} = 28.31$***	
Positive metacognition (PMQ-P)				.70***
Negative metacognition (PMQ-N)				1.46***

* p < .05 (two-tailed). ** p < .01 (two-tailed). ***p < .001 (two-tailed).

4.4.3.4 Identification of cut-off scores

The ROC curve analyses yielded significant AUC for both PMQ subscales, showing people with clinical levels of catastrophising had higher positive metacognitions than those with non-clinical catastrophising 65% of the time (95% CI .61-.70, SE = 0.02, $p < .0001$). Similarly, people with clinical levels of catastrophising had higher negative metacognitions than those with non-clinical catastrophising 74% percent of the time (95% CI .70-.78, SE = 0.02, $p < .0001$). The Youden index suggested a cut-off for the positive metacognitions subscale of

>12 (J = 0.22, sensitivity = 49.8%, specificity = 72.5%), as depicted by a white circle on the ROC curve in Figure 4.2. However, prioritising sensitivity over specificity suggests a PMQ-P score of >9 (sensitivity = 76.9%, specificity = 42.6%), provides a better cut-off, as shown in Table 4.10.

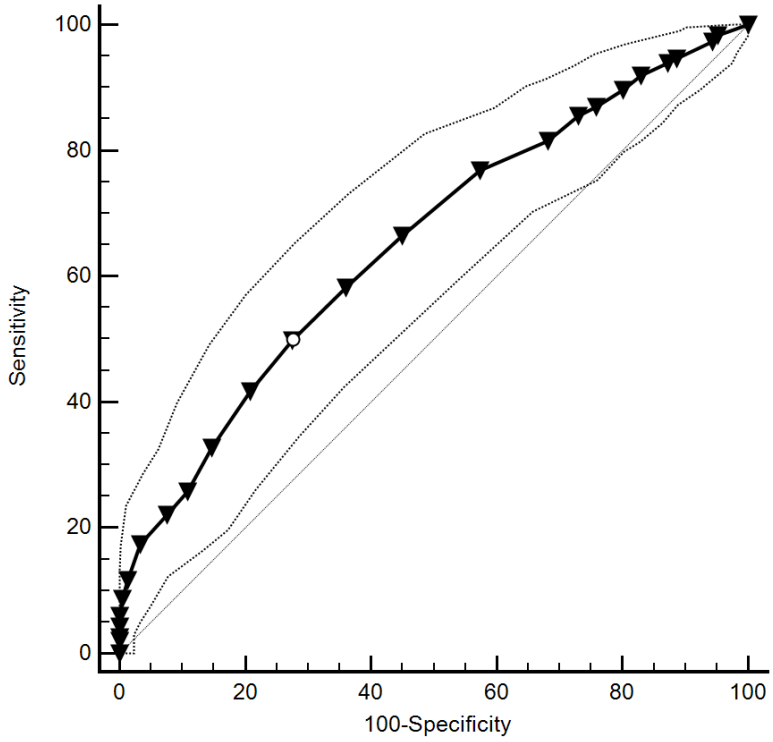


Figure 4.2. Receiver Operating Characteristic (ROC) curve and 95% CI for positive subscale of the Pain Metacognitions Questionnaire (PMQ-P).

Table 4.10. Coordinates of the ROC curve for values of the Pain Metacognitions Questionnaire, positive subscale (PMQ-P)

Score (PMQ-P)	Sensitivity	95% CI	Specificity	95% CI
>0	98.33	96.1 - 99.5	4.74	2.3 - 8.5
>2	94.65	91.5 - 96.9	11.37	7.4 - 16.5
>5	89.63	85.6 - 92.8	19.91	14.7 - 25.9
>7	85.62	81.1 - 89.4	27.01	21.1 - 33.5
>8	81.61	76.7 - 85.8	31.75	25.5 - 38.5
>9	76.92	71.7 - 81.6	42.65	35.9 - 49.6

>12	49.83	44.0 - 55.6	72.51	66.0 - 78.4

Note. Clinical levels of pain catastrophising (PCS>24), n=299; non-clinical catastrophising, n=211.

For the negative metacognitions subscale, the Youden index suggested a cut-off of >19 (J = 0.40, sensitivity = 69.6%, specificity = 70.1%), as depicted by a white circle on the ROC curve in Figure 4.3. However, prioritising sensitivity over specificity suggests a PMQ-N score of >18 (sensitivity = 77.6%, specificity = 59.7%) provides a better cut-off, as shown in Table 4.11.

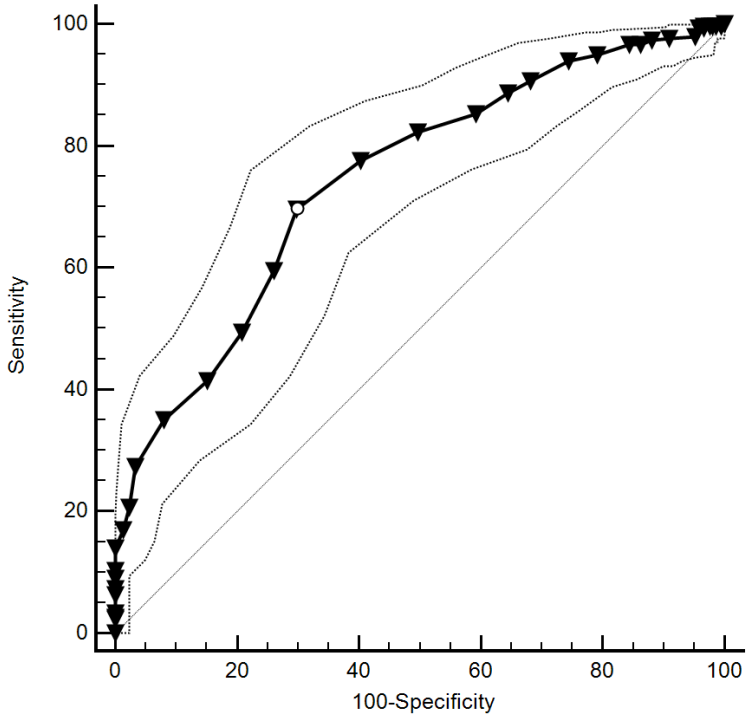


Figure 4.3. Receiver Operating Characteristic (ROC) curve and 95% CI for negative subscale of the Pain Metacognitions Questionnaire (PMQ-N).

Table 4.11. Coordinates of the ROC curve for values of the Pain Metacognitions Questionnaire, negative subscale (PMQ-N)

Score (PMQ-N)	Sensitivity	95% CI	Specificity	95% CI
>0	99.67	98.2 - 100.0	0.47	0.01 - 2.6
>12	94.98	91.9 - 97.2	20.85	15.6 - 27.0
>14	90.64	86.8 - 93.7	31.75	25.5 - 38.5
>16	85.28	80.8 - 89.1	40.76	34.1 - 47.7

>17	82.27	77.5 - 86.4	50.24	43.3 - 57.2
>18	77.59	72.4 - 82.2	59.72	52.8 - 66.4
>19	69.57	64.0 - 74.7	70.14	63.5 - 76.2

4.4.4 Phase 2 Discussion

Results from Study 3 show that the shortened 21-item PMQ retained excellent psychometric properties when tested in another large internet sample of people reporting chronic pain. Tests of construct validity were replicated and extended, with evidence that the PMQ predicts clinical levels of catastrophising and also uniquely predicts PC when several other variables are controlled. Finally, the provision of preliminary cut-offs for clinically meaningful scores on the PMQ pave the way for its use in both research and clinical settings.

4.5 General discussion

This study aimed to develop a psychometrically sound self-report measure of unhelpful metacognitions underlying pain-related rumination, thereby operationalising a new variable to target in pain research and treatment. Worry and rumination are key psychological processes underlying pain catastrophising, which is strongly linked to negative pain and health outcomes. [78] Metacognitions, or beliefs about thinking, have been shown to drive worry and rumination in people with anxiety and mood disorders, [205] suggesting they may underlie similar processes in people with pain. However, despite the existence of a generic self-report measure of metacognition, [364] there is no instrument tailored to the pain experience that also encompasses beliefs pertaining to both anxiety-related worry and depressogenic rumination. This study therefore filled this research-practice gap, showing it is possible to reliably measure pain metacognitions with a self-report instrument.

The resulting Pain Metacognitions Questionnaire (PMQ) is a 21-item bi-dimensional scale comprising a 9-item positive metacognition subscale (PMQ-P) measuring the extent to which people believe thinking a lot about their pain is

helpful, and a 12-item negative metacognition subscale (PMQ-N) measuring how uncontrollable and damaging people believe their thinking about pain to be. Both types of metacognition are unhelpful due to their tendency to facilitate worry and rumination. The scales have good internal consistency reliability (PSI = .86, .87 respectively) and test-retest reliability (r = .76, .72, respectively), as well as minimal floor and ceiling effects, making them suitable for use in both research and clinical settings. As predicted, they also correlate with measures of pain intensity, disability, catastrophising, perseverative thinking, cognitive intrusion of pain, fear of pain, depression, anxiety, metacognition and mindfulness (negative correlation). These associations were mostly of moderate strength, providing good evidence of construct validity but not redundancy. Based on its ability to predict clinical levels of pain catastrophising, a PMQ-P score >9 or PMQ-N score >18 is likely to be clinically meaningful.

The two-dimensional nature of the PMQ is consistent with metacognitive theory, which describes attitudes towards cognition as falling into distinct positive and negative categories. [205,359] It also reflects previously published qualitative results from Study 1. [389] The fact that these qualitative interviews were informed by metacognitive theory probably influenced this dimensionality in that participants were asked about their perceptions of the positive and negative aspects of rumination/worry about pain. The PMQ's dimensionality also mirrors the factor structure of a similar disorder-specific self-report measure of metacognition for people with chronic fatigue syndrome. [222] Although the PMQ's bi-dimensionality rules out the convenience of a single full-scale score, it may be helpful in future clinical applications of the scale. For example, Metacognitive Therapy (MCT), although so far untested in people with chronic pain, targets positive and negative metacognitions in different ways, [206] making the structure of the PMQ useful in any future trials of MCT for people with pain.

Rasch analysis showed that participants found it harder to endorse positive metacognitions than negative ones. This is also consistent with MCT literature showing that in clinical settings it is often harder to elicit positive metacognitions; therefore negative metacognitions are targeted first in MCT to allow time for meta-cognitive awareness to develop before positive beliefs about worry/rumination become more apparent. [206] Considering this, it is unsurprising

that people found it somewhat difficult to endorse positive metacognitions with a self-report questionnaire. The PMQ was nevertheless able to assess these beliefs with minimal floor effects, suggesting it remains a valid measure of positive metacognitions.

Many of the metacognitions reflected in the PMQ items also echo existing theoretical models of pain-related worry. For example, item 2 of the final scale (see Appendix G) describes thinking about pain as a form of problem-solving. This is consistent with the misdirected problem solving model of pain-related worry, which sees worry as an attempt to solve the problem of how to relieve persistent pain. [195] Items 1 and 4 are also consistent with this model in that they describe thinking about pain as a strategy to resolve pain or prevent it from getting worse. Similarly, items 6, 8 and 9 characterise worry/rumination as a form of coping, which is consistent with research depicting pain catastrophising as a coping behaviour, [86] as well as more recent functional analytic accounts of worry about pain as a self-regulation strategy aimed at reducing emotional distress. [178] Item 3 depicting worry/rumination as a strategy to prevent injury is consistent with a commonsense model of pain-related fear, linking avoidance behaviour to representations of pain as a sign of structural damage. [202] Similarly, the uncontrollability of worry/rumination captured by items 10 and 11 the negative subscale is consistent with models of hypervigilance [414] and cognitive intrusion, [383] which depict pain-related stimuli, including thoughts, as difficult to disengage from.

The research reported here has several strengths, including the fact that it used a rigorous evidence-based approach to scale development. While many self-report measures are drafted based on theory and expert opinion, [387] items for the PMQ emerged out of rich qualitative data as well as theory. Secondly, the two validation studies employed large samples that were well powered and largely comprised people with chronic pain rather than being non-clinical cohorts. Thirdly, the psychometric evaluation of PMQ data employed rigorous Rasch analysis based on Item Response Theory (IRT), which has several advantages over Classical Test Theory (CTT) techniques such as factor analysis. For example, IRT allows for a more thorough analysis of individual item functioning as well as producing true interval-level scales rather than ordinal scales produced in CTT. [388] The IRT

techniques used here also allowed for a data-driven approach to optimising response format, with the final 4-point Likert scale derived from an analysis of how participants used the scale. Finally, this study involved two phases or validation, with the final version of the scale tested in a fresh sample rather than relying on a single sample as is common during initial validation of new scales using exploratory factor analysis.

However, this study is not without limitations. Firstly, while the qualitative scale development sample (Study 2, Chapter 3) was a pain clinic sample that is representative of many other clinical samples, the validation samples employed internet samples which had lower pain intensity and disability than many pain clinic samples, [415] despite their clinical levels of catastrophising. This may limit generalisability of results and highlights the need to validate the PMQ in other samples with chronic pain disorders, such as those found in treatment settings where pain diagnoses are assessed by health professionals rather than merely self-reported. Furthermore, present results are susceptible to the biases inherent in all studies using self-report, such as social desirability bias, recall bias and context effects. [388]

Another significant limitation, which highlights the need for further research, is the fact that these three linked studies used cross-sectional designs, notwithstanding the test-retest analysis. This means it is unclear whether the PMQ is sensitive to treatment-related changes in pain metacognitions and therefore whether it can be used as an outcome measure in intervention research. Future validation studies could address this by collecting pre- and post-intervention data, particularly during psychological or multidisciplinary treatments that aim to reduce pain-related rumination and catastrophising. An important question is whether changes in pain catastrophising are associated with changes in pain metacognition (PMQ). Future research using prospective designs is needed to answer this. Prospective research is also needed to refine estimates of clinically meaningful cut-offs since the present suggestions should be considered preliminary and subject to revision.

More broadly, prospective research is needed to test the theoretical model that underlies this new measure, namely a metacognitive model of pain-related rumination. In metacognitive theory, unhelpful positive and negative

metacognitions function as risk factors for subsequent worry/rumination and indeed evidence in psychopathology literature supports this. [209] Future research should therefore test whether baseline PMQ scores predict future episodes of pain rumination or pain catastrophising, as would be expected. A precursor to this could also be to test whether metacognition moderates the relationship between pain intensity and rumination/worry, as would be expected according to metacognitive theory. Lastly, an obvious avenue for future research based on this theoretical work is the development of interventions targeting metacognitions, such as a form of MCT for people with elevated pain catastrophising. Present findings suggest this is warranted and that metacognition is a promising future treatment target.

In summary, this study shows that pain-related metacognitions can be validly and reliably measured using a new self-report instrument. The PMQ can be used in clinical and research settings and operationalises a psychological variable that warrants further investigation as a potential new treatment target in pain research. This has the potential to improve the efficacy of interventions for outcomes such as pain catastrophising and other forms of pain-related distress.

Chapter 5 Study 4 – Rumination mediates between pain intensity and catastrophising most strongly when unhelpful metacognitions are high: A moderated-mediation analysis in adults with chronic non-cancer pain

Chapter Linking Statement

The previous study developed and validated a new measure of pain-related metacognition. This was a prerequisite for being able to conduct quantitative research into the relationship between metacognition and other pain outcomes. The following chapter therefore uses this scale to test how metacognition relates to PC, the key pain construct of interest in this body of research given its well-documented association with negative pain outcomes. In particular, this scale is used to empirically test a model of how pain-related metacognition relates to pain intensity, repetitive negative thinking and PC. Developing and testing a theoretical model of pain-related metacognition is one of the main aims of this thesis since it paves the way for future treatment research that integrates metacognitive components and optimises PC interventions using a person-centred approach.

5.1 Abstract

Objectives: Pain catastrophising (PC) is linked to a range of negative pain, health and treatment outcomes, although debate continues about how best to define and treat it, since most interventions produce only modest benefit. This study aimed to contribute to the theory-driven development of these treatments by exploring the role of rumination in PC, along with the higher order beliefs, called metacognitions, that might shape it.

Methods: A cross-sectional internet sample of 510 people (mean[SD] age = 37.5[12.4] years; 60% female) with chronic non-cancer pain (≥3 months), who had clinically significant levels of PC, completed self-report measures of PC, pain intensity, disability, rumination, depression, anxiety, and pain metacognition. Regression-based moderated-mediation analysis tested the conditional indirect effect of pain intensity on PC via rumination at varying levels of unhelpful pain metacognition.

Results: Rumination partially mediated the effect of pain intensity on PC, accounting for 20% of the total effect. This indirect effect was conditional on both positive and negative metacognition. Higher levels of both forms of unhelpful metacognition strengthened the indirect effect, which was only significant above the 50th percentile for positive metacognitions or above the 60th percentile for negative metacognitions.

Discussion: Both strongly believing that thinking about pain helps one to solve problems or cope with pain (positive metacognition), or that rumination is harmful and uncontrollable (negative metacognition) increases the amount one ruminates as pain increases, which is associated with increased PC. Identifying and modifying these unhelpful pain metacognitions may improve treatments for PC and thereby chronic pain generally.

5.2 Introduction

Pain catastrophising is one of the most widely studied psychological variables in pain science, given its strong prospective links with negative pain and health outcomes [78,93,95] as well as poorer treatment response. [118] Pain catastrophising is often defined as an exaggerated negative mental set brought to bear during actual or anticipated pain, although affective and behavioural components are also involved. [79] The exact nature of PC remains contested, reflected by the lack of a unifying theory linking the various theoretical models of PC together, from appraisal-based formulations to interpersonal accounts such as the communal coping model. [82]

Compounding this theoretical plurality is the fact that a range of different interventions can treat elevated PC. A recent meta-analysis of 79 controlled trials targeting PC found at least nine different interventions showed efficacy (see Study 1). [380] While treatments combining Cognitive Behaviour Therapy (CBT) and exercise seem to perform best, evidence quality is moderate and most treatments only produce a modest effect that may not be clinically meaningful. This suggests the need to develop more effective and efficient interventions for PC, which might include different targeted interventions for different PC sub-types. [192,416] However such treatment development must be theory driven, since the proliferation of pragmatic mixes of treatment components has failed to improve the efficacy of psychological interventions for chronic pain more generally. [120]

The present study aims to address this question by re-examining the PC construct and, in particular, the role of rumination and related moderators. Rumination is one of three facets of PC measured by the dominant Pain Catastrophising Scale (PCS), [83] a facet derived from early research suggesting worry and hypervigilance were the defining features of PC. [84] Supporting this, validation of the PCS showed that rumination accounts for most of its variance [83] and some authors have argued that PC should be seen predominantly as a form of repetitive negative thinking, encompassing worry and rumination generally. [178] This view portrays PC as problematic because of its perseverative nature rather than because certain distorted or unhelpful pain beliefs cause distress.

Interrupting the perseverative process may involve novel treatment targets and interventions. While unhelpful beliefs are most commonly targeted with cognitive restructuring components of CBT, [88] perseverative thinking is largely engaged and maintained by higher order beliefs called metacognitions, according to metacognitive theory. [204,205] For example, implicitly held positive metacognitions, such as "worrying helps me to solve problems", promote perseveration by ascribing it a useful role. Negative metacognitions, such as "my worry is dangerous for me", may also fuel perseveration by triggering worry about worry (meta-worry). This model of repetitive negative thinking has been widely validated in the psychotherapy literature, with the associated intervention – Metacognitive Therapy (MCT) [206] – effective in treating a wide range of anxiety and mood disorders. [224] Other metacognitively-informed interventions, such as Acceptance and Commitment Therapy (ACT) and Mindfulness-Based Stress Reduction (MBSR), also have well-established efficacy, including for pain populations, [229] and for PC in particular. [135,136] However metacognitive beliefs are not targeted by these interventions as explicitly as in MCT.

Despite the efficacy of MCT in treating pathological worry and rumination, metacognition has received little attention in pain research. [358] The exception is a recent study showing positive metacognitions mediate the relationship between neuroticism and PC, [228] and a qualitative study documenting positive and negative metacognitions in people with chronic low back pain. [389] The present study therefore attempted to test a model of how rumination and metacognition are related to PC. Given the dominance of rumination in predicting variance in the PCS, we expected rumination to mediate the relationship between pain intensity and PC (Hypothesis 1). Secondly, since metacognitions are moderators of perseverative thinking in psychopathology samples, [205] we predicted that the same would apply in a pain sample. Specifically, we predicted that pain intensity would have an indirect effect on PC via rumination, but that this effect would be moderated by both positive and negative metacognition (Hypothesis 2). That is, stronger unhelpful metacognitive beliefs about pain-related thinking should be associated with more rumination and therefore higher levels of catastrophising.

5.3 Materials and Methods

5.3.1 Participants

Data for this study were collected during the second validation phase of the Pain Metacognitions Questionnaire (see Study 3, Chapter 4). An internet sample of adults with chronic pain (N=510) was recruited through Amazon Mechanical Turk (MTurk), an online labour market for low cost tasks that can be completed electronically. It has been reliably used in social science and pain research, [383] with participants shown to be more demographically diverse than the university undergraduate samples and general internet samples that are often used. [384,385] To be eligible, participants needed to be ≥18 years old, reside in a country where English is an official language, and report having a chronic pain condition (≥3 months duration). Participants with less than moderate pain, defined as a pain intensity of less than three on a 0-10 numerical rating scale, [381] were screened out.

5.3.2 Measures

The following self-report questionnaires were used, which are described in detail in Chapter 4: Brief Pain Inventory (BPI), [363] Pain Catastrophising Scale (PCS), [83] Perseverative Thinking Questionnaire (PTQ), [185] and the new Pain Metacognitions Questionnaire (PMQ), whose development and validation was the subject of Study 3 (Chapter 4). The Hospital Anxiety and Depression Scale (HADS) [365] was administered to test for confounds in the sensitivity analyses described below. Demographic data gathered in Study 3 were also used in sensitivity analyses. These included: age, gender, marital status, work status, compensation status, education level, and pain duration.

5.3.3 Procedure

This study received ethical approval from the Western Australian Department of Health (SMHS 2014-079) and Curtin University (HR23/2015). Recruitment took place from September to October 2016. Participants responding to an online

advertisement on MTurk were directed to a study link within the Qualtrics™ platform, [366] which contained participant information, informed consent questions, inclusion criteria screening questions, and online versions of the measures described above. Each measure was presented in a separate screen and participants were required to answer all questions to progress through the survey. Participants were free to withdraw at any time and were paid US$2 to participate.

5.3.4 Statistical Analysis

All data were analysed in IBM SPSS Statistics 24 for Macintosh. [406] Simple descriptive statistics were planned for sample demographics, along with bivariate Pearson correlations to quantify associations between variables. Two mediation models were also tested using the PROCESS macro for SPSS, [417] a tool for conducting path analysis using ordinary least squares regression. PROCESS uses bias-corrected bootstrap confidence intervals for inference about indirect effects, overcoming vulnerabilities to irregular sampling distributions that are common in least squares regression. [417] PROCESS has been found to produce equivalent results to structural equation modelling (SEM) for observed variable models such as those tested here. [418]

Two predefined models were used to test the mediation hypotheses above. The simple mediation model of the relationship between pain intensity and PC via rumination (H1) was tested with PROCESS model 4, [417] as depicted in in Figure 5.1. To test the moderated-mediation model – also called a conditional process model –PROCESS model 7 was used, as depicted in Figure 5.2. This tested whether the indirect effect of pain intensity on PC via rumination was conditional on pain metacognition (H2). Separate models were tested with positive metacognitions and negative metacognitions as the moderator since they represent separate dimensions of the PMQ. A confidence interval of 95% and 10,000 bias corrected bootstrap samples were used for all PROCESS tests. Sensitivity analysis was also performed by simultaneously entering several possible confounding variables as covariates into the PROCESS model: age, gender, marital status, compensation status, work status, pain duration, functional disability, depression and anxiety.

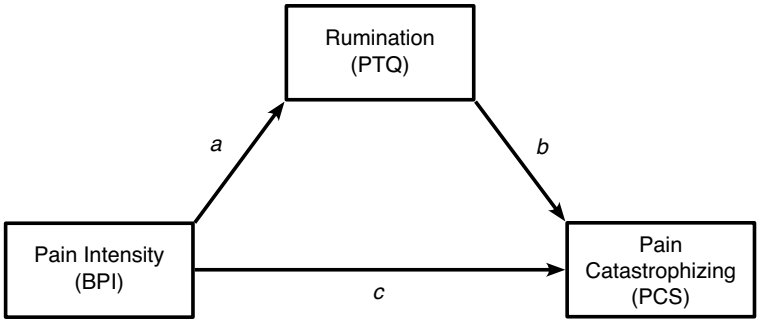


Figure 5.1. Statistical diagram of the simple mediation model

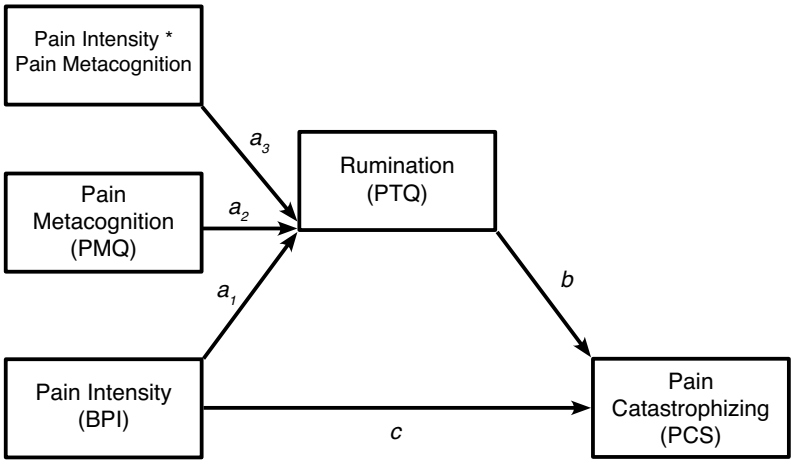


Figure 5.2. Statistical diagram of the moderated mediation model

5.4 Results

5.4.1 Sample characteristics

As described in Study 3, of the 635 people who began the survey, 109 were screened out for not meeting inclusion criteria and 51 only provided partial responses. This left 510 participants included in the final sample. Almost all resided in the United States (*n*=496, 97.3%), although another six countries were represented. Detailed demographic characteristics of the sample were reported in

Chapter 4. Most participants were female (*n*=306, 60%) and the mean age was 37.5 years (*SD* = 12.4). The mean pain duration was 6.43 years (*SD* = 7.44). A large proportion were employed (n=362, 71%) and most were not involved in compensation claims (*n*=449, 88%). All participants had non-cancer pain and the most common site of pain was the lower back (*n*=305, 59.8%). Average scores of the sample on variables in the conditional process model are presented in Table 5.1.

Table 5.1. Means, standard deviations and reliability of measures in mediation models (N = 510)

Outcome	Mean	SD	Interpretation	α
Positive pain metacognitions (PMQ-p)	11.36	5.26	-	.88
Negative pain metacognitions (PMQ-n)	19.66	6.11	-	.87
Pain intensity (BPI)	4.97	1.66	Moderate [a]	.80
Pain Catastrophising (PCS)	26.27	10.75	Clinical [b]	.93
Perseverative thinking (PTQ)	29.60	7.61	-	.96

Note. [a] According to criteria suggested by Anderson [381]; [b] According to criteria suggested by Scott et al. [262]. α: internal consistency reliability using Cronbach's coefficient.

5.4.2 Correlations

There were significant, mostly moderate, associations between all variables in the specified model, as shown in Table 5.2.

Table 5.2. Bivariate Pearson correlations between observed variables (N = 510)

Variable	1.	2.	3.	4.
Positive pain metacognitions (PMQ-P)	–			
Negative pain metacognitions (PMQ-N)	.35***	–		
Pain intensity (BPI-pain)	.23**	.26***	–	
Pain catastrophising (PCS)	.35***	.56***	.41***	–
Perseverative thinking (PTQ)	.21***	.52***	.19***	.50***

* $p < .05$ (two-tailed). ** $p < .01$ (two-tailed). *** $p < .001$ (two-tailed)

5.4.3 Mediation

Results of the simple mediation model (Figure 5.1) are summarised in Table 5.3. Supporting Hypothesis 1, the bias-corrected bootstrap confidence interval for the indirect effect did not cross zero, showing a significant mediation effect of pain intensity on pain catastrophising via rumination. The direct effect (path *c*) remained significant, indicating partial rather than full mediation. Using the ratio of indirect effect to total effect as a measure of effect size, [417] this partial mediation effect accounts for 20% of the total effect (95% bootstrap CI 11% to 29%). This simple mediation model accounts for 35% of the variance in pain catastrophising ($R^2 = .35$, $F(2,507) = 137.55$, $p < .001$).

Table 5.3. Results of regression-based mediation model showing the effect of pain intensity on pain catastrophising via rumination

Normal theory test						
Effect (path)	Coeff.	SE	t	p	LLCI	ULCI
Pain Intensity → Rumination (a)	1.60	0.36	4.41	<.001	0.89	2.31
Rumination → Pain Catastrophising (b)	0.36	0.03	11.87	<.001	0.30	0.42
Pain Intensity → Pain Catastrophising (c)	2.30	0.25	9.09	<.001	1.80	2.80
Bias corrected bootstrap test of indirect effect						
	Effect	Boot SE	BootLLCI	BootULCI	Effect size	
Pain Intensity → Rumination → Pain Catastrophising (a x b)	0.58	0.14	0.31	0.87	.20	

Note. Coeff.: unstandardised regression coefficient; *SE*: standard error; LLCI: lower level of the 95% confidence interval; ULCI: upper level of the 95% confidence interval; Effect size: ratio of the indirect effect to the total effect.

5.4.4 Moderated Mediation

Table 4 shows the results of the moderated mediation model (Figure 5.2), using positive metacognition as the moderator. The significant coefficients for paths a_1, *b* and *c*, along with the significant interaction effect depicted in path a_3, provide support for Hypothesis 2. The bias-corrected bootstrap confidence interval for the overall model remained above zero (index of moderated mediation = 0.09, *SE* = 0.02, Boot LLCI = 0.04, Boot ULCI = 0.14), indicating a significant conditional indirect effect. The model remained significant when possible confounds were simultaneously added as covariates. Table 5.5 shows the conditional indirect effects of pain intensity on catastrophising via rumination at different levels of positive metacognition. This highlights that the indirect effect is not significant at low levels of positive metacognition (10th and 25th percentiles). Figure 5.3 depicts this graphically, showing that the confidence interval is above zero at scores of 11 and above on the PMQ-P (50th percentile). Therefore, rumination partially mediates the relationship between pain and catastrophising, and this effect gets stronger the more people see rumination as helpful.

Table 5.4. Ordinary least squares regression coefficients for conditional indirect effect of pain intensity on pain catastrophising through rumination, with positive metacognition as moderator.

Outcome →	Rumination				Pain Catastrophising			
Predictor	Path	Coeff.	*SE*	*p*	Path	Coeff.	*SE*	*p*
Intercept		33.07	4.18	<.01		4.16	1.45	<.01
Pain Intensity	a_1	-1.70	0.81	.04	*c*	2.30	0.25	<.01
Rumination		-	-	-	*b*	0.36	0.03	<.01
Positive Metacognition	a_2	-0.81	0.33	.02		-	-	-
Pain Intensity x Positive Metacognition	a_3	.24	0.06	<.01		-	-	-
	R^2 = .10				R^2 = .35			
	$F(3,506)=18.69$, $p < .01$				$F(2,507)=137.55$, $p < .01$			

Note. Coeff.: unstandardised regression coefficients; *SE*: standard error.

Table 5.5. Conditional indirect effect of pain intensity on pain catastrophising through rumination at different values of positive metacognition.

Positive Metacognition	Percentile	Effect	Boot SE	Boot LLCI	Boot ULCI
4	10	-0.26	0.28	-0.83	0.26
9	25	0.17	0.18	-0.17	0.52
11	50*	0.35	0.15	0.07	0.64
15	75*	0.70	0.13	0.47	0.97
18	90*	0.96	0.16	0.67	1.30

Note: * Significant effect; SE: standard error; LLCI: lower level of the 95% confidence interval; ULCI: upper level of the 95% confidence interval. Effects are unadjusted for covariates.

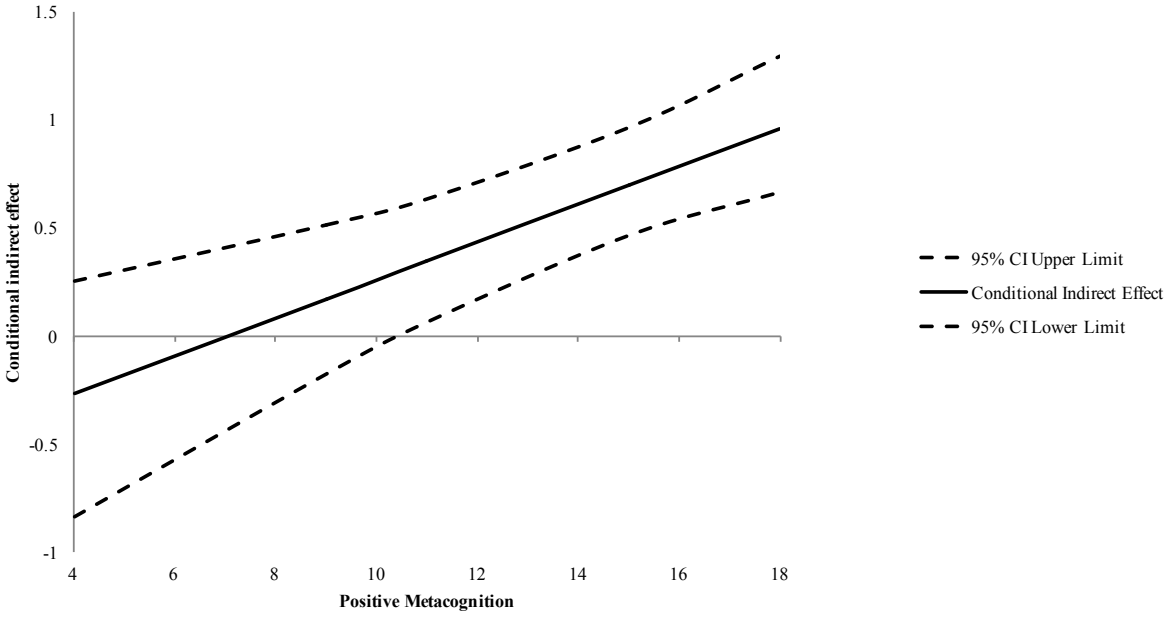


Figure 5.3. The conditional indirect effect of pain intensity on pain catastrophising through rumination at different values of positive metacognition (with bias-corrected bootstrap confidence intervals).

Results of the same tests of the moderated mediation model (Figure 5.2), this time with negative metacognition as moderator, are shown in Table 5.6. While paths *b* and *c* were significant, the pain-rumination path (a_1) was not. However, the interaction path (a_3) was significant, showing that negative metacognition moderates the pain-rumination relationship. The overall model of conditional indirect effect was significant since the bias-corrected bootstrap confidence interval did not cross zero (index of moderated mediation = 0.04, SE = 0.02, Boot LLCI = 0.00, Boot ULCI = 0.08). This further supports Hypothesis 2 and the

model remained significant when possible confounds were simultaneously added as covariates. Table 5.7 documents this conditional indirect effect of pain intensity on catastrophising via rumination at different levels of negative metacognition, which is shown graphically in Figure 5.4. Rumination only mediated between pain and catastrophising at high levels of negative metacognition, with the confidence interval only above zero for scores of 21 (60th percentile) and above on the PMQ-N.

Table 5.6. Ordinary least squares regression coefficients for conditional process model using negative metacognition as moderator.

Outcome →	Rumination				Pain Catastrophising			
Predictor	Path	Coeff.	*SE*	*p*	Path	Coeff.	*SE*	*p*
Intercept		18.12	4.98	<.01		4.16	1.45	<.01
Pain Intensity	a_1	-1.73	0.97	.07	*c*	2.30	0.25	<.01
Rumination		-	-	-	*b*	0.36	0.03	<.01
Negative Metacognition	a_2	0.44	0.24	.07		-	-	-
Rumination x Negative Metacognition	a_3	0.11	0.05	.01		-	-	-
	$R^2 = .27$				$R^2 = .35$			
	$F(3,506) = 61.11, p < .01$				$F(2,507) = 137.55, p < .01$			

Note. Coeff.: unstandardised regression coefficients; *SE*: standard error; R^2: coefficient of determination.

Table 5.7. Conditional indirect effect of pain intensity on pain catastrophising through rumination at different values of negative metacognition

Negative Metacognition	Percentile	Effect	Boot SE	Boot LLCI	Boot ULCI
12	10	-0.13	0.23	-0.60	0.31
16	25	0.03	0.17	-0.31	0.35
20	50	0.19	0.12	-0.04	0.44
23	75*	0.32	0.11	0.11	0.55
27	90*	0.48	0.14	0.20	0.77

Note: * Significant effect; SE: standard error; LLCI: lower level of the 95% confidence interval; ULCI: upper level of the 95% confidence interval. Effects are unadjusted for covariates.

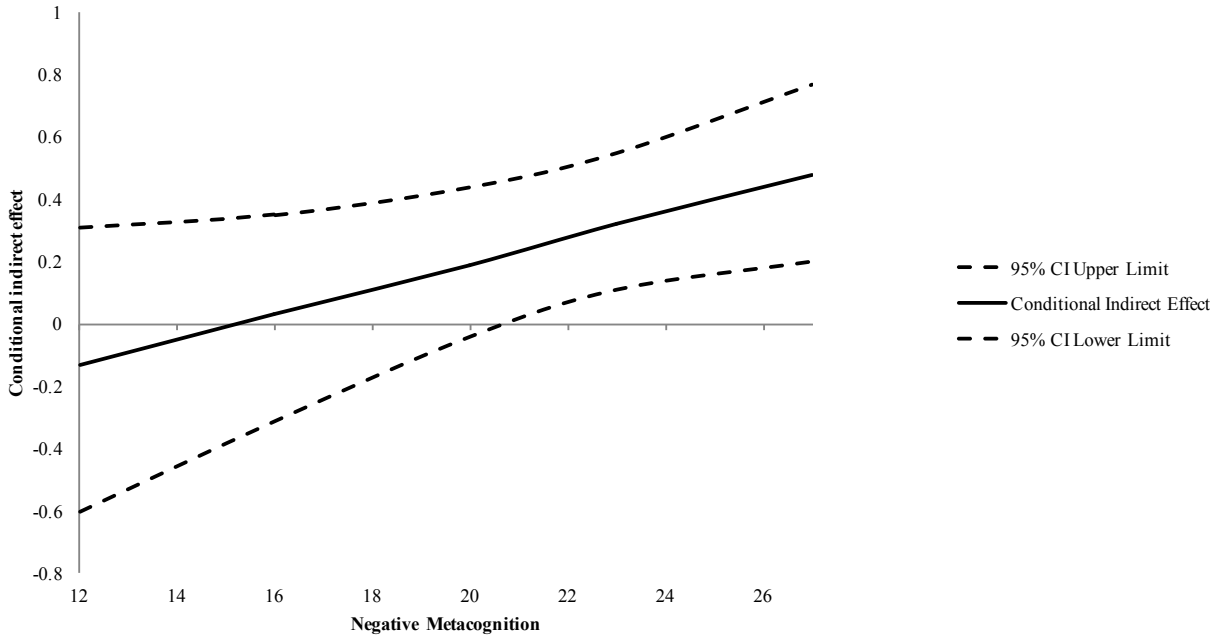


Figure 5.4. The conditional indirect effect of pain intensity on pain catastrophising through rumination at different values of negative metacognition (with bias-corrected bootstrap confidence intervals).

5.5 Discussion

This study aimed to develop a model of how rumination and metacognition relate to PC in an effort to facilitate theory-driven treatment development. Two hypotheses were tested: (1) rumination should mediate the relationship between pain intensity and PC given its strong role in predicting variance in the dominant scale of PC [83]; (2) positive and negative metacognitions should moderate the relationship between pain intensity and rumination, based on theory positing these higher order beliefs as mental templates guiding how much attention is given to threatening internal cues. [204] This moderation was expected to occur alongside the mediation effect, so that the indirect effect of pain on catastrophising through rumination would be conditional on metacognition. Results supported both hypotheses, providing the first known evidence of these relationships.

The simple mediation effect involved partial rather than full mediation, suggesting the direct path from pain intensity to PC is non-redundant when controlling for rumination. This is unsurprising given that the measure of PC used here had three subscales, with rumination being just one alongside magnification and helplessness. Results suggest that the effect of pain on PC is not solely due to rumination. This supports current conceptualisations of PC as a multifaceted construct involving a range of cognitive and affective dimensions,[78] rather than being solely repetitive negative thinking. [178]

Despite early evidence that the rumination subscale of the PCS accounts for most of its variance, [83] there are conflicting reports regarding the role of rumination in PC. For example, a recent study of 844 treatment-seeking adults with chronic pain and similar PC scores to our sample found that the helplessness and magnification subscales were most important, uniquely predicting various measures of pain and functioning, while the rumination subscale did not. [192] However, in another study the same researchers found that reductions in rumination during interdisciplinary pain treatment mediated improvements in pain intensity, disability and depression, while reductions in magnification were less important in predicting outcomes. [193] Present findings similarly suggest that targeting rumination in pain interventions may be one important way to reduce PC, and thereby improve other pain outcomes, [78] but it is not the only way.

These findings, however, do suggest that reducing PC by attenuating rumination should take into account people's attitudes towards their own thinking, or their metacognitions. The significant cross-sectional moderated-mediation models tested here suggest that people experiencing pain may be more likely to ruminate when they have strong unhelpful pain metacognitions, which may in turn increase their PC and associated risk of adverse outcomes,[78] although longitudinal data are needed to confirm this. This is consistent with metacognitive theory [359] and empirical findings. For example, in people with generalised anxiety disorder, the more that people believe worry helps them to solve problems or stay organised, the more likely they are to engage in pathological worry. [419] Similarly, present data suggest the more strongly someone believes that analysing their pain either helps them to solve problems, helps them to cope, or protects them from injury, the more likely they are to think excessively about their pain. Although not as intuitive, the

same occurs for negative metacognitions. The more harmful people believe rumination is for their mood or pain, the more they worry about their rumination, thereby fuelling the cycle of perseverative thinking.

Interestingly, the conditional indirect effect was more pronounced for positive metacognition than negative metacognition, as shown by the larger area above zero in Figure 5.3 than Figure 5.4. In other words, people need to have more exaggerated negative metacognitions (PMQ-N of 21, 60th percentile) than positive metacognitions (PMQ-P of 11, 50th percentile) for pain intensity to have an indirect effect on PC via rumination. This makes sense given that the negative items on the Pain Metacognitions Questionnaire (e.g. "I make my pain worse by analysing it.") are not as exaggerated as those in other metacognitive measures (e.g. "My worrying could make me go mad", "My worrying is dangerous for me") [364]. In a qualitative study of metacognition in people with chronic low back pain, some people described modest negative metacognitions (e.g. "Rumination is pointless") as actually motivating adaptive coping behaviours that reduced rumination, while only more pronounced negative metacognitions fuelled rumination through meta-worry [389]. Present findings support this notion that negative pain metacognitions are only problematic when very elevated, suggesting positive metacognitions should be targeted before negative metacognitions in treatment.

5.5.1 Clinical implications and future research

Indeed, these findings suggest that equipping people living with chronic pain with skills to attenuate high levels of unhelpful rumination might be one way to reduce their PC and thereby improve other pain outcomes such as pain intensity, functional disability, depression and medication misuse. [95] Reducing rumination is already a common treatment target in interventions such as CBT, ACT and MBSR and this study reinforces the rationale for addressing this. Moreover, present findings suggest that explicitly addressing unhelpful metacognitions through Socratic dialogue and behavioural experiments [205] could be another way to reduce rumination or to treat it more effectively. This is a central component in Metacognitive Therapy for anxiety and depression, [206] however MCT has never

been studied in people with chronic pain or elevated PC. Our findings provide a strong rationale for adapting this treatment for people with chronic pain, given that it is in some cases more effective than CBT in treating anxiety and depression, [224]

Just as disorder-specific MCT protocols exist for treating different psychological disorders, [206] MCT for chronic pain should be tailored to the pain-specific metacognitions found in this cohort. [389] Similarly, the attention-training techniques that are integral to MCT may require adaptation given the unique attentional demands found in people with chronic pain. [157,420] It is also possible that addressing pain metacognitions could be incorporated into existing theoretically congruent treatments to increase their efficacy. For example, identifying and modifying unhelpful pain metacognitions could be incorporated into newer CBT protocols that focus on reducing rumination [374] or mindfulness-based interventions that aim to reduce rumination through cognitive decentring. [241] It is likely that targeting metacognition will be most relevant to people prone to unhelpful rumination, which may in itself be only one subgroup of people with chronic pain or perhaps one particular phenotype of PC. Future treatment research is needed to explore this.

5.5.2 Limitations

Along with future treatment studies, further research also needed to overcome limitations in the research design used here. Caution is needed in interpreting the relationships in our conditional process model, given the cross-sectional nature of this study. Mediation models such as this cannot be interpreted as involving causal relationships. Future research using prospective designs is needed to determine whether, for example, pain has a causal effect on rumination. There is also the possibility that variables outside our model accounted for the conditional indirect effects. However, to mitigate this risk, we included possible confounding variables as covariates in the analyses, which did not nullify the effects.

The reliance on self-report measures in this study also introduces risks of error associated with social desirability responding, general response bias, and recall bias. [388] This was lessened by using measures with good psychometric properties, all of which demonstrated excellent reliability in this sample. Finally, while our

sample was large and provided good statistical power, it was a self-selecting internet sample with no independent verification of chronic pain status. Our sample had moderate pain and clinically significant PC; however participants generally reported less severe symptoms than many people attending multidisciplinary pain clinics, [415] which may limit generalisability to tertiary care chronic pain cohorts, for example. Future research should therefore replicate these findings in other clinical settings such as primary care.

5.5.3 Conclusions

This study provides the first empirical evidence that rumination mediates the relationship between pain intensity and PC. This pathway is strongest in people who strongly endorse unhelpful metacognitions about pain-related thinking, such as, “My pain won’t improve unless I analyse it”. This suggests perseverative thinking about pain is an implicit, although misdirected, [195] self-regulation strategy that ultimately backfires. Screening for unhelpful metacognitions, using the Pain Metacognitions Questionnaire, and then replacing these with more helpful metacognitions and associated coping behaviours has the potential to improve the efficacy of treatments for PC and pain outcomes more generally.

Chapter 6 General discussion

Pain is prevalent, costly and complex. Although it is the most frequent reason people seek medical help, most of the time their symptoms resolve. However, for a significant minority of people, especially those who have a combination of several risk factors, pain persists even when no significant tissue pathology can be found. Unsurprisingly, this can place an incredible strain on one's ability to work, sustain rewarding relationships, sleep well, have fun, and maintain a general sense of emotional wellbeing. As a result, people living with chronic pain frequently experience elevated symptoms of depression, anxiety and other kinds of negative affect, and these psychological sequelae can themselves contribute to ongoing pain through various neuro-immune changes including central sensitisation. [40,42,377] A vast body of research over the last 20 years now points to pain catastrophising (PC) as a key risk factor for pain persistence within this biopsychosocial model of pain, although exactly what this style of pain-related cognition is and what we should do about it continues to be debated. [78,178,421–423]

This thesis advances that debate by re-examining the construct of PC in an effort to better target treatment. The four studies presented here arose from the observation that our current suite of interventions for PC has variable content, costs, efficacy, and theoretical rationale, with no clear gold-standard. Before this project came to fruition in its current form, the intention was to trial an early intervention for PC in people with acute pain, given the extensive evidence that PC is a risk marker for future adverse pain outcomes. However, in devising a protocol for an evidence-based early intervention, it became evident that the treatment mechanisms of existing interventions were unclear and this partly reflected heterogeneity in how PC was operationalised. In particular, while incumbent CBT treatments drew on the theoretically plausible appraisal model of PC to target 'catastrophic misinterpretations', the *process* aspects of cognition, particularly the role of repetitive negative thinking, appeared under-developed. Therefore, rather than contribute to the proliferation of novel interventions by trialling 'pain focused Metacognitive Therapy', for example, these insights catalysed an exploration of the ruminative dimension of PC (and by implication its metacognitive features), in the hope that shedding empirical light on the construct itself would lead to better

treatments further down the translational research pipeline. The intention was to heed mounting well-considered calls for more theory-driven intervention research in pain psychology, to create more effective, efficient and well-targeted interventions. [28,126–128,131,424]

Therefore, this research had four aims, which were addressed in the four preceding chapters:

1. Systematically review the PC intervention literature and compare pooled effect sizes of different interventions in order to document the characteristics of interventions with efficacy in reducing PC.
2. Explore the lived experience of worry and rumination among people with elevated PC, including their metacognitive beliefs about pain-related thinking.
3. Develop and validate a self-report measure of pain-related metacognition that can be used in research and clinical settings.
4. Develop and test a model of pain-related metacognition in a sample of people with chronic pain.

In addressing the first of these aims, Study 1 (Chapter 2) involved the first known systematic review and meta-analysis of all randomised controlled trials that measure treatment-related changes in PC. A previous meta-analysis that considered only psychological therapies found that CBT was superior to behaviour therapy. [120] However, the new meta-analysis reported in Chapter 2, which considered all possible interventions, found CBT was just one of several interventions with merit. This meta-analysis found that at least nine different interventions have efficacy compared to usual care: ACT, CBT, EFT, exercise, graded exposure, manual therapy, mindfulness, multimodal treatment, and yoga. However, when only interventions with at least moderate quality evidence were considered, three interventions stood out: ACT, CBT and multimodal treatment. When comparing only the 8 studies that targeted PC as a primary outcome and that included people with high baseline PC, effect sizes were stronger. For example, the efficacy of CBT versus active control at post-test increased from SMD = -0.47 to SMD = -0.84, which represents a change from a medium to a large effect size. The best evidence among these targeted studies was observed for CBT, although

this was likely due to the small sample and lack of similar targeted research using other interventions.

Multimodal interventions that combined CBT and exercise had the biggest effect sizes when all included studies were considered. However, the high heterogeneity and disproportionate influence of one research group's data on the pooled effect reduce confidence in this estimate. If multimodal interventions are set aside, this review found no statistically significant difference between the interventions, which mostly had medium strength effect sizes. However, this lack of statistically significant difference may have been due to low power and there was a trend towards ACT having larger effects than CBT (e.g. SMD = -0.71 vs SMD = -0.25 at post-test compared to usual care). ACT was also the only intervention to have high quality evidence according to GRADE criteria, while another third wave psychological treatment – MBSR – was superior to CBT in a rigorous head-to-head trial. [136]

These findings are pertinent in the context of debate around theoretical models of PC. The appraisal model of PC was implicitly invoked in most of the included CBT-based interventions through their inclusion of cognitive restructuring exercises aimed at PC reduction. However, this is slightly speculative given that it was rare for research reports to articulate how their intervention components were matched to an operationalised definition of PC. Notwithstanding this, as discussed in Chapter 1, ACT interventions are based on the psychological flexibility model and explicitly do not try to change appraisals/beliefs. [240] The focus is instead on reducing the influence of negative automatic appraisals using exercises aimed at increasing 'defusion' from these thoughts. [239] Thus, a key goal in ACT and other third wave psychological treatments is to reduce rumination and worry by increasing mindful awareness. [229] The goal is to increase direct contact with moment-to-moment experience, rather than filtering sensory and affective information through the lens of evaluative thoughts about that experience. Thus, the goal of ACT, to quote the title of a popular self-help ACT book, is to "get out of your mind and into your life". [425]

This has theoretical relevance, because if non-appraisal oriented approaches (e.g. ACT, MBSR) can be as effective as, if not more effective than, an appraisal-oriented approach (e.g. CBT) in reducing PC then it calls into question the

primacy of the appraisal model of PC. Notwithstanding the strong association between pain beliefs and disability even when controlling for PC, [426] findings from this systematic review suggest we can reduce catastrophising very well without necessarily changing what people believe about their pain. Of course, changes in the content and process dimensions of cognition are not mutually exclusive and reductions in PC are likely to involve both aspects. [241] Furthermore, there is evidence that a content-focused intervention like CBT can modify cognitive process variables like mindfulness [427] and acceptance, [428] while process-focused third wave interventions like ACT can change content-level beliefs about pain control. [428] Therefore, the strong performance of ACT and to a lesser extent meditation in reducing PC is not evidence against the relevance of catastrophic beliefs but rather evidence that the process dimensions of PC are also important. These results therefore support more recent RNT models of PC which suggest the perseverative nature of cognition associated with PC is a significant problem, not just the beliefs themselves. [178,195] This, in turn justifies exploration of a factor known to influence RNT but which has received little attention in relation to pain – metacognition.

Study 2 (Chapter 3), therefore, began the empirical exploration of metacognition by interviewing people with CLBP and very high PC (i.e. PCS score > 30) about their beliefs about thinking. This was both the first qualitative study focusing on a purposive sample of people with elevated PC, and the first study to document specific pain metacognitions. As mentioned in Chapter 1, only two previous studies have addressed the relevance of metacognition to PC, and these took a nomothetic approach using instruments designed for non-pain samples. [226,228] The qualitative study reported in Chapter 3 therefore provided novel data about the lived experience of catastrophising and how people make sense of their own pain-related thinking.

The aim of documenting pain metacognitions was realised by reporting an array of beliefs that could be categorised as positive attitudes towards worry/rumination about pain (i.e. positive metacognitions) and negative attitudes towards it (i.e. negative metacognitions). This categorisation is consistent with the S-REF model of metacognition. [203,204] In ascribing a useful role to perseverative thinking, many participants said it was an attempt to solve problems around treatment decisions

and pain flare-ups, while others said it helped them to cope emotionally, feel in control, understand themselves, feel prepared for pain or even prevent injury. This notion of managing and preparing for symptoms parallels the positive metacognitions of people with chronic fatigue syndrome, who describe symptom analysis and self-focused attention as ways of controlling unwanted somatic experiences. [222,223] Furthermore, the belief that rumination/worry helps one to cope emotionally is consistent with Borkovec's avoidance theory of worry [179,180]. It therefore echoes the main tenets of Flink and colleagues' functional-analytic account of PC being a form of emotion regulation whereby abstract ruminative thinking reduces short-term distress by reducing experiential contact with unwanted thoughts, feelings and sensations. [178]

As reported in Study 2, negative cognitions were more prevalent and easier to elicit. Participants described how pain rumination was very difficult to control and could exacerbate their pain, distress and social relationships. Some people also described how these negative metacognitions prompted worry about the negative effects of worry itself, a key process that has also been found to maintain pathological worry in people with generalised anxiety disorder. [419] By contrast, some participants described how breaking out of ruminative cycles (e.g. using behavioural activation) was motivated by negative metacognitions. However, these tended to be more nuanced (e.g. "I don't feel that worrying about it is going to really help me") than the exaggerated negative metacognitions (e.g. "if you sit and think about it, you're doomed") which were sometimes described as triggering meta-worry. This distinction between different degrees of negative metacognition and how they relate to subsequent coping underlines the value of documenting pain-specific metacognitions in this cohort of people in pain rather than inferring them from existing psychopathology research on metacognition which has not documented such a distinction.

Importantly, results from this qualitative study provided an insight into how metacognition might influence thinking processes, as described above in relation to the onset of meta-worry or the interruption of rumination through behavioural activation. The way participants described bouts of perseverative thinking suggests it was influenced by their attitudes towards this type of thinking, which is consistent with the main tenets of metacognitive theory and the S-REF model.

[203,204] Of course, the causal influence of metacognition on rumination can only be inferred somewhat speculatively here because the qualitative data involved retrospective self-reporting, which is susceptible to a range of biases.

Despite this, participant reports of fluctuations in levels of pain rumination are consistent with the state/trait view of PC that has emerged from epidemiological, experimental and daily tracking data.[78] Like state/trait models of anxiety, [429,430] and panic disorder in particular, [431] this view suggests that people have a trait-like disposition towards more or less catastrophising but that levels of PC also fluctuate contextually. [167,191,357,432] The interviews documented in Study 2 gave voice to these fluctuations as people described the processes whereby instances of catastrophising were triggered, maintained and resolved. Therefore, this qualitative study advances our understanding of PC as a dynamic process that has multiple intrapsychic and contextual determinants and correlates.

These qualitative accounts of how metacognitions influenced perseveration or attenuation of pain-related thinking also provide an empirical rationale for the remaining studies in this thesis. Participants' descriptions of how seeing pain rumination as pointless motivated behavioural activation or concrete problem solving implies that metacognition has a moderating effect on the relationship between various triggers (e.g. a pain flare-up) and rumination about that trigger. In other words, it suggests metacognition moderates the relationship between pain intensity and rumination, which is a key feature of the metacognitive model of PC tested in Study 4. However, to test this model quantitatively, an instrument for measuring pain metacognitions was required.

Developing such a tool was the focus of Study 3 (Chapter 4), which describes the development and validation of the Pain Metacognitions Questionnaire (PMQ). This scale development study used the qualitative results from Study 2 to generate items aimed at capturing the dominant metacognitions related to pain. As discussed in Chapter 1, although many metacognitive studies use the MCQ-30 as a generic transdiagnostic measure, it has limited face validity in a pain sample, particularly because it only asks about worry rather than capturing RNT more generally. A strength of the PMQ, therefore, is that it captures both forms of RNT. Its content was also informed by empirical evidence as well as theory, similar to how a cohort-specific measure of metacognition was developed for people with

chronic fatigue [222]. Furthermore, validation of the PMQ was based on two large samples and involved rigorous item evaluation using Rasch analysis (based on item response theory) to create a true interval scale rather than the ordinal scales produced through factor analysis (classical test theory). [403,404]

The resulting 21-item PMQ is two dimensional, measuring positive (9 items) and negative (12 items) pain metacognitions on a four-point Likert scale. Both subscales have good test-retest reliability and internal consistency reliability, making it suitable for both research and clinical applications. As reported in Chapter 4, the PMQ has good construct validity, correlating positively with measures of pain, disability, PC, fear, depression, anxiety, and cognitive intrusion of pain, while correlating negatively with mindfulness, as expected. It was also able to uniquely predict PC when controlling for other variables, including predicting 'patient' status [262] on the PCS. Finally, this study provided preliminary suggestions for cut-offs for the PMQ, with a raw score of 9 for positive metacognition and 18 for negative metacognition indicating increased risk of clinically significant catastrophising. Study 3 therefore showed that it is possible to assess pain metacognitions using a brief self-report questionnaire.

Successful development of this psychometrically sound measure of pain metacognition facilitated the final aim of this thesis – to test a metacognitive model of PC. Study 4 (Chapter 5) therefore reports on a regression-based path analysis of a proposed moderated-mediation model. This model had two main components, derived from a review of existing theory and empirical findings presented in Chapter 1. In particular, the more recent RNT model of PC [178] was examined empirically by testing the hypothesis that the effect of pain intensity on PC would be mediated by RNT. The relationship between pain and PC was chosen because this is where PC fits into the FA model, the most widely studied biopsychosocial model of pain incorporating PC. [70] As discussed in Chapter 1, cognitive behavioural models of RNT [184] and metacognitive theory [205] suggest that how much someone ruminates depends to some extent on the activation of positive and negative metacognitions. In other words, metacognition moderates the degree to which rumination is associated with a given trigger. Therefore, it was predicted that positive and negative metacognitions would moderate the relationship between pain intensity and a transdiagnostic measure of RNT.

As reported in Study 4, these hypotheses were supported, with RNT partially mediating between pain and PC in a simple meditation model. A significant conditional indirect effect was also observed in separate models using positive and negative metacognition as moderators. In other words, pain intensity had an indirect effect on PC via rumination, but the strength of this indirect effect depended on people's levels of unhelpful metacognition. Consistent with theories of metacognition and RNT, this effect was strongest at higher levels of positive and negative metacognition. Strongly believing that thinking about pain helps one to solve problems or cope with pain (positive metacognition), or strongly believing that rumination is harmful and uncontrollable (negative metacognition) increases the amount one ruminates as pain increases, which is then associated with increased PC.

These findings are consistent with theories of RNT and metacognition [184,205] and mirror the qualitative findings in Study 2. However, it must be noted that these qualitative reports suggested a causal relationship between meta-beliefs and rumination, whereas the conditional process analysis in Study 4 is based on cross-sectional data that cannot show causation (as discussed below). Nonetheless, these data provide empirical support for a RNT model of PC that sees the preservative nature of thinking as a key feature of catastrophising. [178] As discussed earlier, this is not to say that individual beliefs (i.e. an appraisal model) are unimportant. Indeed, the fact that Study 4 found only partial mediation of rumination between pain intensity and PC suggests that PC is not merely rumination, even if the ruminative dimension is significant. This is consistent with previous research operationalising PC as more than just the rumination subscale of the PCS. [78,79,93,422]

6.1 Theoretical implications

These novel findings can be incorporated into existing models of PC, such as the FA model [71] and the misdirected problem solving model of worry. [195] Figure 6.1 depicts how the conditional indirect effect of pain in PC via rumination/worry might be added to the FA model. The misdirected problem solving model already focuses on RNT in the form of worry. Therefore, incorporating findings from

Study 4 only involves including the moderating effect of metacognition on the relationship between pain intensity and worry. This is depicted in Figure 6.2.

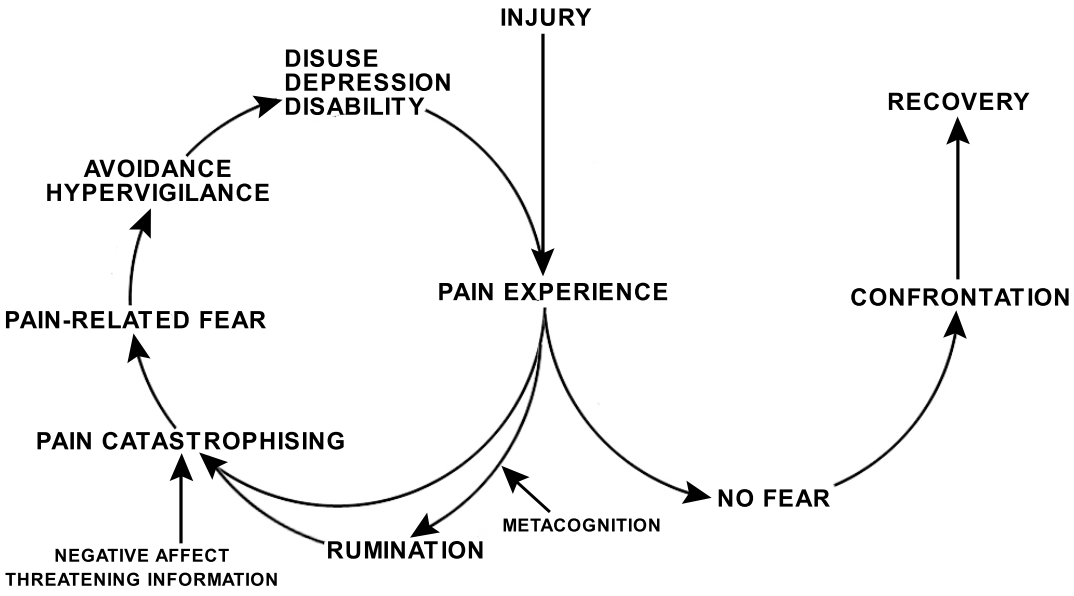


Figure 6.1. Revised version of the fear-avoidance model of pain, showing the indirect effect of pain intensity on pain catastrophising via rumination, dependent on different levels of positive and negative metacognition.

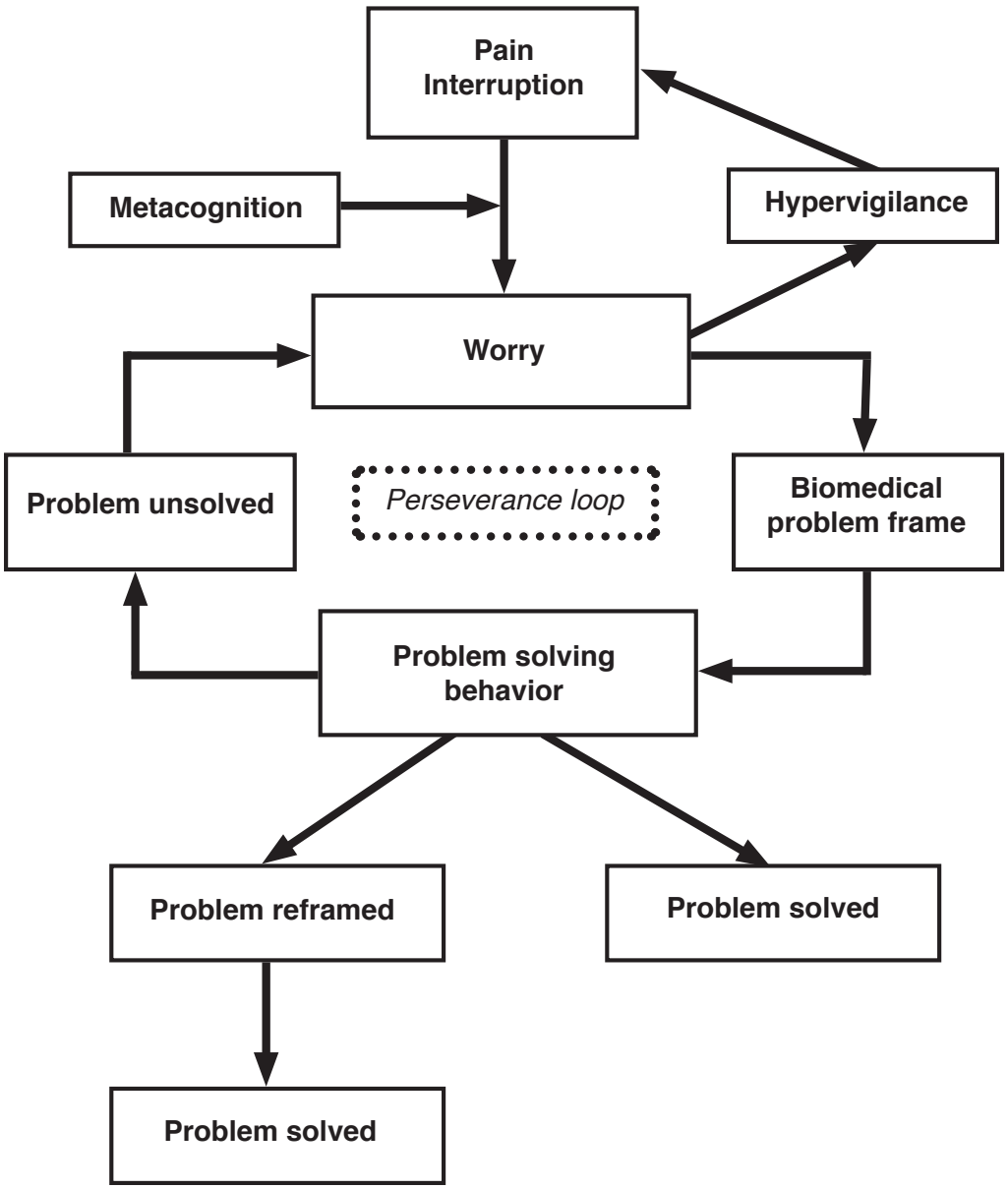


Figure 6.2. Revised version of the misdirected problem solving model of worry in pain, showing the moderating effect of positive and negative metacognition on the relationship between pain and worry.

Incorporating data from the present research into the S-REF model [359] and including findings from other studies in the wider pain literature allows for a more sophisticated, though somewhat speculative, metacognitive model (MCM) of chronic pain to be developed. Figure 6.3 therefore proposes a model of the cognitive attentional syndrome (CAS) in PC, which accounts for the role of metacognition in shaping pain-related thinking and the cascade of attentional, behavioural, and affective processes that stem from it. Although not traditionally

part of the S-REF model, neurophysiological correlates of PC (reviewed in Chapter 1) are also included. These are relevant because of the bidirectional relationship between ongoing pain and neuroplastic changes such as central sensitisation, [40,377]. Including these neurophysiological factors therefore facilitates a more comprehensive formulation of the pain-related CAS. Figure 6.4 provides an example of the MCM using quotes from a participant in the qualitative study (Chapter 3). Conversely, Figure 6.5 proposes a version of the MCM that is characterised by protective metacognitions and associated adaptive coping.

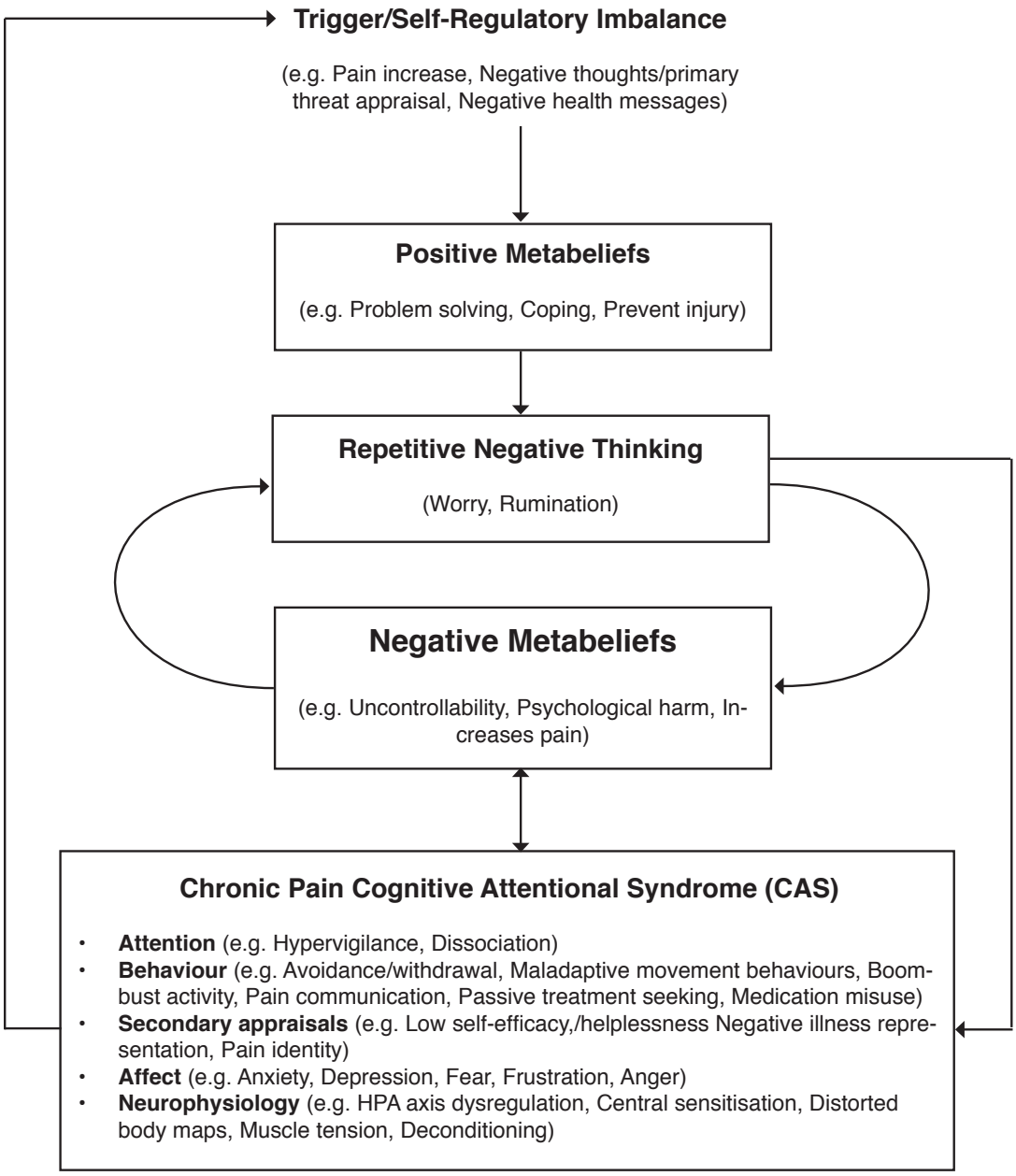


Figure 6.3. Metacognitive model of the cognitive attentional syndrome (CAS) in pain catastrophising.

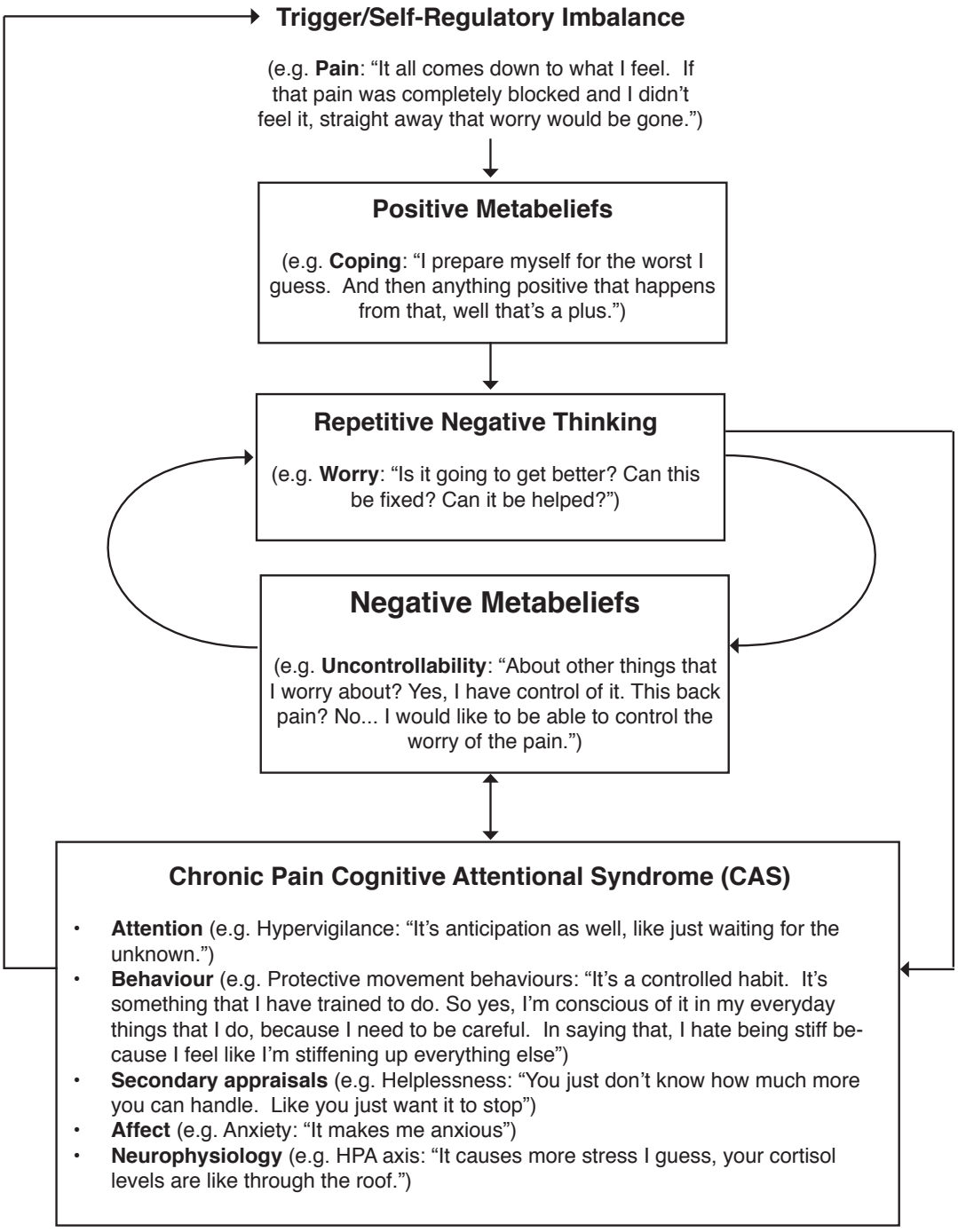


Figure 6.4. Illustrative example of the Metacognitive model of pain catastrophising using quotes from Study 2 participant, "Gail".

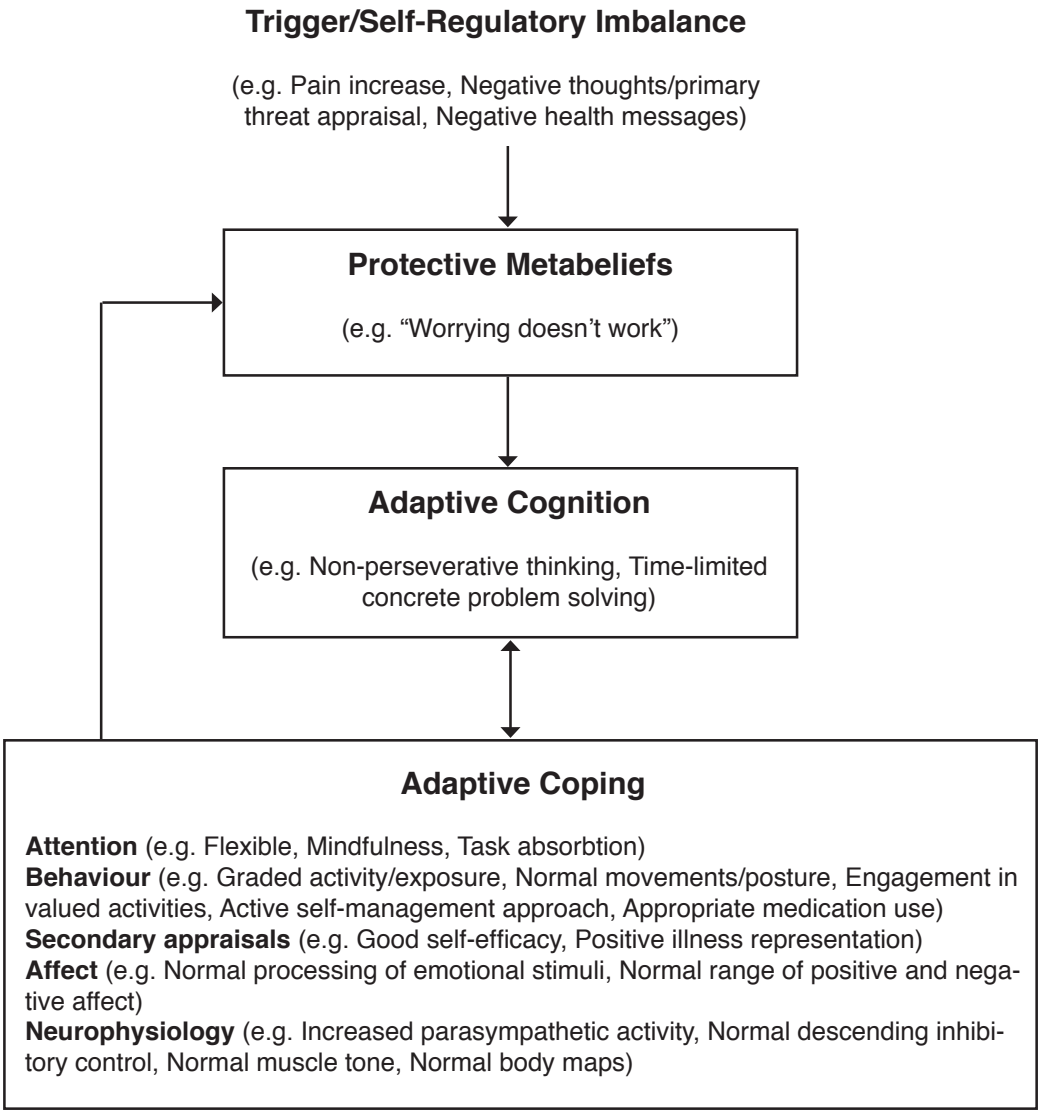


Figure 6.5. Metacognitive model of adaptive coping with chronic pain.

The proposed MCM of chronic pain integrates major elements of the FA model, [71] with the inclusion of affective variables (fear, depression, anxiety), attentional variables (hypervigilance, dissociation), behaviour (avoidance) and neurophysiology (deconditioning). It also includes pain communication behaviour, which is the central component of the communal coping model of PC. [167] The MCM also offers a more detailed version of ineffective problem solving that is relevant to the misdirected problem-solving model of worry. [195] This encompasses all perseverative thinking about a problem where the problem is abstract or poorly defined, including thinking aimed at absolute pain remission, which is the focus the misdirected problem solving model. The proposed MCM also includes

movement behaviours that are pain-provocative and often linked to PC-related fear-avoidance. [376,378,433] Furthermore, the MCM proposed here accounts for the distinction between primary threat appraisals and secondary coping appraisals (e.g. helplessness versus self-efficacy [434]) that have been noted by others and linked to the PCS subscales. [78,149] As previously suggested, a primary negative appraisal of pain is most likely linked to the magnification subscale, where the threat value of the pain is exaggerated. However, the MCM proposed here does not include rumination in the primary appraisal process, contrary to the suggestion of Severeijns and colleagues. [149] Rather it is suggested that rumination may contribute to both the primary appraisal of threat and secondary appraisal of coping ability (e.g. linked to the helplessness scale of the PCS). As shown in the MCM (Figure 6.3), the cascade of maladaptive coping behaviours associated with dwelling on pain likely results in low self-efficacy as these coping attempts fail or backfire.

The link between this sense of helplessness and primary appraisals of pain is usefully articulated in the recently developed commonsense model (CSM) of pain that was discussed in Chapter 1. [76,202] The CSM suggests fear, avoidance and loss of a sense of control stem from difficulties forming a useful representation of pain, which finds fertile ground amidst diagnostic uncertainty or conflicting messages from health professionals. [76,202] This results in emotion-focused and problem-focused coping attempts that are largely ineffective and often backfire. Catastrophising or general perseverative thinking associated with trying to make sense of pain, is one of the lynchpins of this process. Therefore, finding more effective ways to control pain through movement re-training, regulating emotion, and forming helpful illness representations are key aspects of recovery. [202,433] The MCM of chronic pain therefore suggests that challenging unhelpful metacognitions, and thereby reducing RNT and its associated negative coping responses, could be one way of 'gaining control', which is a key process involved in pathways out of pain-related fear and distress. [202,433]

The proposed MCM of chronic pain is not intended to supersede or replace existing theoretical models such as the FA model. It is offered more to bring existing pain research into focus through the specific lens of metacognitive theory and highlight new treatment targets that might be explored. The MCM represents

one way to integrate various theoretical models of PC, but it is not the only way. Furthermore, as a self-regulatory model, this MCM might also be incorporated into broader self-regulatory models such as the recent functional conceptualisation of 'embodied' pain as a defensive action rather than a passive experience. [3,435]

6.2 Strengths and limitations

One of the main strengths of this body of research is that it systematically developed a MCM of chronic non-cancer pain using existing theories to generate hypotheses which were then empirically tested. Furthermore, the data that informed this model were both qualitative and quantitative, responding to a significant gap in the PC literature, which is heavily biased towards quantitative data. More fundamentally, this research aimed to contribute to the broader enterprise of improving treatments for people with chronic non-cancer pain by not rushing to clinical trials but rather focusing on establishing a firm theoretical basis for any such intervention. Given the *a priori* rationale for targeting metacognition based on the narrative review in Chapter 1, it might be tempting to roll out a Phase 1 trial of Metacognitive Therapy [206] in a chronic pain cohort, for example. However, this would have been premature given the lack of an appropriate tool to measure pain-related metacognition and the lack of a pain-specific model of metacognition to guide the development of treatment components. Therefore, this body of research was focused upstream in the translational research pipeline in order to maximise the value of future downstream clinical applications and clinical trials. From an ethical perspective, this also maximises chances that future research participants are offered a well-targeted treatment that can be effectively evaluated.

The strengths of each of the studies comprising this thesis have been discussed in Chapters 2-5. Briefly, key strengths of the qualitative study (Study 1, Chapter 2) were its use of a clinical sample recruited from secondary and tertiary care settings, use of a well-validated qualitative methodology, and adherence to the COREQ design and reporting guidelines. Strengths of the systematic review (Study 2, Chapter 3) included its use of a prospectively registered protocol and adherence to gold standard reporting guidelines (PRISMA) and Cochrane

methodology. The sample was large for a meta-analysis and strict inclusion criteria resulted in generally good quality trials, allowing for greater confidence in the pooled outcomes. One of the significant strengths of the scale development study (Study 3, Chapter 4) is that its development was based not only on theory and expert opinion, as is common, but on direct empirical evidence from qualitative interviews and feedback. Furthermore, it used two large samples that provided good statistical power, and used a rigorous empirical approach based on item response theory to create a psychometrically sound measure appropriate for both research and clinical applications.

However, there are also some limitations of the research that must be acknowledged. The most obvious of these relates to the proposed MCM of PC, which implies a cognitive-behavioural process that unfolds over time, yet it is based on cross-sectional data. This is a pervasive problem with many path analytic studies, [418] and indeed much of the empirical data underlying the S-REF model is cross-sectional, despite some prospective designs validating the model. [209] Furthermore, the cross-sectional data used to validate the PMQ in Study 3 and test the basic MCM of pain in Study 4 was derived from a self-selecting internet sample. This may therefore have limited generalisability to other cohorts such as people seeking treatment in tertiary pain clinics, for example. However, the included sample was almost entirely people with chronic non-cancer pain, and shared many demographic and clinical characteristics with other clinical samples. Despite this, the included samples that had slightly lower pain intensity and disability than comparable samples recruited in tertiary care. [415] Furthermore, while self-report measures are the gold-standard for many pain outcomes, [436] the validity of clinical diagnoses was weakened by not being verified by a clinician.

There are also broader threats to validity that are common among self-report scales, including social desirability bias, general responding bias, poor recall, and inattention. [388] Indeed, issues around social desirability, retrospective reporting and limited insight may have impacted the qualitative findings in Study 2 as well. In particular, levels of metacognitive awareness vary between individuals and in MCT preliminary work is often needed to help people develop metacognitive insight before effective therapy can take place. [206] It is therefore possible that some people were unable to report on latent metacognitions, resulting in some

metacognitions being omitted from item generation in the scale development. However, this is a limitation shared by all self-report scales to some extent and attempts were made to compensate by generating items based on theory and clinical experience, in addition to the results of qualitative interviewing.

Another potential threat to validity was the fact that some of the internet sample – those recruited through Amazon Mechanical Turk (MTurk) – were paid a nominal fee to participate and as such might have responded randomly or fabricated answers purely to complete the tasks and receive payment. However, the theory-consistent patterns of correlations, as well as similar results observed between the MTurk and non-paid snowballing internet samples suggests this was not the case. Therefore, the main limitations of the present research do not involve serious threats to internal validity of the data and subsequent analyses, but rather reflect the need for further research to verify and extend the findings and the interpretations offered here.

6.3 Implications for theory and research

Limitations in the representativeness of samples used here could be addressed in future research by replicating these findings using different samples. For example, it would be useful to validate the PMQ and replicate the moderated-mediation model underlying the MCM, in a clinical treatment-seeking sample from a multidisciplinary pain clinic. Specific cohorts could also be targeted to examine whether the correlational relationships examined here vary between people with different pain conditions (e.g. musculoskeletal pain, neuropathic pain, postoperative pain). It would also be useful to understand how chronicity might impact these relationships. For example, future research could validate the PMQ and the MCM in people without pain or those experiencing acute pain.

A particularly important area for future research will be the use of prospective designs. The relationships between pain, metacognition, rumination and PC should be tested using such designs to shed more light on the temporal and perhaps causal nature of these relationships. Given that metacognitions are somewhat stable in the absence of treatment, [246] it would be expected that levels of unhelpful

metacognition on the PMQ at a given time should predict rumination and PC at a later point in time. For example, in a prospective study of the metacognitive model of depression, Papageorgiou and Wells [209] showed that negative metacognitions prospectively predicted depressive symptoms in a non-clinical sample. Therefore, just as longitudinal studies have established the prognostic value of PC in predicting subsequent pain, disability, distress and various other adverse outcomes, [78] pain metacognition should prospectively predict future levels of rumination and PC, according to the moderated-mediation model validated in Study 4.

The broader MCM proposed above (see Figure 6.3) implies a range of testable hypotheses, including that scores on the PMQ should prospectively predict all the variables listed under 'chronic pain cognitive attentional syndrome'. Using prospective designs to test these hypotheses would not only help elucidate the core tenets of the MCM, it would provide further evidence of the PMQ's construct validity. For example, if scores on the PMQ prospectively predicted clinical levels of PC or a transition to chronicity, this would provide evidence of criterion validity. Therefore, while there is still scope to do further cross-sectional research to test the associations implied within the MCM of pain, the most useful information is likely to come from prospective designs.

Examining treatment-related changes in pain metacognition is another important area for future research using such prospective designs. Existing psychopathology research on MCT suggest metacognitions are modifiable beliefs that are responsive to treatment. [205,207,224] Therefore, scores on the PMQ should change in response to treatments like ACT and MBCT, which implicitly target metacognition. Indeed, given that existing treatments do not exclusively modify theory-consistent process variables, [427,428] one would expect that metacognition would vary as other outcomes vary. Since the S-REF model suggests latent metacognitions are 'activated' by different triggers (e.g. unwanted thoughts, feelings, sensations) and that the ensuing CAS sets up a feedback loop that modulates these triggers, [205,359] it is plausible that a reduction in pain intensity, for example, might result in a reduction in unhelpful metacognition. Future research could easily test these relationships by including the PMQ as a secondary outcome in chronic pain intervention studies. A useful application of this would be to mimic previous treatment mechanism studies in the pain literature that use cross-lagged

designs to examine whether early changes in mediator/process variables predict later changes in primary outcomes. [115,117] For example, future research could use such a design to test whether early reductions in unhelpful metacognition predict later reductions in PC. Again, such research would contribute to further validating both the MCM of pain and the PMQ.

Beyond examining how metacognition varies in response to existing pain interventions, the most obvious form of treatment research that this thesis points towards is the development of metacognitively-focused interventions for people in pain. Present findings suggest this is warranted, although the prospective research described above is required to inform this treatment development. One possibility would be to trial a form of MCT in people with elevated PC using existing MCT protocols developed by Adrian Wells. [206] Given the effectiveness of MCT for a range of anxiety and mood disorders, [224] as well as health concerns such as fear of cancer recurrence, [217,218] there is a sound rationale for this. Such research should begin with pilot studies and case series' before moving to clinical trials, particularly in order to examine whether the protocol is appropriate for people experiencing pain. There are indications that some aspects of traditional MCT such as the attention training technique (ATT) may be less effective in the context of pain. For example, an experimental study using the cold pressor task with undergraduate students found that ATT was better than relaxation training in reducing hypervigilance but was not superior in terms of pain tolerance or pain intensity. [437] While this was only a brief ATT intervention and several theory-consistent effects were noted, [437] it is possible that the attentional demands related to pain make this form of ATT, which uses sound stimuli to reduce self-focused attention, challenging for people in pain. Adaptations to existing MCT protocols may therefore be needed.

Aside from trialling MCT, another way to test whether targeting pain metacognitions can be helpful for people with elevated PC would be to modify other effective treatments. For example, as discussed earlier, third-wave interventions such as ACT, MBSR and MBCT have efficacy for pain, disability, depression, quality of life and PC. [229,233] Furthermore, they implicitly challenge unhelpful metacognitions through cognitive defusion/decentring exercises aimed at showing how evaluative thinking is not always necessary, helpful or

unavoidable. [241] That is, someone practising mindfulness meditation may come to realise they can cope with a flare up without analysing it or worrying about it, but rather by attempting to stay focused on their moment-to-moment experience with a non-judgmental attitude.[438] Much like behavioural experiments lead to cognitive restructuring in CBT, [154] a positive experience using this mindful approach implicitly challenges positive metacognitive beliefs that RNT helps one solve problems or cope with their pain. Similarly, the increasing control over attention that develops with ongoing mindfulness practice serves to implicitly challenge uncontrollability metacognitions.

It is possible that these processes of metacognitive change could be enhanced by more explicitly targeting metacognitions within these existing approaches. For example, metacognitions could be assessed and discussed explicitly using the PMQ and clinical interview, or even through group discussion at the beginning of a mindfulness intervention (e.g. MBCT). The weekly home meditation practice that is foundational to mindfulness interventions could then serve as an explicit behavioural experiment for testing these metacognitions. The practice reflection discussions that are integral components of mindfulness interventions, [238] could then serve as opportunities for explicit cognitive restructuring of these metacognitions, just as metacognitive reattribution is a core feature of MCT. [206] This is one example of how the current research might facilitate an enhancement of current interventions rather than needing to re-invent treatments for PC altogether.

One of the main objections to this from proponents of MCT may be that mindfulness meditation, unlike ATT, could encourage greater interoceptive awareness. This often viewed negatively within MCT because it is reminiscent of 'self-focused attention', which is linked to psychopathology. [205] For example in social anxiety disorder, health anxiety, and panic disorder, a significant maintaining factor is attentional bias towards unwanted interoceptive cues, along with the ensuing RNT about those cues. [439] However, concerns about self-focused attention being exacerbated by meditation have little empirical evidence and this argument conflates hypervigilant attentional bias with mindful awareness. Mindfulness is more than awareness of sensations in the body or thoughts in the mind; it is awareness of these phenomena coupled with an attitude of acceptance

and non-judgment/non-evaluation. The self-focused attention that is criticised in MCT involves awareness coupled with pronounced judgment and evaluation (i.e. RNT). Previous research shows hypervigilance is positively correlated with the 'observing' facet of mindfulness but negatively correlated with the 'non-judging' facet. [73] Indeed, the non-judging facet contributes most of the variance in mindfulness' association with lower PC, anxiety, and depression, [73] suggesting it is the cultivation of an accepting, non-judgmental, non-elaborative attitude that is most important, not where attention is focused.

Furthermore, there is reason to expect that developing a healthy form of self-focused attention, is an important goal for people with chronic pain. Given the intrusive nature of pain on attention, [383] and the neurobiological substrates of this including activation of the salience network, [440] it is perhaps unrealistic to expect an attenuation of interoceptive awareness. Rather, a more helpful goal would be the development of attentional flexibility so that attention can move between interoceptive and exteroceptive cues at will rather than becoming rigidly focused on pain-related stimuli as commonly occurs with hypervigilance. [155] A key strategy for achieving this may be to focus more on non-elaboration of cognition rather than on shifting attention to environmental cues. That is, the attentional training goal for people with pain may be more about the quality and flexibility of the attention rather than the attentional referent.

The value of adaptive self-focused attention in pain management is supported by the accumulating neuroimaging evidence around meditative analgesia. A recent review of this literature suggests that sustained direct monitoring of sensory experience through open monitoring styles of mindfulness meditation is associated with decreased activation of brain regions involved in fear (e.g. amygdala), memory (e.g. hippocampus) and elaborative mental narratives (e.g. dorsolateral prefrontal cortex, medial prefrontal cortex, orbitofrontal cortex). [235] It is suggested that through mindfulness training a decoupling occurs between brains regions associated with the monitoring of sensory experience and those associated with elaborative cognition. [235] Furthermore, it is in fact the enhanced attention to sensation that facilitates this through passive decay of memory-dependent processes underlying higher order thinking. [235] This is supported by evidence that meditation reduces rumination [441] and is well-suited to reducing brain activity

associated with intrinsic attention to pain, a trait that is elevated in people with chronic pain. [440] Taken together, this evidence suggests that mindfulness meditation is a form of attention training appropriate for people in pain, particularly those prone to rumination about their pain. Certainly, there is a stronger evidence base for using mindfulness-based attention training in people with pain than there is for using sound-based techniques used in MCT. Therefore, results from this research suggest integrating metacognitive reattribution exercises into existing mindfulness-based interventions may be an effective way to treat elevated pain rumination, and therefore PC. Future research is needed to explore this; however, larger effect sizes for metacognitively-enhanced mindfulness protocols would be expected.

Another treatment approach for reducing RNT about pain might involve transforming it into more adaptive forms of elaborative cognition rather than seeking to attenuate it altogether. This is one approach taken in newer forms of CBT aimed at addressing RNT, such as Rumination-Focused Cognitive-Behavioural Therapy for Depression (RF-CBT). [374] This approach focuses on replacing abstract, evaluative cognition that is characteristic of RNT, with "concrete, process-focused, and specific thinking" [374(p21)]. This often involves using functional-analysis to facilitate more effective structured problem solving, which is a therapeutic goal emphasised in the RNT models of PC discussed in Chapter 1. [178,195] Therefore, another avenue for extending the findings of this thesis would be to more directly target rumination within CBT interventions, for example by trialling an adapted version of RF-CBT for people with chronic pain and elevated PC.

However, combining cognitive restructuring exercises that are commonly used in CBT for pain with metacognitive reattribution exercises is potentially fraught. Proponents of MCT certainly warn against conflating the two approaches because for people prone to rumination, evidence-testing thoughts easily spirals into maladaptive RNT. [206] Similarly, despite being situated within the cognitive behavioural tradition, RF-CBT does not target specific appraisals or beliefs but rather the maladaptive process of rumination. Therefore, it is suggested that integration of the present findings into existing treatments would most helpfully

involve a focus on cognitive *processes* rather than *contents* where RNT is identified as a significant problem.

This highlights a more fundamental question posed by the present findings. That is, should PC be viewed as a uniform characteristic/process across individuals, or might there be different sub-types of catastrophising? Given the variable response to different interventions by people with elevated PC (see Study 1), along with conflicting evidence surrounding the relative importance of the different PCS subscales in predicting pain outcomes, [192] it seems likely that there are different clinical profiles of catastrophising. The only partial mediation of rumination between pain and PC (see Study 4) may even reflect this heterogeneity. Similarly, the qualitative findings (see Study 2) showed people reported significant differences in how much they engaged in perseverative thinking, even though they all scored very highly on a measure of catastrophising (PCS).

From a functional self-regulatory perspective it is plausible that someone's elevated catastrophising on the PCS could be driven predominantly by the helplessness subscale, for example, while associated pain behaviours might implicitly be oriented towards eliciting support from others to alleviate this sense of helplessness, as suggested by the communal coping model of PC. [162,167] Meanwhile the same full-scale score on the PCS might stem from an elevated magnification subscale driven by exaggerated threat appraisals associated with threatening illness information and biomedical 'damage beliefs' about pain, [349] as suggested by the appraisal model. [79] Conversely someone might not have any particularly 'distorted' beliefs but might just find it difficult to control their RNT and therefore have an elevated PCS score driven by the rumination subscale, as suggested by the RNT model. [178]

Consistent with patient-treatment matching models of pain psychotherapy, [131] these different PC phenotypes would then be best suited to different treatment components focused on their predominant maintaining factors. For example, pain education and cognitive restructuring might help someone with an elevated 'magnification' PCS score more than someone high on the rumination subscale. As discussed in Study 1, the 'Limit, Activate, Enhance' model of psychosocial pain management moderation provides one useful framework for operationalising this. [131] It suggests treatments need to be tailored to: a) limit a person's

maladaptive coping responses, b) activate or increase their healthy behaviours, and c) enhance outcomes by optimising existing strengths. The results presented in this thesis suggest that the integration of metacognition into the assessment and treatment of PC would be most relevant to a PC subtype that is dominated by RNT (e.g. elevated rumination subscale on the PCS or an elevated ECIP score). Rumination would therefore be identified as a 'limit-factor' in the limit-activate-enhance model, as would any unhelpful positive and negative metacognitions that are identified with the PMQ and clinical interview. Conversely, treatment may focus on 'activating' the protective metacognitions that were identified as helpful in Study 2 (e.g. "there's more important things to be done than just continually worrying"). Clearly, further research is needed to explore these possibilities, much like sub-typing research has improved our understanding and treatment of low back pain [442–444]. Developing better ways to assess these specific forms of PC would allow interventions to be better matched to a person's broader clinical phenotype, thereby increasing its efficacy, efficiency and cost effectiveness, which is ultimately the goal of all evidence-based healthcare.

6.4 Clinical implications

Notwithstanding the need to conduct further research to better understand the implications of this research, there are a few clinical recommendations that might be offered already. Firstly, there is not yet a clear gold standard treatment for people with elevated PC so health care professionals can use clinical judgment in selecting a treatment that is well-suited to patient presentation and preference using a person-centred approach. At present, the evidence-based treatments from which to choose include: multimodal treatment (combining CBT and exercise), ACT, and CBT. However, this list is likely to change over time as more research emerges. For example, mindfulness meditation looks likely to have increasing evidence of efficacy as research designs improve. Clinicians should therefore see this as an empirical landscape that is evolving and requires monitoring in order to implement appropriate evidence-based best practice.

Secondly, rumination is clearly an important aspect of PC and deserves clinical attention. Clinicians should be aware that the predominance of appraisal-based

formulations of PC may obscure its ruminative dimensions in individual clients. Where RNT appears pronounced and deserves greater attention within treatment, it may be clinically useful to use more specific scales to assess its response to treatment. The Perseverative Thinking Questionnaire [185] and Experience of Cognitive Intrusion in Pain scale [383] are useful tools in this regard. Although further research is needed to optimise treatment for pain-related rumination, clinicians should consider using existing treatment components that seem to have efficacy, while paying attention to how these can be best matched to patient characteristics. This might include the use of structured problem solving, which is common in existing CBT protocols but could be enhanced by linking it to a functional analysis of pain rumination acting as an ineffective form of problem solving. Other existing treatment components with efficacy include: mindfulness meditation; paced exercise and movement retraining; and goal setting. Cognitive restructuring is unlikely to be as effective as these interventions in the case of elevated perseverative thinking about pain.

Finally, present findings suggest that metacognitions influence people's rumination and worry about their pain, therefore these beliefs warrant clinical attention, although how best to target these is yet to be determined. In people with elevated RNT about their pain, it may be useful to examine metacognitions using the PMQ and clinical interview. The PMQ could be used to develop a collaborative functional analysis of worry/rumination. This is likely to have treatment benefits even in the absence of a well-researched metacognitive intervention for PC. For example, understanding that one might be thinking perseveratively as an implicit coping strategy may de-stigmatise it and reframe this as a understandable feature of the 'normal psychology of pain'. [60,61] This may help alleviate some of the distress that is common in chronic pain. This is also likely to enhance treatment engagement and motivation for self-management, thereby possibly boosting therapeutic outcomes. [445] Of course, these efforts to improve PC outcomes may be hampered by contradictory approaches or messages from other health professionals, especially when health care is fragmented. Therefore, clinicians should work at both the systemic and individual level to improve patient outcomes.

6.5 Conclusions

This research aimed to identify gaps in the research on PC in an effort to advance our understanding of this important chronic pain risk factor. While the appraisal model and to a lesser extent communal coping model have received most attention in PC research, there has been less focus on the process aspects of PC which sees it as a form of repetitive negative thinking. Using both qualitative and quantitative methods, the perseverative aspects of cognition were explored in a series of linked studies. Results suggest that rumination and worry are important features of PC and in fact mediate the relationship between pain intensity and catastrophising. This builds on the emerging evidence to support a RNT model of PC. Moreover, understanding perseverative thinking about pain requires an understanding of the factors that influence it. While these are likely multidimensional, including contextual, behavioural and neurophysiological determinants, the beliefs people hold about their thinking – their metacognitions – appear to be important intrapsychic factors that shape rumination. Present findings suggest people believe thinking a lot about pain can be both helpful and unhelpful. While it is often seen as a way to solve problems, cope with pain or prevent injury, pain rumination is most commonly seen as harmful or uncontrollable. How strongly people hold these beliefs influences how strongly rumination/worry is associated with pain intensity.

This research shows it is possible to measure these pain metacognitions with a purpose-built self-report instrument that is psychometrically sound for both research and clinical applications – the Pain Metacognitions Questionnaire (PMQ). Development and validation of the PMQ allows metacognition to be integrated into existing theoretical models that demonstrate how PC can be maladaptive, such as the fear avoidance model and the misdirected problem solving model of worry. These findings also provide a springboard for proposing a preliminary metacognitive model of chronic pain, situating unhelpful pain metacognitions as key maintaining factors for pain-related distress, and therefore as potential treatment targets. This provides new possibilities for developing more effective and well-targeted pain treatments, either by refining existing interventions or

trialling novel ones, such as Metacognitive Therapy for chronic pain. This collection of studies therefore provides an empirical foundation for future translational research that will hopefully ease the enormous personal and societal burden of pain.

Bibliography

1. Turk DC, Monarch ES. Biopsychosocial perspectives on chronic pain. In: Turk DC, Gatchel RJ, eds. *Psychological Approaches to Pain Management: A Practitioner's Handbook*. 2nd ed. New York: Guilford; 2002:2-29.

2. Merskey H, Bogduk N. *Classification of Chronic Pain: Descriptions of Chronic Pain Syndromes and Definitions of Pain Terms*. 2nd ed. Seattle: IASP Press; 1994.

3. Nicholas MK, Ashton-James C. Embodied pain: grasping a thorny problem? *Pain*. 2017;158(6):993-994. doi:10.1097/j.pain.0000000000000864.

4. Loeser JD, Melzack R. Pain: An overview. *Lancet*. 1999;353:1607–1609.

5. Blyth FM, March LM, Brnabic AJM, Jorm LR, Williamson M, Cousins MJ. Chronic pain in Australia: a prevalence study. *Pain*. 2001;89:127-134.

6. Access Economics. *The High Price of Pain: The Economic Impact of Persistent Pain in Australia*. Sydney: MBF Foundation; University of Sydney Pain Management Research Institute; 2007.

7. Arthritis & Osteoporosis Victoria. *A Problem Worth Solving*. Elsternwick: Arthritis and Osteoporosis Victoria; 2013.

8. IOM Committee on Advancing Pain Research Care. *Relieving Pain in America: A Blueprint for Transforming Prevention, Care, Education, and Research*. Washington DC: Institute of Medicine, National Academies Press; 2011.

9. Eriksen J, Jensen MK, Sjøgren P, Ekholm O, Rasmussen NK. Epidemiology of chronic non-malignant pain in Denmark. *Pain*. 2003;106(3):221-228.

10. Kerr S, Fairbrother G, Crawford M, Hogg M, Fairbrother D, Khor KE.

Patient characteristics and quality of life among a sample of Australian chronic pain clinic attendees. *Intern Med J*. 2004;34(7):403-409.

11. Becker N, Thomsen AB, Olsen AK, Sjøgren P, Bech P, Eriksen J. Pain epidemiology and health related quality of life in chronic non-malignant pain patients referred to a Danish multidisciplinary pain center. *Pain*. 1997;73(3):393-400.

12. Eccleston C. Role of psychology in pain management. *Br J Anaesth*. 2001;87(1):144-152.

13. National Pain Summit Initiative, ANZCA. *National Pain Strategy: Pain Management for All Australians*. Melbourne: National Pain Summit Initiative; 2010.

14. Walsh N, Brooks P, Mieke Hazes J, et al. Standards of Care for Acute and Chronic Musculoskeletal Pain: The Bone and Joint Decade (2000-2010). *Arch Phys Med Rehabil*. 2008;89:1830-1845.

15. Macintyre PE, Schug SA, Scott DA, Visser EJ, Walker SM. *Acute Pain Management: Scientific Evidence (3rd Edition)*. 3rd ed. (Macintyre P, Scott D, Schug S, Visser E, Walker S, eds.). Melbourne: ANZCA & FPM; 2010.

16. Beales D, Fried K, Nicholas M, Blyth F, Finniss D, Moseley GL. Management of musculoskeletal pain in a compensable environment: Implementation of helpful and unhelpful Models of Care in supporting recovery and return to work. *Best Pract Res Clin Rheumatol*. 2016;30(3):445-467. doi:10.1016/j.berh.2016.08.011.

17. Darnall BD, Scheman J, Davin S, et al. Pain Psychology: A Global Needs Assessment and National Call to Action. *Pain Med*. 2016;17(2):250-263. doi:10.1093/pm/pnv095.

18. Siddall P, Cousins M. Persistent pain as a disease entity: Implications for clinical management. *Anesth Analg*. 2004;99:510-520.

19. Cousins MJ, Power I, Smith G. Pain: A persistent problem. *Reg Anesth Pain Med*. 2000;25(1):6-21.

20. Melzack R. Pain and the Neuromatrix in the Brain. *J Dent Educ*. 2001;65(12):1378-1382.

21. Moseley GL. A pain neuromatrix approach to patients with chronic pain. *Man Ther*. 2003;8(3):130-140.

22. Sharpe L, Nicholas MK, Nicholson Perry K. A clinician's quick guide of evidence-based approaches: Chronic pain. *Clin Psychol*. 2015;19:105-106. doi:10.1111/cp.12066.

23. Nicholas MK. When to refer to a pain clinic. *Best Pract Res Clin Rheumatol*. 2004;18(4):613-629. doi:10.1016/j.berh.2004.04.004.

24. Nicholas M, Molloy A, Tonkin L, Beeston L. *Manage Your Pain*. 3rd ed. London: Souvenir Press; 2011.

25. Darnall BD. *Less Pain, Fewer Pills: Avoid the Dangers of Prescription Opioids and Gain Control over Chronic Pain*. Boulder: Bull Publishing; 2014.

26. Burch V, Penman D. *You Are Not Your Pain: Using Mindfulness to Relieve Pain, Reduce Stress, and Restore Well-Being*. New York: Flatiron Books; 2013.

27. Otis JD. *Managing Chronic Pain: A Cognitive-Behavioral Therapy Approach*. Oxford: Oxford University Press; 2007.

28. Skinner M, Wilson HD, Turk DC. Cognitive-Behavioral Perspective and Cognitive-Behavioral Therapy for People With Chronic Pain: Distinctions, Outcomes, and Innovations. *J Cogn Psychother*. 2012;26(2):93-113. doi:10.1891/0889-8391.26.2.93.

29. Bonica JJ. *The Management of Pain*. 2nd ed. Philadelphia: Lea & Febiger; 1990.

30. Gatchel RJ, Peng YB, Peters ML, Fuchs PN, Turk DC. The biopsychosocial approach to chronic pain: scientific advances and future directions. *Psychol Bull*. 2007;133(4):581-624. doi:10.1037/0033-2909.133.4.581.

31. Turk DC. The role of psychological factors in chronic pain. *Acta Anaesthesiol Scand*. 1999;43(9):885–888.

32. Banks SM, Kerns RD. Explaining High Rates of Depression in Chronic Pain: A Diathesis-Stress Framework . *Psychol Bull*. 1996;119(1):95-110.

33. Elliott TE, Renier CM, Palcher JA. Chronic Pain, Depression, and Quality of Life: Correlations and Predictive Value of the SF-36. *Pain Med*. 2003;4(4):331-339.

34. Australian Bureau of Statistics. *National Survey of Mental Health and Wellbeing: Summary of Results, 2007*. Canberra; 2008.

35. Substance Abuse and Mental Health Services Administration. *Results from the 2012 National Survey on Drug Use and Health: Mental Health Findings*. Rockville, MD: NSDUH Series H-47, HHS Publication No. (SMA) 13-4805; 2013.

36. Demyttenaere K, Bruffaerts R, Lee S, et al. Mental disorders among persons with chronic back or neck pain: results from the World Mental Health Surveys. *Pain*. 2007;129(3):332-342. doi:10.1016/j.pain.2007.01.022.

37. Police R, Zhao Y, Russell M, Foster T. A systematic review of the burden, epidemiology, costs and treatment of chronic low back pain. *J Pain*. 2009;10(4, Supplement 1):S5-S5.

38. Asmundson GJG, Katz J. Understanding the co-occurrence of anxiety disorders and chronic pain: State-of-the-art. *Depress Anxiety*. 2009;26(10):888-901. doi:10.1002/da.20600.

39. Turk DC. Psychological Interventions. In: Benzon H, Rathmell J, Wu CL, Turk DC, Argoff C, Hurley R, eds. *Practical Management of Pain*. 5th ed. Philadelphia: Elsevier; 2014:615-628. doi:10.1016/B978-0-323-08340-9.00045-1.

40. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. *Pain*. 2011;152(3 Suppl):S2-15. doi:10.1016/j.pain.2010.09.030.

41. Phillips K, Clauw DJ. Central pain mechanisms in chronic pain states--maybe it is all in their head. *Best Pract Res Clin Rheumatol*. 2011;25(2):141-154. doi:10.1016/j.berh.2011.02.005.

42. Woolf CJ, Salter MW. Neuronal plasticity: increasing the gain in pain. *Science*. 2000;288(5472):1765-1769.

43. Butler DS, Moseley GL. *Explain Pain*. 2nd ed. Adelaide: Noigroup Publications; 2013. doi:10.3138/ptc.58.3.243.

44. Horn S, Munafò M. *Pain: Theory, Research, and Intervention*. Buckingham: Open University Press; 1997.

45. Beecher HK. Pain in Men Wounded in Battle. *Ann Surg*. 1946;123(1):96-105.

46. Breuer J, Freud S. *The Complete Psychological Works of Sigmund Freud, Volume II (1893-1895): Studies on Hysteria*. London: Hogarth Press; 1955.

47. Freud S, Strachey J, Freud A. *The Standard Edition of the Complete Psychological Works of Sigmund Freud*. Vintage; 2001.

48. Engel GL. Psychogenic pain and pain-prone patient. *Am J Med*. 1959;26(6):899-918. doi:10.1016/0002-9343(59)90212-8.

49. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders-Text Revision*. 4th ed. Arlington: American Psychiatric Association; 2000.

50. Aigner M, Bach M. Clinical utility of DSM-IV pain disorder. *Compr Psychiatry*. 1999;40(5):353-357.

51. Dimsdale JE, Creed F, Escobar J, et al. Somatic symptom disorder: an important change in DSM. *J Psychosom Res*. 2013;75(3):223-228. doi:10.1016/j.jpsychores.2013.06.033.

52. Sullivan MD. DSM-IV Pain Disorder: A case against the diagnosis. *Int Rev Psychiatry*. 2000;12(2):91-98.

53. Birket-Smith M, Mortensen EL. Pain in somatoform disorders: Is

Somatoform Pain Disorder a valid diagnosis? *Acta Psychiatr Scand*. 2002;106(2):103-108.

54. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Arlington, VA: American Psychiatric Publishing; 2013.

55. Merskey H. Somatization: or another God that failed. *Pain*. 2009;145(1-2):4-5. doi:10.1016/j.pain.2009.04.031.

56. Lipowski ZJ. Somatization: The concept and its clinical application. *Am J Psychiatry*. 1988;145(11):1358-1368. doi:10.1176/ajp.145.11.1358.

57. Crombez G, Beirens K, Van Damme S, Eccleston C, Fontaine J. The unbearable lightness of somatisation: A systematic review of the concept of somatisation in empirical studies of pain. *Pain*. 2009;145(1-2):31-35. doi:10.1016/j.pain.2009.04.006.

58. Toda K. The term "psychogenic pain" should be abolished or changed to "braingenic pain" (pain whose affected area is in the brain). *Pain Pract*. 2011;11(4):421. doi:10.1111/j.1533-2500.2011.00471.x.

59. Ojala T, Häkkinen A, Karppinen J, Sipilä K, Suutama T, Piirainen A. Although unseen, chronic pain is real—A phenomenological study. *Scand J Pain*. 2015;6:33-40. doi:10.1016/j.sjpain.2014.04.004.

60. Eccleston C. A normal psychology of chronic pain. *Psychologist*. 2011;24(6):422-425. doi:10.1002/14651858.CD003968.pub2.Fearon.

61. Maujean A, Sterling M, Urmston M, Woby SR, Studdert DM. "De-pathologising" the psychological responses to injury and pain. *Musculoskelet Sci Pract*. 2017;96(0):151-159. doi:10.1016/j.msksp.2017.06.005.

62. Vlaeyen JWS, Linton SJ. Fear avoidance and its consequences in musculoskeletal pain: A state of the art. *Pain*. 2000;85(3):317-332.

63. Lethem J, Slade PD, Troup JDG, Bentley G. Outline of a fear-avoidance model of exaggerated pain perceptions. *Behav Res Ther*. 1983;21:401-408.

64. Vlaeyen JWS, Kole-Snijders AMJ, Boeren RGB, van Eek H. Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. *Pain*. 1995;62(3):363-372. doi:10.1016/0304-3959(94)00279-N.

65. Asmundson GJG, Norton PJ, Norton GR. Beyond pain: The role of fear and avoidance in chronicity. *Clin Psychol Rev*. 1999;19(1):97-119.

66. Goubert L, Crombez G, van Damme S. The role of neuroticism, pain catastrophizing and pain-related fear in vigilance to pain: a structural equations approach. *Pain*. 2004;107(3):234-241. doi:10.1016/j.pain.2003.11.005.

67. Vangronsveld K, Peters M, Goossens MEJB, Linton SJ, Vlaeyen JWS. Applying the fear-avoidance model to the chronic whiplash syndrome. *Pain*. 2007;131(3):258-261. doi:10.1016/j.pain.2007.04.015.

68. Leeuw M, Goossens MEJB, Linton SJ, Crombez G, Boersma K, Vlaeyen JWS. The fear-avoidance model of musculoskeletal pain: Current state of scientific evidence. *J Behav Med*. 2007;30(1):77-94.

69. Crombez G, Eccleston C, Van Damme S, Vlaeyen JWS, Karoly P. Fear-avoidance model of chronic pain: The next generation. *Clin J Pain*. 2012;28(6):475-483. doi:10.1097/AJP.0b013e3182385392.

70. Vlaeyen JWS, Linton SJ. Fear-avoidance model of chronic musculoskeletal pain: 12 years on. *Pain*. 2012;153(6):1144-1147. doi:10.1016/j.pain.2011.12.009.

71. Vlaeyen JWS, Linton SJ. Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. *Pain*. 2000;85(3):317-332.

72. Asmundson GJG, Norton PJ, Vlaeyen JWS. Fear-avoidance models of chronic pain: An overview. In: Asmundson GJG, Vlaeyen JWS, Crombez G, eds. *Understanding and Treating Fear of Pain*. Oxford: Oxford University Press; 2004:3-24.

73. Schütze R, Rees CS, Preece M, Schütze M. Low mindfulness predicts pain

catastrophizing in a fear-avoidance model of chronic pain. *Pain*. 2010;148:120-127. doi:10.1016/j.pain.2009.10.030.

74. Norton PJ, Asmundson GJG. Amending the fear-avoidance model of chronic pain: What is the role of physiological arousal? *Behav Ther*. 2003;34(1):17-30.

75. Moseley GL, Vlaeyen JWS. Beyond nociception: The imprecision hypothesis of chronic pain. *Pain*. 2015;156(1):35-38. doi:10.1016/j.pain.0000000000000014.

76. Bunzli S, Smith AJ, Schütze R, O'Sullivan PB. The lived experience of pain-related fear in people with chronic low back pain. In: van Rysewyk S, ed. *Meanings of Pain*. Springer International Publishing; 2016. doi:10.1007/978-3-319-49022-9.

77. Bunzli S, Smith AJ, Schütze R, O'Sullivan PB. Beliefs underlying pain-related fear and how they evolve: A qualitative investigation in people with chronic back pain and high pain-related fear. *BMJ Open*. 2015;5(10):e008847. doi:10.1136/bmjopen-2015-008847.

78. Quartana PJ, Campbell CM, Edwards RR. Pain catastrophizing: A critical review. *Expert Rev Neurother*. 2009;9(5):745-758. doi:10.1586/ern.09.34.

79. Sullivan MJL, Thorn BE, Haythornthwaite JA, et al. Theoretical perspectives on the relation between catastrophizing and pain. *Clin J Pain*. 2001;17(1):52-64.

80. Ellis A. *Reason and Emotion in Psychotherapy*. Michigan: Carol Pub. Group; 1994.

81. Beck AT. *Cognitive Therapy and the Emotional Disorders*. New York: International Universities Press; 1976.

82. Neblett R. Pain catastrophizing: An historical perspective. *J Appl Biobehav Res*. 2017;22(1):1-6. doi:10.1111/jabr.12086.

83. Sullivan MJL, Bishop SR, Pivik J. The Pain Catastrophizing Scale: Development and validation. *Psychol Assess*. 1995;7(4):524-532.

doi:10.1037/1040-3590.7.4.524.

84. Spanos NP, Radtke-Bodorik HL, Ferguson JD, Jones B. The effects of hypnotic susceptibility, suggestions for analgesia, and the utilization of cognitive strategies on the reduction of pain. *J Abnorm Psychol*. 1979;88(3):282-292.

85. Chaves JF, Brown JM. Spontaneous cognitive strategies for the control of clinical pain and stress. *J Behav Med*. 1987;10(3):263-276. doi:10.1007/BF00846540.

86. Rosenstiel a K, Keefe FJ. The use of coping strategies in chronic low back pain patients: relationship to patient characteristics and current adjustment. *Pain*. 1983;17(1):33-44.

87. Vlaeyen JWS, Crombez G. Fear and Pain. *Pain Clin Updat*. 2007;15(6):1-4.

88. Beck JS. *Cognitive Therapy: Basics and Beyond*. New York: The Guilford Press; 1995.

89. Kahl KG, Winter L, Schweiger U. The third wave of cognitive behavioural therapies: what is new and what is effective? *Curr Opin Psychiatry*. 2012;25(6):522-528. doi:10.1097/YCO.0b013e328358e531.

90. Wertli MM, Eugster R, Held U, Steurer J, Kofmehl R, Weiser S. Catastrophizing - A prognostic factor for outcome in patients with low back pain: A systematic review. *Spine J*. 2014;14(11):2639-2657. doi:10.1016/j.spinee.2014.03.003.

91. Linton SJ, Nicholas MK, MacDonald S, et al. The role of depression and catastrophizing in musculoskeletal pain. *Eur J Pain*. 2011;15(4):416-422. doi:10.1016/j.ejpain.2010.08.009.

92. Edwards RR, Cahalan C, Calahan C, Mensing G, Smith MT, Haythornthwaite JA. Pain, catastrophizing, and depression in the rheumatic diseases. *Nat Rev Rheumatol*. 2011;7(4):216-224. doi:10.1038/nrrheum.2011.2.

93. Leung L. Pain catastrophizing: An updated review. *Indian J Psychol Med*.

2012;34(3):204-217. doi:10.4103/0253-7176.106012.

94. Besen E, Gaines B, Linton SJ, Shaw WS. The role of pain catastrophizing as a mediator in the work disability process following acute low back pain. *J Appl Biobehav Res*. 2017;22(1):e12085. doi:10.1111/jabr.12085.

95. Lazaridou A, Franceschelli O, Buliteanu A, Cornelius M, Edwards RR, Jamison RN. Influence of catastrophizing on pain intensity, disability, side effects, and opioid misuse among pain patients in primary care. *J Appl Biobehav Res*. 2017;22(1):e12081. doi:10.1111/jabr.12081.

96. de Boer MJ, Struys MMRF, Versteegen GJ. Pain-related catastrophizing in pain patients and people with pain in the general population. *Eur J Pain*. 2012;16(7):1044-1052. doi:10.1002/j.1532-2149.2012.00136.x.

97. Severeijns R, Vlaeyen JWS, van den Hout MA, Picavet HS. Pain catastrophizing and consequences of musculoskeletal pain: A prospective study in the Dutch community. *J Pain*. 2005;6(2):125-132.

98. Picavet HS, Vlaeyen JWS, Schouten J. Pain catastrophizing and kinesiophobia: Predictors of chronic low back pain. *Am J Epidemiol*. 2002;156(11):1028-1034. doi:10.1093/aje/kwf136.

99. Darnall BD. Pain Psychology and Pain Catastrophizing in the Perioperative Setting: A Review of Impacts, Interventions, and Unmet Needs. *Hand Clin*. 2016;32(1):33-39. doi:10.1016/j.hcl.2015.08.005.

100. Masselin-Dubois A, Attal N, Fletcher D, et al. Are psychological predictors of chronic postsurgical pain dependent on the surgical model? A comparison of total knee arthroplasty and breast surgery for cancer. *J Pain*. 2013;14(8):854-864. doi:10.1016/j.jpain.2013.02.013.

101. Roth ML, Tripp DA, Harrison MH, Sullivan MJL, Carson P. Demographic and psychosocial predictors of acute perioperative pain for total knee arthroplasty. *Pain Res Manag*. 2007;12(3):185-194.

102. Sullivan MJL, Tanzer M, Stanish W, et al. Psychological determinants of problematic outcomes following Total Knee Arthroplasty. *Pain*.

2009;143(1-2):123-129. doi:10.1016/j.pain.2009.02.011.

103. Vissers MM, Bussmann JB, Verhaar JAN, Busschbach JJ V, Bierma-Zeinstra SMA, Reijman M. Psychological factors affecting the outcome of total hip and knee arthroplasty: a systematic review. *Semin Arthritis Rheum*. 2012;41(4):576-588. doi:10.1016/j.semarthrit.2011.07.003.

104. Riddle DL, Wade JB, Jiranek W a, Kong X. Preoperative pain catastrophizing predicts pain outcome after knee arthroplasty. *Clin Orthop Relat Res*. 2010;468(3):798-806. doi:10.1007/s11999-009-0963-y.

105. Burns JW, Moric M. Psychosocial factors appear to predict postoperative pain: Interesting, but how can such information be used to reduce risk? *Tech Reg Anesth Pain Manag*. 2011;15(3):90-99. doi:10.1053/j.trap.2011.08.001.

106. Cohen J. *Statistical Power Analysis for the Behavioral Sciences.* 2nd ed. Hillsdale, NJ: Lawrence Erlbaum Associates; 1988.

107. Dowsey MM, Choong PFM. Predictors of Pain and Function Following Total Joint Replacement. In: Kinov P, ed. *Arthroplasty - Update*. Rijeka: InTech; 2013:67-99. doi:10.5772/53245.

108. Liu SS, Buvanendran A, Rathmell JP, et al. A cross-sectional survey on prevalence and risk factors for persistent postsurgical pain 1 year after total hip and knee replacement. *Reg Anesth Pain Med*. 2012;37(4):415-422. doi:10.1097/AAP.0b013e318251b688.

109. Riddle DL, Jiranek WA, Hayes CW. Using a validated algorithm to judge the appropriateness of total knee arthroplasty in the United States: A multi-center longitudinal cohort study. *Arthritis Rheumatol*. 2014;66(8):2134-2143. doi:10.1002/art.38685.

110. Sun EC, Darnall BD, Baker LC, Mackey S. Incidence of and Risk Factors for Chronic Opioid Use Among Opioid-Naive Patients in the Postoperative Period. *JAMA Intern Med*. 2016;176(9):1286. doi:10.1001/jamainternmed.2016.3298.

111. Burns JW, Day MA, Thorn BE. Is reduction in pain catastrophizing a

therapeutic mechanism specific to cognitive-behavioral therapy for chronic pain? *Transl Behav Med*. 2012;2(1):22-29. doi:10.1007/s13142-011-0086-3.

112. Turner JA, Holtzman S, Mancl L. Mediators, moderators, and predictors of therapeutic change in cognitive-behavioral therapy for chronic pain. *Pain*. 2007;127(3):276-286. doi:10.1016/j.pain.2006.09.005.

113. Vowles KE, McCracken LM, Eccleston C. Processes of change in treatment for chronic pain: The contributions of pain, acceptance, and catastrophizing. *Eur J Pain*. 2007;11:779-787.

114. Smeets RJ, Vlaeyen JWS, Kester AD, Knottnerus JA. Reduction of pain catastrophizing mediates the outcome of both physical and cognitive-behavioral treatment in chronic low back pain. *J Pain*. 2006;7(4):261-271. doi:10.1016/j.jpain.2005.10.011.

115. Burns JW, Kubilus A, Bruehl S, Harden RN, Lofland K. Do changes in cognitive factors influence outcome following multidisciplinary treatment for chronic pain? A cross-lagged panel analysis. *J Consult Clin Psychol*. 2003;71(1):81-91. doi:10.1037/0022-006X.71.1.81.

116. Spinhoven P, Ter Kuile M, Kole-Snijders AMJ, Hutten Mansfeld M, Den Ouden D-J, Vlaeyen JWS. Catastrophizing and internal pain control as mediators of outcome in the multidisciplinary treatment of chronic low back pain. *Eur J Pain*. 2004;8(3):211-219. doi:10.1016/j.ejpain.2003.08.003.

117. Racine M, Moulin DE, Nielson WR, et al. The reciprocal associations between catastrophizing and pain outcomes in patients being treated for neuropathic pain : a cross-lagged panel analysis study. *Pain*. 2016;157(9):1946-1953.

118. Wertli MM, Burgstaller JM, Weiser S, Steurer J, Kofmehl R, Held U. Influence of Catastrophizing on Treatment Outcome in Patients With Nonspecific Low Back Pain. *Spine (Phila Pa 1976)*. 2014;39(3):263-273. doi:10.1097/BRS.0000000000000110.

119. Basler HD, Jakle C, Kroner-Herwig B. Incorporation of cognitive-behavioral treatment into the medical care of chronic low back patients: a

controlled randomized study in German pain treatment centers. *Patient Educ Couns*. 1997;31(2):113-124.

120. Williams AC de C, Eccleston C, Morley S. Psychological therapies for the management of chronic pain (excluding headache) in adults. *Cochrane Database Syst Rev*. 2012;11(CD007407). doi:10.1002/14651858.CD007407.pub3.

121. Broderick JE, Keefe FJ, Bruckenthal P, et al. Nurse practitioners can effectively deliver pain coping skills training to osteoarthritis patients with chronic pain: A randomized, controlled trial. *Pain*. 2014;155(9):1743-1754. doi:10.1016/j.pain.2014.05.024.

122. Alda M, Luciano J V, Andres E, et al. Effectiveness of cognitive behaviour therapy for the treatment of catastrophisation in patients with fibromyalgia: a randomised controlled trial. *Arthritis Res Ther*. 2011;13:1-13.

123. Riddle DL, Keefe FJ, Nay WT, McKee D, Attarian DE, Jensen MP. Pain coping skills training for patients with elevated pain catastrophizing who are scheduled for knee arthroplasty: a quasi-experimental study. *Arch Phys Med Rehabil*. 2011;92(6):859-865. doi:10.1016/j.apmr.2011.01.003.

124. Eccleston C, Williams AC de C, Morley S. Psychological therapies for the management of chronic pain (excluding headache) in adults. *Cochrane Database Syst Rev*. 2009;2:Art. No.: CD007407. doi:10.1002/14651858.CD007407.pub2.

125. Morley S, Eccleston C, Williams AC de C. Systematic review and meta-analysis of randomized controlled trials of cognitive behaviour therapy and behaviour therapy for chronic pain in adults, excluding headache. *Pain*. 1999;80:1-13.

126. Day MA, Thorn BE. Using theoretical models to clarify shared and unique mechanisms in psychosocial pain treatments: a commentary on McCracken and Morley's theoretical paper. *J Pain*. 2014;15(3):237-238. doi:10.1016/j.jpain.2014.01.486.

127. Day MA, Thorn BE, Burns JW. The Continuing Evolution of

Biopsychosocial Interventions for Chronic Pain. *J Cogn Psychother*. 2012;26(2):114-129. doi:10.1891/0889-8391.26.2.114.

128. Thorn BE, Burns JW. Common and specific treatment mechanisms in psychosocial pain interventions: the need for a new research agenda. *Pain*. 2011;152(4):705-706. doi:10.1016/j.pain.2010.12.017.

129. Kazdin AE. Understanding how and why psychotherapy leads to change. *Psychother Res*. 2009;19(4-5):418-428. doi:10.1080/10503300802448899.

130. Kazdin AE. Mediators and mechanisms of change in psychotherapy research. *Annu Rev Clin Psychol*. 2007;3:1-27. doi:10.1146/annurev.clinpsy.3.022806.091432.

131. Day MA, Ehde DM, Jensen MP. Psychosocial Pain Management Moderation: The Limit, Activate, and Enhance Model. *J Pain*. 2015;16(10):947-960. doi:10.1016/j.jpain.2015.07.003.

132. Nicholas MK. Obstacles to recovery after an episode of low back pain; the "usual suspects" are not always guilty. *Pain*. 2010;148(3):363-364. doi:10.1016/j.pain.2009.12.007.

133. Gardner-Nix J, Backman S, Barbati J, Grummitt J. Evaluating distance education of a mindfulness-based meditation programme for chronic pain management. *J Telemed Telecare*. 2008;14(2):88-92.

134. Day MA, Thorn BE, Ward LC, et al. Mindfulness-based cognitive therapy for the treatment of headache pain: a pilot study. *Clin J Pain*. 2014;30(2):152-161. doi:10.1097/AJP.0b013e318287a1dc.

135. Luciano J V., Guallar J a., Aguado J, et al. Effectiveness of group acceptance and commitment therapy for fibromyalgia: A 6-month randomized controlled trial (EFFIGACT study). *Pain*. 2014;155(4):693-702. doi:10.1016/j.pain.2013.12.029.

136. Turner JA, Anderson ML, Balderson BH, Cook AJ, Sherman KJ, Cherkin DC. Mindfulness-based stress reduction and cognitive-behavioral therapy for chronic low back pain: similar effects on mindfulness, catastrophizing,

self-efficacy, and acceptance in a randomized controlled trial. *Pain*. 2016;157:2434-2444. doi:10.1097/j.pain.0000000000000635.

137. Sullivan MJL, Stanish W, Waite H, Tripp DA. Catastrophizing, pain, and disability in patients with soft-tissue injuries. *Pain*. 1998;77(3):253-260.

138. Vasey MW, Borkovec TD. A catastrophizing assessment of worrisome thoughts. *Cognit Ther Res*. 1992;16(5):505-520. doi:10.1007/BF01175138.

139. Flor H, Behle DJ, Birbaumer N. Assessment of pain-related cognitions in chronic pain patients. *Behav Res Ther*. 1993;31(1):63-73.

140. Lefebvre MF. Cognitive distortion and cognitive errors in depressed psychiatric and low back pain patients. *J Consult Clin Psychol*. 1981;49(4):517-525. doi:10.1037/0022-006X.49.4.517.

141. Klaassen M, Ter Kuile MM. Development and initial validation of the vaginal penetration cognition questionnaire (VPCQ) in a sample of women with vaginismus and dyspareunia. *J Sex Med*. 2009;6(6):1617-1627. doi:10.1111/j.1743-6109.2009.01217.x.

142. Vlaeyen JWS, Geurts SM, Kole-Snijders AM, Schuerman JA, Groenman NH, van Eek H. What do chronic pain patients think of their pain? Towards a pain cognition questionnaire. *Br J Clin Psychol*. 1990;29:383-394. doi:10.1111/j.2044-8260.1990.tb00901.x.

143. Sullivan MJL. *The Pain Catastrophizing Scale: User Manual*. Montreal: University of Montreal; 2004.

144. Chibnall JT, Tait RC. Confirmatory factor analysis of the Pain Catastrophizing Scale in African American and Caucasian Workers' Compensation claimants with low back injuries. *Pain*. 2005;113(3):369-375. doi:10.1016/j.pain.2004.11.016.

145. Osman A, Barrios FX, Kopper BA, Hauptmann W, Jones J, O'Neill E. Factor structure, reliability, and validity of the Pain Catastrophizing Scale. *J Behav Med*. 1997;20(6):589-605.

146. Van Damme S, Crombez G, Bijttebier P, Goubert L, Van Houdenhove B. A

confirmatory factor analysis of the Pain Catastrophizing Scale: invariant factor structure across clinical and non-clinical populations. *Pain*. 2002;96(3):319-324.

147. van Damme S, Crombez G, Vlaeyen JWS, Goubert L, Van den Broeck A, Van Houdenhove B. The Pain Catastrophizing Scale: Psychometric characteristics and norms. *Gedragstherapie*. 2000;33(3):209-220.

148. Lazarus RS, Folkman S. Transactional theory and research on emotions and coping. *Eur J Pers*. 1987;1(3):141-169.

149. Severeijns R, Vlaeyen JWS, van den Hout MA. Do we need a communal coping model of pain catastrophizing? An alternative explanation. *Pain*. 2004;111(3):226-229. doi:10.1016/j.pain.2004.07.024.

150. Davey GCL, Levy S. Catastrophic worrying: Personal inadequacy and a perseverative iterative style as features of the catastrophizing process. *J Abnorm Psychol*. 1998;107(4):576-586. doi:10.1037/0021-843X.107.4.576.

151. Thorn BE. *Cognitive Therapy for Chronic Pain: A Step-by-Step Guide*. New York: Guilford Press; 2004.

152. Darnall B, Sturgeon JA, Kao M-C, Hah J, Mackey SC. From Catastrophizing to Recovery: a pilot study of a single-session treatment for pain catastrophizing. *J Pain Res*. 2014;7:219-226. doi:10.2147/JPR.S62329.

153. Crombez G, Eccleston C, Vlaeyen JWS, Vansteenwegen D, Lysens R, Eelen P. Exposure to physical movement in low back pain patients: Restricted effects of generalization. *Heal Psychol*. 2002;21(6):573-578.

154. Bennett-Levy J, Butler G, Fennell M, Hackman A, Mueller M, Westbrook D. *Oxford Guide to Behavioural Experiments in Cognitive Therapy*. Oxford: Oxford University Press; 2004.

155. Asmundson GJG, Wright KD, Hadjistavropoulos HD. Hypervigilance and attentional fixedness in chronic musculoskeletal pain: Consistency of findings across modified stroop and dot-probe tasks. *J Pain*. 2005;6(8):497-506.

156. Bar-Haim Y, Lamy D, Pergamin L, Bakermans-Kranenburg MJ, Van IJzendoorn MH. Threat-related attentional bias in anxious and nonanxious individuals: a meta-analytic study. *Psychol Bull*. 2007;133(1):1-24. doi:10.1037/0033-2909.133.1.1.

157. Crombez G, Van Ryckeghem DML, Eccleston C, van Damme S. Attentional bias to pain-related information: a meta-analysis. *Pain*. 2013;154(4):497-510. doi:10.1016/j.pain.2012.11.013.

158. van Damme S, Crombez G, Eccleston C. Disengagement from pain: The role of catastrophic thinking about pain. *Pain*. 2004;107(1-2):70-76.

159. Dear BF, Sharpe L, Nicholas MK, Refshauge KM. The psychometric properties of the dot-probe paradigm when used in pain-related attentional bias research. *J Pain*. 2011;12(12):1247-1254. doi:10.1016/j.jpain.2011.07.003.

160. Sullivan MJL, Tripp DA, Santor D. Gender differences in pain and pain behavior: The role of catastrophizing. *Cognit Ther Res*. 2000;24(1):121-134.

161. Lyons RF, Sullivan MJL, Ritvo PG. *Relationships in Chronic Illness and Disability*. Thousand Oaks, Calif.: Sage Publications; 1995.

162. Sullivan MJL, Martel MO, Tripp DA, Savard A, Crombez G. The relation between catastrophizing and the communication of pain experience. *Pain*. 2006;122(3):282-288. doi:10.1016/j.pain.2006.02.001.

163. Buenaver LF, Edwards RR, Haythornthwaite JA. Pain-related catastrophizing and perceived social responses: Inter-relationships in the context of chronic pain. *Pain*. 2007;127(3):234-242. doi:10.1016/j.pain.2006.08.018.

164. Keefe FJ, Lipkus I, Lefebvre JC, et al. The social context of gastrointestinal cancer pain: A preliminary study examining the relation of patient pain catastrophizing to patient perceptions of social support and caregiver stress and negative responses. *Pain*. 2003;103:151-156.

165. Boothby JL, Thorn BE, Overduin LY, Ward LC. Catastrophizing and perceived partner responses to pain. *Pain*. 2004;109(3):500-506.

166. Waxman SE, Tripp DA, Flamenbaum R, et al. The mediating role of depression and negative partner responses in chronic low back pain and relationship satisfaction. *J Pain*. 2008;9(5):434-442. doi:10.1016/j.jpain.2007.12.007.

167. Burns JW, Gerhart JI, Post KM, et al. The Communal Coping Model of Pain Catastrophizing in Daily Life: A Within-Couples Daily Diary Study. *J Pain*. 2015;16(11):1163-1175. doi:10.1016/j.jpain.2015.08.005.

168. Flink IK, Engman L, Thomtén J, Linton SJ. The role of catastrophizing in vulvovaginal pain: Impact on pain and partner responses over time. *J Appl Biobehav Res*. 2017;22(1):e12093. doi:10.1111/jabr.12093.

169. Main CJ, Keefe FJ, Jensen MP, Vlaeyen JWS, Vowles KE, Fordyce WE, eds. *Fordyce's Behavioral Methods for Chronic Pain and Illness*. Philadelphia: IASP Press; 2014.

170. Edwards RR, Kronfli T, Haythornthwaite JA, Smith MT, McGuire L, Page GG. Association of catastrophizing with interleukin-6 responses to acute pain. *Pain*. 2008;140(1):135-144. doi:10.1016/j.pain.2008.07.024.

171. Quartana PJ, Buenaver LF, Edwards RR, Klick B, Haythornthwaite J a, Smith MT. Pain catastrophizing and salivary cortisol responses to laboratory pain testing in temporomandibular disorder and healthy participants. *J Pain*. 2010;11(2):186-194. doi:10.1016/j.jpain.2009.07.008.

172. Goodin BR, McGuire L, Allshouse M, et al. Associations between catastrophizing and endogenous pain-inhibitory processes: sex differences. *J Pain*. 2009;10(2):180-190. doi:10.1016/j.jpain.2008.08.012.

173. Seminowicz DA, Davis KD. Cortical responses to pain in healthy individuals depends on pain catastrophizing. *Pain*. 2006;120(3):297-306. doi:10.1016/j.pain.2005.11.008.

174. Kucyi A, Moayedi M, Weissman-Fogel I, et al. Enhanced medial prefrontal-

default mode network functional connectivity in chronic pain and its association with pain rumination. *J Neurosci*. 2014;34(11):3969-3975. doi:10.1523/JNEUROSCI.5055-13.2014.

175. Trost Z, Strachan E, Sullivan MJL, Vervoort T, Avery AR, Afari N. Heritability of pain catastrophizing and associations with experimental pain outcomes. *Pain*. 2015;156(3):514-520. doi:10.1097/01.j.pain.0000460326.02891.fc.

176. George SZ, Wallace MR, Wright TW, et al. Evidence for a biopsychosocial influence on shoulder pain: Pain catastrophizing and catechol-O-methyltransferase (COMT) diplotype predict clinical pain ratings. *Pain*. 2008;136(1-2):53-61. doi:10.1016/j.pain.2007.06.019.

177. Campbell CM, Edwards RR. Mind-body interactions in pain: the neurophysiology of anxious and catastrophic pain-related thoughts. *Transl Res*. 2009;153(3):97-101. doi:10.1016/j.trsl.2008.12.002.

178. Flink IK, Boersma K, Linton SJ. Pain catastrophizing as repetitive negative thinking: A development of the conceptualization. *Cogn Behav Ther*. 2013;42(3):215-223. doi:10.1080/16506073.2013.769621.

179. Borkovec TD, Inz J. The nature of worry in generalized anxiety disorder: A predominance of thought activity. *Behav Res Ther*. 1990;28(2):153-158. doi:10.1016/0005-7967(90)90027-G.

180. Borkovec TD, Alcaine O, Behar E. Avoidance theory of worry and generalized anxiety disorder. In: Heimberg RG, Turk CL, Mennin DS, eds. *Generalized Anxiety Disorder: Advances in Research and Practice*. Vol 2004. New York: Guildford Press; 2004:77-108.

181. Provencher MD, Dugas MJ, Ladouceur R. Efficacy of problem-solving training and cognitive exposure in the treatment of generalized anxiety disorder: A case replication series. *Cogn Behav Pract*. 2004;11(4):404-414. doi:10.1016/S1077-7229(04)80057-9.

182. Creamer M, Forbes D, Phelps A, Humphreys L. *Treating Traumatic Stress : Conducting Imaginal Exposure in PTSD*. Melbourne: Australian Centre for

Posttraumatic Mental Health; 2007.

183. van der Heiden C, ten Broeke E. The When, Why, and How of Worry Exposure. *Cogn Behav Pract*. 2009;16(4):386-393. doi:10.1016/j.cbpra.2008.11.003.

184. Davey GCL, Meeten F. The perseverative worry bout: A review of cognitive, affective and motivational factors that contribute to worry perseveration. *Biol Psychol*. 2016. doi:10.1016/j.biopsycho.2016.04.003.

185. Ehring T, Zetsche U, Weidacker K, Wahl K, Schönfeld S, Ehlers A. The Perseverative Thinking Questionnaire (PTQ): Validation of a content-independent measure of repetitive negative thinking. *J Behav Ther Exp Psychiatry*. 2011;42(2):225-232. doi:10.1016/j.jbtep.2010.12.003.

186. Nolen-Hoeksema S. The response styles theory. In: Papageorgiou C, Wells A, eds. *Depressive Rumination: Nature, Theory and Treatment*. West Sussex, UK: Wiley; 2004:107-123.

187. Watkins ER. Constructive and unconstructive repetitive thought. *Psychol Bull*. 2008;134(2):163-206. doi:10.1037/0033-2909.134.2.163.

188. Harvey AG, Watkins ER, Mansell W, Shafran R. *Cognitive Behavioural Processes across Psychological Disorders: A Transdiagnostic Approach to Research and Treatment*. New York: Oxford University Press; 2004.

189. Ehring T, Watkins ER. Repetitive Negative Thinking as a Transdiagnostic Process. *Int J Cogn Ther*. 2008;1(3):192-205. doi:10.1680/ijct.2008.1.3.192.

190. Trick L, Watkins ER, Windeatt S, Dickens C. The association of perseverative negative thinking with depression, anxiety and emotional distress in people with long term conditions: A systematic review. *J Psychosom Res*. 2016;91:89-101. doi:10.1016/j.jpsychores.2016.11.004.

191. Darnall BD, Sturgeon JA, Cook KF, et al. Development and Validation of a Daily Pain Catastrophizing Scale. *J Pain*. 2017;18(9):1139-1149. doi:10.1016/j.jpain.2017.05.003.

192. Craner JR, Gilliam WP, Sperry JA. Rumination, Magnification, and

Helplessness: How do Different Aspects of Pain Catastrophizing Relate to Pain Severity and Functioning? *Clin J Pain*. 2016;32(12):1028-1035. doi:10.1097/AJP.0000000000000355.

193. Gilliam W, Craner JR, Morrison EJ, Sperry JA. The Mediating Effects of the Different Dimensions of Pain Catastrophizing on Outcomes in an Interdisciplinary Pain Rehabilitation Program. *Clin J Pain*. 2017;33(5):443-451. doi:10.1097/AJP.0000000000000419.

194. Martin LL, Tesser A. Some ruminative thoughts. In: Wyer RS, ed. *Ruminative Thoughts*. Mahwah, NJ: Lawrence Erlbaum Associates; 1996:1-47.

195. Eccleston C, Crombez G. Worry and chronic pain: A misdirected problem solving model. *Pain*. 2007;132(3):233-236. doi:10.1016/j.pain.2007.09.014.

196. Aldrich S, Eccleston C, Crombez G. Worrying about chronic pain: Vigilance to threat and misdirected problem solving. *Behav Res Ther*. 2000;38:457-470. doi:10.1016/S0005-7967(99)00062-5.

197. Flink IK, Boersma K, MacDonald S, Linton SJ. Understanding catastrophizing from a misdirected problem-solving perspective. *Br J Health Psychol*. 2012;17(2):408-419. doi:10.1111/j.2044-8287.2011.02044.x.

198. Eccleston C, Crombez G, Aldrich S, Stannard C. Worry and chronic pain patients: a description and analysis of individual differences. *Eur J Pain*. 2001;5(3):309-318. doi:10.1053/eujp.2001.0252.

199. Sullivan MJL, Thorn BE, Rodgers WM, Ward LC. Path model of psychological antecedents to pain experience: Experimental and clinical findings. *Clin J Pain*. 2004;20(3):164-173.

200. Leventhal H, Meyer D, Nerenz D. The common sense model of illness danger. In: Rachman S, ed. *Medical Psychology*. New York: Pergamon; 1980:7-30.

201. Hagger MS, Orbell S. A Meta-Analytic Review of the Common-Sense

Model of Illness Representations. *Psychol Health*. 2003;18(2):141-184. doi:10.1080/0887044031000813321.

202. Bunzli S, Smith AJ, Schütze R, Lin IB, O'Sullivan PB. Making sense of low back pain and pain-related fear. *J Orthop Sport Phys Ther*. 2017;47(9):628-636. doi:10.2519/jospt.2017.7434.

203. Wells A. *Emotional Disorders and Metacognition: Innovative Cognitive Therapy*. Chichester: Wiley; 2000.

204. Wells A, Matthews G. *Attention and Emotion: A Clinical Perspective*. Erlbaum. Hove: Psychology Press; 1994.

205. Fisher PL, Wells A. *Metacognitive Therapy: Distinctive Features*. New York: Routledge; 2009.

206. Wells A. *Metacognitive Therapy for Anxiety and Depression*. New York: Guilford Press; 2009.

207. Wells A, Fisher PL. Metacognitive Therapy: Theoretical Background and Model of Depression. In: Wells A, Fisher PL, eds. *Treating Depression: MCT, CBT and Third Wave Therapies*. Chichester: John Wiley & Sons; 2016:144-168.

208. Wells A. Meta-Cognition & worry: A cognitive model of Generalized Anxiety Disorder. *Behav Cogn Psychother*. 1995;23:301–320.

209. Papageorgiou C, Wells A. A Prospective Test of the Clinical Metacognitive Model of Rumination and Depression. *Int J Cogn Ther*. 2009;2(2):123-131.

210. Hagen R, Hjemdal O, Solem S, et al. Metacognitive Therapy for Depression in Adults : A Waiting List Randomized Controlled Trial with Six Months Follow-Up. *Front Psychol*. 2017;8:1-10. doi:10.3389/fpsyg.2017.00031.

211. Rees CS, Koesveld K van. An open trial of group metacognitive therapy for obsessive-compulsive disorder. *J Behav Ther*. 2008.

212. Fisher PL, Wells A. Metacognitive therapy for obsessive–compulsive disorder: A case series. *J Behav Ther Exp*. 2008.

213. Wells A, Colbear JS. Treating Posttraumatic Stress Disorder With Metacognitive Therapy: A Preliminary Controlled Trial. *J Clin Psychol*. 2012;68(4):373-381. doi:10.1002/jclp.20871.

214. Wells A, Walton D, Lovell K, Proctor D. Metacognitive Therapy Versus Prolonged Exposure in Adults with Chronic Post-traumatic Stress Disorder: A Parallel Randomized Controlled Trial. *Cognit Ther Res*. 2015;39(1):70-80. doi:10.1007/s10608-014-9636-6.

215. Bailey R, Wells A. Metacognitive Therapy in the Treatment of Hypochondriasis: A Systematic Case Series. *Cognit Ther Res*. 2014;38(5):541-550. doi:10.1007/s10608-014-9615-y.

216. Bailey R, Wells A. Metacognitive beliefs moderate the relationship between catastrophic misinterpretation and health anxiety. *J Anxiety Disord*. 2015;34:8-14. doi:10.1016/j.janxdis.2015.05.005.

217. Fisher PL, Byrne A, Salmon P. Metacognitive Therapy for Emotional Distress in Adult Cancer Survivors: A Case Series. *Cognit Ther Res*. 2017. doi:10.1007/s10608-017-9862-9.

218. Fisher PL, McNicol K, Young B, Smith E, Salmon P. Alleviating Emotional Distress in Adolescent and Young Adult Cancer Survivors: An Open Trial of Metacognitive Therapy. *J Adolesc Young Adult Oncol*. 2015;4(2):64-69. doi:10.1089/jayao.2014.0046.

219. Fardell JE, Thewes B, Turner J, et al. Fear of cancer recurrence : a theoretical review and novel cognitive processing formulation. *J Cancer Surviv*. 2016;10:663-673. doi:10.1007/s11764-015-0512-5.

220. Nikčević A V., Spada MM. Metacognitions about smoking: A preliminary investigation. *Clin Psychol Psychother*. 2010;17(6):536-542. doi:10.1002/cpp.689.

221. Nikčević A V., Caselli G, Wells A, Spada MM. The Metacognitions about Smoking Questionnaire: Development and psychometric properties. *Addict Behav*. 2015;44:102-107. doi:10.1016/j.addbeh.2014.11.004.

222. Fernie B a., Maher-Edwards L, Murphy G, Nikčević A V., Spada MM. The Metacognitions about Symptoms Control Scale: Development and concurrent validity. *Clin Psychol Psychother*. 2015;22(5):443-449. doi:10.1002/cpp.1906.

223. Maher-Edwards L, Fernie B a., Murphy G, Nikcevic AV, Spada MM. Metacognitive factors in chronic fatigue syndrome. *3395*. 2012;557:552-557. doi:10.1002/cpp.757.

224. Normann N, Van Emmerik A a P, Morina N. The efficacy of metacognitive therapy for anxiety and depression: A meta-analytic review. *Depress Anxiety*. 2014;31:402-411. doi:10.1002/da.22273.

225. Sadeghi R, Mokhber N, Mahmoudi LZ, Asgharipour N, Seyfi H. A systematic review and meta-analysis on controlled treatment trials of metacognitive therapy for anxiety disorders. *J Res Med Sci*. 2015;20(9):901-909. doi:10.4103/1735-1995.170632.

226. Yoshida T, Molton IR, Jensen MP, et al. Cognitions, metacognitions, and chronic pain. *Rehabil Psychol*. 2012;57(3):207-213. doi:10.1037/a0028903.

227. Wells A, Hout V Den, Silva D, Davies MI. The Thought Control Quesionnaire: A measure of individual differences in the control of unwanted thoughts. *Behav Res Ther*. 1994;32(8):871-878.

228. Spada MM, Gay H, Nikčević A V., Fernie BA, Caselli G. Meta-cognitive beliefs about worry and pain catastrophising as mediators between neuroticism and pain behaviour. *Clin Psychol*. 2016;20:138-146. doi:10.1111/cp.12081.

229. Veehof MM, Trompetter HR, Bohlmeijer ET, Schreurs KMG. Acceptance- and mindfulness-based interventions for the treatment of chronic pain: a meta-analytic review. *Cogn Behav Ther*. 2016;45(1):5-31. doi:10.1080/16506073.2015.1098724.

230. Veehof MM, Oskam M-J, Schreurs KMG, Bohlmeijer ET. Acceptance-based interventions for the treatment of chronic pain: a systematic review and meta-analysis. *Pain*. 2011;152(3):533-542.

doi:10.1016/j.pain.2010.11.002.

231. Bawa FLM, Mercer SW, Atherton RJ, et al. Does mindfulness improve outcomes in patients with chronic pain? Systematic review and meta-analysis. *Br J Gen Pract*. 2015;65(635):e387-400. doi:10.3399/bjgp15X685297.

232. Teixeira ME. Meditation as an intervention for chronic pain: An integrative review. *Holist Nurs Pract*. 2008;22(4):225-234.

233. Hilton L, Hempel S, Ewing BA, et al. Mindfulness Meditation for Chronic Pain: Systematic Review and Meta-analysis. *Ann Behav Med*. 2017;51(2):199-213. doi:10.1007/s12160-016-9844-2.

234. Chiesa A, Serretti A. Mindfulness-based interventions for chronic pain: a systematic review of the evidence. *J Altern Complement Med*. 2011;17(1):83-93. doi:10.1089/acm.2009.0546.

235. Grant JA. Meditative analgesia: the current state of the field. *Ann N Y Acad Sci*. 2014;1307:55-63. doi:10.1111/nyas.12282.

236. Bishop SR, Lau M, Shapiro SL, et al. Mindfulness: A proposed operational definition. *Clin Psychol Sci Pract*. 2004;11(3):230-241.

237. Beck AT, Rush AJ, Shaw BF, Emery G. *Cognitive Therapy of Depression*. New York: Guilford Press; 1979.

238. Segal Z V, Williams JMG, Teasdale JD. *Mindfulness-Based Cognitive Therapy for Depression*. 2nd ed. New York: Guilford Press; 2013.

239. Hayes SC, Wilson KG, Strosahl KD. *Acceptance and Commitment Therapy: An Experiential Approach to Behavior Change*. New York: Guilford Press; 1999.

240. McCracken LM, Morley S. The psychological flexibility model: A basis for integration and progress in psychological approaches to chronic pain management. *J Pain*. 2014;15(3):221-234. doi:10.1016/j.jpain.2013.10.014.

241. Day MA, Jensen MP, Ehde DM, Thorn BE. Toward a theoretical model for

mindfulness-based pain management. *J Pain*. 2014;15(7):691-703. doi:10.1016/j.jpain.2014.03.003.

242. Day MA, Smitherman A, Ward LC, Thorn BE. An investigation of the associations between measures of mindfulness and pain catastrophizing. *Clin J Pain*. 2015;31(3):222-228. doi:10.1097/AJP.0000000000000102.

243. Day MA, Smitherman A, Thorn BE, Ward LC. Are mindfulness and pain catastrophizing unique constructs? *J Pain*. 2014;15(4):S16. doi:10.1016/j.jpain.2014.01.066.

244. Bunzli S, Watkins R, Smith AJ, Schütze R, O'Sullivan PB. Lives on hold: A qualitative synthesis exploring the experience of chronic low-back pain. *Clin J Pain*. 2013;29(10):907-916. doi:10.1097/AJP.0b013e31827a6dd8.

245. Smith JA, Osborn M. Pain as an assault on the self: An interpretative phenomenological analysis of the psychological impact of chronic benign low back pain. *Psychol Health*. 2007;22(5):517-534. doi:10.1080/14768320600941756.

246. Cartwright-Hatton S, Wells A. Beliefs about Worry and Intrusions: The Meta-Cognitions Questionnaire and its Correlates. *J Anxiety Disord*. 1997;11(3):279-296. doi:10.1016/S0887-6185(97)00011-X.

247. Watkins ER. Depressive Rumination and Co-Morbidity: Evidence for Brooding as a Transdiagnostic Process. *J Ration Cogn Ther*. 2009;27(3):160-175. doi:10.1007/s10942-009-0098-9.

248. D'Eon JL, Harris CA, Ellis JA. Testing factorial validity and gender invariance of the Pain Catastrophizing Scale. *J Behav Med*. 2004;27(4):361-372.

249. Moher D, Liberati A, Tetzlaff J, Altman DG, Grp P. Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement (Reprinted from Annals of Internal Medicine). *PLoS Med*. 2009;6(7):e1000097. doi:10.1371/journal.pmed.1000097.

250. Schütze R, Rees CS, Smith AJ, O'Sullivan PB. How effectively can pain

catastrophizing be reduced in adults with non-cancer pain? A systematic review and meta-analysis. PROSPERO. http://www.crd.york.ac.uk/PROSPERO/display_record.asp?ID=CRD42016042761. Published 2016.

251. Higgins J, Green S. *Cochrane Handbook for Systematic Reviews of Interventions Version 5.1.0*. The Cochrane Collaboration; 2011.

252. Rosen L, Suhami R. The art and science of study identification: a comparative analysis of two systematic reviews. *BMC Med Res Methodol*. 2016;16(1):24. doi:10.1186/s12874-016-0118-2.

253. CADTH. Strings attached: CADTH database search filters. Ottawa. https://www.cadth.ca/resources/finding-evidence/. Published 2016. Accessed August 16, 2016.

254. Scottish Intercollegiate Guidelines Network. SIGN Search Filters. http://www.sign.ac.uk/methodology/filters.html. Published 2015. Accessed August 16, 2016.

255. Veritas Health Innovation. Covidence systematic review software. 2016.

256. Borenstein, M., Hedges, L., Higgins, J., & Rothstein H. Comprehensive meta-analysis: A computer program for meta-analysis. 2014.

257. Higgins J, Altman DG, Gotzsche PC, et al. The Cochrane Collaboration's tool for assessing risk of bias in randomised trials. *BMJ*. 2011;343(oct18 2):d5928--d5928. doi:10.1136/bmj.d5928.

258. Yates SL, Morley S, Eccleston C, Williams AC de C. A scale for rating the quality of psychological trials for pain. *Pain*. 2005;117(3):314-325. doi:10.1016/j.pain.2005.06.018.

259. Fewtrell MS, Kennedy K, Singhal A, et al. How much loss to follow-up is acceptable in long-term randomised trials and prospective studies? *Arch Dis Child*. 2008;93(6):458-461. doi:10.1136/adc.2007.127316.

260. Carpenter JR, Kenward MG. *Missing Data in Randomised Controlled Trials - a Practical Guide*. Birmingham: National Institute for Health

Research; 2008.

261. Higgins JPT, Thompson SG, Deeks JJ, Altman DG. Measuring inconsistency in meta-analyses. *BMJ Br Med J*. 2003;327:557-560. doi:10.1136/bmj.327.7414.557.

262. Scott W, Wideman TH, Sullivan MJL. Clinically meaningful scores on pain catastrophizing before and after multidisciplinary rehabilitation: a prospective study of individuals with subacute pain after whiplash injury. *Clin J Pain*. 2014;30(3):183-190. doi:10.1097/AJP.0b013e31828eee6c.

263. Sullivan MJL, Adams H, Ellis T. Targeting Catastrophic Thinking to Promote Return to Work in Individuals With Fibromyalgia. *J Cogn Psychother*. 2012;26(2):130-143. doi:http://dx.doi.org/10.1891/0889-8391.26.2.130.

264. Burns LC, Ritvo SE, Ferguson MK, Clarke H, Seltzer Z, Katz J. Pain catastrophizing as a risk factor for chronic pain after total knee arthroplasty: A systematic review. *J Pain Res*. 2015;8:21-32. doi:10.2147/JPR.S64730.

265. Wideman TH, Adams H, Sullivan MJL. A prospective sequential analysis of the fear-avoidance model of pain. *Pain*. 2009;145(1):45-51. doi:10.1016/j.pain.2009.04.022.

266. Egger M, Davey Smith G, Schneider M, Minder C. Bias in meta-analysis detected by a simple, graphical test. *Br Med J*. 1997;315(7109):629-634. doi:10.1136/bmj.316.7129.469.

267. Duval S, Tweedie R. Trim and Fill: A Simple Funnel-Plot-Based Method. *Biometrics*. 2000;56(June):455-463. doi:10.1111/j.0006-341x.2000.00455.x.

268. Guyatt GH, Oxman AD, Vist GE, et al. GRADE: An Emerging Consensus on Rating Quality of Evidence and Strength of Recommendations. *Br Med J*. 2008;336(April):924-926. doi:10.1136/bmj.39489.470347.AD.

269. Schünemann HJ, Brożek J, Guyatt GH, Oxman AD. *GRADE Handbook for Grading Quality of Evidence and Strength of Recommendation*. October 20.

GRADE Working Group; 2013.

270. Abbott AD, Tyni-Lenne R, Hedlund R. Early rehabilitation targeting cognition, behavior, and motor function after lumbar fusion: a randomized controlled trial. *Spine J*. 2010;35(8):848-857.

271. Abrahamsen R, Zachariae R, Svensson P. Effect of hypnosis on oral function and psychological factors in temporomandibular disorders patients. *J Oral Rehabil*. 2009;36(8):556-570. doi:10.1111/j.1365-2842.2009.01974.x.

272. Amris K, Waehrens EE, Christensen R, Bliddal H, Danneskiold-Samsoe B, Group IMproS. Interdisciplinary rehabilitation of patients with chronic widespread pain: primary endpoint of the randomized, nonblinded, parallel-group IMPROvE trial. *Pain*. 2014;155(7):1356-1364. doi:http://dx.doi.org/10.1016/j.pain.2014.04.012.

273. Bennell KL, Ahamed Y, Jull G, et al. Physical Therapist-Delivered Pain Coping Skills Training and Exercise for Knee Osteoarthritis: Randomized Controlled Trial. *Arthritis Care Res*. 2016;68(5):590-602. doi:10.1002/acr.22744.

274. Bergeron S, Khalife S, Dupuis MJ, McDuff P. A randomized clinical trial comparing group cognitive-behavioral therapy and a topical steroid for women with dyspareunia. *J Consult Clin Psychol*. 2016;84(3):259-268. doi:http://dx.doi.org/10.1037/ccp0000072.

275. Brattberg G. Self-administered EFT (Emotional Freedom Techniques) in individuals with fibromyalgia: a randomized trial. *Integr Med*. 2008;7(4):30-35.

276. Bromberg J, Wood ME, Black RA, Surette DA, Zacharoff KL, Chiauzzi EJ. A Randomized Trial of a Web-Based Intervention to Improve Migraine Self-Management and Coping. *Headache*. 2012;52(2):244-261. doi:10.1111/j.1526-4610.2011.02031.x.

277. Buhrman M, Fältenhag S, Ström L, Andersson G. Controlled trial of Internet-based treatment with telephone support for chronic back pain. *Pain*.

2004;111(3):368-377. doi:10.1016/j.pain.2004.07.021.

278. Buhrman M, Fredriksson A, Edstrom G, et al. Guided Internet-delivered cognitive behavioural therapy for chronic pain patients who have residual symptoms after rehabilitation treatment: Randomized controlled trial. *Eur J Pain*. 2013;17(5):753-765. doi:http://dx.doi.org/10.1002/j.1532-2149.2012.00244.x.

279. Buhrman M, Syk M, Burvall O, Hartig T, Gordh T, Andersson G. Individualized Guided Internet-delivered Cognitive-Behavior Therapy for Chronic Pain Patients With Comorbid Depression and Anxiety: A Randomized Controlled Trial. *Clin J Pain*. 2015;31(6):504-516. doi:http://dx.doi.org/10.1097/AJP.0000000000000176.

280. Buhrman M, Nilsson-Ihrfeldt E, Jannert M, Ström L, Andersson G. Guided internet-based cognitive behavioural treatment for chronic back pain reduces pain catastrophizing: a randomized controlled trial. *J Rehabil Med*. 2011;43(6):500-505.

281. Buhrman M, Skoglund A, Husell J, et al. Guided internet-delivered acceptance and commitment therapy for chronic pain patients: A randomized controlled trial. *Behav Res Ther*. 2013;51(6):307-315. doi:http://dx.doi.org/10.1016/j.brat.2013.02.010.

282. Carpenter KM, Stoner SA, Mundt JM, Stoelb B. An on-line self-help CBT intervention for chronic lower back pain. *Clin J Pain*. 2012;28(1):14-22. doi:http://dx.doi.org/10.1097/AJP.0b013e31822363db.

283. Carson JW, Carson KM, Jones KD, Bennett RM, Wright CL, Mist SD. A pilot randomized controlled trial of the Yoga of Awareness program in the management of fibromyalgia. *Pain*. 2010;151(2):530-539. doi:10.1016/j.pain.2010.08.020.

284. Castel A, Cascon R, Padrol A, Sala J, Rull M. Multicomponent cognitive-behavioral group therapy with hypnosis for the treatment of fibromyalgia: Long-Term outcome. *J Pain*. 2012;13(3):255-265. doi:10.1016/j.jpain.2011.11.005.

285. Castel A, Castro S, Fontova R, et al. Body mass index and response to a multidisciplinary treatment of fibromyalgia. *Rheumatol Int*. 2015;35(2):303-314. doi:10.1007/s00296-014-3096-x.

286. Castel A, Fontova R, Montull S, et al. Efficacy of a multidisciplinary fibromyalgia treatment adapted for women with low educational levels: a randomized controlled trial. *Arthritis Care Res*. 2013;65(3):421-431. doi:10.1002/acr.21818.

287. Chiauzzi E, Zacharoff KL, Bond KS, Yiu EC, Wood ME. PainACTION.com: an interactive self-management web site for chronic back pain patients. *Pain Med*. 2010;11(2):307-308.

288. De Boer MJ, Versteegen GJ, Vermeulen KM, Sanderman R, Struys MM. A randomized controlled trial of an Internet-based cognitive-behavioural intervention for non-specific chronic pain: an effectiveness and cost-effectiveness study. *Eur J Pain*. 2014;18(10):1440-1451. doi:10.1002/ejp.509.

289. Dowd H, Hogan MJ, McGuire BE, et al. Comparison of an Online Mindfulness-based Cognitive Therapy Intervention With Online Pain Management Psychoeducation: A Randomized Controlled Study. *Clin J Pain*. 2015;31(6):517-527. doi:http://dx.doi.org/10.1097/AJP.0000000000000201.

290. Ersek M, Turner JA, Cain KC, Kemp CA. Results of a randomized controlled trial to examine the efficacy of a chronic pain self-management group for older adults [ISRCTN11899548]. *Pain*. 2008;138(1):29-40. doi:10.1016/j.pain.2007.11.003.

291. Fritsche G, Frettloh J, Huppe M, et al. Prevention of medication overuse in patients with migraine. *Pain*. 2010;151(2):404-413. doi:http://dx.doi.org/10.1016/j.pain.2010.07.032.

292. Gallagher L, McAuley J, Moseley GL. A Randomized-controlled Trial of Using a Book of Metaphors to Reconceptualize Pain and Decrease Catastrophizing in People With Chronic Pain. *Clin J Pain*. 2013;29(1):20-25. doi:10.1097/AJP.0b013e3182465cf7.

293. Garland EL, Gaylord SA, Palsson O, Faurot K, Douglas Mann J, Whitehead WE. Therapeutic mechanisms of a mindfulness-based treatment for IBS: effects on visceral sensitivity, catastrophizing, and affective processing of pain sensations. *J Behav Med*. 2012;35(6):591-602. doi:10.1007/s10865-011-9391-z.

294. Geraets JJ, Goossens ME, de Groot IJ, et al. Effectiveness of a graded exercise therapy program for patients with chronic shoulder complaints. *Aust J Physiother*. 2005;51(2):87-94.

295. Gustavsson C, Denison E, Koch L. Self-management of persistent neck pain: a randomized controlled trial of a multi-component group intervention in primary health care. *Eur J Pain*. 2010;14:630.e1-630.e11. doi:10.1016/j.ejpain.2009.10.004.

296. Helminen E-E, Sinikallio SH, Valjakka AL, Vaisanen-Rouvali RH, Arokoski JPA. Effectiveness of a cognitive-behavioural group intervention for knee osteoarthritis pain: A randomized controlled trial. *Clin Rehabil*. 2015;29(9):868-881. doi:http://dx.doi.org/10.1177/0269215514558567.

297. Huang G, Travison TG, Edwards RR, Basaria S. Effects of Testosterone Replacement on Pain Catastrophizing and Sleep Quality in Men with Opioid-Induced Androgen Deficiency. *Pain Med*. 2016;18(6):1070-1076. doi:http://dx.doi.org/10.1093/pm/pnw159.

298. Hutting N, Bart Staal J, Engels JA, Heerkens YF, Detaille SI, Nijhuis-Van Der Sanden MWG. Effect evaluation of a self-management programme for employees with complaints of the arm, neck or shoulder: A randomised controlled trial. *Occup Environ Med*. 2015;72(12):852-861. doi:10.1136/oemed-2015-103089.

299. Ittersum MW, Wilgen CP, Schans CP, Lambrecht L, Groothoff JW, Nijs J. Written Pain Neuroscience Education in Fibromyalgia: A Multicenter Randomized Controlled Trial. *Pain Pract*. 2014;14(8):689-700. doi:10.1111/papr.12137.

300. Kristjánsdóttir OB, Fors EA, Eide E, et al. A smartphone-based intervention with diaries and therapist-feedback to reduce catastrophizing and increase

functioning in women with chronic widespread pain: randomized controlled trial. *J Med Internet Res*. 2013;15(1):e5.1-e5.22. doi:10.2196/jmir.2249.

301. la Cour P, Petersen M. Effects of Mindfulness Meditation on Chronic Pain: A Randomized Controlled Trial. *Pain Med*. 2015;16(4):641-652. doi:10.1111/pme.12605.

302. Leeuw M, Goossens ME, Breukelen GJ, et al. Exposure in vivo versus operant graded activity in chronic low back pain patients: results of a randomized controlled trial. *Pain*. 2008;138(1):192-207. doi:10.1016/j.pain.2007.12.009.

303. Mannion AF, Müntener M, Taimela S, Dvorak J. A randomized clinical trial of three active therapies for chronic low back pain. *Spine (Phila Pa 1976)*. 1999;24(23):2435-2448.

304. Martínez M, Miró E, Sánchez A, et al. Cognitive-behavioral therapy for insomnia and sleep hygiene in fibromyalgia: a randomized controlled trial. *J Behav Med*. 2014;37(4):683-697. doi:10.1007/s10865-013-9520-y.

305. Meeus M, Nijs J, Oosterwijck J, Alsenoy V, Truijen S. Pain physiology education improves pain beliefs in patients with chronic fatigue syndrome compared with pacing and self-management education: a double-blind randomized controlled trial. *Arch Phys Med Rehabil*. 2010;91(8):1153-1159. doi:10.1016/j.apmr.2010.04.020.

306. Monticone M, Ambrosini E, Cazzaniga D, et al. Adults with idiopathic scoliosis improve disability after motor and cognitive rehabilitation: results of a randomised controlled trial. *Eur Spine J*. 2016;25(10):3120-3129. doi:http://dx.doi.org/10.1007/s00586-016-4528-y.

307. Monticone M, Ambrosini E, Rocca B, Cazzaniga D, Liquori V, Foti C. Group-based task-oriented exercises aimed at managing kinesiophobia improved disability in chronic low back pain. *Eur J Pain*. 2016;20(4):541-551. doi:10.1002/ejp.756.

308. Monticone M, Ambrosini E, Rocca B, et al. Group-based multimodal exercises integrated with cognitive-behavioural therapy improve disability,

pain and quality of life of subjects with chronic neck pain: A randomized controlled trial with one-year follow-up. *Clin Rehabil*. 2016;31(6):742-752. doi:http://dx.doi.org/10.1177/0269215516651979.

309. Monticone M, Ferrante S, Teli M, et al. Management of catastrophising and kinesiophobia improves rehabilitation after fusion for lumbar spondylolisthesis and stenosis. A randomised controlled trial. *Eur Spine J*. 2014;23(1):87-95. doi:10.1007/s00586-013-2889-z.

310. Moustafa IM, Diab AA. The addition of upper cervical manipulative therapy in the treatment of patients with fibromyalgia: a randomized controlled trial. *Rheumatol Int*. 2015;35(7):1163-1174. doi:http://dx.doi.org/10.1007/s00296-015-3248-7.

311. Müller R, Gertz KJ, Molton IR, et al. Effects of a Tailored Positive Psychology Intervention on Well-Being and Pain in Individuals With Chronic Pain and a Physical Disability: A Feasibility Trial. *Clin J Pain*. 2016;32(1):32-44. doi:http://dx.doi.org/10.1097/AJP.0000000000000225.

312. Naylor MR, Keefe FJ, Brigidi B, Naud S, Helzer JE. Therapeutic Interactive Voice Response for chronic pain reduction and relapse prevention. *Pain*. 2008;134(3):335-345. doi:http://dx.doi.org/10.1016/j.pain.2007.11.001.

313. Nicholas MK, Asghari A, Sharpe L, et al. Cognitive exposure versus avoidance in patients with chronic pain: adherence matters. *Eur J Pain*. 2014;18(3):424-437. doi:http://dx.doi.org/10.1002/j.1532-2149.2013.00383.x.

314. Nicholas MK, Asghari A, Blyth FM, et al. Self-management intervention for chronic pain in older adults: A randomised controlled trial. *Pain*. 2013;154(6):824-835. doi:http://dx.doi.org/10.1016/j.pain.2013.02.009.

315. Nordin CA, Michaelson P, Gard G, Eriksson MK. Effects of the Web Behavior Change Program for Activity and Multimodal Pain Rehabilitation: Randomized Controlled Trial. *J Med Internet Res*. 2016;18(10):e265.1-e265.18.

316. Oerlemans S, Cranenburgh O, Herremans PJ, Spreeuwenberg P, Dulmen S.

Intervening on cognitions and behavior in irritable bowel syndrome: A feasibility trial using PDAs. *J Psychosom Res*. 2011;70(3):267-277. doi:10.1016/j.jpsychores.2010.09.018.

317. Overmeer T, Peterson G, Landen Ludvigsson M, Peolsson A. The effect of neck-specific exercise with or without a behavioral approach on psychological factors in chronic whiplash-associated disorders: A randomized controlled trial with a 2-year follow-up. *Medicine (Baltimore)*. 2016;95(34):e4430. doi:http://dx.doi.org/10.1097/MD.0000000000004430.

318. Picard P, Jusseaume C, Boutet M, Dualé C, Mulliez A, Aublet-Cuvellier B. Hypnosis for Management of Fibromyalgia. *Int J Clin Exp Hypn*. 2013;61(1):111-123. doi:10.1080/00207144.2013.729441.

319. Rodriguez Torres J, Cabrera Martos I, Torres Sanchez I, Ortiz Rubio A, Diaz Pelegrina A, Valenza MC. Results of an Active Neurodynamic Mobilization Program in Patients With Fibromyalgia Syndrome: A Randomized Controlled Trial. *Arch Phys Med Rehabil*. 2015;96(10):1771-1778. doi:10.1016/j.apmr.2015.06.008.

320. Ruehlman LS, Karoly P, Enders C. A randomized controlled evaluation of an online chronic pain self management program. *Pain*. 2012;153(2):319-330. doi:10.1016/j.pain.2011.10.025.

321. Schofield P. Evaluating Snoezelen for relaxation within chronic pain management. *Br J Nurs*. 2002;11(12):812-821.

322. Somers TJ, Blumenthal JA, Guilak F, et al. Pain coping skills training and lifestyle behavioral weight management in patients with knee osteoarthritis: A randomized controlled study. *Pain*. 2012;153(6):1199-1209. doi:http://dx.doi.org/10.1016/j.pain.2012.02.023.

323. Sterling M, Vicenzino B, Souvlis T, Connelly LB. Dry-needling and exercise for chronic whiplash-associated disorders: a randomized single-blind placebo-controlled trial. *Pain*. 2015;156(4):635-643. doi:10.1097/01.j.pain.0000460359.40116.c1.

324. Ter Kuile MM, Melles RJ, Tuijnman-Raasveld CC, Groot HE, Lankveld J.

Therapist-Aided Exposure for Women with Lifelong Vaginismus: Mediators of Treatment Outcome: A Randomized Waiting List Control Trial. *J Sex Med*. 2015;12(8):1807-1819. doi:10.1111/jsm.12935.

325. Ter Kuile MM, Spinhoven P, Linssen AC, van Houwelingen HC. Cognitive coping and appraisal processes in the treatment of chronic headaches. *Pain*. 1996;64(2):257-264. doi:10.1016/0304-3959(95)00135-2.

326. Tetsunaga T, Tanaka M, Ozaki T. Efficacy of tramadol-acetaminophen tablets in low back pain patients with depression. *J Orthop Sci*. 2015;20(2):281-286. doi:10.1007/s00776-014-0674-4.

327. Thorn BE, Day MA, Burns J, et al. Randomized trial of group cognitive behavioral therapy compared with a pain education control for low-literacy rural people with chronic pain. *Pain*. 2011;152(12):2710-2720. doi:10.1016/j.pain.2011.07.007.

328. Trompetter H, Bohlmeijer ET, Veehof MM, Schreurs K. Internet-based guided self-help intervention for chronic pain based on Acceptance and Commitment Therapy: A randomized controlled trial. *J Behav Med*. 2015;38(1):66-80. doi:10.1007/s10865-014-9579-0.

329. Trudeau K, Pujol L, DasMahapatra P, Wall R, Black R, Zacharoff K. A randomized controlled trial of an online self-management program for adults with arthritis pain. *J Behav Med*. 2015;38(3):483-496. doi:10.1007/s10865-015-9622-9.

330. Turner JA, Clancy S. Comparison of operant behavioral and cognitive-behavioral group treatment for chronic low back pain. *J Consult Clin Psychol*. 1988;56(2):261-266.

331. Turner JA, Mancl L, Aaron LA. Short- and long-term efficacy of brief cognitive-behavioral therapy for patients with chronic temporomandibular disorder pain: a randomized, controlled trial. *Pain*. 2006;121(3):181-194. doi:10.1016/j.pain.2005.11.017.

332. Turner JA, Mancl L, Huggins KH, Sherman JJ, Lentz G, LeResche L. Targeting temporomandibular disorder pain treatment to hormonal

fluctuations: a randomized clinical trial. *Pain*. 2011;152(9):2074-2084. doi:10.1016/j.pain.2011.05.005.

333. Vallejo MA, Ortega J, Rivera J, Comeche MI, Vallejo-Slocker L. Internet versus face-to-face group cognitive-behavioral therapy for fibromyalgia: A randomized control trial. *J Psychiatr Res*. 2015;68:106-113. doi:http://dx.doi.org/10.1016/j.jpsychires.2015.06.006.

334. van der Maas LCC, Koke A, Pont M, et al. Improving the Multidisciplinary Treatment of Chronic Pain by Stimulating Body Awareness: A Cluster-randomized Trial. *Clin J Pain*. 2015;31(7):660-669. doi:10.1097/AJP.0000000000000138.

335. Vlaeyen JWS, Teeken-Gruben NJ, Goossens ME, et al. Cognitive-educational treatment of fibromyalgia: a randomized clinical trial. I. Clinical effects. *J Rheumatol*. 1996;23(7):1237-1245.

336. Vonk F, Verhagen AP, Twisk JW, Koke AJ, Luiten MW, Koes BW. Effectiveness of a behaviour graded activity program versus conventional exercise for chronic neck pain patients. *Eur J Pain*. 2009;13(5):533-541. doi:10.1016/j.ejpain.2008.06.008.

337. Weiner DK, Moore CG, Morone NE, Lee ES, Kwoh CK. Efficacy of Periosteal Stimulation for Chronic Pain Associated With Advanced Knee Osteoarthritis: A Randomized, Controlled Clinical Trial. *Clin Ther*. 2013;35(11):1704-1720. doi:10.1016/j.clinthera.2013.09.025.

338. Williams AC de C, Richardson PH, Nicholas MK, et al. Inpatient vs. outpatient pain management: results of a randomised controlled trial. *Pain*. 1996;66(1):13-22.

339. Zautra AJ, Davis MC, Reich JW, et al. Comparison of cognitive behavioral and mindfulness meditation interventions on adaptation to rheumatoid arthritis for patients with and without history of recurrent depression. *J Consult Clin Psychol*. 2008;76(3):408-421. doi:10.1037/0022-006x.76.3.408.

340. Marshall PWM, Kennedy S, Brooks C, Lonsdale C. Pilates exercise or

stationary cycling for chronic nonspecific low back pain: does it matter? A randomized controlled trial with 6-month follow-up. *Spine (Phila Pa 1976)*. 2013;38(15):E952-E959. doi:10.1097/BRS.0b013e318297c1e5.

341. Moseley GL, Nicholas MK, Hodges PW. A Randomized Controlled Trial of Intensive Neurophysiology Education in Chronic Low Back Pain. *Clin J Pain*. 2004;20(5):324-330.

342. Gustavsson C, Denison E, Koch L. Self-management of persistent neck pain: two-year follow-up of a randomized controlled trial of a multicomponent group intervention in primary health care. *Spine (Phila Pa 1976)*. 2011;36(25):2105-2115. doi:10.1097/BRS.0b013e3182028b04.

343. Flink IK, Boersma K, Linton SJ. Changes in Catastrophizing and Depressed Mood During and After Early Cognitive Behaviorally Oriented Interventions for Pain Changes in Catastrophizing and Depressed Mood During and After Early Cognitive Behaviorally Oriented Interventions for Pain. *Cogn Behav Ther*. 2017;43(4):332-341. doi:10.1080/16506073.2014.940605.

344. Schulz KF, Altman DG, Moher D. CONSORT 2010 statement: updated guidelines for reporting parallel group randomized trials. *Ann Intern Med*. 2010;152(11):726.

345. ICMJE, De Angelis C, Drazen JM, et al. Clinical Trial Registration : A Statement from the International Committee of Medical Journal Editors. *N Engl J Med*. 2004;351(12):1250-1251. doi:10.1056/NEJMe048225.

346. Jacobson NS, Truax P. Clinical Significance : A Statistical Approach to Denning Meaningful Change in Psychotherapy Research. *J Consult Clin Psychol*. 1991;59(1):12-19.

347. Fernandes L, Storheim K, Lochting I, Grotle M. Cross-cultural adaptation and validation of the Norwegian Pain Catastrophizing Scale in patients with low back pain. *BMC Musculoskelet Disord*. 2012;13(1):111. doi:10.1186/1471-2474-13-111.

348. Sullivan MJL, Adams H, Rhodenizer T, Stanish W. A psychosocial risk

factor-targeted intervention for the prevention of chronic pain and disability following whiplash injury. *Phys Ther*. 2006;86(1):8-18.

349. Bunzli S, Smith AJ, Watkins R, Schütze R, O'Sullivan PB. "What Do People who Score Highly on the Tampa Scale of Kinesiophobia Really Believe? A Mixed Methods Investigation in People with Chronic Non Specific Low Back Pain. *Clin J Pain*. 2015;31(7):621-632. doi:10.1097/AJP.0000000000000143.

350. Asmundson GJG, Fetzner MG, Deboer LB, Powers MB, Otto MW, Smits JAJ. Let's get physical: A contemporary review of the anxiolytic effects of exercise for anxiety and its disorders. *Depress Anxiety*. 2013;30(4):362-373. doi:10.1002/da.22043.

351. Nijs J, Kosek E, Van Oosterwijck J, Meeus M. Dysfunctional endogenous analgesia during exercise in patients with chronic pain: to exercise or not to exercise? *Pain Physician*. 2012;15(3):ES205-ES213.

352. Morley S, Williams AC de C, Eccleston C. Examining the evidence about psychological treatments for chronic pain: time for a paradigm shift? *Pain*. 2013;154(10):1929-1931. doi:10.1016/j.pain.2013.05.049.

353. Crofford LJ. Psychological aspects of chronic musculoskeletal pain. *Best Pract Res Clin Rheumatol*. 2015;29(1):147-155. doi:10.1016/j.berh.2015.04.027.

354. Sullivan MJL, Rodgers WM, Kirsch I. Catastrophizing, depression and expectancies for pain and emotional distress. *Pain*. 2001;91(1-2):147-154. doi:10.1016/S0304-3959(00)00430-9.

355. Flink IK, Boersma K, Linton SJ. Catastrophizing moderates the effect of exposure in vivo for back pain patients with pain-related fear. *Eur J Pain*. 2010;14(8):887-892. doi:10.1016/j.ejpain.2010.02.003.

356. Gardner-Nix J, Barbati J, Grummitt J, Pukal S, Raponi Newton R. Exploring the effectiveness of a Mindfulness-based chronic pain management course delivered simultaneously to on-site and off-site patients using telemedicine. *Mindfulness (N Y)*. 2012;5(3):223-231.

doi:10.1007/s12671-012-0169-3.

357. Turner JA, Aaron LA. Pain-related catastrophizing: What is it? *Clin J Pain*. 2001;17(1):65-71.

358. Schütze R. Re-thinking over-thinking pain: What can metacognition add to our understanding of pain catastrophising? *Clin Psychol*. 2016;20:147-153. doi:10.1111/cp.12105.

359. Wells A, Matthews G. Modelling cognition in emotional disorder: The S-REF model. *Behav Res Ther*. 1996;34(11-12):881-888. doi:10.1016/S0005-7967(96)00050-2.

360. Osborn M, Rodham K. Insights into pain: A review of qualitative research. *Rev Pain*. 2010;4(1):2-7. doi:10.1177/204946371000400102.

361. O'Reilly M, Parker N. "Unsatisfactory Saturation": A critical exploration of the notion of saturated sample sizes in qualitative research. *Qual Res*. 2012;13(2):190-197. doi:10.1177/1468794112446106.

362. Smith JA, Osborn M. Interpretative phenomenological analysis. In: Smith JA, ed. *Qualitative Psychology: A Practical Guide to Research Methods*. 2nd ed. London: Sage; 2008:53-80.

363. Cleeland CS, Ryan KM. Pain assessment: Global use of the Brief Pain Inventory. *Ann Acad Med Singapore*. 1994;23(2):129-138.

364. Wells A, Cartwright-Hatton S. A short form of the metacognitions questionnaire: Properties of the MCQ-30. *Behav Res Ther*. 2004;42(4):385-396. doi:10.1016/S0005-7967(03)00147-5.

365. Zigmond AS, Snaith RP. Hospital Anxiety and Depression Scale (HADS): *Ann Gen Psychiatry*. 1983;67:361-370. doi:10.1186/1744-859X-7-4.

366. Qualtrics. Qualtrics Research Suite. 2013.

367. Smith JA, Flowers P, Larkin M. *Interpretative Phenomenological Analysis: Theory, Method and Research*. Vol 9. London: Sage; 2009.

368. QSR International. NVivo for Mac qualitative data analysis software. 2014.

369. Tong A, Sainsbury P, Craig J. Consolidated criteria for reporting qualitative research (COREQ): A 32-item checklist for interviews and focus groups. *Int J Qual Heal Care*. 2007;19(6):349-357. doi:10.1093/intqhc/mzm042.

370. Taylor B, Carswell K, Williams AC de C. The interaction of persistent pain and post-traumatic re-experiencing: A qualitative study in torture survivors. *J Pain Symptom Manage*. 2013;46(4):546-555. doi:10.1016/j.jpainsymman.2012.10.281.

371. Osborn M, Smith JA. The fearfulness of chronic pain and the centrality of the therapeutic relationship in containing it: An interpretative phenomenological analysis. *Qual Res Psychol*. 2008;5(4):276-288. doi:10.1080/14780880701826143.

372. Kirkham JA, Smith JA, Havsteen-Franklin D. Painting pain: An interpretative phenomenological analysis of representations of living with chronic pain. *Heal Psychol*. 2015;34(4):398-406. doi:10.1037/hea0000139.

373. Snelgrove S, Edwards S, Liossi C. A longitudinal study of patients' experiences of chronic low back pain using interpretative phenomenological analysis: Changes and consistencies. *Psychol Health*. 2013;28(2):121-138.

374. Watkins ER. *Rumination-Focused Cognitive-Behavioral Therapy for Depression*. New York: Guildford Press; 2016.

375. Moseley GL, Butler DS. Fifteen years of explaining pain: The past, present and future. *J Pain*. 2015;16(9):807-813. doi:10.1016/j.jpain.2015.05.005.

376. Vibe Fersum K, O'Sullivan P, Skouen JSS, et al. Efficacy of classification-based cognitive functional therapy in patients with non-specific chronic low back pain: A randomized controlled trial. *Eur J Pain*. 2013;17(6):916-928. doi:10.1002/j.1532-2149.2012.00252.x.

377. Adams LM, Turk DC. Psychosocial factors and central sensitivity syndromes. *Curr Rheumatol Rev*. 2015;11(2):96-108. doi:10.2174/1573397111666150619095330.

378. O'Sullivan P. Diagnosis and classification of chronic low back pain

disorders: Maladaptive movement and motor control impairments as underlying mechanism. *Man Ther*. 2005;10(4):242-255.

379. Salomons T V., Moayedi M, Erpelding N, Davis KD. A brief cognitive-behavioural intervention for pain reduces secondary hyperalgesia. *Pain*. 2014;155(8):1446-1452. doi:10.1016/j.pain.2014.02.012.

380. Schütze R, Rees CS, Smith AJ, Slater H, Campbell JM, O'Sullivan P. How can we best reduce pain catastrophizing in adults with chronic non-cancer pain? A systematic review and meta-analysis. *J Pain*. 2017;epub ahead. doi:https://doi.org/10.1016/j.jpain.2017.09.010.

381. Anderson KO. Role of cutpoints: why grade pain intensity? *Pain*. 2005;113:5-6. doi:10.1016/j.pain.2004.10.024.

382. Carver RP. Reading Rate: Theory, Research, and Practical Implications. *J Read*. 1992;36(2):84-95.

383. Attridge N, Crombez G, Van Ryckeghem DML, Keogh E, Eccleston C. The Experience of Cognitive Intrusion of Pain: scale development and validation. *Pain*. 2015;156:1978-1990. doi:10.1097/j.pain.0000000000000257.

384. Buhrmester M, Kwang T, Gosling SD. Amazon's Mechanical Turk: A New Source of Inexpensive, Yet High-Quality, Data? *Perspect Psychol Sci*. 2011;6(1):3-5. doi:10.1177/1745691610393980.

385. Paolacci G, Chandler J. Inside the Turk: Understanding Mechanical Turk as a Participant Pool. *Curr Dir Psychol Sci*. 2014;23(3):184-188. doi:10.1177/0963721414531598.

386. Electronic Persistent Pain Outcomes Collaboration. *Patient Outcomes in Pain Management*. Wollongong; 2014.

387. Streiner DL, Norman GR. *Health Measurement Scales: A Practical Guide to Their Development and Use*. 4th ed. Oxford: Oxford University Press; 2008.

388. DeVellis RF. *Scale Development: Theory and Applications*. 3rd ed. London:

Sage; 2012.

389. Schütze R, Rees CS, Slater H, Smith AJ, O'Sullivan PB. "I call it stinkin' thinkin'": A qualitative analysis of metacognition in people with chronic low back pain and elevated catastrophising. *Br J Health Psychol*. 2017;22(3):463-480. doi:10.1111/bjhp.12240.

390. Tan G, Jensen MP, Thornby JI, Shanti BF. Validation of the Brief Pain Inventory for chronic nonmalignant pain. *J Pain*. 2004;5(2):133-137. doi:10.1016/j.jpain.2003.12.005.

391. Pallant JF, Bailey CM. Assessment of the structure of the Hospital Anxiety and Depression Scale in musculoskeletal patients. *Health Qual Life Outcomes*. 2005;3:82. doi:10.1186/1477-7525-3-82.

392. Snaith RP. Hospital Anxiety and Depression scale (HADS). *Health Qual Life Outcomes*. 2003;1:29. doi:10.1186/1477-7525-1-29.

393. Miller RP, Kori SH, Todd DD. The Tampa Scale for Kinesiophobia. *Unpubl Rep*. 1991.

394. Roelofs J, Sluiter JK, Frings-Dresen MH, et al. Fear of movement and (re)injury in chronic musculoskeletal pain: Evidence for an invariant two-factor model of the Tampa Scale for Kinesiophobia across pain diagnoses and Dutch, Swedish, and Canadian samples. *Pain*. 2007;131:181-190.

395. Cook AJ, Brawer PA, Vowles KE. The fear-avoidance model of chronic pain: Validation and age analysis using structural equation modeling. *Pain*. 2006;121(3):195-206.

396. Crombez G, Vlaeyen JWS, Heuts PH, Lysens R. Pain-related fear is more disabling than pain itself: Evidence on the role of pain-related fear in chronic back pain disability. *Pain*. 1999;80:329-339.

397. French DJ, France CR, Vigneau F, French JA, Evans RT. Fear of movement/(re)injury in chronic pain: A psychometric assessment of the original English version of the Tampa Scale for Kinesiophobia (TSK). *Pain*. 2007;127:42-51.

398. Brown KW, Ryan RM. The benefits of being present: Mindfulness and its role in psychological well-being. *J Pers Soc Psychol*. 2003;84(4):822-848. doi:10.1037/0022-3514.84.4.822.

399. Guy W. *ECDEU Assessment Manual for Psychopharmacology*. Rockville, MD; 1976.

400. Hurst H, Bolton J. Assessing the clinical significance of change scores recorded on subjective outcome measures. *J Manipulative Physiol Ther*. 2004;27(1):26-35. doi:10.1016/j.jmpt.2003.11.003.

401. Farrar JT, Young JP, LaMoreaux L, Werth JL, Poole RM. Clinical importance of changes in chronic pain intensity measured on an 11-point numerical pain rating scale. *Pain*. 2001;94(2):149-158.

402. Dworkin RH, Turk DC, Wyrwich KW, et al. Interpreting the clinical importance of treatment outcomes in chronic pain clinical trials: IMMPACT recommendations. *J Pain*. 2008;9(2):105-121. doi:10.1016/j.jpain.2007.09.005.

403. Pallant JF, Tennant A. An introduction to the Rasch measurement model: An example using the Hospital Anxiety and Depression Scale (HADS). *Br J Clin Psychol*. 2007;46(1):1-18. doi:10.1348/014466506X96931.

404. Tennant A, Conaghan PG. The Rasch measurement model in rheumatology: What is it and why use it? When should it be applied, and what should one look for in a Rasch paper? *Arthritis Care Res*. 2007;57(8):1358-1362. doi:10.1002/art.23108.

405. Wright BD. Rating scale model (RSM) or partial credit model (PCM). *Rasch Meas Trans*. 1998;12(3):641-642.

406. IBM Corp. IBM SPSS Statistics for Macintosh. 2016.

407. Wertli MM, Rasmussen-Barr E, Weiser S, Bachmann LM, Brunner F. The role of fear avoidance beliefs as a prognostic factor for outcome in patients with nonspecific low back pain: A systematic review. *Spine J*. 2014;14(5):816-836. doi:10.1016/j.spinee.2013.09.036.

408. Bjelland I, Dahl AA, Haug TT, et al. The validity of the Hospital Anxiety and Depression Scale. An updated literature review. *J Psychosom Res*. 2002;52(2):69-77. doi:10.1016/S0022-3999(01)00296-3.

409. Swets JA. Measuring the Accuracy of Diagnostic Systems. *Science (80-)*. 1988;240(4857):1285-1293.

410. Zweig MH, Campbell G. Receiver-operating characteristic (ROC) plots: A fundamental evaluation tool in clinical medicine. *Clin Chem*. 1993;39(4):561-577. doi:ROC; Receiver-Operating Characteristic; SDT; Signal Detection Theory.

411. Hanley AJ, McNeil JB. The Meaning and Use of the Area under a Receiver Operating Characteristic (ROC) Curve. *Radiology*. 1982;143:29-36. doi:10.1148/radiology.143.1.7063747.

412. Zhou X-H, Obuchowski NA, McClish DK. *Statistical Methods in Diagnostic Medicine*. Hoboken, NJ: John Wiley & Sons; 2011. doi:10.1002/9780470906514.

413. Youden WJ. Index for rating diagnostic tests. *Cancer*. 1950;3(1):32-35.

414. van Damme S, Crombez G, Eccleston C, Roelofs J. The role of hypervigilance in the experience of pain. In: Asmundson GJ, Vlaeyen JW, Crombez G, eds. *Understanding and Treating Fear of Pain*. Chichester: Wiley & Sons; 2004:71-90.

415. Tardif H, Arnold C, Hayes C, Eagar K. Establishment of the Australasian Electronic Persistent Pain Outcomes Collaboration. *Pain Med*. 2016;18(6):1007-1018. doi:10.1093/pm/pnw201.

416. Suso-ribera C, García-palacios A, Botella C, Ribera-canudas MV. Pain Catastrophizing and Its Relationship with Health Outcomes : Does Pain Intensity Matter ? *Pain Res Manag*. 2017;2017:9762864. doi:10.1155/2017/9762864.

417. Hayes AF. *Introduction to Mediation, Moderation, and Conditional Process Analysis: A Regression-Based Approach*. New York: Guildford Press; 2013.

418. Hayes AF, Montoya AK, Rockwood NJ. The analysis of mechanisms and their contingencies: PROCESS versus structural equation modeling. *Australas Mark J*. 2017;25(1):76-81. doi:10.1016/j.ausmj.2017.02.001.

419. Wells A. A metacognitive model and therapy for Generalized Anxiety Disorder. *Clin Psychol Psychother*. 1999;6(2):86-95.

420. Sharpe L, Ianiello M, Dear BF, Nicholson Perry K, Refshauge KM, Nicholas MK. Is there a potential role for attention bias modification in pain patients? Results of 2 randomised, controlled trials. *Pain*. 2012;153(3):722-731. doi:10.1016/j.pain.2011.12.014.

421. Gatchel RJ. Introduction to the "Special Issue on Pain Catastrophizing." *J Appl Biobehav Res*. 2017;22(1):e12088. doi:10.1111/jabr.12088.

422. Martorella G, Côté J, Choinière M. Pain catastrophizing: a dimensional concept analysis. *J Adv Nurs*. 2008;63(4):417-426. doi:10.1111/j.1365-2648.2008.04699.x.

423. Sharpe L, Johnson A. Pain-related catastrophizing: we know it is important but why and how can we change it?: commentary on a paper by de Boer et al. (this issue). *Eur J Pain*. 2012;16(7):951-952. doi:10.1002/j.1532-2149.2012.00161.x.

424. Nicholas MK, Linton SJ, Watson PJ, Main CJ. Early Identification and Management of Psychological Risk Factors ("Yellow Flags") in Patients With Low Back Pain: A Reappraisal. *Phys Ther*. 2011;91(5):737-753. doi:10.2522/ptj.20100224.

425. Hayes SC, Smith SX. *Get Out of Your Mind and Into Your Life: The New Acceptance and Commitment Therapy*. Oakland: New Harbinger Publications; 2005.

426. Turner JA, Jensen MP, Romano JM. Do beliefs, coping, and catastrophizing independently predict functioning in patients with chronic pain? *Pain*. 2000;85:115-125. doi:10.1016/S0304-3959(99)00259-6.

427. Cassidy EL, Atherton RJ, Robertson N, Walsh DA, Gillett R. Mindfulness,

functioning and catastrophizing after multidisciplinary pain management for chronic low back pain. *Pain*. 2012;153(3):644-650. doi:10.1016/j.pain.2011.11.027.

428. Wetherell JL, Afari N, Rutledge T, et al. A randomized, controlled trial of acceptance and commitment therapy and cognitive-behavioral therapy for chronic pain. *Pain*. 2011;152(9):2098-2107. doi:10.1016/j.pain.2011.05.016.

429. Spielberger CD, Gorsuch RL, Lushene RE. *Manual for the State-Trait Anxiety Inventory*. Palo Alto: Consulting Psychologists Press; 1970.

430. Cattell RB, Scheier IH. *The Meaning and Measurement of Neuroticism and Anxiety*. New York: Ronald Press; 1961.

431. Clark DM. A cognitive model of panic attacks. In: Rachman S, Maser JD, eds. *Panic: Psychological Perspectives*. Hillsdale, NJ: Lawrence Erlbaum Associates; 1988:71-89.

432. Affleck G, Urrows S, Tennen H, Higgins P. Daily coping with pain from rheumatoid arthritis: patterns and correlates. *Pain*. 1992;51(2):221-229. doi:10.1016/0304-3959(92)90263-B.

433. Caneiro J, Smith A, Rabey M, Moseley GL, O'Sullivan P. Process of Change in Pain-Related Fear: Clinical Insights From a Single-Case of Persistent Back Pain Managed With Cognitive Functional Therapy. *J Orthop Sport Phys Ther*. 2017;47(9):637-651. doi:10.2519/jospt.2017.7371.

434. Nicholas MK. The pain self-efficacy questionnaire: Taking pain into account. *Eur J Pain*. 2007;11(2):153-163.

435. Tabor A, Keogh E, Eccleston C. Embodied pain— negotiating the boundaries of possible action. *Pain*. 2017;158(6):1007-1011. doi:10.1097/j.pain.0000000000000875.

436. Dworkin RH, Turk DC, Farrar JT, et al. Core outcome measures for chronic pain clinical trials: IMMPACT recommendations. *Pain*. 2005;113:9-19.

437. Sharpe L, Nicholson Perry K, Rogers P, Dear BF, Nicholas MK, Refshauge

KM. A comparison of the effect of attention training and relaxation on responses to pain. *Pain*. 2010;150(3):469-476. doi:10.1016/j.pain.2010.05.027.

438. Day MA, Thorn BE, Rubin NJ. Mindfulness-based cognitive therapy for the treatment of headache pain: A mixed-methods analysis comparing treatment responders and treatment non-responders. *Complement Ther Med*. 2014;22(2):278-285. doi:10.1016/j.ctim.2013.12.018.

439. Mor N, Winquist J. Self-focused attention and negative affect: A meta-analysis. *Psychol Bull*. 2002;128(4):638-662.

440. Kucyi A, Davis KD. The dynamic pain connectome. *Trends Neurosci*. 2014;38(2):86-95. doi:10.1016/j.tins.2014.11.006.

441. Jain S, Shapiro SL, Swanick S, et al. A randomized controlled trial of mindfulness meditation versus relaxation training: Effects on distress, positive states of mind, rumination, and distraction. *Ann Behav Med*. 2007;33(1):11-21. doi:10.1207/s15324796abm3301_2.

442. Rabey M, Smith AJ, Beales D, Slater H, O'Sullivan PB. Differing Psychologically Derived Clusters in People With Chronic Low Back Pain are Associated With Different Multidimensional Profiles. *Clin J Pain*. 2016;32(12):1015-1027. doi:10.1097/AJP.0000000000000363.

443. Hill JC, Whitehurst DGT, Lewis M, et al. Comparison of stratified primary care management for low back pain with current best practice (STarT Back): a randomised controlled trial. *Lancet*. 2011;378(9802):1560-1571. doi:10.1016/S0140-6736(11)60937-9.

444. Beneciuk JM, Robinson ME, George SZ. Subgrouping for Patients With Low Back Pain: A Multidimensional Approach Incorporating Cluster Analysis and the STarT Back Screening Tool. *J Pain*. 2015;16(1):19-30. doi:10.1016/j.jpain.2014.10.004.

445. Carr JL, Moffett J a K, Sharp DM, Haines DR. Is the Pain Stages of Change Questionnaire (PSOCQ) a useful tool for predicting participation in a self-management programme? Further evidence of validity, on a sample of UK

pain clinic patients. *BMC Musculoskelet Disord*. 2006;7:101. doi:10.1186/1471-2474-7-101.

Appendix A Example of systematic review search strategy (Study 1)

MEDLINE search strategy	
1	randomized controlled trial.pt.
2	controlled clinical trial.pt.
3	randomized.ab.
4	placebo.ab.
5	drug therapy.fs.
6	randomly.ab.
7	trial.ab.
8	groups.ab.
9	1 or 2 or 3 or 4 or 5 or 6 or 7 or 8
10	exp animals/ not humans.sh.
11	9 not 10
12	exp catastrophization/
13	exp pain/
14	12 and 13
15	"Pain Catast* Scale".af.
16	"Coping Strategies Questionnaire".af.
17	"Pain?Related Self Statements Scale".af.
18	"Catastrophic Thoughts about Pain Scale".af.
19	"Pain Cognition List".af.
20	"Profile of Chronic Pain".af.
21	"Vaginal Penetration Cognition Questionnaire".af.
22	"Cognitive Error Questionnaire".af. and pain.mp.
23	15 or 16 or 17 or 18 or 19 or 20 or 21 or 22
24	"pain catastrophi?ing".af.
25	(pain adj catastrophi*).mp. [mp=title, abstract, original title, name of substance word, subject heading word, keyword heading word, protocol supplementary concept word, rare disease supplementary concept word, unique identifier]
26	14 or 23 or 24 or 25

27	11 and 26

Appendix B Characteristics of 79 studies included in systematic review (Study 1)

Study ID (country) [citation]	Pain condition	Experimental (duration / contact)	Control (duration / contact)	Sample demographics	Experimental facilitator	Experimental format (# sessions)	PC Scale	PC primary outcome
Abbott 2010 (Sweden) [270]	Low back pain	CBT + exercise (3mo / 4.8hr)	Home exercise (3mo / 0.3hr)	Experimental: n=53 (35f); age 50.3yr; pain duration NA. Control: n=54 (31f); age 51yr; pain duration NA.	Physiotherapist	Individual face-to-face + home exercise (4)	PCS	No
Abrahamsen 2009 (Denmark) [271]	Temporomandibular disorders	Hypnosis (5wk / 4hr)	Individual Relaxation (5wk / 4hr)	Experimental: n=20 (20f); age 40.9yr; pain duration 10.6yr. Control: n=20 (20f); age 38.6yr; pain duration 13.2yr.	Dentist	Individual face-to-face (4)	CSQ	No
Alda 2011 (Spain) [122]	Fibromyalgia	CBT (9wk / 15hr)	1. Standard medication (9wk / NA) 2. TAU	Experimental: n=57 (54f); age 46.4yr; pain duration 12.9yr.	"Therapist"	Group face-to-face + homework (10)	PCS	Yes

				Control 1: n=56 (52f); age 47.1yr; pain duration 11.2yr. Control 2: n=56 (53f); age 47yr; pain duration 11.7yr.				
Amris 2014 (Denmark) [272]	Fibromyalgia	CBT (2wk / 35hr)	Waitlist (2wk / NA)	Experimental: n=96 (96f); age 44.4yr; pain duration 12.9yr. Control: n=95 (95f); age 44.2yr; pain duration 11.2yr.	Multidisciplinary: Rheumatologist + Psychologist + Nurse + Occupational therapist + Physiotherapist	Group face-to-face (NA)	CSQ	No
Basler 1997 (Germany) [119]	Low back pain	CBT (12wk / 30hr)	TAU (12wk / NA)	Combined: n=94 (71f); age 49.3yr; pain duration 10.8yr.	Clinical psychologist	Group face-to-face + homework (12)	PRSS	Yes
Bennell 2016 (Australia) [273]	Knee osteoarthritis	CBT + Exercise (12wk / 11.7hr)	1. CBT (12wk / 7.5hr) 2. Individual Exercise (12wk / 2.5hr)	Experimental: n=73 (44f); age 64.6yr; pain duration 5.5yr. Control 1: n=74 (45f); age 63yr; pain duration 5.5yr. Control 2: n=75 (44f); age 62.7yr; pain duration 6yr.	Physiotherapist	Individual face-to-face + homework (10)	PCS	No

Bergeron 2016 (Canada) [274]	Vestibulodynia (provoked)	CBT (13wk / 20hr)	Topical steroid (13wk / NA)	Experimental: n=52 (52f); age 27.8yr; pain duration 6.4yr. Control: n=45 (45f); age 26.1yr; pain duration 4.6yr.	Clinical psychologist	Group face-to-face + homework (10)	PCS	No
Brattberg 2008 (Sweden) [275]	Fibromyalgia	Emotional Freedom Techniques (8wk / NA)	Waitlist (8wk / NA)	Experimental: n=43 (43f); full sample age 43.8yr. Control: n=43 (43f); full sample age 43.8yr.	None	Internet + homework	PCS	No
Broderick 2014 (USA) [121]	Osteoarthritis (knee/hip)	CBT (10wk / 5-7.5hr)	TAU (10wk / NA)	Experimental: n=129 (129f); age 68yr; pain duration 14yr. Control: n=128 (128f); age 66.4yr; pain duration 13.6yr.	Nurse practitioner	Individual face-to-face/telephone + homework (≤4)	CSQ	Yes
Bromberg 2012 (USA) [276]	Migraine	Internet CBT (4wk / NA)	TAU (4k / NA)	Experimental: n=94 (83f); age 43.4yr; pain duration 2.5yr. Control: n=95 (82f); age 41.9yr; pain duration 2.5yr.	None	Internet + homework	PCS	No
Buhrman 2004 (Sweden) [277]	Low back pain	Internet CBT (8wk / ≤1hr)	Waitlist (8wk / NA)	Experimental: n=22 (14f); age	"Student therapist"	Internet + phone + homework (6)	CSQ	Yes

				43.5yr; pain duration 8.8yr. Control: n=29 (18f); age 45yr; pain duration 10.5yr.				
Buhrman 2011 (Sweden) [280]	Back pain	Internet CBT (12wk / NA)	Waitlist (12wk / NA)	Experimental: n=26 (19f); age 43.5yr; pain duration 12.1yr. Control: n=28 (18f); age 42.9yr; pain duration 13.1yr.	Clinical psychologist	Internet + email + homework (8)	CSQ	Yes
Buhrman 2013a (Sweden) [281]	Mixed chronic pain	Internet ACT (7wk / <1hr)	Internet discussion (7wk / 0hr)	Experimental: n=38 (21f); age 48.8yr; pain duration 13.1yr. Control: n=38 (24f); age 49.3yr; pain duration 17.4yr.	Student psychologist	Internet + email/phone + homework (7)	CSQ	No
Buhrman 2013b (Sweden) [278]	Mixed chronic pain	Internet CBT (8wk / NA)	Internet discussion (8wk / 0hr)	Experimental: n=36 (26f); age 39.9yr; pain duration 6.4yr. Control: n=36 (26f); age 40.2yr; pain duration 6.1yr.	Student psychologist	Internet + email + homework (8)	CSQ	Yes

Buhrman 2015 (Sweden) [279]	Mixed chronic pain + depression / anxiety	Internet CBT (8wk / <1hr)	Internet discussion (8wk / 0hr)	Experimental: n=28 (24f); age 54.1yr; pain duration NA. Control: n=24 (20f); age 46.8yr; pain duration NA.	Student psychologist	Internet + email/phone + homework (8)	CSQ	No
Carpenter 2012 (USA) [282]	Low back pain	Internet CBT (3wk / 0hr)	Waitlist (3wk / 0hr)	Combined: n=141 [70 Experimental, 71 Control] (114f); age 42.5yr; pain duration 8.64yr.	None	Internet + homework (6)	PCS	No
Carson 2010 (USA) [283]	Fibromyalgia	Yoga (8wk / 16hr)	Waitlist (8wk / 0.1hr)	Experimental: n=25 (25f); age 51.4yr; pain duration 10.6yr. Control: n=28 (25f); age 55.8yr; pain duration 12.5yr.	Yoga teacher	Group face-to-face + homework (8)	CSQ	No
Castel 2012 (Spain) [284]	Fibromyalgia	CBT + Standard medication + Hypnosis (14wk / 28hr)	1. CBT + Standard medication (14wk / 28hr) 2. TAU: Standard medication (14wk / NA)	Experimental: n=29 (28f); age 50.2yr; pain duration 12.5yr. Control: n=34 (32f); age 50yr; pain duration 13.6yr. Control: n=30 (30f); age 48.7yr;	Psychologist	Group face-to-face + homework (14)	CSQ	Yes

				pain duration 11.6yr.				
Castel 2013 (Spain) [286]	Fibromyalgia	CBT + physical therapy + standard medication (12wk / 48hr)	TAU: Standard medication (12wk / NA)	Experimental: n=81 (81f); age 49yr; pain duration 12.9yr. Control: n=74 (74f); age 48.8yr; pain duration 10.8yr.	Multidisciplinary: Clinical psychologist + Physiotherapist	Group face-to-face + homework (24)	CSQ	Yes
Castel 2015 (Spain) [285]	Fibromyalgia	CBT + physical therapy + standard medication (12wk / 48hr)	TAU: Standard medication (12wk / NA)	Combined: n=130 [69 intervention, 61 control] (130f); age 49.2yr; pain duration 11.8yr.	Multidisciplinary: Clinical psychologist + Physiotherapist	Group face-to-face + homework (24)	CSQ	Yes
Chiauzzi 2010 (USA) [287]	Back pain	Internet CBT (6mo / 0hr)	Education booklet (6mo / 0hr)	Experimental: n=95 (64f); age 47.3yr; pain duration NA. Control: n=104 (70f); age 45yr; pain duration NA.	None	Internet + homework (13)	PCS	Yes
De Boer 2014 (Netherlands) [288]	Mixed chronic pain	Internet CBT (16wk / NA)	Group CBT (16wk / 16hr)	Experimental: n=38 (15f); age 50.6yr; pain duration 9.8yr. Control: n=34 (17f); age 53.2yr; pain duration 7.4yr.	Psychologist	Internet + homework + email (16)	PCS	Yes
Dowd 2015 (Ireland) [289]	Mixed chronic pain	Internet mindfulness	Internet education	Combined: n=124 [62 intervention, 62	None	Internet + homework (12)	PCS	No

		(6wk / 0hr)	(6wk / 0hr)	control] (112f); age 44.5yr; pain duration 10.2yr (intervention), 11.5yr (control).				
Ersek 2008 (USA) [290]	Mixed chronic pain (older adult)	CBT (7wk / 10.5hrs)	Education book (7wk / NA)	Experimental: n=133 (116f); age 81.9yr; pain duration NA. Control: n=123 (101f); age 81.8yr; pain duration NA.	Nurse or Clinical psychologist	Group face-to-face + homework (7)	CSQ	No
Fritsche 2010 (Germany) [291]	Migraine	Group education (5wk / 10hr)	Written education (5wk / NA)	Experimental: n=79 (74f); age 47.7yr; pain duration 26.1yr. Control: n=71 (62f); age 48.4yr; pain duration 20yr.	Psychotherapist	Group face-to-face + homework (5)	PRSS	No
Gallagher 2013 (Australia) [292]	Mixed chronic pain	Written pain neuroscience education (3wk / 0hr)	Written CBT advice (3wk / 0hr)	Experimental: n=40 (26f); age 42yr; pain duration 2.1yr. Control: n=39 (22f); age 45yr; pain duration 2.6yr.	None	Written information	PCS	Yes
Garland 2012 (USA) [293]	Abdominal pain (Irritable Bowel Syndrome)	Mindfulness-Based Stress Reduction (8wk / 20hr)	Support group (8wk / 20hr)	Experimental: n=36 (36f); age 44.7yr; pain duration NA.	Certified MBSR instructor	Group face-to-face + homework (9)	CSQ	No

				Control: n=39 (39f); age 40.9yr; pain duration NA.				
Geraets 2005 (Netherlands) [294]	Shoulder pain	Graded exercise (12wk / 18hr)	TAU (12wk / NA)	Experimental: n=87 (44f); age 51.2yr; pain duration NA. Control: n=89 (52f); age 53.3yr; pain duration NA.	Physiotherapist	Group face-to-face + homework (19)	PCCL	No
Gustavsson 2010 (Sweden) [295]	Neck pain	CBT (20wk / 12hr)	Individual physiotherapy (12wk / NA)	Experimental: n=77 (69f); age 45.7yr; pain duration NA. Control: n=79 (70f); age 45.7yr; pain duration NA.	Physiotherapist	Group face-to-face + homework (8)	CSQ	No
Helminen 2015 (Finland) [296]	Knee osteoarthritis	CBT (6wk / 12hr)	TAU (6wk / NA)	Experimental: n=55 (39f); age 64.5yr; pain duration 6.6yr. Control: n=56 (38f); age 62.8yr; pain duration 8.9yr.	Multidisciplinary: Psychologist + Physiotherapist	Group face-to-face + homework (6)	PCS	No
Huang 2016 (USA) [297]	Mixed chronic pain (men with opioid induced low testosterone)	Testosterone replacement gel (3mo / NA)	Placebo gel (3mo / NA)	Experimental: n=43 (0f); age 47yr; pain duration NA. Control: n=41 (0f); age 50yr; pain duration NA.	Physician	Individual face-to-face + self-administration	PCS	No

Hutting 2015* (Netherlands) [298]	Arm, neck or shoulder pain	CBT (6wk / 15hr)	TAU (6wk / NA)	Experimental: n=64 (53f); age 45yr; pain duration NA. Control: n=53 (36f); age 47.7yr; pain duration NA.	"Moderator"	Group face-to-face + homework (6)	PCS	No
Ittersum 2014 (Belgium) [299]	Fibromyalgia	Written pain neuroscience education (6wk / NA)	Written relaxation advice	Experimental: n=53 (50f); age 47.6yr; pain duration 8.5yr. Control: n=52 (48f); age 45.8yr; pain duration 8yr.	None	Written information	PCS	Yes
Kristjánsdóttir 2013 (Norway) [300]	Fibromyalgia / Widespread pain	Smartphone ACT (4wk / 1hr)	Internet education (4wk / NA)	Experimental: n=70 (70f); age 44.6yr; pain duration 13.1yr. Control: n=70 (70f); age 43.8yr; pain duration 15.5yr.	Smartphone + Psychologist or Nurse	Smartphone + SMS feedback + homework	PCS	Yes
laCour 2015 (Denmark) [301]	Mixed chronic pain	Mindfulness-Based Stress Reduction (8wk / 31.5hr)	Waitlist (8wk / NA)	Experimental: n=55 (48f); age 48.8yr; pain duration 11.8yr. Control: n=54 (45f); age 46.5yr; pain duration 7.83yr**.	Certified MBSR teacher	Group face-to-face + homework (10)	CSQ	No

Leeuw 2008 (Netherlands) [302]	Low back pain	Exposure in vivo (NA / 16hr)	Graded exercise (NA / 26hr)	Experimental: n=42 (22f); age 46.4yr; pain duration 8.1yr. Control: n=43 (19f); age 44.2yr; pain duration 9yr.	Multidisciplinary: Psychologist + Physiotherapist + Occupational therapist	Individual face-to-face (16)	PCS	No
Luciano 2014 (Spain) [135]	Fibromyalgia	ACT (8wk / 20hr)	1. Standard medication (8wk / NA) 2. Waitlist	Experimental: n=51 (49f); age 48.9yr; pain duration 14.1yr. Control 1: n=52 (51f); age 47.8yr; pain duration 11.4yr. Control 2: n=53 (50f); age 48.3yr; pain duration 13yr.	Clinical psychologist	Group face-to-face + homework (8)	PCS	No
Mannion 1999 (Switzerland) [303]	Low back pain	Individual physiotherapy exercise + manual therapy (3mo / 13hr)	1. Group aerobic exercise (3mo / 26hr) 2. Group 'David Back Clinic' exercise (3mo / 26hr)	Experimental: n=49 (30f); age 46.3yr; pain duration 10yr. Control 1: n=50 (27f); age 45.2yr; pain duration 9.7yr. Control 2: n=49 (27f); age 43.7yr; pain duration 13yr.	Physiotherapist	Individual face-to-face + homework (26)	CSQ	No
Marshall 2013 (Australia) [340]	Low back pain	Specific trunk exercise Pilates (8wk / 24hr)	Cycling (8wk / 24hr)	Experimental: n=32 (20f); age	Exercise instructor	Group face-to-face (24)	PCS	No

				36.2yr; pain duration 9.5yr. Control: n=32 (20f); age 36.2yr; pain duration 11.1yr.				
Martínez 2014 (Spain) [304]	Fibromyalgia	CBT (6wk / 9hr)	Sleep advice (6wk / 9hr)	Experimental: n=30 (30f); age 46.5yr; pain duration 4.2yr. Control: n=29 (29f); age 48.7yr; pain duration 5.9yr.	"Therapist"	Group face-to-face (6)	PCS	No
Meeus 2010 (Belgium) [305]	Widespread pain + Chronic Fatigue Syndrome	Pain neuroscience education (1day / 0.5hr)	Pacing education (1day / 0.5hr)	Experimental: n=24 (22f); age 38.3yr; pain duration NA. Control: n=24 (18f); age 42.3yr; pain duration NA.	Physiotherapist	Individual face-to-face (1)	PCS	No
Monticone 2014 (Italy) [309]	Low back pain (post lumbar fusion surgery)	Exercise + CBT (4wk / 38hr)	Exercise (4wk / 30hr)	Experimental: n=65 (44f); age 58.8yr; pain duration 2.8yr. Control: n=65 (34f); age 55.9yr; pain duration 2.4yr.	Multidisciplinary: Physiotherapist + Psychologist	Individual face-to-face	PCS	No
Monticone 2016a (Italy) [306]	Back pain (idiopathic scoliosis)	Physiotherapy exercise + CBT (20wk / 30hr)	General physiotherapy (20wk / 20hr)	Experimental: n=65 (48f); age 51.6yr; pain duration 3.2yr.	Multidisciplinary: Physiotherapist + Psychologist	Individual face-to-face (20)	PCS	No

				Control: n=65 (46f); age 51.7yr; pain duration 3yr.				
Monticone 2016b (Italy) [307]	Low back pain (non-specific)	Exercise + CBT (5wk / 15hr)	Exercise (5wk / 10hr)	Experimental: n=75 (47f); age 53.2yr; pain duration 1.8yr. Control: n=75 (45f); age 53.8yr; pain duration 1.9yr.	Multidisciplinary: Physiotherapist + Clinical psychologist	Group face-to-face	PCS	No
Monticone 2016c (Italy) [308]	Neck pain (non-specific)	Exercise + CBT (10wk / 20hr)	Exercise (10wk / 10hr)	Experimental: n=85 (61f); age 53.8yr; pain duration 1.9yr. Control: n=85 (60f); age 52yr; pain duration 2.1yr.	Multidisciplinary: Physiotherapist + Psychologist	Group face-to-face + homework	PCS	No
Moseley 2004 (Australia) [341]	Low back pain	Pain neuroscience education (15d / 3hr)	Back anatomy education (15d / 3hr)	Experimental: n=31 (18f); age 42yr; pain duration 2.4yr. Control: n=27 (25f); age 45yr; pain duration 2.5yr.	Physiotherapist	Individual face-to-face + homework (1)	PCS	Yes
Moustafa 2015 (Egypt) [310]	Fibromyalgia	CBT + Exercise + Neck manipulation (12wk / 89hr)	CBT + Exercise + Sham neck manipulation (12wk / 89hr)	Experimental: n=60 (60f); age 53.5yr; pain duration NA. Control: n=60 (60f); age 51.4yr; pain duration NA.	Physiotherapist	Group/individual face-to-face + homework	PCS	No

Müller 2016 (Switzerland) [311]	Mixed chronic pain + physical disability (SCI / MS / Neuromuscular disease)	Internet positive psychology (8wk / 0hr)	Writing control (8wk / 0hr)	Experimental: n=51 (34f); age 58.9yr; pain duration NA. Control: n=45 (33f); age 59.9yr; pain duration NA.	None	Internet	PCS	No
Naylor 2008 (USA) [312]	Musculoskeletal pain	CBT + automated phone maintenance (7mo / 16.5hr)	CBT (7mo / 16.5hr)	Experimental: n=26 (23f); age 47yr; pain duration 13.6. Control: n=25 (21f); age 46yr; pain duration 8.6yr.	"Therapist"	Group face-to-face + automated phone messages + homework	CSQ	Yes
Nicholas 2013 (Australia) [314]	Mixed chronic pain (older adult)	Exercise + CBT (4wk / 16hr)	1. Exercise + discussion (4wk / 16hr) 2. Waitlist	Experimental: n=49 (32f); age 74.6yr; pain duration 17.2. Control 1: n=53 (37f); age 72.4yr; pain duration 14.9yr. Control 2: n=39 (20f); age 75yr; pain duration 11.2yr.	Multidisciplinary: Clinical psychologist + Physiotherapist + Registered Nurse	Group face-to-face + homework (8)	PRSS	No
Nicholas 2014 (Australia) [313]	Mixed chronic pain	CBT + Exposure (3wk / 120hr)	CBT + Distraction (3wk / 120hr)	Experimental: n=66 (34f); age 42yr; pain duration 5.6.	Multidisciplinary: Clinical psychologist + Physiotherapist + Nurse +	Group face-to-face + homework (15)	PRSS	No

				Control: n=74 (41f); age 43.2yr; pain duration 6.5yr.	Rehabilitation advisor + Pain medicine specialist			
Nordin 2016 (Sweden) [315]	Musculoskeletal pain	Multimodal (acupuncture, TENS, medication, CBT, manual therapy) + Internet CBT (4mo / NA)	Multimodal (acupuncture, TENS, medication, CBT, manual therapy) (4mo / NA)	Experimental: n=60 (47f); age 44yr; pain duration 6.6. Control: n=49 (37f); age 42yr; pain duration 6.5yr.	Multidisciplinary: Nurse + Occupational therapist + Physician+ Physiotherapist + Psychologist	Individual/group face-to-face + Internet	CSQ	No
Oerlemans 2011 (Netherlands) [316]	Abdominal pain (Irritable Bowel Syndrome)	SMS CBT (4wk / NA)	TAU (4wk / NA)	Experimental: n=38 (34f); age 35.9yr; pain duration NA. Control: n=38 (29f); age 40.6yr; pain duration NA.	Psychologist	Phone SMS	PCS	Yes
Overmeer 2016 (Sweden) [317]	Neck pain (WAD)	Neck specific exercises + CBT (12wk / NA)	1. Neck specific exercises (12wk / NA) 2. General home exercise (12wk / NA)	Experimental: n=68 (44f); age 40yr; pain duration 1.7yr Control 1: n=67 (51f); age 39yr; pain duration 1.6. Control 2: n=59 (31f); age 43.4yr; pain duration 1.6yr.	Physiotherapist	Individual face-to-face	PCS	No

Picard 2013 (France) [318]	Fibromyalgia	Hypnosis (2mo / 5hr)	Waitlist (2mo / NA)	Experimental: n=31 (30f); age 48.1yr; pain duration 9.7. Control: n=31 (29f); age 49.3yr; pain duration 6.8.	Psychologist	Individual face-to-face + homework (5)	CSQ	No
Rodríguez Torres 2015 (Spain) [319]	Fibromyalgia	Manual therapy (Neurodynamic mobilization) (8wk / 16hr)	TAU + Written education (8wk / NA)	Experimental: n=24 (19f); age 53yr; pain duration NA. Control: n=24 (20f); age 53.1yr; pain duration NA.	Physiotherapist	Individual face-to-face (16)	PCS	No
Ruehlman 2012 (USA) [320]	Mixed chronic pain	Internet CBT (6wk / 0hr)	TAU (6wk / NA)	Experimental: n=162 (combined 195f); combined age 44.9yr; pain duration NA. Control: n=143 (combined 195f); combined age 44.9yr; pain duration NA.	None	Internet + homework	PCP-EA	No
Schofield 2002* (UK) [321]	Mixed chronic pain	Snoezelen sensory stimulation (1mo / 6hr)	Progressive muscle relaxation (1mo / 6hr)	Experimental: n=43 (NA f); age 48.2yr; pain duration 6yr. Control: n=30 (NA f); age 48yr; pain duration 7.5yr.	Clinical nurse specialist	Individual face-to-face	CSQ	No

Smeets 2006 (Netherlands) [114]	Low back pain	Exercise + CBT (10wk / 79hr)	1. Exercise (10wk / 52.5hr) 2. CBT (10wk / 26.5hr) 3. Waitlist (10wk / 0hr)	Experimental: n=55 (21f); age 41.6yr; pain duration 4.7yr Control 1: n=52 (21f); age 43yr; pain duration 4.8. Control 2: n=55 (33f); age 42yr; pain duration 5.8yr. Control 3: n=49 (25f); age 40.6yr; pain duration 3.7yr.	Multidisciplinary: Physiotherapist or Occupational therapist + Clinical psychologist or Social worker	Group face-to-face + homework	PCL	No
Somers 2012 (USA) [322]	Knee osteoarthritis + overweight	CBT + weight loss program (24wk / 90hr)	1. CBT (24wk / 18hr) 2. Weight loss program (24wk / 72hr) 3. TAU (24wk / NA)	Experimental: n=62 (57f); age 57.5yr; pain duration NA Control 1: n=60 (40f); age 58.1yr; pain duration NA. Control 2: n=59 (47f); age 58.3yr; pain duration NA. Control 3: n=51 (40f); age 57.9yr; pain duration NA.	Multidisciplinary: Clinical psychologist + Exercise physiologist	Group face-to-face + homework	CSQ	No
Spinhoven 2004 (Netherlands) [116]	Low back pain (non-specific)	Behavioural activation + CBT (10wk / 150hr)	1. Behavioural activation + group discussion	Experimental: n=59 (37f); age 39.7yr; pain duration 8.2yr.	Multidisciplinary: Physiotherapist + Occupational therapist +	Group face-to-face + homework	PCCL	Yes

			(10wk / 150hr) 2. Waitlist (10wk / NA)	Control 1: n=58 (38f); age 39.2yr; pain duration 10.7yr. Control 2: n=31 (19f); age 41.1yr; pain duration 11.3yr.	Clinical psychologist			
Sterling 2015 (Australia) [323]	Neck pain (WAD)	Dry needling + exercise (6wk / 13hr)	Sham needling + exercise (6wk / 13hr)	Experimental: n=40 (24f); age 41.5yr; pain duration 1.7yr. Control: n=40 (30f); age 41.7yr; pain duration 1.3yr.	Physiotherapist	Individual face-to-face + homework (16)	PCS	No
Ter Kuile 1996 (Netherlands) [325]	Headache	Self-hypnosis + cognitive restructuring (7wk / 7hr)	Autogenic relaxation (7wk / 7hr)	Combined: n=143 [Experimental=75, Control=68] (89f); age 34.3yr; pain duration 11.1yr.	Psychologist	Individual face-to-face + homework (7)	CSQ	No
Ter Kuile 2015 (Netherlands) [324]	Vaginismus (lifelong)	Graded exposure (6wk / 10hr)	Waitlist (6wk / NA)	Experimental: n=35 (35f); age 28.5yr; pain duration 9.6yr. Control: n=35 (35f); age 29.3yr; pain duration 11.2yr.	Psychologist	Individual face-to-face + homework (5)	VPCQ	No

Tetsunaga 2015 (Japan) [326]	Low back pain + depression	Medication: Tramadol + Acetaminophen (8wk / NA)	NSAID medication: Celecoxib (8wk / NA)	Experimental: n=35 (22f); age 65.4yr; pain duration 3.8yr. Control: n=35 (22f); age 62.3yr; pain duration 4.5yr.	Physician	Individual face-to-face + home administration (4)	PCS	Yes
Thorn 2011 (USA) [327]	Mixed chronic pain + low SES	CBT (10wk / 15hr)	Education (10wk / 15hr)	Experimental: n=49 (35f); age 50.2yr; pain duration 20.6yr. Control: n=34 (30f); age 55.4yr; pain duration 19.5yr.	Clinical psychologist	Group face-to-face + homework (10)	PCS	No
Trompetter 2015 (Netherlands) [328]	Mixed chronic pain	Internet ACT (3mo / NA)	1. Expressive writing (3mo / NA) 2. Waitlist (3mo / NA)	Experimental: n=82 (63f); age 52.9yr; pain duration NA. Control 1: n=79 (60f); age 52.3yr; pain duration NA. Control 2: n=77 (58f); age 53.2yr; pain duration NA.	Student psychologist	Internet + email + homework	PCS	No
Trudeau 2015 (USA) [329]	Arthritis (osteoarthritis / rheumatoid arthritis)	Internet CBT (1mo / NA)	Waitlist (1mo / NA)	Experimental: n=113 (75f); age 50.3yr; pain duration NA.	None	Internet + homework	PCS	Yes

				Control: n=115 (81f); age 49.6yr; pain duration NA.				
Turner 1988 (USA) [330]	Low back pain	CBT (8wk / 16hr)	1. Behaviour therapy (8wk / 16hr) 2. Waitlist (8wk / 0hr)	Combined: n=81 [Experimental=26, Control1=30, Control2=25] (30f); age 46yr; pain duration 6.2yr.	Clinical psychologist	Group face-to-face + homework (8)	CEQ	Yes
Turner 2006 (USA) [331]	Temporomandibular disorders	CBT (8wk / NA)	Education (8wk / NA)	Experimental: n=61 (Combined 111f); age 39.3yr; pain duration combined 1.2yr. Control: n=65 (Combined 111f); age 35.4yr; pain duration combined 1.2yr.	Clinical psychologist	Individual face-to-face + phone + homework	CSQ	No
Turner 2011 (USA) [332]	Temporomandibular disorders	CBT + hormone education (6mo / 4.25hr)	1. CBT (6mo / 4.25hr) 2. Contraceptive pill (6mo / NA)	Experimental: n=60 (60f); age 29.1yr; pain duration 5yr. Control: n=57 (57f); age 25.4yr; pain duration 5.	Dental hygienist	Individual face-to-face + homework (8)	CSQ	No
Turner 2016 (2016) [136]	Low back pain	Mindfulness (MBSR) (8wk / 16hr)	1. CBT (8wk / 16hr) 2. TAU (8wk / 0hr)	Experimental: n=116 (71f); age 50yr; pain duration NA.	Meditation teacher	Group face-to-face + homework (8)	PCS	No

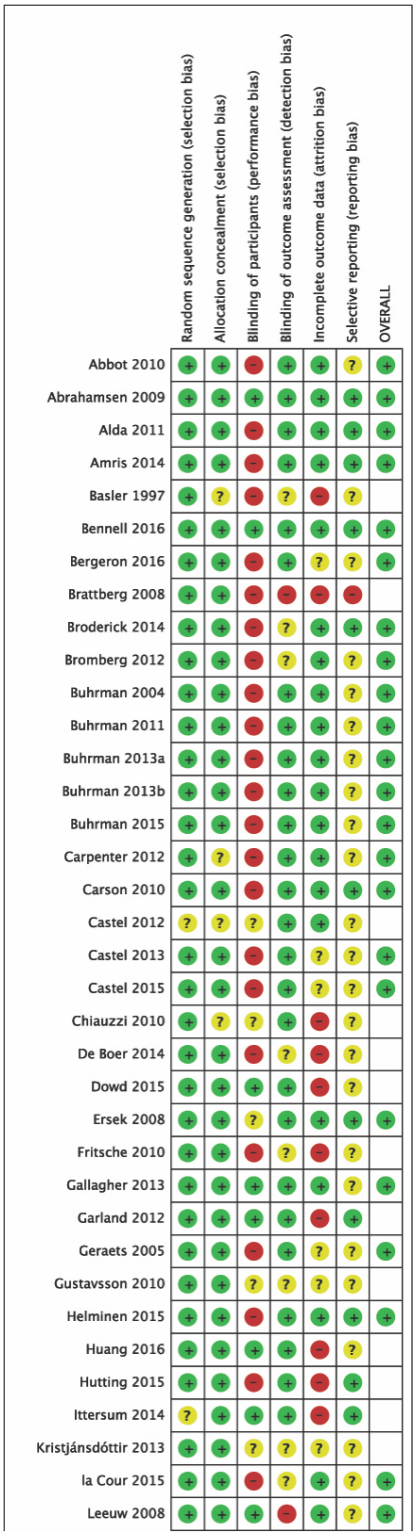
				Control 1: n=112 (66f); age 49.1yr; pain duration NA. Control 2: n=113 (87f**); age 48.9yr; pain duration NA.				
Vallejo 2015 (Spain) [333]	Fibromyalgia	CBT [internet] (10wk / NA)	1. CBT [group] (10wk / 20hr) 2. Waitlist (10wk / 0hr)	Experimental: n=20 (20f); age 49.8yr; pain duration 13.8. Control 1: n=20 (20f); age 53.5yr; pain duration 14.9. Control 2: n=20 (20f); age 51.3yr; pain duration 12.4.	Clinical psychologist	Internet + email + homework (10)	PCS	No
Van der Maas 2015 (Netherlands) [334]	Mixed musculoskeletal pain	CBT + Psychomotor Therapy (12wk / 109hr)	CBT (12wk / 94hr)	Experimental: n=49 (45f); age 38.6yr; pain duration NA. Control: n=45 (32f); age 45.4yr; pain duration NA.	Multidisciplinary: Psychologist + Physiotherapist or Occupational therapist	Group face-to-face (36)	PCS	No
Vlaeyen 1996 (Netherlands) [335]	Fibromyalgia	Education + exercise + cognitive restructuring (6wk / 42hr)	1. Education + exercise + group discussion (6wk / 42hr) 2. Wailist	Experimental: n=46 (46f); age 44.6yr; pain duration 10.4. Control 1: n=39 (34f); age 44.6yr; pain duration 10.2.	Psychologist	Group face-to-face + homework (10)	PCL	No

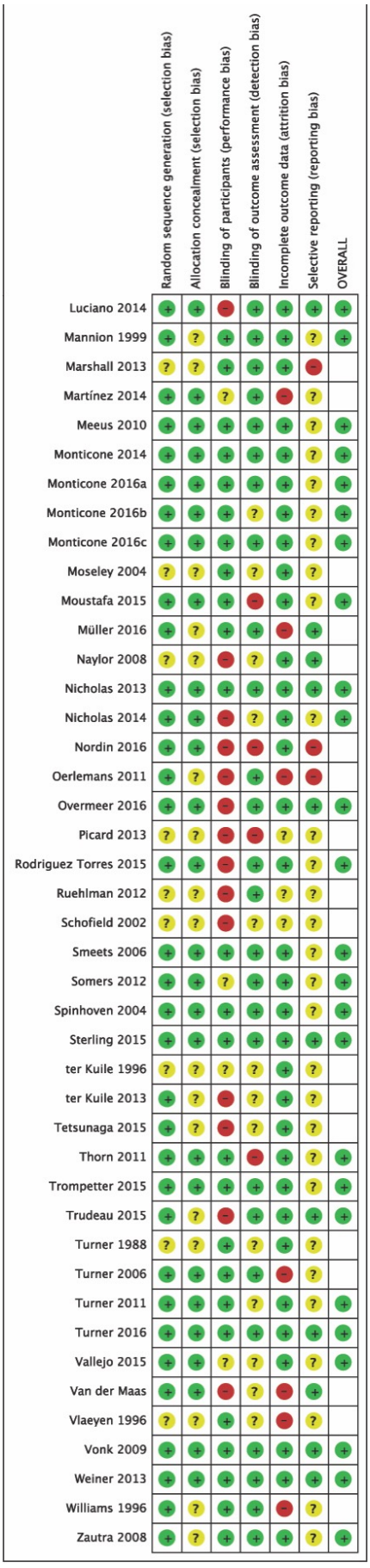
			(6wk / 0hr)	Control 2: n=40 (35f); age 42.8yr; pain duration 9.9.				
Vonk 2009 (Netherlands) [336]	Neck pain (non-specific)	Graded activity (9wk / 3.3hr)	Conventional exercise (9wk / 5.6)	Experimental: n=68 (43f); age 45.7yr; pain duration 5yr. Control: n=71 (43f); age 45.7yr; pain duration 4.5yr.	Physiotherapist	Individual face-to-face + homework	PCS	No
Weiner 2013 (USA) [337]	Knee osteoarthritis	Acupuncture periosteal stimulation + boosters (12wk / 8.5hr)	1. Acupuncture periosteal stimulation + sham boosters (12wk / 8.5hr) 2. Sham periosteal stimulation therapy (12wk / 5hr)	Experimental: n=63 (8f); age 67.1yr; pain duration 5.7yr. Control 1: n=64 (10f); age 65.8yr; pain duration 6.2yr. Control 2: n=63 (11f); age 66.8yr; pain duration 7.2.	Acupuncturist	Individual face-to-face (10)	CSQ	No
Williams 1996 (UK) [338]	Mixed chronic pain	Multimodal CBT [inpatient] (4wk / 126hr)	1. Multimodal CBT [outpatient] (8wk / 28hr) 2. Waitlist (8wk / 0hr)	Experimental: n=43 (23f); age 48.7yr; pain duration 8.4yr. Control 1: n=45 (22f); age 50.4yr; pain duration 7.7yr.	Multidisciplinary: Anaesthetist + Clinical psychologist + Physiotherapist + Occupational therapist + Nurse	Group face-to-face (20)	CSQ	No

				Control 2: n=33 (19f); age 51.1yr; pain duration 7.2yr.				
Zautra 2008 (USA) [339]	Rheumatoid arthritis	Mindfulness (8wk / 16hr)	1. CBT (8wk / 16hr) 2. Education (8wk / 16hr)	Experimental: n=47 (27f); age 55.9yr; pain duration 10.9yr. Control 1: n=52 (36f); age 54.4yr; pain duration 15yr. Control 2: n=44 (34f); age 52.1yr; pain duration 11.8yr.	Psychologist	Group face-to-face + homework (8)	CSQ	No

Note: * Insufficient data for meta-analysis; ** Statistically significant difference between groups at baseline; NA = not available; CBT = Cognitive Behaviour Therapy; TAU=Treatment as usual; ACT = Acceptance and Commitment Therapy; PCCL= Pain Coping and Cognition List, PCS= Pain Catastrophising Scale; CSQ = Coping Strategies Questionnaire; PRSS = Pain-Related Self-Statements Scale; PCL=Pain Cognition List; VPCQ = Vaginal Penetration Cognition Questionnaire; CEQ= Cognitive Errors Questionnaire.

Appendix C Risk of bias summary for studies included in systematic review (Study 1)




Random sequence generation (selection bias)
Allocation concealment (selection bias)
Blinding of participants (performance bias)
Blinding of outcome assessment (detection bias)
Incomplete outcome data (attrition bias)
Selective reporting (reporting bias)
OVERALL
Luciano 2014
Mannion 1999
Marshall 2013
Martínez 2014
Meeus 2010
Monticone 2014
Monticone 2016a
Monticone 2016b
Monticone 2016c
Moseley 2004
Moustafa 2015
Müller 2016
Naylor 2008
Nicholas 2013
Nicholas 2014
Nordin 2016
Oerlemans 2011
Overmeer 2016
Picard 2013
Rodriguez Torres 2015
Ruehlman 2012
Schofield 2002
Smeets 2006
Somers 2012
Spinhoven 2004
Sterling 2015
ter Kuile 1996
ter Kuile 2013
Tetsunaga 2015
Thorn 2011
Trompetter 2015
Trudeau 2015
Turner 1988
Turner 2006
Turner 2011
Turner 2016
Vallejo 2015
Van der Maas
Vlaeyen 1996
Vonk 2009
Weiner 2013
Williams 1996
Zautra 2008

Appendix D Semi-structured interview schedule (Study 2)

1. Could you begin by telling me about your experience of pain?
    - Prompts:
        i. What does it feel like?
        ii. How has pain impacted your life?

2. What goes through your mind when you're in pain?
    - Prompts:
        i. Thoughts, images, memories

3. How do you try to cope with your pain?
    - Prompts:
        a. What kinds of things do you do?
        b. What kind of mental strategies do you use?

4. On this questionnaire (PCS) you agreed quite a bit with statements like (*read strongly endorsed items*) … Could you tell me more about that?

5. At times we all get stuck thinking about something over and over, a bit like getting a song stuck in your head. One word for this is rumination. Worrying about things in the future or dwelling on things that have happened are both kinds of rumination. Do you ever find yourself ruminating about pain, or getting stuck thinking about it?
    - Prompts:
        i. What kinds of things do you think about when you're in pain?
        ii. Do you ruminate more about the future or about the past?

6. Are there any advantages to thinking a lot about pain?
    - Prompts:
        i. If you could give up thinking about about pain would you?
        ii. Does thinking a lot about pain help you to deal with it?
        iii. What would happen if you didn't pay much attention to thoughts about pain?
        iv. Is there a purpose or goal somewhere in the back of your mind when you're analyzing things? What are you trying to achieve by thinking about it?

7. Are there any disadvantages to thinking a lot about pain?
    - Prompts:
        i. How does it affect your pain experience?

ii. How does it affect how you feel about yourself?
iii. Could anything bad happen if you kept ruminating like this?

8. How much control do you have over whether you think about pain or not?

9. How do you know when to stop thinking about pain?

10. Could you tell me about a time in the last few days when you found yourself thinking a lot about pain or worrying about it?
    - Prompts:
        i. What triggered you to think about it?
        ii. What thoughts or images were going through your mind?
        iii. Where was your attention when that was happening?
        iv. How did you feel while this was happening?
        v. What happened next?

11. Is there anything else you would like to tell me that might help me to understand your experience of pain better?

Appendix E Examples of metacognitions elicited during interviews with scale development sample (Study 3)

Metacognitions		Examples
Positive Thinking about pain is helpful	Helps me solve problems (pain relief)	"What the problem solving is, it's just to work through the exercises to see if they'll help the pain." (16. Back)
	Helps me solve problems (general)	"There's lots of options and, when there's options, I need to think about what's the best option" (12. Knee)
	Protects me from injury	"So you have a bit of caution about doing things… You're not putting yourself at risk." (19. Back)
	Prepares me	"I guess it's more trying to predict what could happen so I'm pre-empting things I guess" (013. Back)
	Helps me cope	"I'd rather think than not think. I don't know how to explain it to you, but when I think I know where I'm going. If I don't think it's like I'm lost." (20. Back)
	Avoidance	"When you think about lots of things, even if you've got pain there you can't feel that because you're thinking already about something else." (07. Back)
Negative Thinking about pain is unhelpful	Makes my pain worse	"If I think about my pain, it makes it worse." (06. Back)
	Increases distress	"You just get worse and worse and worse and into depression. I suppose you just get suicidal." (02. Knee)
	Damages my relationships	"It not only grabs you in one way, it also can destroy your family in others. It's a very nasty type of thing." (09. Back)
	Uncontrollable	"You can't help it. Especially when you're sitting there on your own… It's uncontrollable – you just think about it because it's constantly there. It does, it starts

		consuming yourself, consumes your thoughts" (17. Back)
	Makes me worry more (meta-worry)	"I spend so much time thinking about it and then, on top of that, thinking that I shouldn't be thinking about it. Then on top of that, thinking I shouldn't be beating myself up about thinking about it. It just gets ridiculous." (18. Back)
	Pointless	"To me, there's more important things to be done than just continually worrying about things. That, to me, is sort of like ifs, buts and maybes. You know, you just try and make decisions and get on with things." (08. Back)

Note. Participant identification numbers shown in parentheses, along with sub-sample membership (Back = chronic back pain sample, N=15; Knee = total knee replacement sample, N=5).

Appendix F Demographic and pain characteristics of initial scale validation sample (Study 3)

Variable	Frequency	% Sample
Gender		
Male	279	32.3
Female	585	67.7
Marital status		
Married	409	47.3
Defacto	89	10.3
Single	356	41.2
Widow	10	1.2
Compensation claim		
No	803	92.9
Yes, workers	28	3.2
Yes, motor	10	1.2
Yes, other	23	2.7
Work status		
Full-time paid	397	45.9
Part-time paid	145	16.8
Unemployed (pain-related)	81	9.4
Unemployed (not pain-related)	32	3.7
Leave (pain-related)	8	.9
Studying	68	7.9
Retraining	2	.2
Retired	34	3.9
Home duties	39	4.5
Voluntary work	2	.2
Other	56	6.5
Education		
<10 years schooling	10	1.2
10 years schooling	47	5.4
12 years schooling	197	22.8
Apprenticeship/technical	121	14.0
University Bachelor degree	314	36.3

University postgraduate degree	175	20.3
Pain site		
Head/face	41	4.7
Neck	187	21.6
Upper limb (including shoulder, elbow, hands)	181	20.9
Chest	39	4.5
Upper back	125	14.5
Abdomen	20	2.3
Lower back	347	40.2
Lower limb (including, hip, knee, ankle)	307	35.5
Pelvis/genitals	36	4.2
Widespread pain (all over my body)	126	14.6

Note. N=864. Participants could endorse more than one pain site.

Appendix G Pain Metacognitions Questionnaire (PMQ)

Pain Metacognitions Questionnaire (PMQ)

We all think about pain in different ways. Please help us to understand your attitudes towards your own thoughts about pain. Rate how much you agree or disagree with the statements below. There are no right or wrong answers.

0 = Strongly Disagree
1 = Disagree
2 = Agree
3 = Strongly Agree

1.	**My pain won't improve unless I analyse it.**	0	1	2	3
2.	**When I'm thinking about my pain I'm trying to problem solve it.**	0	1	2	3
3.	**Thinking a lot about my pain protects me from getting injured.**	0	1	2	3
4.	**My pain would get worse if I didn't think about it a lot.**	0	1	2	3
5.	**Analyzing my pain prepares me for the worst.**	0	1	2	3
6.	**Focusing on the bad things about my pain helps me to enjoy the good things more.**	0	1	2	3
7.	**My pain won't sneak up on me as long as I keep thinking about it.**	0	1	2	3
8.	**Thinking a lot about my pain helps me to cope with it.**	0	1	2	3
9.	**Thinking about my pain helps me to understand myself.**	0	1	2	3
10.	**When I start thinking about my pain, it's impossible to stop.**	0	1	2	3
11.	**I don't try to stop thinking about my pain because my thoughts seem to have a life of their own.**	0	1	2	3
12.	**Thinking about my pain all the time makes me feel depressed.**	0	1	2	3
13.	**I'd be happier if I stopped thinking about pain.**	0	1	2	3
14.	**I feel stressed if I think a lot about my pain.**	0	1	2	3
15.	**I would be less anxious if I didn't focus on my pain as much.**	0	1	2	3
16.	**I make my pain worse by analysing it.**	0	1	2	3
17.	**I must block out my thoughts about pain.**	0	1	2	3
18.	**It's important to control my thoughts about pain.**	0	1	2	3
19.	**I worry about the negative effects of thinking too much about my pain.**	0	1	2	3
20.	**I get caught in a vicious cycle of thinking about my pain and then thinking about how I wish I could stop thinking about it.**	0	1	2	3
21.	**When I find myself brooding on my pain, it starts me thinking about how I'm just making things worse.**	0	1	2	3

(Scoring instructions: Sum items 1-9 for positive metacognitions subscale score (PMQ-P); sum items 10-21 for negative metacognitions subscale score (PMQ-N). Further assessment is indicated for a PMQ-P score >9 or PMQ-N score >18.)

Appendix H Demographic and pain characteristics of second scale validation sample (Study 3)

Variable	Frequency	%
Gender		
Male	204	40.0
Female	306	60.0
Marital status		
Married	270	52.9
Defacto	11	2.2
Single	226	44.3
Widow	3	0.6
Compensation claim		
No	449	88.0
Yes, workers	36	7.1
Yes, motor	10	2.0
Yes, other	15	2.9
Work status		
Full-time paid	292	57.3
Part-time paid	70	13.7
Unemployed (pain-related)	29	5.7
Unemployed (not pain-related)	19	3.7
Leave (pain-related)	4	.8
Studying	16	3.1
Retired	20	3.9
Home duties	40	7.8
Voluntary work	4	.8
Other	16	3.1
Education		
<10 years schooling	2	0.4
10 years schooling	7	1.4
12 years schooling	134	26.3
Apprenticeship/technical	90	17.6
University Bachelor degree	208	40.8

University postgraduate degree	69	13.5
Pain frequency		
Always present (intensity constant)	25	4.9
Always present (intensity varies)	229	44.9
Often present	133	26.1
Occasionally present	95	18.6
Rarely present	28	5.5
Pain diagnosis		
Yes	210	41.2
No	300	58.8
Psychological diagnosis		
Yes	98	19.2
No	412	80.8
Pain site		
Head/face	29	5.7
Neck	90	17.6
Upper limb (including shoulder, elbow, hands)	101	19.8
Chest	9	1.8
Upper back	73	14.3
Abdomen	20	3.9
Lower back	205	40.2
Lower limb (including, hip, knee, ankle)	156	30.6
Pelvis/genitals	12	2.4
Widespread pain (all over my body)	39	7.6

Note. N=510. Participants could endorse more than one pain site.

Appendix I Evidence of ethical approval

Government of **Western Australia**
Department of **Health**
South Metropolitan Health Service

Human Research Ethics Committee

dm
9 December 2014

Mr Robert Schutze
6/6 Richardson Street
WEST PERTH WA 6005

Dear Mr Schutze,

Project Title: Development and Validation of the Pain Rumination and Metacognitions Inventory (PRAMI)
HREC Reference: 14/79

Thank you for attending the South Metropolitan Health Service (SMHS) Human Research Ethics Committee (HREC) meeting on 2nd December 2014 and for answering Members' questions on the above project.

Document/s
• Research Transition Management Form 14-79 • WA Health Ethics Application Form Date: 18/11/2014 • Research Protocol, November 2014 • PRAMI flyer, study 1 (November 2014, version 2) • PRAMI flyer, study 2 (November 2014, version 2) • PRAMI flyer, study 3 (November 2014, version 2) • PRAMI Participant Information Form – Study 1 (November 2014, version 2) • PRAMI Participant Information Form – Study 2 (November 2014, version 2) • PRAMI Participant Information Form – Study 3 (November 2014, version 2) • Draft Interview Schedule – Semi-Structured Interview Schedule (November 2014) • Draft PRAMI Items – Pain Rumination and Metacognitions Inventory (PRAMI) (November 2014) • Validation Measures – Pain Catastrophizing Scale. Michael JL Sullivan, 1995 • Brief Pain Inventory, undated • MCT Institute Meta-cognitions Questionnaire 30 MCQ-30 – Adrian Wells and Samantha Cartwright-Hatton (1999) • Hospital Anxiety and Depression Scale, undated • Tampa Scale of Kinesiophobia, undated • Pain Vigilance and Awareness Questionnaire, undated. • Chronic Pain Acceptance Questionnaire, undated • Mindful Attention Awareness Scale, undated.

Approval of this project from SMHS HREC, is valid to December 2017 and on the basis of compliance with the 'Conditions of HREC Approval for a Research Project' (attached). The following project specific conditions also apply.

When the draft appendices *A. Semi-Structured Interview Schedule* and *B. Pain Rumination and Metacognitions Inventory (PRAMI)* have been finalised for use in Phase 2 of the study, this document will need to be submitted to the Human Research Ethics Committee as a protocol amendment and gain approval before use.

546d.docx

Research and Governance & Ethics Office
Fremantle Hospital
Demountable 3, G Block
Postal Address: PO BOX 480 FREMANTLE WA 6959
Telephone: (08) 94312929 Facsimile: (08) 94313930
http://www.fhhs.health.wa.gov.au ABN 13 992 250 709

MEMORANDUM

Curtin University

To:	A/Prof Clare Rees School of Psychology and Speech Pathology
CC:	
From	Professor Peter O'Leary, Chair HREC
Subject	Reciprocal ethics approval Approval number: HR23/2015
Date	05-Feb-15

Office of Research and Development
Human Research Ethics Office

TELEPHONE 9266 2784
FACSIMILE 9266 3793
EMAIL hrec@curtin.edu.au

Thank you for your application submitted to the Human Research Ethics Office for the project: 6078
Development and validation of the Pain Rumination and Metacognitions Inventory (PRAMI)

Your application has been approved through Curtin University Human Research Ethics Committee (HREC) through a reciprocal approval process with the lead HREC.

The lead HREC for this project has been identified as South Metropolitan Health Service HREC

Approval number from the lead HREC is noted as: HREC Ref:14/76

Please note the following conditions of approval:

1. Approval is granted from **05-Feb-15** to **31-Dec-17**
2. Research must be conducted as stated in the approved protocol.
3. Any amendments to the approved protocol must be approved by the Ethics Office.
4. An annual progress report must be submitted to the Ethics Office annually, on the anniversary of approval.
5. All adverse events must be reported to the Ethics Office.
6. A completion report must be submitted to the Ethics Office on completion of the project.
7. Data must be stored in accordance with WAUSDA and Curtin University policy.
8. The Ethics Office may conduct a randomly identified audit of a proportion of research projects approved by the HREC.

Should you have any queries about the consideration of your project please contact the Ethics Support Officer for your faculty, or the Ethics Office at hrec@curtin.edu.au or on 9266 2784. All human research ethics forms and guidelines are available on the ethics website.

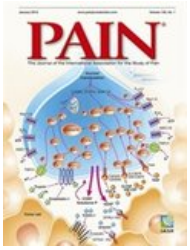
Yours sincerely

Professor Peter O'Leary
Chair, Human Research Ethics Committee

Appendix J Permission to reproduce published material

Copyright Clearance Center RightsLink®

Home | Account Info | Help



Title: Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art

Author: Johan Vlaeyen and Steven Linton

Publication: Pain

Publisher: Wolters Kluwer Health, Inc.

Date: Jan 1, 2000

Logged in as:
Robert Schutze
Curtin University, School of Psychology & Speech Pathology
Account #: 3001067895
LOGOUT

Order Completed

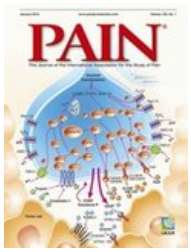
Thank you for your order.

This Agreement between Curtin University, School of Psychology & Speech Pathology -- Robert Schutze ("You") and Wolters Kluwer Health, Inc. ("Wolters Kluwer Health, Inc.") consists of your license details and the terms and conditions provided by Wolters Kluwer Health, Inc. and Copyright Clearance Center.

Your confirmation email will contain your order number for future reference.

Printable details.

License Number	4180550572547
License date	Sep 01, 2017
Licensed Content Publisher	Wolters Kluwer Health, Inc.
Licensed Content Publication	Pain
Licensed Content Title	Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art
Licensed Content Author	Johan Vlaeyen and Steven Linton
Licensed Content Date	Jan 1, 2000
Licensed Content Volume	85
Licensed Content Issue	3
Type of Use	Dissertation/Thesis
Requestor type	Individual
Portion	Figures/table/illustration
Number of figures/tables/illustrations	1
Figures/tables/illustrations used	Figure 2
Author of this Wolters Kluwer article	No
Title of your thesis / dissertation	Thinking about thinking about pain: Development of a measure and model of pain-related metacognition
Expected completion date	Sep 2017
Estimated size(pages)	320
Requestor Location	Curtin University, School of Psychology & Speech Pathology 6/6 Richardson Street West Perth, Western Australia 6005 Australia Attn: Robert Schutze
Billing Type	Invoice



Title: Worry and chronic pain: A misdirected problem solving model

Author: Christopher Eccleston and Geert Crombez

Publication: Pain

Publisher: Wolters Kluwer Health, Inc.

Date: Dec 1, 2007

Order Completed

Thank you for your order.

This Agreement between Curtin University, School of Psychology & Speech Pathology -- Robert Schutze ("You") and Wolters Kluwer Health, Inc. ("Wolters Kluwer Health, Inc.") consists of your license details and the terms and conditions provided by Wolters Kluwer Health, Inc. and Copyright Clearance Center.

Your confirmation email will contain your order number for future reference.

Printable details.

License Number	4180550087898
License date	Sep 01, 2017
Licensed Content Publisher	Wolters Kluwer Health, Inc.
Licensed Content Publication	Pain
Licensed Content Title	Worry and chronic pain: A misdirected problem solving model
Licensed Content Author	Christopher Eccleston and Geert Crombez
Licensed Content Date	Dec 1, 2007
Licensed Content Volume	132
Licensed Content Issue	3
Type of Use	Dissertation/Thesis
Requestor type	Individual
Portion	Figures/table/illustration
Number of figures/tables/illustrations	1
Figures/tables/illustrations used	Figure 1
Author of this Wolters Kluwer article	No
Title of your thesis / dissertation	Thinking about thinking about pain: Development of a measure and model of pain-related metacognition
Expected completion date	Sep 2017
Estimated size(pages)	320
Requestor Location	Curtin University, School of Psychology & Speech Pathology 6/6 Richardson Street West Perth, Western Australia 6005 Australia Attn: Robert Schutze
Billing Type	Invoice
Billing address	Curtin University, School of Psychology & Speech Pathology

RightsLink®

Home | Account Info | Help

Book: Treating Depression: MCT, CBT and Third Wave Therapies
Author: Adrian Wells (Editor), Peter Fisher (Editor)
Publisher: John Wiley and Sons
Date: Dec 1, 2015

Logged in as:
Robert Schutze
Curtin University, School of Psychology & Speech Pathology
Account #:
3001067895
LOGOUT

Order Completed

Thank you for your order.

This Agreement between Curtin University, School of Psychology & Speech Pathology -- Robert Schutze ("You") and John Wiley and Sons ("John Wiley and Sons") consists of your license details and the terms and conditions provided by John Wiley and Sons and Copyright Clearance Center.

Your confirmation email will contain your order number for future reference.

Printable details.

License Number	4180551140968
License date	Sep 01, 2017
Licensed Content Publisher	John Wiley and Sons
Licensed Content Publication	Wiley Books
Licensed Content Title	Treating Depression: MCT, CBT and Third Wave Therapies
Licensed Content Author	Adrian Wells (Editor), Peter Fisher (Editor)
Licensed Content Date	Dec 1, 2015
Licensed Content Pages	464
Type of use	Dissertation/Thesis
Requestor type	University/Academic
Format	Print and electronic
Portion	Figure/table
Number of figures/tables	1
Original Wiley figure/table number(s)	Figure 6.1
Will you be translating?	No
Title of your thesis / dissertation	Thinking about thinking about pain: Development of a measure and model of pain-related metacognition
Expected completion date	Sep 2017
Expected size (number of pages)	320
Requestor Location	Curtin University, School of Psychology & Speech Pathology 6/6 Richardson Street West Perth, Western Australia 6005 Australia Attn: Robert Schutze
Publisher Tax ID	EU826007151
Billing Type	Invoice
Billing address	Curtin University, School of Psychology & Speech Pathology

WILEY

Title: Re-thinking over-thinking pain: What can metacognition add to our understanding of pain catastrophising?

Author: Robert Schütze

Publication: Clinical Psychologist

Publisher: John Wiley and Sons

Date: Nov 17, 2016

Order Completed

Thank you for your order.

This Agreement between Curtin University, School of Psychology & Speech Pathology -- Robert Schutze ("You") and John Wiley and Sons ("John Wiley and Sons") consists of your license details and the terms and conditions provided by John Wiley and Sons and Copyright Clearance Center.

Your confirmation email will contain your order number for future reference.

Printable details.

License Number	4180560196428
License date	Sep 01, 2017
Licensed Content Publisher	John Wiley and Sons
Licensed Content Publication	Clinical Psychologist
Licensed Content Title	Re-thinking over-thinking pain: What can metacognition add to our understanding of pain catastrophising?
Licensed Content Author	Robert Schütze
Licensed Content Date	Nov 17, 2016
Licensed Content Pages	7
Type of use	Dissertation/Thesis
Requestor type	Author of this Wiley article
Format	Print and electronic
Portion	Full article
Will you be translating?	No
Title of your thesis / dissertation	Thinking about thinking about pain: Development of a measure and model of pain-related metacognition
Expected completion date	Sep 2017
Expected size (number of pages)	320
Requestor Location	Curtin University, School of Psychology & Speech Pathology 6/6 Richardson Street West Perth, Western Australia 6005 Australia Attn: Robert Schutze
Publisher Tax ID	EU826007151
Billing Type	Invoice
Billing address	Curtin University, School of Psychology & Speech Pathology 6/6 Richardson Street

WILEY

Title: 'I call it stinkin' thinkin'': A qualitative analysis of metacognition in people with chronic low back pain and elevated catastrophizing

Author: Robert Schütze,Clare Rees,Helen Slater,Anne Smith,Peter O'Sullivan

Publication: British Journal of Health Psychology

Publisher: John Wiley and Sons

Date: Apr 3, 2017

Logged in as:
Robert Schutze
Curtin University, School of Psychology & Speech Pathology
Account #:
3001067895
LOGOUT

Order Completed

Thank you for your order.

This Agreement between Curtin University, School of Psychology & Speech Pathology -- Robert Schutze ("You") and John Wiley and Sons ("John Wiley and Sons") consists of your license details and the terms and conditions provided by John Wiley and Sons and Copyright Clearance Center.

Your confirmation email will contain your order number for future reference.

Printable details.

License Number	4180551495864
License date	Sep 01, 2017
Licensed Content Publisher	John Wiley and Sons
Licensed Content Publication	British Journal of Health Psychology
Licensed Content Title	'I call it stinkin' thinkin'': A qualitative analysis of metacognition in people with chronic low back pain and elevated catastrophizing
Licensed Content Author	Robert Schütze,Clare Rees,Helen Slater,Anne Smith,Peter O'Sullivan
Licensed Content Date	Apr 3, 2017
Licensed Content Pages	18
Type of use	Dissertation/Thesis
Requestor type	Author of this Wiley article
Format	Print and electronic
Portion	Full article
Will you be translating?	No
Title of your thesis / dissertation	Thinking about thinking about pain: Development of a measure and model of pain-related metacognition
Expected completion date	Sep 2017
Expected size (number of pages)	320
Requestor Location	Curtin University, School of Psychology & Speech Pathology 6/6 Richardson Street West Perth, Western Australia 6005 Australia Attn: Robert Schutze

Appendix K Published version of Chapter 1

Clinical Psychologist

Clinical Psychologist **20** (2016) 147–153

COMMENTARY

Re-thinking over-thinking pain: What can metacognition add to our understanding of pain catastrophising?

Robert SCHÜTZE

School of Psychology and Speech Pathology, Curtin University, Perth, Western Australia, Australia

Key words
chronic pain, metacognition, metacognitive therapy, pain catastrophising, repetitive negative thinking, worry.

Correspondence
Robert Schütze, School of Psychology and Speech Pathology, Curtin University, Perth, Western Australia, Australia.
Email: r.schutze@curtin.edu.au

doi:10.1111/cp.12105

Abstract

Pain catastrophising is one of the most widely studied psychological constructs in pain science, given its association with increased pain intensity, disability, emotional distress, psychopathology, and pain-facilitating neurobiological changes in people with chronic pain. Yet despite being a key target in evidence-based biopsychosocial treatments, it is still not entirely clear what pain catastrophising is or how best to treat it. This paper argues that the contents of catastrophic thinking have been over-emphasised in incumbent cognitive behavioural interventions and that pain catastrophising is most usefully conceptualised as a form of repetitive negative thinking. However, what is missing from this view of pain-related rumination is an understanding of how higher order cognitive processes, such as metacognition, might moderate it. Spada and colleagues' paper in this issue of Clinical Psychologist begins to shed light on this subject, providing the first clear evidence that metacognitive beliefs about worry are strongly associated with pain catastrophising. These exciting results and their implications for future directions in pain research are discussed in the context of mounting calls for more theory-driven psychological interventions for chronic pain.

Psychologists have a pivotal role to play in helping people living with persistent, or chronic pain, an often debilitating experience that affects around one in five people and costs the Australian economy alone around $55 billion each year (Arthritis & Osteoporosis Victoria, 2013). As the dominant biopsychosocial model of pain suggests (Gatchel, Peng, Peters, Fuchs, & Turk, 2007), there is overwhelming evidence that psychological factors are intrinsically related to the development and maintenance of persistent pain through their influence on behaviour and nervous system functioning (Butler & Moseley, 2013). Moreover, the experience of living with unrelenting pain often has negative psychological sequelae—most commonly depression and anxiety—that warrant psychological intervention in their own right (Turk, 2014). Therefore, psychological therapy for persistent pain is now a mainstay of evidence-based multidisciplinary treatment, with high quality reviews consistently showing these interventions reliably improve pain, disability, and mood compared to usual medical care (Eccleston et al., 2014; Morley, Eccleston, & Williams, 1999; Williams, Eccleston, & Morley, 2012).

The problem is that as the evidence for pain psychology accumulates, effect sizes have dwindled, with the latest Cochrane review suggesting cognitive behaviour therapy (CBT), for which there is currently the most evidence, has only small effects, and these often disappear at long-term follow up (Williams et al., 2012). Furthermore, it is not clear which interventions are best suited to working with clients in pain, because there is mounting evidence that so-called third treatments such as acceptance and commitment therapy (ACT; Hayes,

Funding: This research was supported by Fremantle Hospital Medical Research Foundation in the form of a Bellberry Medical Research Scholarship to the author.
Conflict of interest: None.

Wilson, & Strosahl, 1999) and mindfulness-based stress reduction (MBSR; Kabat-Zinn, 1990) are as effective as incumbent CBT approaches (Veehof, Trompetter, Bohlmeijer, & Schreurs, 2016; Wetherell et al., 2011). This has led to widespread calls to focus future treatment research on mechanisms of change in order to dismantle and improve these psychological therapies so that we can better tailor them to individual presentations (Day & Thorn, 2014; Morley & Williams, 2015; Skinner, Wilson, & Turk, 2012), a development mirrored in the broader psychotherapy literature (Kazdin, 2009).

However, beyond merely uncovering the moderators and mediators of treatment effects, this initiative to improve both the efficacy and efficiency of psychological pain interventions must also deepen our understanding of the pain experience and the psychological constructs we target in therapy. In light of this, the paper by Spada and colleagues in the current issue (Spada, Gay, Nikčević, Fernie, & Caselli, 2015) provides an exciting new insight into perhaps one of the most widely studied psychological variables in pain science—pain catastrophising (PC). Spada et al.'s large cross-sectional study has provided our first insight into the role that metacognitive beliefs might play in shaping PC, which can broadly be defined as the tendency to exaggerate and ruminate on the threat value of pain (Sullivan, Thorn, et al., 2001). In a community sample of 308 people, Spada et al. found that positive metacognitive beliefs about worry (e.g., "worrying helps me to get things sorted out in my mind") mediated the relationship between neuroticism and PC. Meanwhile, negative metacognitions (e.g., "my worrying is dangerous for me") mediated the relationship between PC and self reported maladaptive pain behaviours.

This new insight into PC is significant given that it has become a key predictor of poor pain outcomes and therefore an important target of treatment. For example, elevated PC is associated with greater pain intensity (Leung, 2012), disability (Picavet, Vlaeyen, & Schouten, 2002), emotional distress (Sullivan, Rodgers, & Kirsch, 2001), and health-care utilisation (de Boer, Struys, & Versteegen, 2012). Furthermore, PC plays a central role in the fear avoidance model of pain (Vlaeyen & Linton, 2000, 2012), one of the most influential psychological models of common persistent musculoskeletal pain disorders such as chronic low back pain (CLBP), whiplash, and fibromyalgia (Goubert, Crombez, & van Damme, 2004; Vangronsveld, Peters, Goossens, Linton, & Vlaeyen, 2007; Vlaeyen, Kole-Snijders, Boeren, & van Eek, 1995).

There is also evidence that PC is an important process variable in pain interventions. Changes in PC predict treatment outcomes across a range of interventions, including CBT (Burns, Day, & Thorn, 2012), ACT (Vowles, McCracken, & Eccleston, 2007), physiotherapist-led exercise therapy (Smeets, Vlaeyen, Kester, & Knottnerus, 2006), and multidisciplinary treatment (Burns, Kubilus, Bruehl, Harden, & Lofland, 2003; Spinhoven et al., 2004). Finally, experimental data show that PC is associated with a range of pain-facilitating biological processes: dysregulation of the hypothalamic-pituitary axis that is linked to central nervous system sensitisation (Edwards et al., 2008; Quartana et al., 2010); reduced descending inhibitory control of pain through endogenous opioid pathways (Goodin et al., 2009); increased activation of brain areas associated with affective aspects of pain (Seminowicz & Davis, 2006); and pain-facilitating changes in functional connectivity of the brain's default mode network (Kucyi et al., 2014).

What Is PC and How Can We Change It?

While these associations obviously show that PC is important in shaping pain outcomes, it is less clear exactly what PC is (Turner & Aaron, 2001). Despite prima facie associations with depression and anxiety, PC cannot be easily reduced to either of these constructs. For example, regression studies show that PC predicts unique variance in various pain outcomes even when depression and anxiety are controlled (Sullivan, Stanish, Waite, Sullivan, & Tripp, 1998). Offering some insight into how PC has been operationalized, the most widely used and well-validated measure of PC—the Pain Catastrophizing Scale (PCS; Sullivan, Bishop, & Pivik, 1995)—suggests it has three distinct facets that are reflected in its subscales: exaggeration of the threat value of pain (*magnification* subscale); perseverative thinking about pain (*rumination* subscale); and the underestimation of one's coping ability (*helplessness* subscale).

The historical trend towards treating elevated PC by targeting the content of catastrophic cognitions (Williams & McCracken, 2004) suggests there has been an emphasis on the magnification facet of PC in the pain literature. This may be unsurprising given that at face value PC clearly evokes the spectre of Aaron Beck's cognitive theory of emotional disorders (Beck, 1976), where catastrophising is a cognitive distortion involving unhelpful and/or unrealistic beliefs (e.g., "If I can't get rid of this pain I'll end up in a wheelchair"). Indeed, detailed protocols for cognitive restructuring in the cognitive therapy tradition of Beck (Beck, Rush, Shaw, & Emery, 1979) have been developed for PC (Thorn, 2004) and CBT for pain most commonly aims to replace catastrophic pain cognitions with more helpful beliefs and coping statements (Otis, 2007). High quality reviews

suggest that compared to usual care these approaches robustly reduce PC, with a medium effect size: standardised mean difference −0.53 (Williams et al., 2012).

The success of cognitive restructuring in reducing PC may, on the one hand, support appraisal and schema-based models of PC that suggest the problem lies with unhelpful beliefs (Sullivan, Thorn, et al., 2001). On the other hand, these effects are rather modest and no better than interventions such as exercise which do not explicitly target beliefs (Smeets et al., 2006). One clue to unravelling this puzzle may lie in evidence that validation studies of the PCS show that the magnification and helplessness subscales, which most explicitly target belief content, only account for 10 and 8% of its variance, respectively (Sullivan et al., 1995). By contrast, the largest proportion of variance in this dominant measure of PC is accounted for by rumination (41%), and this subscale is the strongest predictor of pain and disability (Sullivan et al., 1998). The dominant, and somewhat neglected, role of rumination suggests that PC should be viewed from the perspective of both cognitive *contents* and cognitive *processes* (Day, Jensen, Ehde, & Thorn, 2014). Indeed, Flink and colleagues argue convincingly for a process-oriented view of PC as a form of repetitive negative thinking whose putative function is to reduce negative affect (Flink, Boersma, & Linton, 2013).

The distinction between thought contents and processes has become important in distinguishing so-called second wave CBT approaches from third-wave acceptance and mindfulness-based approaches in the broader psychotherapy literature (Öst, 2008). Third-wave interventions particularly target cognitive processes such as rumination and worry by effecting metacognitive changes in the way people view and respond to uncomfortable internal events such that intrusive thoughts, for example, come to be seen as passing mental events that can be tolerated, rather than upsetting facts requiring extended attention or concerted avoidance. Disorders maintained by unhelpful cognitive processes like rumination are remarkably responsive to third wave interventions, as the extensive evidence for mindfulness-based cognitive therapy (MBCT) in treating recurrent depression attests (Segal, Williams, & Teasdale, 2013).

We might expect the success of metacognitive approaches such as MBCT in reducing depressive rumination to translate to PC, where rumination accounts for the most variance. Indeed, this is what emerging evidence suggests. MBCT in headache patients reduces PC with a large effect size of $d = -0.97$ compared to waitlist control (Day, Thorn, et al., 2014), while numerous other studies show that mindfulness-based approaches can reliably reduce catastrophising (Gardner-Nix, Backman, Barbati, & Grummitt, 2008; Veehof et al., 2016). The same holds true for ACT, which shares an emphasis on changing the way people relate to their catastrophic thoughts rather than changing the thoughts themselves (Hayes et al., 1999). A recent high quality controlled trial found that compared to waitlist, group ACT reduced PC with a large effect size of $d = -0.89$ (Luciano et al., 2014). The success of these third-wave treatments in improving pain outcomes by targeting cognitive processes rather than cognitive contents suggests that metacognition plays an important role in explaining and modifying PC.

A Role for Metacognitive Beliefs

Although largely ignored until Spada et al.'s study in this issue, an obvious progression for this exploration of metacognition in PC is the application of another metacognitively focused intervention that has proven efficacy in treating anxiety and mood disorders—Adrian Wells' metacognitive therapy (MCT; Wells, 2009). Based on the self-regulatory executive function (S-REF) model of emotional disorder (Wells & Matthews, 1994, 1996), MCT focuses squarely on the cognitive processes of rumination and worry as forms of perseverative thinking, which is seen as the linchpin of anxiety and depression. The S-REF model suggests that it is not the content of our unhelpful, catastrophic thoughts that makes us anxious, but rather the fact that we focus too much attention on them (Fisher & Wells, 2009). Importantly, it is the beliefs we hold about our thoughts—our metacognitions—that determine how much attention we give them. For example, a common metacognition that maintains worry in generalised anxiety disorder (GAD) is the belief, "worrying helps me to solve problems" (Wells, 1999). In MCT, positive metacognitive beliefs about worry like this become key targets in treatment and are challenged through Socratic dialogue and behavioural experiments (Wells, 2009). Clients are guided into discovering that perseverative thinking about future threats (i.e., worry) is a self-regulation strategy that backfires and can be replaced with more adaptive strategies.

This formulation of worry as a form of maladaptive coping in people with GAD shares some important features with a prominent model of worry in the pain literature—Eccleston and Crombez's (2007) misdirected problem-solving model, as depicted in Fig. 1. The relevance of this model of worry to our discussion of PC is supported by recent evidence of a significant relationship between measures of worry and PC (Day, Smitherman, Ward, & Thorn, 2015). Eccleston and Crombez's (2007) model suggests that worry is an attempt to alleviate pain-related distress by finding a way to remove the

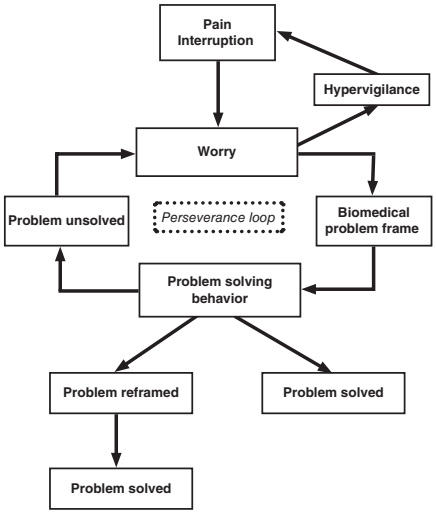


Figure 1 Misdirected problem-solving model of pain-related worry (Eccleston & Crombez, 2007).

pain. They argue that worry persists in a perseverance loop because the problem is poorly framed in terms of reducing pain from a biomedical perspective, rather than as a problem of how to live a valued life despite pain.

This model is consistent with the psychological flexibility model of pain (McCracken & Morley, 2014) that underlies ACT for pain (Dahl & Lundgren, 2006). However, what is missing from this model, particularly in terms of its treatment implications, is a more thorough articulation of the relationship between "pain interruption" and "worry," depicted in Fig. 1. Wells' metacognitive model of GAD speaks directly to this relationship, suggesting it is a person's beliefs about the usefulness of worry that moderates the relationship between an unwanted internal event, such as an intrusive thought or a sensation, and extended analysis of it as a coping response (Wells, 1999). Incorporating this insight from the S-REF model into the misdirected problem-solving model of pain-related worry, it might be proposed that positive metacognitive beliefs (e.g., "worrying helps me to solve problems") will mediate, or at least moderate, the relationship between pain and worry, offering another avenue for intervention aside from problem reframing.

The study by Spada et al. in this issue offers some preliminary support for such as proposal. They found that positive metacognitions about worry mediate between neuroticism and PC. This broadly suggests that how much a person focuses negatively on their pain depends on how helpful they believe worrying to be. Of course, this evidence is far from conclusive. For one thing, they used a non-pain sample and although the relationship between PC and other pain outcomes seems to be similar in clinical and non-clinical samples (de Boer et al., 2012), replication of this finding in a sample of people with persistent pain is an obvious next step in the quest to better understand PC by exploring its metacognitive dimensions.

Similarly, while the relationship between neuroticism and PC is interesting, exploring the role of metacognition in the more fundamental relationship between pain intensity and PC is required. Low levels of mindfulness—another metacognitive variable—have previously been found to moderate the relationship between pain and PC in a persistent pain sample (Schütze, Rees, Preece, & Schütze, 2010). Definitions of mindfulness as a present-focused, non-elaborative form of awareness (Bishop et al., 2004; Kabat-Zinn, 1990) suggests that it would be incompatible with a strong belief in the benefits of worry. Indeed a key element of MCT, alongside challenging positive worry metacognitions, is the cultivation of detached mindfulness (Wells, 2009), although not through meditation as in other mindfulness interventions. Therefore, there is good reason to expect positive metacognitive beliefs about worry to moderate, if not mediate, the relationship between pain and PC, opening the way for exploring how these metacognitions could be targeted in treatments for PC, as opposed to targeting the catastrophic thoughts themselves.

While appealing, the foregoing argument for the relevance of Wells' MCT to PC is simplistic and incomplete. For one thing, it omits the important role of unhelpful behaviours and attentional processes as pillars of the so-called cognitive attentional syndrome (CAS) that maintains distress in emotional disorders (Wells & Matthews, 1994, 1996). A possible pain-related CAS might therefore include well-documented unhelpful behaviours such as avoidance and endurance coping (Andrews, Strong, & Meredith, 2012), as well as attentional factors such as hypervigilance (van Damme, Crombez, Eccleston, & Roelofs, 2004) and attentional bias (Crombez, Van Ryckeghem, Eccleston, & van Damme, 2013). The other obvious omission from this discussion is the important role of negative metacognitive beliefs in maintaining worry and rumination. The fact that Spada et al.'s study found the strongest correlations between PC and negative metacognitions about worry ($r = 0.52$) suggests that these beliefs about the uncontrollability and harms associated with worry are key to understanding and modifying perseverative thinking about pain.

Clearly, further research in this area is needed to explore these relationships and articulate a metacognitive model of PC. There is a strong case for this, given: (1) the importance of PC in predicting poorer pain outcomes; (2) the centrality of rumination in our gold standard measure of PC; (3) the strong evidence that third-wave treatments aimed at interrupting rumination are effective in treating PC; and (4) the fact that Wells' S-REF theory and associated MCT focuses explicitly on interrupting rumination in a novel way—by targeting the beliefs people hold about their thinking. A precursor to articulating this metacognitive model of PC will be ensuring we have an acceptable way to measure metacognition in PC, whether through the existing metacognitions questionnaire (Cartwright-Hatton & Wells, 1997; Wells & Cartwright-Hatton, 2004) or a new pain-specific instrument. While it is possible that a metacognitive model of PC might result in trialling a tailored form of MCT for persistent pain, it is unlikely that MCT will be the new breakthrough treatment for chronic pain. As recent commentaries note, pain psychotherapy research could benefit from consolidation rather than a proliferation of new interventions (Day & Thorn, 2014; Morley & Williams, 2015; Skinner et al., 2012). This entails a focus on better understanding treatment mechanisms, including the shared mechanisms in existing efficacious treatments, and on improving these treatments so that they can be better targeted to specific pain phenotypes. Ultimately, this necessitates clarifying important constructs such as PC so that future generations of pain psychologists can contribute more significantly to easing the enormous individual, social, and economic burden of persistent pain.

References

Andrews, N. E., Strong, J., & Meredith, P. J. (2012). Activity pacing, avoidance, endurance, and associations with patient functioning in chronic pain: a systematic review and meta-analysis. *Archives of Physical Medicine and Rehabilitation*, *93*(11), 2109–2121.e7. doi:10.1016/j.apmr.2012.05.029

Arthritis & Osteoporosis Victoria (2013). *A problem worth solving*. Elsternwick, Australia: Arthritis and Osteoporosis Victoria.

Beck, A. T. (1976). *Cognitive therapy and the emotional disorders*. New York, NY: International Universities Press.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York, NY: Guilford Press.

Bishop, S. R., Lau, M., Shapiro, S., Carlson, L., Anderson, N. D., Carmody, J., … Devins, G. (2004). Mindfulness: A proposed operational definition. *Clinical Psychology: Science and Practice*, *11*(3), 230–241. doi:10.1093/clipsy.bph077

Burns, J. W., Day, M. A., & Thorn, B. E. (2012). Is reduction in pain catastrophizing a therapeutic mechanism specific to cognitive-behavioral therapy for chronic pain? *Translational Behavioral Medicine*, *2*(1), 22–29. doi:10.1007/s13142-011-0086-3

Burns, J. W., Kubilus, A., Bruehl, S., Harden, R. N., & Lofland, K. (2003). Do changes in cognitive factors influence outcome following multidisciplinary treatment for chronic pain? A cross-lagged panel analysis. *Journal of Consulting and Clinical Psychology*, *71*(1), 81–91. doi:10.1037/0022-006X.71.1.81

Butler, D., & Moseley, G. L. (2013). *Explain pain* (2nd ed.). Adelaide, Australia: Noigroup Publications. doi:10.3138/ptc.58.3.243

Cartwright-Hatton, S., & Wells, A. (1997). Beliefs about worry and intrusions: The meta-cognitions questionnaire and its correlates. *Journal of Anxiety Disorders*, *11*(3), 279–296. doi:10.1016/S0887-6185(97)00011-X

Crombez, G., Van Ryckeghem, D. M. L., Eccleston, C., & van Damme, S. (2013). Attentional bias to pain-related information: a meta-analysis. *Pain*, *154*(4), 497–510. doi:10.1016/j.pain.2012.11.013

Dahl, J. A., & Lundgren, T. (2006). Acceptance and commitment therapy (ACT) in the treatment of chronic pain. In R. A. Baer (Ed.), *Mindfulness-based treatment approaches: Clinician's guide to evidence base and applications* (pp. 285–306). London, UK: Academic Press.

Day, M. A., Jensen, M. P., Ehde, D. M., & Thorn, B. E. (2014). Toward a theoretical model for mindfulness-based pain management. *Journal of Pain*, *15*(7), 691–703. doi:10.1016/j.jpain.2014.03.003

Day, M. A., Smitherman, A., Ward, L. C., & Thorn, B. E. (2015). An investigation of the associations between measures of mindfulness and pain catastrophizing. *Clinical Journal of Pain*, *31*(3), 222–228. doi:10.1097/AJP.0000000000000102

Day, M. A., & Thorn, B. E. (2014). Using theoretical models to clarify shared and unique mechanisms in psychosocial pain treatments: A commentary on McCracken and Morley's theoretical paper. *Journal of Pain*, *15*(3), 237–238. doi:10.1016/j.jpain.2014.01.486

Day, M. A., Thorn, B. E., Ward, L. C., Rubin, N., Hickman, S. D., Scogin, F., & Kilgo, G. R. (2014). Mindfulness-based cognitive therapy for the treatment of headache pain: a pilot study. *Clinical Journal of Pain*, *30*(2), 152–161. doi:10.1097/AJP.0b013e318287a1dc

de Boer, M. J., Struys, M. M. R. F., & Versteegen, G. J. (2012). Pain-related catastrophizing in pain patients and people with pain in the general population. *European Journal of Pain*, *16*(7), 1044–1052. doi:10.1002/j.1532-2149.2012.00136.x

Eccleston, C., & Crombez, G. (2007). Worry and chronic pain: A misdirected problem solving model. *Pain*, *132*(3), 233–236. doi:10.1016/j.pain.2007.09.014

Eccleston, C., Fisher, E., Craig, L., Duggan, G. B., Rosser, B. A., & Keogh, E. (2014). Psychological therapies (Internet-delivered) for the management of chronic pain in adults. *Cochrane Database of Systematic Reviews*, *2*, CD010152. doi:10.1002/14651858.CD010152.pub2

Edwards, R. R., Kronfli, T., Haythornthwaite, J. A., Smith, M. T., McGuire, L., & Page, G. G. (2008). Association of catastrophizing with interleukin-6 responses to acute pain. *Pain*, *140*(1), 135–144. doi:10.1016/j.pain.2008.07.024

Fisher, P., & Wells, A. (2009). *Metacognitive therapy: Distinctive features*. New York, NY: Routledge.

Flink, I. L., Boersma, K., & Linton, S. J. (2013). Pain catastrophizing as repetitive negative thinking: A development of the conceptualization. *Cognitive Behaviour Therapy*, *42*(3), 215–223. doi:10.1080/16506073.2013.769621

Gardner-Nix, J., Backman, S., Barbati, J., & Grummitt, J. (2008). Evaluating distance education of a mindfulness-based meditation programme for chronic pain management. *Journal of Telemedicine and Telecare*, *14*(2), 88–92. doi:10.1258/jtt.2007.070811

Gatchel, R. J., Peng, Y. B., Peters, M. L., Fuchs, P. N., & Turk, D. C. (2007). The biopsychosocial approach to chronic pain: Scientific advances and future directions. *Psychological Bulletin*, *133*(4), 581–624. doi:10.1037/0033-2909.133.4.581

Goodin, B. R., McGuire, L., Allshouse, M., Stapleton, L., Haythornthwaite, J., Burns, N., … Edwards, R. R. (2009). Associations between catastrophizing and endogenous pain-inhibitory processes: Sex differences. *Journal of Pain*, *10*(2), 180–90. doi:10.1016/j.jpain.2008.08.012

Goubert, L., Crombez, G., & van Damme, S. (2004). The role of neuroticism, pain catastrophizing and pain-related fear in vigilance to pain: A structural equations approach. *Pain*, *107*(3), 234–241. doi:10.1016/j.pain.2003.11.005

Hayes, S. C., Bond, F. W., Barnes-holmes, D., & Austin, J. (1999). *Acceptance and mindfulness at work: Applying acceptance and commitment therapy and relational frame theory to organizational behavior management*. Haworth Press.

Kabat-Zinn, J. (1990). *Full catastrophe living: Using the wisdom of your body and mind to face stress, pain, and illness*. New York, NY: Delta.

Kazdin, A. E. (2009). Understanding how and why psychotherapy leads to change. *Psychotherapy Research: Journal of the Society for Psychotherapy Research*, *19*(4–5), 418–428. doi:10.1080/10503300802448899

Kucyi, A., Moayedi, M., Weissman-Fogel, I., Goldberg, M. B., Freeman, B. V., Tenenbaum, H. C., & Davis, K. D. (2014). Enhanced medial prefrontal-default mode network functional connectivity in chronic pain and its association with pain rumination. *Journal of Neuroscience*, *34*(11), 3969–3975. doi:10.1523/JNEUROSCI.5055-13.2014

Leung, L. (2012). Pain catastrophizing: An updated review. *Indian Journal of Psychological Medicine*, *34*(3), 204–217. doi:10.4103/0253-7176.106012

Luciano, J. V., Guallar, J. A., Aguado, J., López-Del-Hoyo, Y., Olivan, B., Magallón, R., … Garcia-Campayo, J. (2014). Effectiveness of group acceptance and commitment therapy for fibromyalgia: A 6-month randomized controlled trial (EFFIGACT study). *Pain*, *155*(4), 693–702. doi:10.1016/j.pain.2013.12.029

McCracken, L. M., & Morley, S. (2014). The psychological flexibility model: A basis for integration and progress in psychological approaches to chronic pain management. *Journal of Pain*, *15*(3), 221–234. doi:10.1016/j.jpain.2013.10.014

Morley, S., Eccleston, C., & Williams, A. C. de C. (1999). Systematic review and meta-analysis of randomized controlled trials of cognitive behaviour therapy and behaviour therapy for chronic pain in adults, excluding headache. *Pain*, *80*, 1–13. doi:10.1016/S0304-3959(98)00255-3

Morley, S., & Williams, A. (2015). New developments in the psychological management of chronic pain. *Canadian Journal of Psychiatry*, *60*(4), 168–175. doi:10.1177/070674371506000403

Öst, L. G. (2008). Efficacy of the third wave of behavioral therapies: A systematic review and meta-analysis. *Behaviour Research and Therapy*, *46*(3), 296–321. doi:10.1016/j.brat.2007.12.005

Otis, J. D. (2007). *Managing chronic pain: A cognitive-behavioral therapy approach*. Oxford, UK: Oxford University Press.

Picavet, H. S., Vlaeyen, J. W. S., & Schouten, J. (2002). Pain catastrophizing and kinesiophobia: Predictors of chronic low back pain. *American Journal of Epidemiology*, *156*(11), 1028–1034. doi:10.1093/aje/kwf136

Quartana, P. J., Buenaver, L. F., Edwards, R. R., Klick, B., Haythornthwaite, J. A., & Smith, M. T. (2010). Pain catastrophizing and salivary cortisol responses to laboratory pain testing in temporomandibular disorder and healthy participants. *Journal of Pain*, *11*(2), 186–94. doi:10.1016/j.jpain.2009.07.008

Schütze, R., Rees, C., Preece, M., & Schütze, M. (2010). Low mindfulness predicts pain catastrophizing in a fear-avoidance model of chronic pain. *Pain*, *148*, 120–127. doi:10.1016/j.pain.2009.10.030

Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2013). *Mindfulness-based cognitive therapy for depression* (2nd ed.). New York, NY: Guilford Press.

Seminowicz, D. A., & Davis, K. D. (2006). Cortical responses to pain in healthy individuals depends on pain catastrophizing. *Pain*, *120*(3), 297–306. doi:10.1016/j.pain.2005.11.008

Skinner, M., Wilson, H. D., & Turk, D. C. (2012). Cognitive-behavioral perspective and cognitive-behavioral therapy for people with chronic pain: Distinctions, outcomes, and innovations. *Journal of Cognitive Psychotherapy*, *26*(2), 93–113. doi:10.1891/0889-8391.26.2.93

Smeets, R. J., Vlaeyen, J. W. S., Kester, A. D., & Knottnerus, J. A. (2006). Reduction of pain catastrophizing mediates the outcome of both physical and cognitive-

behavioral treatment in chronic low back pain. *Journal of Pain*, *7*(4), 261–271. doi:10.1016/j.jpain.2005.10.011

Spada, M. M., Gay, H., Nikčević, A. V., Fernie, B. A., & Caselli, G. (2015). Meta-cognitive beliefs about worry and pain catastrophising as mediators between neuroticism and pain behaviour. *Clinical Psychologist*, *20*, 138–146. doi:10.1111/cp.12081

Spinhoven, P., Ter Kuile, M., Kole-Snijders, A. M. J., Hutten Mansfeld, M., Den Ouden, D.-J., & Vlaeyen, J. W. S. (2004). Catastrophizing and internal pain control as mediators of outcome in the multidisciplinary treatment of chronic low back pain. *European Journal of Pain*, *8*(3), 211–219. doi:10.1016/j.ejpain.2003.08.003

Sullivan, M. J., Bishop, S. R., & Pivik, J. (1995). The pain catastrophizing scale: Development and validation. *Psychological Assessment*, *7*(4), 524–532. doi:10.1037/1040-3590.7.4.524

Sullivan, M. J., Rodgers, W. M., & Kirsch, I. (2001). Catastrophizing, depression and expectancies for pain and emotional distress. *Pain*, *91*(1–2), 147–154. doi:10.1016/S0304-3959(00)00430-9

Sullivan, M. J., Stanish, W., Waite, H., Sullivan, M., & Tripp, D. A. (1998). Catastrophizing, pain, and disability in patients with soft-tissue injuries. *Pain*, *77*(3), 253–260. doi:10.1016/S0304-3959(98)00097-9

Sullivan, M. J., Thorn, B. E., Haythornthwaite, J. A., Keefe, F., Martin, M., Bradley, L. A., & Lefebvre, J. C. (2001). Theoretical perspectives on the relation between catastrophizing and pain. *Clinical Journal of Pain*, *17*(1), 52–64. doi:10.1097/00002508-200103000-00008

Thorn, B. E. (2004). *Cognitive therapy for chronic pain: A step-by-step guide*. New York, NY: Guilford Press.

Turk, D. C. (2014). Psychological interventions. In H. Benzon, J. Rathmell, C. L. Wu, D. C. Turk, C. Argoff, & R. Hurley (Eds.), *Practical management of pain* (5th ed., pp. 615–628). Philadelphia, PA: Elsevier. doi:10.1016/B978-0-323-08340-9.00045-1

Turner, J. A., & Aaron, L. A. (2001). Pain-related catastrophizing: What is it? *Clinical Journal of Pain*, *17*(1), 65–71. doi:10.1097/00002508-200103000-00009

van Damme, S., Crombez, G., Eccleston, C., & Roelofs, J. (2004). The role of hypervigilance in the experience of pain. In G. J. Asmundson, J. W. Vlaeyen, & G. Crombez (Eds.), *Understanding and treating fear of pain* (pp. 71–90). Chichester, UK: Wiley & Sons.

Vangronsveld, K., Peters, M., Goossens, M. E. J. B., Linton, S., & Vlaeyen, J. W. S. (2007). Applying the fear-avoidance model to the chronic whiplash syndrome. *Pain*, *131*(3), 258–261. doi:10.1016/j.pain.2007.04.015

Veehof, M. M., Trompetter, H. R., Bohlmeijer, E. T., & Schreurs, K. M. G. (2016). Acceptance- and mindfulness-based interventions for the treatment of chronic pain: a meta-analytic review. *Cognitive Behaviour Therapy*, *6073*-(March), 1–27. doi:10.1080/16506073.2015.1098724

Vlaeyen, J. W. S., Kole-Snijders, A. M. J., Boeren, R. G. B., & van Eek, H. (1995). Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. *Pain*, *62*(3), 363–372. doi:10.1016/0304-3959(94)00279-N

Vlaeyen, J. W. S., & Linton, S. (2000). Fear avoidance and its consequences in musculoskeletal pain: A state of the art. *Pain*, *85*, 317–332. doi:10.1016/S0304-3959(99)00242-0

Vlaeyen, J. W. S., & Linton, S. J. (2012). Fear-avoidance model of chronic musculoskeletal pain: 12 years on. *Pain*, *153*(6), 1144–1147. doi:10.1016/j.pain.2011.12.009

Vowles, K. E., McCracken, L. M., & Eccleston, C. (2007). Processes of change in treatment for chronic pain: The contributions of pain, acceptance, and catastrophizing. *European Journal of Pain*, *11*, 779–787. doi:10.1016/j.ejpain.2006.12.007

Wells, A. (1999). A metacognitive model and therapy for generalized anxiety disorder. *Clinical Psychology & Psychotherapy*, *6*(2), 86–95. doi:10.1002/(SICI)1099-0879(199905)6:2<86::AID-CPP189>3.0.CO;2-S

Wells, A. (2009). *Metacognitive therapy for anxiety and depression*. New York, NY: Guilford Press.

Wells, A., & Cartwright-Hatton, S. (2004). A short form of the metacognitions questionnaire: Properties of the MCQ-30. *Behaviour Research and Therapy*, *42*(4), 385–396. doi:10.1016/S0005-7967(03)00147-5

Wells, A., & Matthews, G. (1994). *Attention and emotion: A clinical perspective*. Hove, UK: Psychology Press.

Wells, A., & Matthews, G. (1996). Modelling cognition in emotional disorder: The S-REF model. *Behaviour Research and Therapy*, *34*(11–12), 881–888. doi:10.1016/S0005-7967(96)00050-2

Wetherell, J. L., Afari, N., Rutledge, T., Sorrell, J. T., Stoddard, J. A., Petkus, A. J., … Atkinson, J. H. (2011). A randomized, controlled trial of acceptance and commitment therapy and cognitive-behavioral therapy for chronic pain. *Pain*, *152*(9), 2098–2107. doi:10.1016/j.pain.2011.05.016

Williams, A. C. D. C., Eccleston, C., & Morley, S. (2012). Psychological therapies for the management of chronic pain (excluding headache) in adults. *Cochrane Database of Systematic Reviews*, 11, CD007407. doi:10.1002/14651858.CD007407.pub3

Williams, A. C. de C., & McCracken, L. M. (2004). Cognitive-behavioral therapy for chronic pain: An overview with specific reference to fear and avoidance. In G. J. Asmundson, J. W. Vlaeyen, & G. Crombez (Eds.), *Understanding and treating fear of pain* (pp. 293–312). Oxford, UK: Oxford University Press.

Appendix L Publication acceptance letter for Study 1

Date: 20 Oct 2017
To: "Robert Michael Schutze" r.schutze@curtin.edu.au,rob@wisdomhealth.com.au
From: "The Journal of Pain" eesserver@eesmail.elsevier.com
Reply To: "The Journal of Pain" jpain@jpain.us
Subject: Your Submission JPAIN-D-17-00388R1

Ms. Ref. No.: JPAIN-D-17-00388R1
Title: How can we best reduce pain catastrophizing in adults with chronic non-cancer pain? A systematic review and meta-analysis
The Journal of Pain

Dear Dr. Schutze:

Thank you for submitting "How can we best reduce pain catastrophizing in adults with chronic non-cancer pain? A systematic review and meta-analysis" to The Journal of Pain. I am pleased to say your manuscript has been accepted for publication.

Prior to publication, you will receive page proofs from Elsevier Science, which publishes The Journal for the American Pain Society. Proofs are made available to you electronically, in PDF file format. You will receive notification via e-mail.

COVER ART: If you have any images that you believe would be suitable for use on the journals cover, please submit these electronically to Julie Eisele, JOP Managing Editor, at jpain@jpain.us.

You may track your manuscript's post-acceptance progress by registering your name at the link below:

http://authors.elsevier.com/TrackPaper.html

Thank you for submitting this interesting paper.

Sincerely yours,

Mark Jensen, PhD

Editor-in-Chief
The Journal of Pain

Appendix M Published version of Study 2

British Journal of Health Psychology (2017)

The British Psychological Society

www.wileyonlinelibrary.com

'I call it stinkin' thinkin'': A qualitative analysis of metacognition in people with chronic low back pain and elevated catastrophizing

Robert Schütze[1]*, Clare Rees[1], Helen Slater[2], Anne Smith[2] and Peter O'Sullivan[2]

[1]School of Psychology and Speech Pathology, Curtin University, Perth, Western Australia, Australia

[2]School of Physiotherapy and Exercise Science, Curtin University, Perth, Western Australia, Australia

Objectives. Pain catastrophizing is widely studied in quantitative pain research because of its strong link with poor pain outcomes, although the exact nature of this construct remains unclear. Focusing on its ruminative dimension, the present qualitative study aimed to explore a nascent aspect of pain catastrophizing – metacognition – by documenting people's attitudes towards rumination and examining how these metacognitions might influence the course it takes.

Design. Qualitative interview study.

Methods. Semi-structured interviews were conducted in a tertiary care setting with 15 adults experiencing chronic (≥6 months) low back pain who scored highly (≥30) on the Pain Catastrophising Scale. Transcripts were analysed using interpretative phenomenological analysis.

Results. The first aim of documenting pain metacognitions revealed both positive (e.g., 'thinking helps me to cope') and negative (e.g., 'rumination is uncontrollable') attitudes towards pain rumination. These were often held simultaneously, creating internal conflict. The second aim of exploring the influence of metacognition on rumination showed that both negative and positive metacognitions could fuel perseverative thinking. However, more nuanced negative metacognitions (e.g., 'worry is pointless') could help to end episodes of rumination by motivating the use of concrete problem-solving or active coping behaviours.

Conclusions. While most participants described pain rumination as uncontrollable and harmful, dwelling on pain could be helpful when focused on tangible and solvable problems, thereby translating into adaptive coping behaviours that eventually interrupt rumination. Future treatments may be more effective if they are based on individualized formulations of pain catastrophizing that focus on its perseverative nature and implicit function.

*Correspondence should be addressed to Robert Schütze, School of Psychology and Speech Pathology, Curtin University, GPO Box U1987, Perth, WA 6845, Australia (email: r.schutze@curtin.edu.au).

DOI:10.1111/bjhp.12240

Statement of contribution

What is already known on this subject?

- Chronic pain affects one in five people, and psychological coping responses are key targets within gold standard biopsychosocial interventions.
- People who have elevated pain catastrophizing tend to have worse pain outcomes, including increased pain, disability, and emotional distress.
- What people believe about their own thinking (i.e., their metacognitions) influences how much they worry or ruminate.

What does this study add?

- This is the first qualitative study exploring metacognitions in people with chronic pain and the first to target a purposive sample of people with elevated pain catastrophizing.
- People with elevated pain catastrophizing often see rumination as uncontrollable and harmful but may simultaneously believe it helps them to solve problems or feel prepared for future threats.
- Pain catastrophizing is not a stable and enduring trait but fluctuates both within and across individuals in response to pain, context, metacognitive beliefs about rumination, and coping behaviours.

Pain is a multidimensional subjective experience involving complex interactions between biological, psychological, genetic, and social factors, as described by the dominant biopsychosocial model of pain (Turk & Monarch, 2002). Chronic pain – defined as pain that persists beyond 3–6 months (Merskey & Bogduk, 1994) – is strongly associated with poor self-rated health, lower quality of life, and psychopathology (Crofford, 2015). There is growing evidence that psychological factors influence maladaptive behavioural and nervous system changes that in themselves may perpetuate pain (Cousins, Power, & Smith, 2000). One of the most widely studied of these psychological constructs is pain catastrophizing (PC).

Broadly defined, PC is a negative psychological response to pain and has various dimensions, including a tendency to ruminate about the pain experience, exaggerate its threat value, and underestimate one's ability to cope with it (Quartana, Campbell, & Edwards, 2009; Sullivan, Thorn *et al.*, 2001). Although not all people in pain catastrophize, elevated PC predicts a range of negative outcomes, including greater pain intensity (Leung, 2012), functional disability (Edwards *et al.*, 2011), emotional distress (Sullivan, Rodgers, & Kirsch, 2001), health care utilization (De Boer, Struys, & Versteegen, 2012), and time off work (Wertli *et al.*, 2014). It is also an important process variable in treatment, mediating or moderating the effects of evidence-based interventions such as cognitive behaviour therapy (CBT), exercise, and exposure *in vivo* (Flink, Boersma, & Linton, 2010; Smeets, Vlaeyen, Kester, & Knottnerus, 2006). This makes elevated PC a key treatment target in gold standard multidisciplinary pain rehabilitation programmes (Williams, Eccleston, & Morley, 2012).

However, ambivalence about how best to modify PC is evidenced by the fact that a range of interventions – from exercise to cognitive therapy – are about equally effective, although effect sizes are generally modest (Smeets *et al.*, 2006). The most evidence exists for CBT, with medium effects of $d = .53$ to $d = .63$ in reducing PC (Williams *et al.*, 2012). However, emerging evidence suggests that mindfulness-based approaches, which address the rumination aspect of PC as a form of repetitive negative thinking rather than addressing the content of catastrophic thoughts, may have larger effect sizes of $d = .62$ to $d = .94$ (Day *et al.*, 2014; Gardner-Nix, Barbati, Grummitt, Pukal, & Raponi Newton, 2012). Indeed, a recent high-quality trial comparing CBT and mindfulness-based

stress reduction (MBSR) found MBSR was superior in reducing PC (Turner *et al.*, 2016). Given this heterogeneity of treatments for PC, their modest effect sizes, and the existence of several competing theoretical models of PC, there have been calls to re-examine this construct so that more effective and efficient interventions might be developed (Flink, Boersma, & Linton, 2013; Schütze, 2016; Turner & Aaron, 2001).

In the light of this, two recent models focus particularly on the function of PC. Eccleston and Crombez (2007) articulate a model of pain-related worry that suggests it is a form of misdirected problem-solving that ultimately backfires. Similarly, Flink *et al.* (2013) argue that PC should be renamed 'catastrophic worry' to prioritize the rumination process over the contents of cognition. They describe PC as a form of *repetitive negative thinking* whose putative function is to reduce negative affect, partly by keeping cognition abstract. Indeed, the perseverative thinking aspect of PC warrants further investigation, given the dominance of the rumination subscale in accounting for most variance in the most common measure of PC – the Pain Catastrophising Scale – (Sullivan, Bishop, & Pivik, 1995), as well as the encouraging effect sizes emerging for treatments that specifically target the cognitive process of rumination in PC[1] (Day *et al.*, 2014; Gardner-Nix *et al.*, 2012).

Exploring rumination in PC implicates metacognition, or the beliefs people hold about their own thinking (Flink *et al.*, 2013; Schütze, 2016). So-called third wave psychological interventions that aim to disrupt rumination through cognitive defusion/decentring techniques encourage metacognitive change, where unhelpful metacognitive beliefs such as, 'thoughts are facts' give way to metacognitions like, 'thoughts are passing mental events' (McCracken & Morley, 2014; Segal, Williams, & Teasdale, 2013). This allows thoughts to be selectively – rather than rigidly – attended to through the cultivation of mindfulness.

However, arguably the most complete account of the role of metacognition in maintaining rumination is the Self-Regulatory Executive Function (S-REF) model of emotional disorder (Wells & Matthews, 1994, 1996). The S-REF model forms the basis of Metacognitive Therapy (MCT; Wells, 2009), which was originally developed to treat perseverative worry in generalized anxiety disorder but has efficacy for numerous disorders of anxiety and depression, possibly even surpassing the efficacy of CBT (Normann, Van Emmerik, & Morina, 2014).

A distinctive feature of MCT is that people's beliefs about their own thinking are key drivers of rumination, which is regarded as a self-regulation strategy. For example, a positive metacognitive belief about worry such as 'worrying helps me to solve problems' fuels perseverative thinking by ascribing it a useful function, while a negative metacognition such as 'my worry is uncontrollable' may also fuel such rumination by causing one to abandon efforts to disengage from it (Fisher & Wells, 2009). These metacognitions become targets for treatment in MCT. Recently, a correlational study provided the first evidence that these metacognitions are important aspects of PC. In a non-clinical student sample, Spada and colleagues found that positive metacognitions mediated between neuroticism and PC, while negative metacognitions mediated between PC and self-reported pain behaviours (Spada, Gay, Nikčević, Fernie, & Caselli, 2016).

Given these emerging findings and the theoretical relevance of metacognition to functional models of PC, this study aimed to further explore metacognition in PC. A qualitative phenomenological approach was taken to gather rich data about

[1] *Consistent with this argument, PC will be defined in this article as repetitive/perseverative thinking about pain. Although worry and rumination are often distinguished from each other based on the contents of cognition focusing on the future versus the past, respectively (Watkins, 2016), for parsimony both forms of perseverative thinking will be treated as instances of PC and referred to as* pain rumination.

metacognition from the idiographic perspective of people with CLBP and elevated PC. This idiographic approach is important given that, despite extensive quantitative research on PC (Leung, 2012), as well as qualitative research on various aspects of chronic pain more generally (Osborn & Rodham, 2010), there are no qualitative studies targeting a purposive sample of people with elevated PC, who are likely to engage in frequent rumination. Focusing on the ruminative aspect of PC, the aims of this study were therefore to (1) document beliefs (metacognitions) about pain-related rumination in people with a tendency towards this type of thinking and (2) explore how these metacognitions might influence the course of pain rumination.

Method

Participants

Fifteen Caucasian adults with CLBP (duration ≥6 months) who were seeking treatment from a medical specialist (orthopaedic surgeon, neurosurgeon, or pain medicine specialist) were recruited. Small, purposive samples are typically sought for interpretative phenomenological analysis (IPA) to maintain idiographic focus on individuals' experiences. The current sample constituted a fairly large sample for this methodology, which does not use the contested notion of saturation (O'Reilly & Parker, 2012) in guiding sample size (Smith & Osborn, 2008). People with elevated PC were selected because elevated PC is often a treatment target (Quartana *et al.*, 2009), so understanding how metacognition operates in this cohort is a starting point for improving their clinical outcomes in the future. The following exclusion criteria applied the following: insufficient English language proficiency for interview; likely cognitive interference due to high-dose opiate medication (>100 mg daily oral morphine equivalent); scoring below 30 on the Pain Catastrophising Scale, above which is a suggested threshold for 'severe' or clinically significant PC (Sullivan, 2004).

Fifty-eight people were screened, with 43 excluded for scoring below 30 on the PCS. Table 1 summarizes key demographic information for the included participants, as well as several quantitative pain outcomes based on the following psychometrically sound self-report measures: Brief Pain Inventory (BPI; Cleeland & Ryan, 1994) measuring pain intensity and pain interference; Pain Catastrophising Scale (PCS; Sullivan *et al.*, 1995) measuring pain catastrophizing; Meta-Cognitions Questionnaire (MCQ-30; Wells & Cartwright-Hatton, 2004) measuring metacognition; and Hospital Anxiety Depression Scale (HADS; Zigmond & Snaith, 1983) measuring anxiety and depression.

Recruitment and data collection

This study received ethical approval from the Western Australian Department of Health (SMHS registration 2014-079) and was undertaken between March and September, 2015. Nursing staff at a private neurosurgery practice (Neurospine and Brain) and a public hospital orthopaedic surgery clinic (Fremantle Hospital) gave interested patients a screening questionnaire (PCS) and information sheet describing the study as aiming to explore how people think when they are in pain. These people were contacted for further screening by the first author, who had no prior relationship with any participants. Eligible consenting participants were interviewed in their homes. Other consenting participants were recruited in the waiting room of a public hospital pain clinic (Sir Charles Gairdner Hospital), where they were interviewed in a consultation room. All participants

Table 1. Participant demographics and outcomes on self-report measures of pain and psychological functioning

Pseudonym	Gender	Age (yrs)	Marital status	Employment	Pain duration (years)	Pain intensity	Pain interference	Pain catastrophizing	Meta-cognition	Anxiety	Depression
Gail	Female	32	Married	Part time	3.5	7.2	7.3	47	75	10	8
Rachel	Female	64	Married	Retired	4	7.5	7.7	30	44	7	12
Ramo	Male	55	Married	Sick leave	.5	6.0	7.9	42	61	13	6
Pauline	Female	58	Married	Home duties	47	4.0	7.9	38	51	5	7
Rhonda	Female	53	Single	None – pain	10	9.0	9.1	51	91	11	12
Edna	Female	71	Widow	None – pain	10	3.8	3.3	47	70	7	4
Kate	Female	35	Married	Part time	8	6.5	9.1	40	67	14	11
Sandra	Female	36	Married	Full time	1.5	5.8	7.9	35	62	12	4
Daniel	Male	60	Married	None – pain	5	7.0	7.9	35	30	2	3
Jack	Male	76	Married	Retired	1.5	3.2	0.7	30	56	5	1
Jacinta	Female	55	Married	None – pain	26	6.2	6.9	30	93	12	12
Kingsley	Male	46	De facto	None – pain	12	8.0	9.7	46	91	15	5
Carly	Female	33	De facto	Full time	3.5	4.2	8.3	31	67	14	12
Jason	Male	65	Married	Retired	44	4.8	6.9	30	58	5	7
Marlene	Female	40	Single	Part time	1	6.2	6.1	32	36	8	6
Mean *(SD)*	–	51.9 (14.3)	–	–	8.9 (11.7)	6.9 (1.7)	7.1 (2.3)	37.6 (7.4)	63.5 (19.1)	9.3 (4.0)	7.3 (3.7)

Note. The following self-report instruments were used, with higher scores reflecting worse symptoms: Brief Pain Inventory measuring pain intensity (possible score 0–10) and pain interference (possible score 0–10); Pain Catastrophising Scale measuring pain catastrophizing (possible score 0–52); Meta-Cognitions Questionnaire 30 (possible score 0–120) measuring metacognition; and Hospital Anxiety Depression Scale measuring anxiety (possible score 0–21) and depression (possible score 0–21). Employment status 'None – pain' refers to being unable to work because of pain.

completed self-report measures and informed consent on a tablet computer using Qualtrics™ software (Qualtrics, 2013).

Interviews were audio-taped and lasted 41–76 minutes, using a semi-structured interview schedule as a loose structure but guided by the principle of sensitivity to context (Smith, Flowers, & Larkin, 2009). The schedule (see Appendix S1) was developed based on a broad reading of the pain literature, discussion among the research team based on clinical and research experience, and using guidelines for eliciting metacognitions in Metacognitive Therapy (Wells, 2009), including the 'metacognitive profiling' interview (Wells, 2000). Three participants had a family member present, while the rest were interviewed alone. No repeat interviews were conducted, and no participants requested changes to interview transcripts or the summary of findings that was sent to them. No participants dropped out of the study.

Analysis

Interviews were transcribed verbatim, and transcript data were managed using NVivo software (QSR International, 2014). Analysis was conducted by the first author according to the IPA process described by Smith *et al.* (2009), but adapted for electronic coding within NVivo. Consistent with IPA guidelines (Smith *et al.*, 2009), the analytic process was iterative, moving from the specific idiographic content of the interviews to their abstracted themes and back to the idiographic again. This 'hermeneutic circle' involved shifting between the emic/insider perspective and the etic/outsider perspective (Smith *et al.*, 2009). The abstracting part of the process also explicitly invoked broader contextual information from the pain science and psychological literature, responding to an identified gap in qualitative pain research (Osborn & Rodham, 2010) by looking for convergence and divergence with existing quantitative pain research.

Validity

Validity of findings was ensured by following guidelines for qualitative research documented in the Consolidated Criteria for Reporting Qualitative Research (COREQ; Tong, Sainsbury, & Craig, 2007). Following IPA guidelines (Smith *et al.*, 2009), a second author (C.R.) also audited all interview transcripts, stages of coding, field notes, and coding trees to ensure internal coherency of findings (Smith *et al.*, 2009). Findings were discussed among all authors and incorporated into the final analysis. Finally, the recognition of subjectivity that is central to qualitative research involves making explicit the interpretative stance and background of the researchers (Tong *et al.*, 2007). All investigators have previously published qualitative research and share a biopsychosocial view of pain along with both an academic and clinical interest in PC. The first author is a male doctoral student and clinical psychologist; the second author is a psychology research academic and clinical psychologist; the remaining authors are pain science research academics and musculoskeletal physiotherapists.

Results

All participants (represented with pseudonyms below) were able to reflect on their pain-related thinking and report on not only what they thought about in relation to pain, but

also to varying degrees on their attitudes towards this process and how it related to their pain and functioning. Interpretative analysis revealed a number of themes related to the two study aims of documenting pain metacognitions and exploring their influence on pain rumination. These are described below and summarized in Table 2.

Aim 1: Documenting pain metacognitions

All participants described holding opinions about their pain-related thinking that revealed both positive and negative attitudes towards rumination. These were often held simultaneously, creating internal tension as people navigated their psychological responses to pain. The most commonly reported positive attitude towards thinking extensively about pain was a belief that it helped participants to solve problems. This was described in terms of both the intention behind thinking about pain and its consequences:

> *Carly:* I guess I'm trying to problem solve, which is failing, but I persist in trying it anyway. I mean, to some degree it helps if I'm in pain and thinking about it makes me stand up instead of sitting down, then that quite often helps. So there is some payoff. But a lot of it is just a lot of useless worry.

Here Carly describes her pain rumination as a kind of implicit self-regulation strategy, aiming to solve the problem of being in pain. However, she demonstrates an ambivalence towards this strategy that was echoed by several participants (e.g., Gail, Rachel, Ramo, Kate, Jacinta). On the one hand, Carly suggests that thinking about pain can have a 'pay-off', but on the other hand, she describes it as 'failing'. This passage reveals the variable nature of rumination. For Carly, helpful rumination translates into behaviour ('thinking about it makes me stand up') which produces a tangible effect ('that quite often helps'). She contrasts this to 'useless worry', which by implication has no tangible behavioural output or benefit. There is a degree of anguish in Carly's recognition of the tensions between her positive and negative metacognitions, describing her strategy as failing but persisting anyway. She goes on to describe how this dialectic often results in more perseverative thinking, a kind of meta-worry.

> *Carly:* I spend so much time thinking about it and then, on top of that, thinking that I shouldn't be thinking about it. Then on top of that, thinking I shouldn't be beating myself up about thinking about it. It just gets ridiculous.

Jacinta shared the view that pain rumination functions as a form of problem-solving. Like Carly, she depicts this as somewhat fraught.

> *Jacinta:* What the problem solving is, it's just to work through the exercises to see if they'll help the pain. . . When I've got pain, I keep thinking I'll do it and have the operation just to get rid of the pain but then they can't guarantee I'll be out of pain so that plays on my mind.

The temporal focus of Jacinta's thinking is important in this extract. Jacinta's analysis of her exercises is concretely linked to a current behaviour ('it's just to work through the exercises') and this seems to constrain her thinking. By contrast, contemplating a treatment decision about surgery involves future behaviour and a range of distal contextual factors, raising the spectre of uncertainty ('they can't guarantee I'll be out of

Table 2. Summary of interpretative coding

Study aims	Themes	Salient codes
Documenting pain metacognitions	Positive attitudes towards rumination	Rumination as problem solving
		Rumination prepares me
		Rumination helps me to cope
	Negative attitudes towards rumination	Uncontrollability of rumination
		Psychological harm
		Rumination exacerbates pain
		Social harms of rumination
		No point ruminating
How does metacognition influence pain rumination?	Rumination facilitators	Pain and pain reminders trigger rumination
		Anticipating pain triggers rumination
		Positive metacognitions fuel rumination
		Harm metacognitions trigger meta-worry
		Uncontrollability metacognitions keep rumination unchecked
	Overcoming rumination	Pain relief ends rumination
		Distraction relieves rumination
		Reframing relieves rumination
		Switching to problem solving is helpful
		Acceptance relieves rumination
		Seeing rumination as pointless helps overcome it

pain'). This uncertainty impedes a decision, leaving Jacinta's original problem unsolved and so rumination persists ('that plays on my mind'). The other factor that distinguishes Jacinta's two descriptions of pain-related thinking is the goal itself. Her goal in the first example is more explorative and realistic, being aimed at pain modulation ('to see if they'll help the pain'), whereas her goal in the second example is complete pain remission, an absolute and ambitious target given her 26-year pain history ('just to get rid of the pain'). Jacinta's experience again highlights the variable nature of pain-related rumination in terms of intention, temporal focus, and outcome.

In contrast to seeing rumination as a problem-solving strategy – most commonly framed in terms of pain relief – some participants described it as a coping strategy yielding a sense of preparedness or control.

> *Gail:* If you think the worst and it doesn't get there, that's a positive. But if you're constantly thinking positive thoughts then anything bad happens, then you're not ready, you're not prepared for that. So I prepare myself for the worst I guess. It sounds horrible. And then anything positive that happens from that, well that's a plus.
> *Marlene:* I'd rather think than don't think. I don't know how to explain it to you, but when I think, I know where I'm going. If I don't think it's like I'm lost.

Despite these expressions of the benefits of rumination, positive metacognitions were overwhelmingly eclipsed by participants' pejorative statements about perseverative thinking. During the interviews, it was much easier to elicit negative metacognitions than positive ones. In fact, when asked about the benefits of thinking about pain, many people said 'nothing', although upon prompting most endorsed some of the positive metacognitions noted above. On one level, this reveals that people find it relatively difficult to explain why they ruminate, despite having insight into what they think about.

Negative attitudes towards rumination were easily elicited and these fell into five main categories: uncontrollability; psychological harm; pain exacerbation; social harm; and ineffectiveness. Kate and Jason describe how pain-related thinking is difficult to control:

> *Kate:* It's just second nature to me. . . There's not much that will stop you thinking about it.
> *Jason:* It's insidious. It's just covert and voom.

Jason's account suggests that rumination creeps up on you and then suddenly accelerates. The word 'voom' onomatopoeically evokes imagery of an explosion that cannot be contained. The word 'covert' portrays rumination as something separate from the self but also hidden and somewhat sinister in that its stealth implies an intent to attack.

This sense of the rumination being threatening was more explicit in the ubiquitously expressed belief that it causes psychological harm, most commonly in the form of depression, but also stress, anxiety, anger, and cognitive interference.

> *Jason:* It drags you down. . . I call it stinkin' thinkin'. There's always another phrase that comes – 'misery-go-round'.
> *Rachel:* I think you can get very depressed if you do, definitely. I've had – as I said, prior to this – black moments that I've been thinking about. 'This pain is never going to go away; I'm ruining everyone's lives.' That's why I just try and not think about it. . . I think if you sit and think about it, you're doomed.

Another harm described by about half the sample was that rumination exacerbates pain itself:

> *Rachel:* If I think about my pain, it makes it worse.
> *Kate:* It makes me notice the pain more, yes.

Kate went on to describe how this spiral of negativity creates social harm, a theme also powerfully evoked by Rhonda:

> *Rhonda:* It not only grabs you in one way, it also can destroy your family in others. It's a very nasty type of thing. If it doesn't destroy your body it can also destroy your relationships, your closest friends, your family. It can tear you apart. . . You have to put it out of your head, but it's constantly there. It never leaves you alone, it's torment, it's actually torment to you. That's all I can say. You're tormented for the rest of your life.

Rhonda's language again represents rumination as an entity separate from the self ('grabs you'; 'never leaves you alone'), echoing Jason's description of a covert force. Here, its harmful nature is more exaggerated, with images of destruction ('destroy your body', 'destroy your family') and malevolent intent ('very nasty').

Prolonged thinking about pain was therefore experienced in different ways by this group of people. Its negative features – uncontrollable and harmful – were obvious to all, while for some people, thinking about pain was a strategy for problem-solving or coping. These positive and negative metacognitions about rumination were sometimes simultaneously held, creating tension and at times prompting further rumination.

Aim 2: How does metacognition influence pain rumination?

This highlights a crucial feature of pain rumination in these 15 people. Rather than being static and uniform, participants described rumination as fluctuating in content, impact, intensity, and duration. For example, despite scoring highly on the PCS, a quantitative measure of pain catastrophizing, some participants said thoughts about pain were fleeting:

> *Daniel:* I don't have those sort of thoughts much anymore. . . Like when you ask me questions like this, that's when it comes up or when I'm talking to other people. That's when it comes up.

However, for many participants, thinking about pain seemed to occupy a large proportion of their time, energy, and attention.

> *Kingsley:* I'm constantly in pain and constantly thinking about the pain level that I'm in and how I can get out of it. . . So you're pretty much constantly, constantly thinking about it.
> *Carly:* Yeah, it's pretty much ticking over all the time. It's got its own special part of my brain that is devoted entirely to that, I think.

Despite this variability in time spent thinking about pain, there was striking congruence in what initially triggered thoughts about pain. These included hearing other people talk about pain, anticipating pain-provoking activities, functional limitations, and, most commonly, pain itself:

> *Edna:* Oh yes, I think mainly when the pain is severe you do think.

Conversely, participants who described pain as triggering rumination also said it eased in response to pain relief.

> *Gail:* If that pain was completely blocked and I didn't feel it, straight away that worry would be gone.

Gail's comment suggests a mechanistic relationship between pain and rumination that is consistent with her uncontrollability metacognition ('About other things that I worry about? Yes, I have control of it. This back pain? No'.). Yet she goes on to describe how even in the absence of pain, her attention is oriented towards future pain ('It [worry] just lingers. Yeah, it's anticipation as well, like just waiting for the unknown'.). This is reminiscent of Gail's earlier comment that rumination prepares her for future threats ('I prepare myself for the worst, I guess'). This shows how a positive metacognition (prepares me) and a negative metacognition (uncontrollable) both facilitate rumination about pain: The 'prepares me' positive metacognition implicitly motivates increased attention to and processing of pain-related stimuli, and the 'uncontrollable' negative metacognition compounds inertia against attempts to disrupt rumination. This ultimately leads to a perseverative loop where both rumination and pain are maintained or exacerbated ('If I dwell on it too much it's just going to get ten times worse'), which in turn become triggers for further rumination.

Carly described experiencing a similar vicious cycle of pain and rumination, which, like Gail, could even be triggered by the absence of pain.

> *Carly:* If I'm having a period of little or no pain, then there will inevitably come a moment where I realise that I'm in little or no pain and scan for it and become suspicious of the fact that it's not there. I know that it's coming back. Then I start thinking oh, okay, right, I'm having a good period right now. Why am I having a good period? What have I done to make this pain good right now? Is it because I saw the physio yesterday or because I slept well? Yeah, then I'm analysing as to why it's not there.

This hypervigilance seems to be fuelled by her positive 'problem-solving' metacognition ('I'm trying to problem solve'). However, earlier Carly described how the tension between her positive and negative metacognitions often resulted in more rumination, in the form of meta-worry ('That kind of gets into a bit of a spiral about feeling bad about thinking about the pain'). This seems to stem from her negative metacognition that rumination is harmful:

> *Carly:* I've been told by various people that chronic pain is kind of a strengthening of these neural pathways and, I guess, short-circuiting of – your brain just saying all right, you're in pain now. You need to be able to weaken those pathways. So continually thinking about it is obviously not going to succeed in breaking those pathways down. So to some degree, I feel like it's self-perpetuating and I'm not helping myself by thinking about it.

Given this negative view of pain-related thinking, Carly's recognition of her thinking becomes a trigger for worrying about her own worry. Therefore, this negative metacognition acts as a potent facilitator of rumination.

However, despite reporting these episodes of perseverative thinking, Gail and Carly also described periods of diminished rumination and pain, which reinforces the observation that pain rumination is a dynamic process, or transient state, rather than an enduring trait. Highlighting this, several participants described how they disrupted or averted cycles of rumination. For example, just as Carly contrasted helpful perseverative thinking (which has a 'pay-off') against 'useless worry', Pauline describes how she consciously shifts her thinking towards the more helpful style.

> *Pauline:* Sometimes, I think okay, well instead of worrying about it, try to find a solution to it. So there is that. I try to sort of think what can I do to make it better, rather than just worrying about it. Because I don't feel that worrying about it is going to really help me. But trying to find a solution, rather than worrying about it. . . To me, there's more important things to be done than just continually worrying about things. That, to me, is sort of like ifs, buts and maybes. You know, you just try and make decisions and get on with things.

Pauline insightfully differentiates worry from problem-solving, even highlighting the linguistic features of worry ('ifs, buts and maybes') and implicitly linking the abstract nature of worry to its lack of utility ('I don't feel that worrying about it is going to really help me'). This is reminiscent of the Jacinta's description of contemplating future surgery, where uncertainty and abstraction fuelled rumination ('that plays on my mind'). By contrast, Pauline depicts problem-solving as concrete, present-focused, and oriented towards a behavioural response ('what can I do to make it better').

An important feature of Pauline's path out of rumination is her negative attitude towards worry ('there's more important things to be done than just continually worrying'). This seems to motivate her to avoid getting stuck in a perseverative loop. As she says elsewhere, 'Sometimes I do worry but I try not to, I try to take my mind off it in other ways'. To this end, she describes using self-management behaviours such as deep breathing, meditation, walking, and distraction through Sudoku puzzles.

This shows how a more nuanced negative metacognition about rumination as merely ineffective can be protective, prompting an adaptive coping response that involves concrete thinking and helpful action, in contrast to more overtly negative 'harm metacognitions' that trigger meta-worry in Carly's case. For Pauline, seeing rumination as pointless and thereby translating it into action has a positive impact on her pain experience, which subsequently diminishes the initial impetus to think about pain.

> *Pauline:* If I'm concentrating on something else and get my mind off the pain, it eases off quicker. . . I'll just start deep breathing. I'll go back to the Sudoku and it just slowly eases off.

Rachel demonstrates a similar protective metacognition, expressing the view that rumination is both pointless and modifiable:

> *Rachel:* I just can't sit and think, 'I'm in pain, God, I'm in pain.' I don't want to do that. I don't want to do that. I just get up and do it. . . I think, 'No, I'm going to get up and do something,' even if it's I've got to go for a little walk. I go out and see the chookies [chickens] or the roos [kangaroos]. Just little things that stop me thinking.

She goes on to describe how taking action not only breaks the cycle of rumination, it also eases her pain, which she attributes to the pain modulating effect of both external attention and movement. Other participants described similar experiences, most

commonly using distraction to disrupt rumination, although some people described how meditation and acceptance of their pain and thoughts allowed them to ride out flare-ups without fuelling more distress.

This group of people therefore described rumination as a process that fluctuates in response to external and internal triggers, changing levels of pain, and different coping behaviours. Their beliefs about rumination – or metacognitions – seemed to influence this process by motivating attempts to transform it into a helpful form of thinking (problem-solving) or shift attention away from thoughts in the case of protective metacognitions such as 'rumination is pointless'. Conversely, metacognition could fuel an unhelpful form of rumination when people believed it was uncontrollable, dangerous, or helped them to cope.

Discussion

This qualitative study of 15 people with CLBP and elevated PC aimed to provide an insight into people's attitudes towards their pain-related thinking and how these metacognitions influence the course of rumination. The most common positive metacognition was that rumination was a form of problem-solving and further analysis revealed that this was only experienced as helpful if the thinking was focused on concrete and solvable problems in the short term, and if it translated into self-management behaviours. Otherwise rumination was commonly seen as unhelpful, uncontrollable, and detrimental in its impact on one's mental state, relationships, and pain levels. In exploring the influence of metacognition on pain rumination, it became apparent that rumination itself is a dynamic process that varies both within and across individuals. Some people, particularly those with strong uncontrollability metacognitions, described rumination as mainly triggered and resolved by pain itself. Others, holding metacognitions that rumination is pointless but modifiable, made active attempts to attenuate unproductive thinking by transforming it into effective problem-solving, taking action or refocusing their attention.

Although this is the first qualitative study employing a purposive sample of people with elevated catastrophizing, several of the themes found here are consistent with existing qualitative pain literature. For example, the negative metacognitions documented here that focus on the danger and harms associated with rumination echo previous qualitative accounts of fear, worry, distress, and depression among people living with chronic pain (Bunzli, Smith, Schütze, & O'Sullivan, 2015; Kirkham, Smith, & Havsteen-Franklin, 2015; Osborn & Rodham, 2010; Osborn & Smith, 2008; Smith & Osborn, 2007; Snelgrove, Edwards, & Liossi, 2013; Taylor, Carswell, & Williams, 2013).

These findings also qualitatively articulate many of the empirical findings of quantitative research into PC. For example, several participants said PC increased the intensity or salience of their pain, echoing the strong empirical association between PC and increased pain intensity (Leung, 2012). Similarly, negative metacognitions describing the deleterious effect that rumination has on mental health mirror the robust correlation between PC and depression and anxiety in the pain literature (Quartana *et al.*, 2009). Furthermore, the sense of rumination's uncontrollability that participants described appears consistent with emerging evidence of neuroplastic changes in the brains of people prone to ruminating about pain, especially increased functional connectivity within the default mode network, a network associated with mind wandering (Kucyi *et al.*, 2014).

Importantly, the present findings are consistent with theoretical models emphasizing the functional aspects of PC. For example, the prevalent metacognition describing rumination as a problem-solving strategy is reminiscent of Eccleston and Crombez's (2007) *misdirected problem-solving model* of pain-related worry. Their contention that a perseverance loop of worry stems from problems being poorly framed, especially in terms of pain relief, was exemplified by people such as Jacinta, who described how uncertainty around whether surgery would get rid of her pain made it play on her mind. This was contrasted to instances where she framed the problem more narrowly in scope and temporal focus, such as thinking about whether her exercises were helping her pain on a given day. Although Eccleston and Crombez's model portrays searching for pain relief as the lynch pin of worry perseveration, participants here described being able to steer away from endless rumination as long as their search for pain relief was helpfully framed. For example, 'what can I do to help the pain right now' rather than 'how can I get rid of this pain once and for all'.

This is also consistent with recent models of rumination as a transdiagnostic process in psychopathology. For example, Watkins' (2016) Rumination-Focused Cognitive Behaviour Therapy for Depression aims to help people 'shift from unconstructive rumination to constructive rumination' (Watkins, 2016, p. 11), such as problem-solving. In our sample, helpful rumination was limited in duration because it produced decisions that articulated naturally into behavioural outputs that shifted attention away from thoughts of the self and onto activity, thereby improving pain and distress.

Our findings also support the Flink *et al.* (2013) model of PC as repetitive negative thinking – which they rename 'catastrophic worry' – characterized by abstract cognition and whose putative function is to reduce negative affect. Present findings support this idea that the essential feature of PC is its perseverative nature, as evidenced by descriptions of it as a 'misery-go-round' (Jason) and 'pretty much ticking over all the time' (Carly). The clear articulation of positive metacognitions in this study provides the first qualitative support for Flink *et al.*'s suggestion that catastrophic worry is, 'positively reinforced through metacognitions about dwelling on the problem as beneficial for finding a solution to the problem' (Flink *et al.*, 2013, p. 218).

Indeed, the stories of these 15 people with chronic back pain seem to support the broader contention that metacognition, or what people believe about their own thinking, influences how much they ruminate. In Metacognitive Therapy, the basic tenet linking metacognition to perseverative thinking is the notion that positive and negative metacognitions both trigger and reinforce rumination in various ways because the ultimate function of this type of thinking is self-regulation (Fisher & Wells, 2009). Our participants' reflections on how their thinking related to attempts to reduce pain, make treatment decisions, and prepare for future threats all illustrate this basic self-regulatory function. Although similar findings have been documented for people with mental health conditions and addictions (Fisher & Wells, 2009; Nikčević & Spada, 2010), this is the first qualitative evidence that the S-REF model and metacognition more broadly are relevant in the context of pain-related cognition.

These findings suggest that, although several factors are likely to influence pain rumination, there may be value in targeting metacognitive beliefs explicitly in people who think excessively about pain and future research is needed to explore this. Although metacognitive changes are an implicit feature of acceptance- and mindfulness-based interventions, present findings suggest the need to create individualized formulations of the function of different metacognitions in sustaining perseverative thinking for different people. For example, targeting positive metacognitive beliefs might be ineffective for

someone with predominantly uncontrollability or psychological harm type metacognitions. The present findings raise the possibility that an adapted form of Metacognitive Therapy (Wells, 2009) may be an effective way to treat elevated PC for some people. This may not be a simple application of incumbent MCT protocols, but could incorporate other evidence-based treatment components such as pain neuroscience education (Moseley & Butler, 2015) and exposure-based movement retraining (Fersum, O'Sullivan, Skouen, Smith, & Kvåle, 2013), although further research is needed to explore this.

On a practical level, the experiences of these 15 people demonstrate that scores on quantitative measures such as the Pain Catastrophising Scale should be interpreted cautiously by clinicians. Our findings highlight that pain rumination, and PC in general, is more state-like than trait-like and may require multiple assessment points for validity. Clinicians would benefit from using such psychometric instruments as interview tools to facilitate dialogue and assessment from a functional-analytic perspective to see how cognitive and behavioural coping responses vary in different contexts. This contextual approach, and evidence of the variability of rumination within and across individuals in this study, may also reduce the stigma sometimes associated with pain-related distress, where people with pain can feel marginalized in treatment settings if psychological factors are identified as contributing to their pain problem (Ojala *et al.*, 2015).

Conclusions

This study provides the first qualitative account of pain-related metacognition. It shows that metacognitive beliefs influence how people with high levels of PC respond to pain, including how much they ruminate. This advances our understanding of the functional aspect of PC, lending support for recent conceptualizations of PC as primarily a form of repetitive negative thinking. These insights may inform the development of more targeted interventions for people who experience poor pain outcomes associated with episodes of unhelpful rumination about their pain.

Acknowledgements

The authors are grateful to all participants for their time, as well as staff who helped with recruitment: Fremantle Hospital Orthopedic Clinic and Pre-Admission Clinic (Dr Piers Yates, Joanne Morrish, Jennifer Harvey, Dr Stephanie Davies, Michelle Fulwood), Sir Charles Gairdner Hospital Department of Pain Management (Dr Roger Goucke, Dr Mark Schütze, Jane Royle), and Neurospine and Brain (Dr Andrew Miles, Kate Moore). This research was financially supported by Spinnaker Health Research Foundation in the form of a Bellberry Medical Research Scholarship to the first author.

Conflict of interest

All authors declare no conflict of interest.

References

Bunzli, S., Smith, A., Schütze, R., & O'Sullivan, P. (2015). Beliefs underlying pain-related fear and how they evolve: A qualitative investigation in people with chronic back pain and high pain-related fear. *Britisb Medical Journal Open*, *5*(10), e008847. doi:10.1136/bmjopen-2015-008847

Cleeland, C. S., & Ryan, K. M. (1994). Pain assessment: Global use of the brief pain inventory. *Annals of the Academy of Medicine, Singapore*, *23*, 129–138.

Cousins, M. J., Power, I., & Smith, G. (2000). Pain: A persistent problem. *Regional Anesthesia and Pain Medicine*, *25*(1), 6–21.

Crofford, L. J. (2015). Psychological aspects of chronic musculoskeletal pain. *Best Practice & Research Clinical Rheumatology*, *29*(1), 147–155. doi:10.1016/j.berh.2015.04.027

Day, M. A., Thorn, B. E., Ward, L. C., Rubin, N., Hickman, S. D., Scogin, F., & Kilgo, G. R. (2014). Mindfulness-based cognitive therapy for the treatment of headache pain: A pilot study. *Clinical Journal of Pain*, *30*(2), 152–161. doi:10.1097/AJP.0b013e318287a1dc

de Boer, M. J., Struys, M. M. R. F., & Versteegen, G. J. (2012). Pain-related catastrophizing in pain patients and people with pain in the general population. *European Journal of Pain*, *16*, 1044–1052. doi:10.1002/j.1532-2149.2012.00136.x

Eccleston, C., & Crombez, G. (2007). Worry and chronic pain: A misdirected problem solving model. *Pain*, *132*, 233–236. doi:10.1016/j.pain.2007.09.014

Edwards, R. R., Cahalan, C., Calahan, C., Mensing, G., Smith, M. T., & Haythornthwaite, J. A. (2011). Pain, catastrophizing, and depression in the rheumatic diseases. *Nature Reviews. Rheumatology*, *7*, 216–224. doi:10.1038/nrrheum.2011.2

Fersum, K. V., O'Sullivan, P., Skouen, J. S., Smith, A., & Kvåle, A. (2013). Efficacy of classification-based cognitive functional therapy in patients with non-specific chronic low back pain: A randomized controlled trial. *European Journal of Pain*, *17*, 916–928. doi:10.1002/j.1532-2149.2012.00252.x

Fisher, P., & Wells, A. (2009). *Metacognitive therapy: Distinctive features*. New York, NY: Routledge.

Flink, I. L., Boersma, K., & Linton, S. J. (2010). Catastrophizing moderates the effect of exposure *in vivo* for back pain patients with pain-related fear. *European Journal of Pain*, *14*, 887–892. doi:10.1016/j.ejpain.2010.02.003

Flink, I. L., Boersma, K., & Linton, S. J. (2013). Pain catastrophizing as repetitive negative thinking: A development of the conceptualization. *Cognitive Behaviour Therapy*, *42*, 215–223. doi:10.1080/16506073.2013.769621

Gardner-Nix, J., Barbati, J., Grummitt, J., Pukal, S., & Raponi Newton, R. (2012). Exploring the effectiveness of a Mindfulness-based chronic pain management course delivered simultaneously to on-site and off-site patients using telemedicine. *Mindfulness*, *5*, 223–231. doi:10.1007/s12671-012-0169-3

Kirkham, J. A., Smith, J. A., & Havsteen-Franklin, D. (2015). Painting pain: An interpretative phenomenological analysis of representations of living with chronic pain. *Health Psychology*, *34*, 398–406. doi:10.1037/hea0000139

Kucyi, A., Moayedi, M., Weissman-Fogel, I., Goldberg, M. B., Freeman, B. V., Tenenbaum, H. C., & Davis, K. D. (2014). Enhanced medial prefrontal-default mode network functional connectivity in chronic pain and its association with pain rumination. *Journal of Neuroscience*, *34*, 3969–3975. doi:10.1523/JNEUROSCI.5055-13.2014

Leung, L. (2012). Pain catastrophizing: An updated review. *Indian Journal of Psychological Medicine*, *34*, 204–217. doi:10.4103/0253-7176.106012

McCracken, L. M., & Morley, S. (2014). The psychological flexibility model: A basis for integration and progress in psychological approaches to chronic pain management. *Journal of Pain*, *15*, 221–234. doi:10.1016/j.jpain.2013.10.014

Merskey, H., & Bogduk, N. (1994). *Classification of chronic pain: Descriptions of chronic pain syndromes and definitions of pain terms* (2nd ed.). Seattle, WA: IASP Press.

Moseley, G. L., & Butler, D. S. (2015). Fifteen years of explaining pain: The past, present and future. *Journal of Pain*, *16*, 807–813. doi:10.1016/j.jpain.2015.05.005

Nikčević, A. V., & Spada, M. M. (2010). Metacognitions about smoking: A preliminary investigation. *Clinical Psychology and Psychotherapy*, *17*, 536–542. doi:10.1002/cpp.689

Normann, N., Van Emmerik, A. A. P., & Morina, N. (2014). The efficacy of metacognitive therapy for anxiety and depression: A meta-analytic review. *Depression and Anxiety*, *31*, 402–411. doi:10.1002/da.22273

Ojala, T., Häkkinen, A., Karppinen, J., Sipilä, K., Suutama, T., & Piirainen, A. (2015). Although unseen, chronic pain is real—A phenomenological study. *Scandinavian Journal of Pain*, *6*, 33–40. doi:10.1016/j.sjpain.2014.04.004

O'Reilly, M., & Parker, N. (2012). "Unsatisfactory Saturation": A critical exploration of the notion of saturated sample sizes in qualitative research. *Qualitative Research*, *13*, 190–197. doi:10.1177/1468794112446106

Osborn, M., & Rodham, K. (2010). Insights into pain: A review of qualitative research. *Reviews in Pain*, *4*(1), 2–7. doi:10.1177/204946371000400102

Osborn, M., & Smith, J. A. (2008). The fearfulness of chronic pain and the centrality of the therapeutic relationship in containing it: An interpretative phenomenological analysis. *Qualitative Research in Psychology*, *5*, 276–288. doi:10.1080/14780880701826143

QSR International (2014). NVivo for Mac qualitative data analysis software.

Qualtrics (2013) *Qualtrics research suite*. Provo, UT: Author.

Quartana, P. J., Campbell, C. M., & Edwards, R. R. (2009). Pain catastrophizing: A critical review. *Expert Review of Neurotherapeutics*, *9*, 745–758. doi:10.1586/ern.09.34

Schütze, R. (2016). Re-thinking over-thinking pain: What can metacognition add to our understanding of pain catastrophising? *Clinical Psychologist*, *20*, 147–153. doi:10.1111/cp.12105

Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2013). *Mindfulness-based cognitive therapy for depression* (2nd ed.). New York, NY: Guilford Press.

Smeets, R. J., Vlaeyen, J. W. S., Kester, A. D., & Knottnerus, J. A. (2006). Reduction of pain catastrophizing mediates the outcome of both physical and cognitive-behavioral treatment in chronic low back pain. *Journal of Pain*, *7*, 261–271. doi:10.1016/j.jpain.2005.10.011

Smith, J. A., Flowers, P., & Larkin, M. (2009). *Interpretative phenomenological analysis: Theory, method and research* (Vol. 9). London, UK: Sage.

Smith, J. A., & Osborn, M. (2007). Pain as an assault on the self: An interpretative phenomenological analysis of the psychological impact of chronic benign low back pain. *Psychology & Health*, *22*, 517–534. doi:10.1080/14768320600941756

Smith, J. A., & Osborn, M. (2008). Interpretative phenomenological analysis. In J. A. Smith (Ed.), *Qualitative psychology: A practical guide to research methods* (2nd ed., pp. 53–80). London, UK: Sage.

Snelgrove, S., Edwards, S., & Liossi, C. (2013). A longitudinal study of patients' experiences of chronic low back pain using interpretative phenomenological analysis: Changes and consistencies. *Psychology & Health*, *28*(2), 121–138.

Spada, M. M., Gay, H., Nikčević, A. V., Fernie, B. A., & Caselli, G. (2016). Meta-cognitive beliefs about worry and pain catastrophising as mediators between neuroticism and pain behaviour. *Clinical Psychologist*, *20*, 138–146. doi:10.1111/cp.12081

Sullivan, M. J. (2004). *The pain catastrophizing scale: User manual*. Montreal, Canada: University of Montreal.

Sullivan, M. J., Bishop, S. R., & Pivik, J. (1995). The pain catastrophizing scale: Development and validation. *Psychological Assessment*, *7*, 524–532. doi:10.1037/1040-3590.7.4.524

Sullivan, M. J., Rodgers, W. M., & Kirsch, I. (2001). Catastrophizing, depression and expectancies for pain and emotional distress. *Pain*, *91*, 147–154. doi:10.1016/S0304-3959(00)00430-9

Sullivan, M. J., Thorn, B. E., Haythornthwaite, J. A., Keefe, F., Martin, M., Bradley, L. A., & Lefebvre, J. C. (2001). Theoretical perspectives on the relation between catastrophizing and pain. *Clinical Journal of Pain*, *17*(1), 52–64.

Taylor, B., Carswell, K., & Williams, A. C. (2013). The interaction of persistent pain and post-traumatic re-experiencing: A qualitative study in torture survivors. *Journal of Pain and Symptom Management*, *46*, 546–555. doi:10.1016/j.jpainsymman.2012.10.281

Tong, A., Sainsbury, P., & Craig, J. (2007). Consolidated criteria for reporting qualitative research (COREQ): A 32-item checklist for interviews and focus groups. *International Journal for Quality in Health Care*, *19*, 349–357. doi:10.1093/intqhc/mzm042

Turk, D. C., & Monarch, E. S. (2002). Biopsychosocial perspectives on chronic pain. In D. C. Turk & R. J. Gatchel (Eds.), *Psychological approaches to pain management: A practitioner's handbook* (2nd ed., pp. 2–29). New York, NY: Guilford.

Turner, J. A., & Aaron, L. A. (2001). Pain-related catastrophizing: What is it? *Clinical Journal of Pain*, *17*(1), 65–71.

Turner, J. A., Anderson, M. L., Balderson, B. H., Cook, A. J., Sherman, K. J., & Cherkin, D. C. (2016). Mindfulness-based stress reduction and cognitive-behavioral therapy for chronic low back pain: Similar effects on mindfulness, catastrophizing, self-efficacy, and acceptance in a randomized controlled trial. *Pain*, *157*, 2434–2444. doi:10.1097/j.pain.0000000000000635

Watkins, E. R. (2016). *Rumination-focused cognitive-behavioral therapy for depression*. New York, NY: Guildford Press.

Wells, A. (2000). *Emotional disorders and metacognition: Innovative cognitive therapy*. Chichester, UK: Wiley.

Wells, A. (2009). *Metacognitive therapy for anxiety and depression*. New York, NY: Guilford Press.

Wells, A., & Cartwright-Hatton, S. (2004). A short form of the metacognitions questionnaire: Properties of the MCQ-30. *Behaviour Research and Therapy*, *42*, 385–396. doi:10.1016/S0005-7967(03)00147-5

Wells, A., & Matthews, G. (1994). *Attention and emotion: A clinical perspective*. Hove, UK: Psychology Press.

Wells, A., & Matthews, G. (1996). Modelling cognition in emotional disorder: The S-REF model. *Behaviour Research and Therapy*, *34*, 881–888. doi:10.1016/S0005-7967(96)00050-2

Wertli, M. M., Eugster, R., Held, U., Steurer, J., Kofmehl, R., & Weiser, S. (2014). Catastrophizing - A prognostic factor for outcome in patients with low back pain: A systematic review. *Spine Journal*, *14*, 2639–2657. doi:10.1016/j.spinee.2014.03.003

Williams, A. C., Eccleston, C., & Morley, S. (2012). Psychological therapies for the management of chronic pain (excluding headache) in adults. *Cochrane Database of Systematic Reviews*, (11), CD007407. doi: 10.1002/14651858.CD007407.pub3

Zigmond, A. S., & Snaith, R. P. (1983). Hospital Anxiety and Depression Scale (HADS). *Annals of General Psychiatry*, *67*, 361–370. doi:10.1186/1744-859X-7-4

Received 21 November 2016; revised version received 5 March 2017

Supporting Information

The following supporting information may be found in the online edition of the article:

Appendix S1. Semi-structured interview schedule.